

2026



# CANADA'S PREMIER VETERINARY CONFERENCE



**JAN  
29-31  
2026**

The Westin  
Harbour Castle  
TORONTO, ON

## CONFERENCE PROCEEDINGS

PART ONE



ONTARIO  
VETERINARY  
MEDICAL  
ASSOCIATION

**#OVMA2026**



## 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.

**Janice Honda, DVM**  
2026 Conference Chair  
Ontario Veterinary Medical Association

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*Veterinary Consultant, Elanco Animal Health*

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*Veterinary Specialist, Animal Hospital of Cambridge*

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# 2026 OVMA CONFERENCE – SPEAKER BIOS

## SMALL ANIMAL PROGRAM



### Dawn Crandell, DVM, DVSc, DACVECC

Veterinary Medical Director, Head of Critical Care, Toronto Animal Health Partners

Dr. Crandell graduated from the Ontario Veterinary College in 1990 and completed an internship in small animal medicine and surgery at UPEI. She worked at a general small animal practice in Thunder Bay for 3 years, then full time work as an emergency veterinarian and practice manager in southern Ontario. She returned to OVC in 2001 to do a 3-year residency in emergency medicine and critical care, obtaining board certification in 2004. Subsequently she worked as a critical care specialist full time in Toronto, and is now is the veterinary medical director at Toronto Animal Health Partners. Her professional special interests outside of critical care are the advancement of recognition and treatment of patient stress and fear in the veterinary clinical environment and promoting an evidence-based approach to veterinary medicine. When not immersed in a veterinary hospital, she loves to be outdoors away from asphalt, concrete and cars, practices and promotes gardening with native plants to help heal our urban environment, and champions safer cycling in Toronto.



### Caitlin Grant, DVM, DVSc, Dip ECVN

Veterinary Specialist, Animal Hospital of Cambridge

Dr. Grant attended the Ontario Veterinary College (OVC) and graduated with honours in 2014. After graduation, she became an associate veterinarian in a private, mixed animal practice. In 2017, she returned to OVC and completed a nutrition residency through the European College of Veterinary and Comparative Nutrition (ECVCN) and earned a Doctor of Veterinary Science (DVSc) degree in September 2020. Dr. Grant then joined OVC as Assistant Professor and held the Nestle Purina Professorship in Companion Animal Nutrition. She was service chief for the OVC-HSC Clinical Nutrition Service and taught nutrition to students in all four phases of the DVM program. She currently works in private practice in Cambridge Ontario providing nutrition consults to pet owners and provides in clinic as well as telemedicine nutrition consultations to pet owners across Ontario and to veterinarians nationwide.



### Peter Helmer, DVM, DABVP-Avian Practice

Assistant Professor, Lincoln Memorial University College of Veterinary Medicine

Peter Helmer is a graduate of the Ontario Veterinary College, University of Guelph and a Diplomate of the American Board of Veterinary Practitioners, Avian Practice. After 25 years in private practice he recently joined the faculty at Lincoln Memorial University in Harrogate, TN. He is a frequent speaker at veterinary CE conferences and has authored numerous publications including journal articles and textbook chapters. Dr Helmer is also a consultant for Avian/Exotic pet medicine at Antech Diagnostics.





## Lissie Henderson, BVSc (Hons), DipECVS

Small Animal Soft Tissue Surgeon

Dr Lissie Henderson graduated with honours from the University of Bristol in 2011. She went on to complete a rotating Internship at the University of Bristol and subsequently worked as a clinical research assistant at the University of Liverpool. During this time she undertook data collection and the analysis for projects and published in several veterinary scientific journals. Dr Henderson then undertook a surgical internship and residency program in small animal surgery at the University of Edinburgh and became a board-certified Diplomate of the European College of Veterinary Surgeons in 2020. She is passionate about her continued learning and the teaching of veterinary students, interns and residents. Dr Henderson enjoys all aspects of small animal soft tissue surgery and has a particular interest in endocrine surgery and interventional procedures.



## Alison Little, DVM, DACVIM (Neurology)

Neurologist, Toronto Animal Health Partners

Dr. Alison Little graduated from the Ontario Veterinary College in 2014. Following graduation, she completed five years of advanced training via a Small Animal Rotating Internship at the Veterinary Emergency Clinic in Toronto, a Specialty Neurology Internship at Southeast Veterinary Neurology in Miami, Florida and a Neurology/Neurosurgery Residency at Mississippi State University. She then returned home to Toronto and joined the Animal Health Partners team in 2020.



## Jenna Manacki, DVM, Residency Trained in Clinical Nutrition

Secretary, Canadian Academy of Veterinary Nutrition

Dr. Manacki completed her undergraduate studies at the University of Guelph. She attended Ross University School of Veterinary Medicine on the beautiful island of St. Kitts and completed her clinical training at the University of Saskatchewan. After graduating, Dr. Manacki practiced companion animal medicine in both Arizona and Ontario. After four years in private practice, Dr. Manacki pursued a clinical nutrition residency at the University of Missouri, finishing in 2022. Dr. Manacki currently runs a clinical nutrition service at the Veterinary Emergency Clinic (VEC) in Toronto. Dr. Manacki is a proud member of the Canadian Academy of Veterinary Nutrition.



## Connie Mosley, Dr.med.vet., DACVAA, CVA

Veterinary Consultant, Elanco Animal Health

Dr. Conny Mosley is a board-certified anesthesiologist and currently the Veterinary Consultant for Pain & Internal Medicine for Elanco Canada. She is certified in acupuncture, served on the WSAVA Global Pain Council and is the founding director of the Canadian Association of Veterinary Cannabinoid Medicine. Conny has held faculty positions at North Carolina State University, Oregon State University and Ontario Veterinary College and has been an anesthesiologist in a private specialty practice, where she also ran an Integrative Veterinary Pain Clinic. She continues to consult on pain cases. She is a passionate advocate for ethical and practical veterinary care and pain management. Her contributions to the field include publications, lectures, and a deep commitment to both teaching and learning.



## Craig Mosley, DVM, MSc, DACVAA

Staff Veterinarian, VCA Canada 404 Veterinary Emergency and Referral Hospital

Dr. Craig Mosley graduated from the Ontario Veterinary College at the University of Guelph where he also completed a residency and Master's of Science program in veterinary anesthesia. Dr. Mosley has been actively involved in many facets of veterinary medicine since graduation including; mixed animal practice, emergency medicine, critical care, teaching, management and of course, anesthesia in both private and academic practices throughout North America. Dr. Mosley's varied experiences have provided him with the foundation for his practical and "real-world" approach to anesthesia and pain management.



## Lynne O'Sullivan, DVM, DVSc, DACVIM (Cardiology)

Professor, Department of Companion Animals, Atlantic Veterinary College, University of Prince Edward Island

Dr. Lynne O'Sullivan received her DVM from AVC, UPEI; Cardiology residency training and DVSc degree from OVC, University of Guelph; and board certification in Cardiology from ACVIM in 2003. She was a faculty member at the University of Guelph for 15 years before returning to the east coast to join the faculty at AVC, UPEI. Her research interests have been in dilated cardiomyopathy and imaging, and her passion is teaching both DVM students and residents. She's been the recipient of the Carl J. Norden Distinguished Teacher Award, the Canadian Veterinary Medical Association Teacher of the Year Award, and the Boehringer Ingelheim Clinical Teaching Award. Outside of veterinary medicine, she can be found on the pool deck, side lines, or ski slopes cheering on her active sons, or spending time with her husband and her pets at home.



## Karen L. Overall, MA, VMD, PhD, DACVB

Professor, Behavioural Medicine, Department of Biomedical Sciences, Atlantic Veterinary College, UPEI

Dr. Karen L. Overall received her BA, MA and VMD degrees from the University of Pennsylvania and a PhD degree from the University of Wisconsin-Madison. She did her residency training in veterinary behavioural medicine at the University of Pennsylvania and is a Diplomate of the American College of Veterinary Behaviourists (DACVB). Dr. Overall is a Professor of Behavioural Medicine at Atlantic Veterinary College, UPEI where she created the clinical, didactic and research program which trains veterinary students, residents, and graduate students. Dr. Overall lectures at meetings and veterinary schools worldwide and consults internationally with governments, NGOs and working dog and welfare organizations. She is the author of hundreds of scholarly publications, textbook chapters, commentaries, et cetera and the texts Clinical Behavioural Medicine for Small Animals (1997; Elsevier) and Manual of Clinical Behavioural Medicine for Dogs and Cats (2013; Elsevier) and of the DVD, Humane Behavioural Care for Dogs: Problem Prevention and Treatment (2013; Elsevier). She is the editor-in-chief for Journal of Veterinary Behaviour: Clinical Applications and Research (Elsevier). Dr. Overall has been named the North American Veterinary Conference (NAVC) Small Animal Speaker of the Year and was named one of the The Bark's 100 Best and Brightest - Bark Magazine's list of the 100 most influential people in the dog world over the past 25 years. Her research interests include psychopharmacological treatments of anxiety and new drug development, behavioural genetics of anxiety disorders, and effects of early trauma on the behavioural development and later behavioural pathology of kittens and puppies.





## Jane Pegg, DVM, MS, DAVDC

Founder & Medical Director, Ascentra Veterinary Dentistry and Oral Surgery

Dr. Pegg is a board-certified vet dentist and founder of Ascentra VDOS and Transcend Vet CE. She's passionate about empowering veterinary teams through hands-on training and advancing pet care through excellence in dentistry and oral surgery.



## Charlie Pye, DVM, DVSc, DACVD

Associate Professor Dermatology, Atlantic Veterinary College

Dr Charlie Pye grew up outside of London, England and moved to Prince Edward Island, which she now calls home, at the age of eighteen. There she attended the University of Prince Edward Island where she completed a three-year bachelor of science undergraduate degree majoring in Biology. She went on to receive her Doctorate of Veterinary Medicine from the Atlantic Veterinary College, PEI. She then moved to Saskatoon to complete a one-year rotating internship at the Western College of Veterinary Medicine. Following her internship, she travelled back across the country for a Dermatology Residency at the Ontario Veterinary College. While at OVC, she also completed her Doctorate of Veterinary Science degree specializing in *Pseudomonas aeruginosa* bacterial biofilms. After completing her residency she began working at Guelph Veterinary Specialty Hospital in Guelph, Ontario. During her time in Guelph she continued to travel back to PEI to teach the veterinary students at the Atlantic Veterinary College a few times a year. As of May 2018 she joined the team at the Atlantic Veterinary College and established the first ever Dermatology service at AVC. She has lectured all over North America and has contributed to multiple journals and textbooks. She is also currently the treasurer for the Canadian Academy of Veterinary Dermatology and an advisor for the Canadian Pre Veterinary Medical Association. In her spare time she enjoys camping, crafting and spending time with her husband, daughter and son. She is also "owned" by two dogs (both of which have allergies!).



## Debbie Reynolds, BVSc, BSc, DACVS-SA

Specialist Surgeon, Toronto Animal Health Partners

Dr. Debbie Reynolds completed a Veterinary Degree at the University of Queensland in 1999 at which time she began working as a mixed animal veterinarian in rural Australia. After chasing horses and cattle around the countryside for 3 years, she decided to pursue a small animal surgery specialty. The pursuit of a surgery residency brought her to North America, initially Washington State for a rotating internship which was followed by a research fellowship studying stem cells before being accepted to a surgery residency at Ontario Veterinary College. Following completion of a residency in 2012, she began working in Toronto as specialist surgeon providing both orthopedic and soft tissue surgery. She joined Toronto Animal Health Partners in 2019 and performs both orthopedic and soft tissue surgery with a special interest in joint replacements (total hip replacement) and minimally invasive surgery.



### Dan Riskin, PhD

Renowned evolutionary biologist, award-winning TV presenter, and bestselling author Dr. Riskin has spent over a decade making science accessible, engaging, and fun. Known for co-hosting Discovery Canada's Daily Planet, hosting Animal Planet's Monsters Inside Me, and serving as CTV's Science and Technology Specialist, his passion and curiosity inspire audiences worldwide. An expert on bat biomechanics with a PhD from Cornell, Riskin left academia to focus on science outreach, appearing regularly on television and radio, and leading wildlife tours to destinations like Borneo, the Amazon, and the Galapagos. He has hosted documentaries for National Geographic, CBC, W5, and more, and been interviewed by the likes of Anderson Cooper, Gayle King, and Craig Ferguson—who called him "my favourite scientist." Dr. Riskin is the author of *Mother Nature is Trying to Kill You* and the children's book *Fiona the Fruit Bat*, and publishes the popular science newsletter *The Bat Signal*. With humour, charisma, and boundless enthusiasm, he sparks audiences' curiosity and inspires them to explore, discover, and dream big.



### Shoshana Verton-Shaw

RVN, VTS (Nutrition), RLAT

Shoshana graduated from the Veterinary Technology program at University of Guelph, Ridgetown Campus with honors and as the recipient of the Award for Proficiency in Canine and Feline Nutrition in 2007. She became the first Registered Veterinary Technician in Ontario to achieve her Veterinary Technician Specialty in Nutrition in 2015 and has been active with the Academy of Veterinary Nutrition Technicians since. In 2026, furthering her dedication to scientific advancements in veterinary medicine, Shoshana successfully achieved her Registered Laboratory Animal Technician certification with the Canadian Association of Laboratory Animal Science. Joining the Ontario Veterinary College (OVC) Clinical Nutrition and Pet Nutrition Research teams in 2019, supporting the Clinical Nutrition Service, patient care, student learning and research. In 2018, Shoshana also joined Fanshawe College first to develop courses, then as a part-time faculty for their new animal health programs. In 2026, she changed roles at OVC to manage the new Hill's Pet Weight Care Program. Shoshana enjoys sharing and igniting a passion for veterinary nutrition in her peers, with a special interest in canine and feline obesity management and performance dog nutrition.

## EQUINE PROGRAM



### Jamie Kopper, DVM, PhD, DACVIM-LAIM, DACVECC-LA

Assistant Professor, Equine Medicine, Iowa State University

Dr. Jamie Kopper is board certified in large animal internal medicine and emergency/critical care. She is currently on the equine internal medicine team at Iowa State University and the large animal chief medical officer for the hospital. Clinically, she enjoys treating emergency and critical care patients, particularly those with gastrointestinal disease.



### Kyla Ortved, DVM, PhD, DACVS, DACVSMR

Associate Professor of Large Animal Surgery, New Bolton Center, University of Pennsylvania

Dr. Kyla Ortved is an Associate Professor of Large Animal Surgery at New Bolton Center, University of Pennsylvania in Kennett Square, PA. She received her DVM degree from the University of Guelph in 2006 and completed her large animal surgical residency training at Cornell University in 2010. Kyla became boarded with the American College of Veterinary Surgeons in 2011. Following her residency, Kyla went on to obtain a PhD in gene therapy for equine cartilage repair at Cornell. In February 2016, Kyla became boarded with the American College of Veterinary Sports Medicine and Rehabilitation. She joined the large animal surgery faculty at New Bolton Center in 2016 as an equine orthopedic surgeon and was named the Jacques Jenny Endowed Chair of Orthopedic Surgery in 2019. Her research program focuses on understanding the pathophysiology of equine osteoarthritis and developing gene and cell-based therapies to improve cartilage repair and prevent osteoarthritis.

## FOCUS ON INFECTION PROGRAM



### Maureen Anderson, DVM, DVSc, PhD, DACVIM

Lead Veterinarian – Animal Health & Welfare, OMAFRA

Maureen Anderson is a graduate of the Ontario Veterinary College and is ACVIM board-certified in large animal internal medicine. Her graduate and post-graduate work had a strong focus on infectious disease control and zoonotic diseases in particular. She is currently Lead Veterinarian - Animal Health and Welfare at the Ontario Ministry of Agriculture, Food and Agribusiness, where she continues to work in areas bridging animal and public health, including rabies and antimicrobial stewardship, and co-leads the companion animal Ontario Animal Health Network (OAHN).



### Ashley Spencer, DVM, MHSc, DACVIM

Assistant Professor, Ontario Veterinary College, University of Guelph

Dr. Ashley Spencer is a board-certified specialist in Small Animal Internal Medicine with a growing focus on infectious diseases of dogs and cats. She earned her DVM from the Ontario Veterinary College, completed a Master of Health Science in Community Health and Epidemiology at the University of Toronto, and undertook specialty training at North Carolina State University. Her background includes clinical practice, public health, and epidemiology, and she is currently launching research on vector-borne diseases in Ontario dogs. Dr. Spencer is passionate about advancing the understanding of infectious diseases in veterinary medicine and sharing practical, evidence-based approaches.



### Scott Weese, DVM, DVSc, DACVIM

Professor, Ontario Veterinary College, University of Guelph

Dr. Weese is a veterinary internist and Professor at the Ontario Veterinary College, University of Guelph, Director of the University of Guelph Centre for Public Health and Zoonoses, Chief of Infection Control at the Ontario Veterinary College Health Sciences Centre, and is Chair of the WHO Advisory Group for Critically Important Antimicrobials in Human Medicine. He runs the infectious disease website WormsAndGermsBlog (<http://www.wormsandgermsblog.com>)

## PRACTICE MANAGEMENT PROGRAM:



### Angie Arora, MSW, RSW

Founder and Veterinary Social Worker, Arora Wellness

Angie Arora is the founder of Arora Wellness, where she helps animal care organizations and professionals improve professional wellbeing. As a Veterinary Social Worker and Certified Compassion Fatigue Specialist, she provides trauma-informed therapeutic coaching, training, and courses to veterinary hospitals, animal shelters, and wildlife and conservation organizations working in high-stress, trauma-exposed environments. Angie is a member of the World Small Animal Veterinary Association's Professional Wellness Committee and serves as faculty with blendVET, the first DEIB certification program for veterinary professionals. She previously sat on the inaugural Board of Directors for the International Association of Veterinary Social Work and was Governance Chair for the Canadian Collective for Equity in Veterinary Medicine. An international speaker, Angie has presented at leading conferences such as WSAVA, CVMA, OVMA, OAVT, ACVIM, Fetch dvm360, the International Veterinary Social Work Summit, MCVMA Rise, and The UK Vet Congress, where she advocates for trauma-informed approaches to professional wellbeing. She holds a Bachelor of Social Work from Toronto Metropolitan University and a Master of Social Work from York University and is certified in Emotional CPR (National Empowerment Centre), Compassion Fatigue (Traumatology Institute), and has completed ICF coaching training with the Mind Rebel Academy.



### Megan Brashear, BS, RVT, VTS (ECC)

Senior Manager, Veterinary Nursing, Purdue University Veterinary Hospital, West Lafayette Indiana

Megan Brashear, BS, RVT, VTS (ECC) graduated in 2000 with a BS in Veterinary Technology and obtained her Veterinary Technician Specialty in Emergency/Critical Care in 2004. She has enjoyed working in emergency and critical care since 2000 and is the Senior Manager of Veterinary Nursing at the Purdue University Veterinary Hospital in West Lafayette, Indiana. Here, Megan truly enjoys the opportunity to work with veterinary nursing supervisors as well as teach and train technicians and students in the hospital. She loves to travel and lecture sharing her knowledge with veterinary technicians and veterinary nurses around the world.



### Christopher Doherty, DVM, MBA, CBV

Assistant Director, Strategic Business Research and Outreach, American Veterinary Medical Association

Dr. Chris Doherty is Assistant Director for Strategic Business Research and Outreach at the AVMA. Dr. Doherty obtained his veterinary degree at the Ontario Veterinary College, his MBA at McMaster University's DeGroote School of Business, and is a Chartered Business Valuator. In his role with the AVMA, he analyzes data pertaining to new and early career veterinarians, as well as the overall population of veterinarians, and works in tandem with the rest of the Veterinary Economics Division team to aid in the translation of research findings into tools, resources, and actionable items that veterinarians can utilize and implement in their practices and their careers.





## Andy Roark, DVM, MS

CEO/Founder, Uncharted Veterinary Conference

Dr. Andy Roark is a practicing veterinarian in Greenville SC and the founder of the Uncharted Veterinary Conference. He has received the VMX Practice Management Speaker of the Year Award four times, the WVC Practice Management Educator of the Year Award, the Outstanding Young Alumni Award from the University of Florida's College of Veterinary Medicine, and the Veterinarian of the Year Award from the South Carolina Association of Veterinarians.



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# SMALL ANIMAL PROGRAM

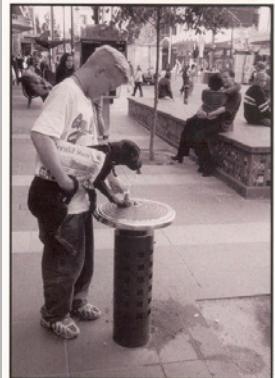
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2001

# HOW DO BEHAVIOURAL MEDICATIONS WORK?

SMALL ANIMAL PROGRAM | BEHAVIOUR

 Karen L. Overall, MA, VMD, PhD, DACVB



## How do behavioural medications work?

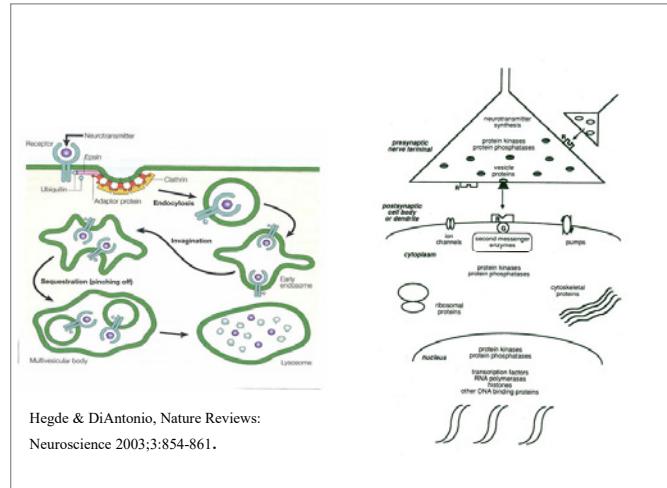
Karen L. Overall, MA, VMD, PhD, Diplomate ACVB  
AVC UPEI  
Charlottetown, PE, Canada

[koverall@upei.ca](mailto:koverall@upei.ca)

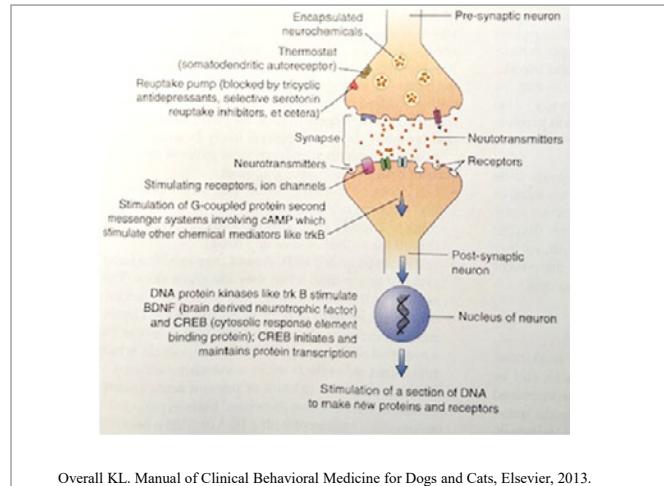
What do we **live for**,  
if it is not to make **life**  
less difficult for each other?

[George Eliot]

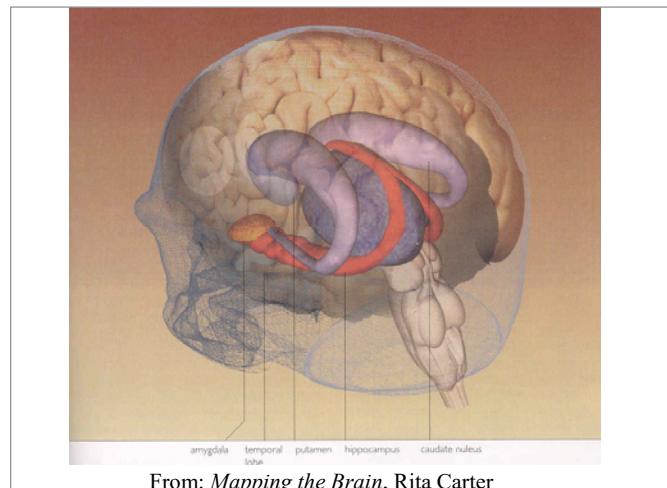
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3



4

## Medications affecting GABA

5

## Benzodiazepines

- diazepam (Valium)
- clorazepate (Tranxene)
- chlordiazepoxide (Librax)
- alprazolam (Xanax)
- oxazepam (Serax)
- lorazepam
- midazolam

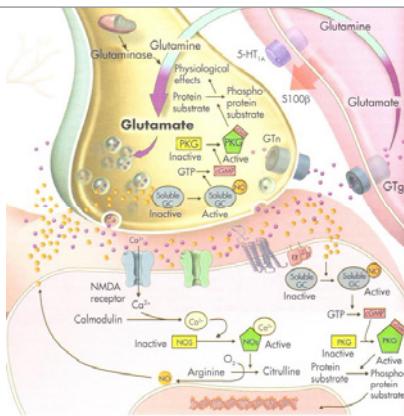
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## Benzodiazepines

7

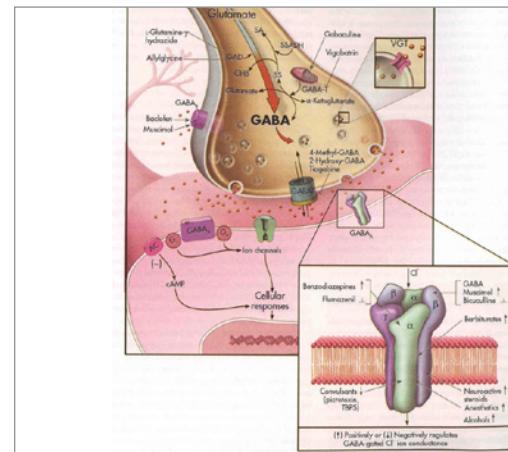
## Gamma amino butyric acid (GABA) and glutamate effects

8



The American Psychiatry Publishing Textbook of Psychopharmacology, 3rd Edition, Schatzberg and Nemeroff, 2004.

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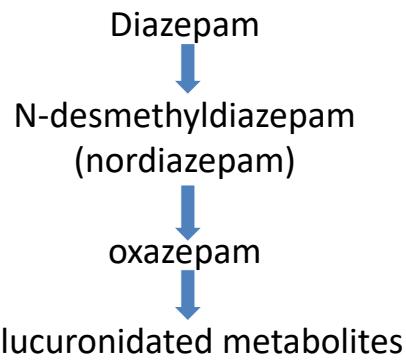


American Psychiatric Publishing Textbook of Psychopharmacology

10

What about hepatic toxicity in cats given diazepam?

11

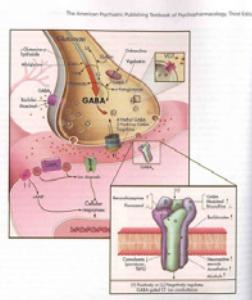


Note: alprazolam entirely skips this pathway and instead has hydroxylated intermediate metabolites that are then glucuronidated.

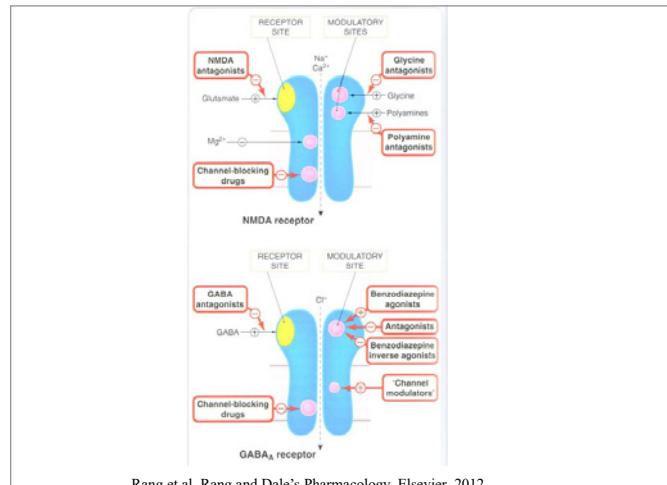
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### GABA-A receptors

- GABA-A receptors are predominant receptors for GABAergic neurotransmission; binding of GABA and GABA-A receptors increases neuronal membrane conductance for  $\text{Cl}^-$  ions leading to membrane hyperpolarization
- pentobarbital and other barbiturates allosterically enhance GABAergic neurotransmission by increasing the mean channel open time in a GABA-dependent fashion
- diazepam and other benzodiazepines enhance the actions of GABA at the GABA-A receptor by increasing the probability of  $\text{Cl}^-$  channel opening and  $\text{Cl}^-$  conductance



13



Rang et al. Rang and Dale's Pharmacology, Elsevier, 2012.

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### Half-lives of benzodiazepines & their intermediate metabolites in humans

Drug	$t_{1/2}$ parent compound	$t_{1/2}$ metabolite	Overall duration of action
triazolam	2-4 h	2 h	Ultra short; < 6 h
oxazepam	8-12 h		Short: 12-18 h
alprazolam	6-12 h	6 h	Medium: 24 h
diazepam	24-40 h	60 h	Long: 24-48 h
clonazepam	50 h		Long: 24-48 h

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### Diazepam in domestic animals

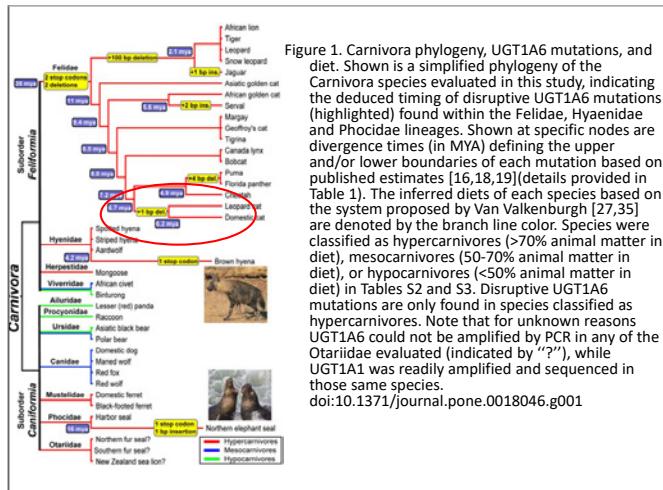
Animal	Diazepam $t_{1/2}$	$t_{1/2}$ N-desmethyl-diazepam
horse	24-48 h	51-120 h
cat	5.5 h	21 h
dog	3.2 h	3.6 h

16

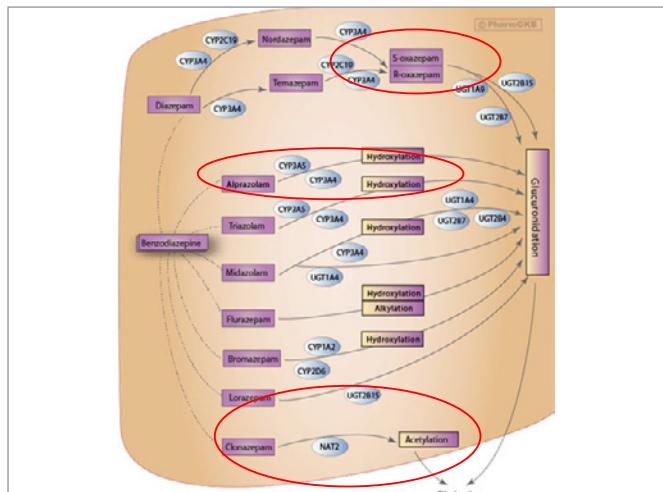


What about hepatic toxicity in cats given diazepam?

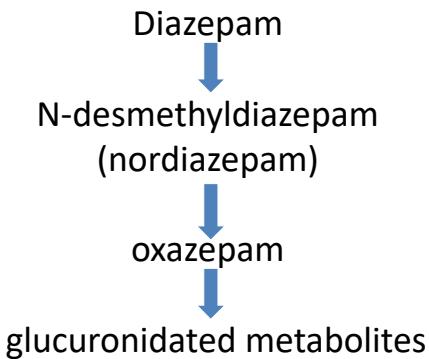
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Note: alprazolam entirely skips this pathway and instead has hydroxylated intermediate metabolites that are then glucuronidated.

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## Benzodiazepines and hepatotoxicity

- A role for N-desmethyl diazepam and UGT metabolism
  - oxazepam and alprazolam do not go through this pathway
- A role for lipophillia
- A role for accurate diagnosis (eg, not 'aggression')
- What about feline metabolism?
- How do we dose: per cat v per kg?
- Generic v. brand name differences?
- What about the international experience?

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## BZ uses in cats

- Fear prior to coming to vet's
- Fear / panic engendered while at vet's
- Grief and anorexia (oxazepam)
- Stress / anxiety / anorexia in new social situation (oxazepam, alprazolam)
- Aggression between cats for victim cat (oxazepam, alprazolam)

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## Relevant dosages for cats

- Alprazolam: 0.0125-0.025 mg/kg po prn q 4-6 h (give 2 h & 30 minutes before provocative circumstance)
- Lorazepam: 0.125-0.25 mg/kg po q. 12-24 h
- Oxazepam: 0.2-0.4 mg/kg po q 12-24 h
- Diazepam: 0.2-0.4 mg/kg po q 12-24 h (last choice)
- Start low; be aware with any medication with a long half-life repeat dosages will be additive

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## BZ uses in dogs

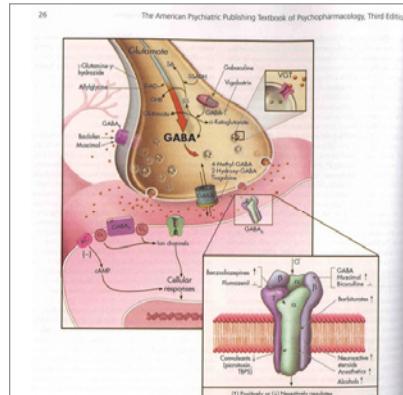
- Storms / noise – preventative (alprazolam >clonazepam diazepam > other BZ)
- Panic / extreme distress unpredictable storm / noise (alprazolam)
- Fear of car rides (alprazolam)
- Fear of vet visit (alprazolam)
- Fear generated by vet visits (alprazolam)
- Generalized or situational anxiety (alprazolam + loperamide – MDR1 concerns; oxazepam; clonazepam)

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## BZ dosages in dogs

- Alprazolam – 0.01-0.1 mg / kg po as often as q. 4-6 h prn
  - For anticipated problem given 1<sup>st</sup> ½ dose 2 h before and 2<sup>nd</sup> ½ dose 30 minutes before to take advantage of intermediate metabolites and combined half-lives
- Oxazepam – 0.5-2.0 mg / kg po q. 4-6 h
- Clonazepam - 0.01-0.1 mg/kg starting dose and go up to ~0.5 mg/kg but no more than 4 mg/day (small dogs may have surprisingly high dosages)
- Diazepam – 0.5-2.0 mg / kg po q. 4-6 h prn (generally a last choice – sedative)

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Flumazenil: 0.008-0.04 mg/kg IV, IM, submucosal, endotracheal or rectal for profound BZ induced sedation

The American Psychiatry Publishing Textbook of Psychopharmacology, 3rd Edition, Schatzberg and Nemeroff, 2004.

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## Neuroactive / excitatory amino acids

- *glutamate* - *glutamate decarboxylase (GAD)* = enzyme involved in metabolism
- GAD is responsible for *GABA* synthesis and is diminished in interneurons in discrete regions of the epileptogenic cortex and hippocampus

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## Considering EAA and why glutamate may be a culprit

- potentially, to sensitize excitatory synapses
- synaptic remodeling associated with this sensitization may underlie psychiatric and cognitive conditions
- increased in neurocytotoxic states (e.g., stroke)
- increased in human, rodent, primate, cat epileptogenic cerebral cortex, as is GAD
- increased in epileptic hippocampi and increases before seizure to potentially excitotoxic levels
- increased, possibly, in impulsive states
- with serine and aspartate increased in human status epilepticus

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## Gabapentinoids

*Gabapentin and pregabalin*

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## Gabapentin uses in humans

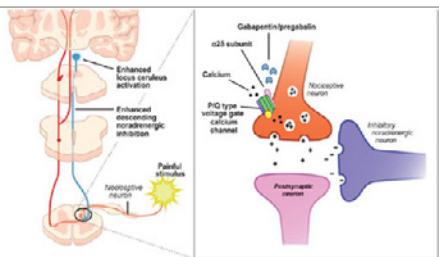
- Used in the treatment of:
  - Epilepsy – adjunctive for partial seizures with only mild side effects (sedation, dizziness, ataxia)
  - Neuropathic pain
  - Movement disorders
  - Migraine
  - Anxiety disorders – panic disorder, social phobia
  - Bipolar disorder
  - Substance abuse and withdrawal

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## Gabapentin uses and potential uses in dogs

- Seizures – adjuvant
- OCD – adjuvant
- Anxiety – primary or adjuvant in non-specific anxiety and/or as seen in GAD

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- Gabapentin selectively binds to the  $\alpha 2\delta$  subunit of voltage gated calcium channels (cerebral cortex, superficial dorsal horn, cerebellum, hippocampus) and inhibits calcium influx through these channels.
- This inhibits the evoked release of glutamate, aspartate, substance P, and calcitonin generated peptide (CGRP) from the primary afferent nerve fibres in pain pathways and reduces neuronal hyper excitability.
- Gabapentin also acts on spinal  $\alpha 2$  adrenoreceptor to produce analgesia by activating the descending spinal nor-adrenergic system by releasing noradrenaline.

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## Gabapentin

- Originally considered GABA analog
- Does not act as GABA precursor, agonist or antagonist
- Increases brain and intracellular GABA by a amino acid active transporter at the BBB and via multiple enzymatic regulatory mechanisms among which is thought to be *the increase of GAD*
- Increases non-synaptic GABA release from glia
- Good modulator of glutamate metabolism through binding Ca++ receptors ( $\alpha 2\delta$  subunit binding) in excitatory neurons*
- Also modulates Na+ channels and increases whole blood serotonin concentrations*
- Not well excreted with renal failure

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## Gabapentin dosage and 'side' effects

### Undesirable effects:

- Few because no active metabolites (40% at most(av = 32%) converted in dogs and may depend on dose; none in cats/humans; few in rodents) and no plasma binding, hepatic metabolism or CPY 450 autoinductions (so....kidney sees the entire amount of the parent compound in cats, humans and majority of it in dogs, rodents)
- $t_{1/2} = 6-8$  in humans (dogs 4-6 h; cats 4 h)
- Dose 1-3 times a day (*dogs to start or for pain: 10-20 mg/kg; cats to start or for pain: 5-10 mg/kg; dogs for anxiety: to 30-50 mg/kg; cats for anxiety: to 10-20 mg/kg to start*) (depending on condition in humans dose is 600-3,600 mg/d)

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## Pregabalin

- Linear pharmacokinetics – more predictable since the dose mirrors concentration, fewer adverse effects
- Bonqat – Zoetis US – for cats (5 mg/kg)
- For dogs – 4+ mg/kg

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## Adverse effects

- May be dose-dependent
- Increased appetite
- Sedation (usually transient – must distinguish from catching up on sleep because you are exhausted)
- Ataxia
- Agitation at high dosages in some patients
- Euphoria and complete lack of judgement in rare patients
- Tremors at high dosages in rare patients

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## Fluoxetine and clomipramine

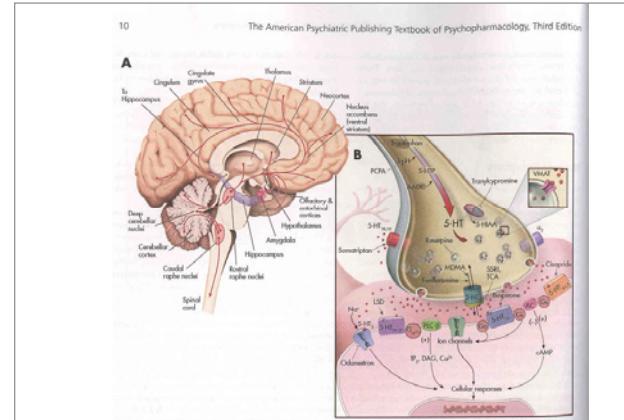
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## Medication affecting 5-HT and NE/NA

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## Serotonin pathways

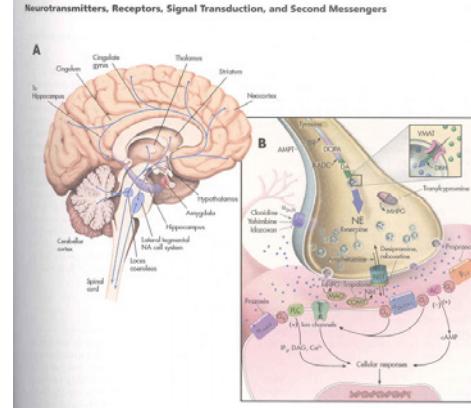
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## Norepinephrine pathways

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The American Psychiatry Publishing Textbook of Psychopharmacology, 3rd Edition, Schatzberg and Nemeroff, 2004.

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## Selective serotonin re-uptake inhibitors (SSRIs)

- fluoxetine (Prozac)
- sertraline (Zoloft)
- paroxetine (Paxil)
- fluvoxamine (Luvox)
- citalopram (Celexa)
- escitalopram (Lexapro)

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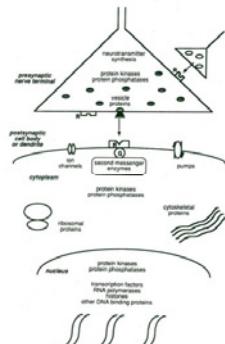
## Tricyclic antidepressants (TCAs)

- desipramine
- nortriptyline (Pamelor)
- imipramine (Tofranil)
- amitriptyline (Elavil)
- clomipramine (Anafranil; Clomicalm)

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Phases of plasticity and long-term potentiation (LTP) [specific process that occurs in learning]

- activity-dependent increase in synaptic efficacy that can last days to weeks
- induced by repeated, high frequency stimulation of neurons, particularly in the hippocampus
- 2 phases:
  - early protein synthesis independent phase that enhances information exchange, i.e., neurotransmission
  - late phase that's blocked by protein synthesis inhibitors and requires production of new proteins, i.e., new receptors

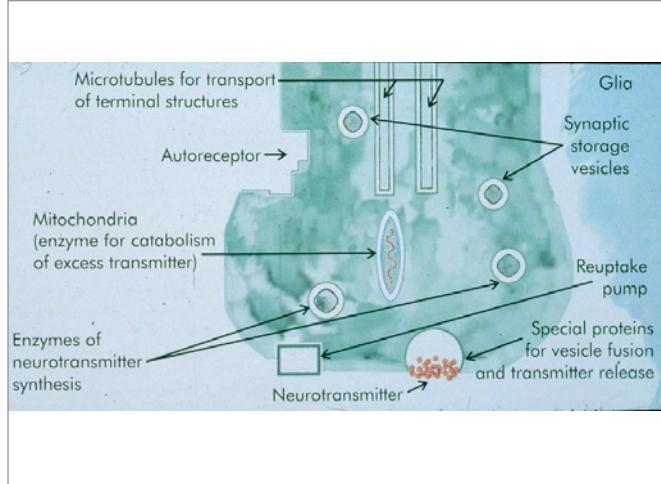


45

## Model for mode of action of TCAs and SSRIs: short-term effects

- TCAs and SSRIs cause short-term increases in 5-HT and NE by inhibiting reuptake and breakdown of relevant amines
- involves the somatodendritic auto-receptor in pre-synaptic neuron that controls the rate of firing
- initial decrease in rate of firing results, but there is overall increased receptor saturation
- increased saturation stimulates B-adrenergic coupled cAMP system
- profound effects then depend on changes induced in long-term treatment

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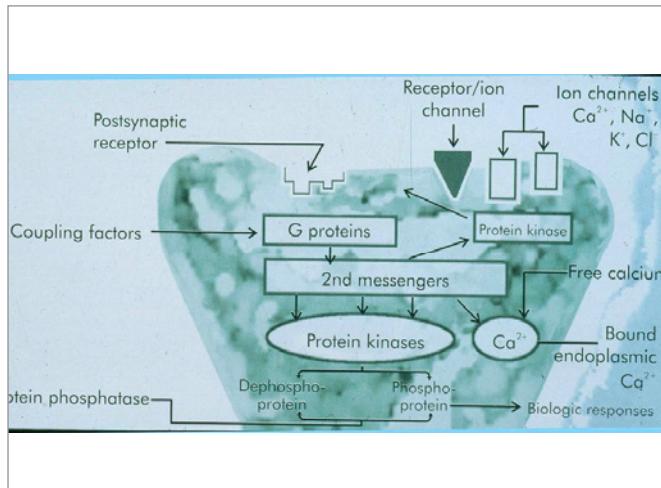


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## Model for mode of action of TCAs and SSRIs involves long-term, nuclear effects

- Stimulation of post-synaptic receptors increase the cAMP signal transduction pathway; protein kinase (PKA) increases moves into the cell nucleus
- PKA translocation increases cytosolic element binding protein (CREB) - **CREB may be the real post-receptor target for these drugs**
- increases in CREB lead to increases in brain derived neurotrophic factor (BDNF) (and vice versa)
- BDNF enhances growth of 5-HT and NE neurons, protects them from neurotoxic damage, and remodels receptors, altering function through mRNA transcriptional alterations in translated protein products (e.g., receptors)
- This may mean that we may need lower doses of medication than commonly used once stable....serotonin is the messenger, not the final target.**

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## TCAs, intermediate metabolites, and relative effects on NA & 5-HT

Compound	Metabolite	NA	5-HT
desipramine		++	+
imipramine	desipramine	+++	++
amitriptyline	nortriptyline	++	++
nortriptyline		+	+
clomipramine	desmethyl-clomipramine	++	+++

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TCAs and SSRIs	
• TCAs:	• SSRIs:
• Dosing for cats is generally 0.5 – 1.0 mg / kg po q. 12 h.	• Dosing for cats is generally 0.5 mg / kg po q. 24 h.
• For dogs dosing starts at 1.0 mg / kg po q. 12 h.	• For dogs dosing starts at 1.0 mg / kg po q. 24 h.
• For CMI need a target dose of 2-3 mg / kg po q. 12 h.	• Must treat for at least 8 weeks.
• Must treat for at least a month, and for CMI, 8 weeks.	

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Receptor subtype	Region of brain with mRNA activity
5-HT <sub>1A</sub>	Hippocampus (dentate gyrus, CA1, CA3), lateral septum, raphe nuclei, entorhinal cortex, central amygdala
5-HT <sub>1B</sub>	Globus pallidus, substantia nigra (pars reticula), olfactory pretectal nuclei, dorsal subiculum, superior colliculi (superficial layer)
5-HT <sub>1D</sub>	Globus pallidus, substantia nigra, caudate, putamen, accumbens nuclei, frontal cortex
5-HT <sub>2A</sub>	Claustro, olfactory tubercle, frontal cortex, neocortex (layer IV), raphe nuclei
5-HT <sub>2B</sub>	Cortex, amygdala, caudate, hypothalamus
5-HT <sub>2C</sub>	Choroid plexus, substantia nigra, globus pallidus, neocortex (layer III), hippocampus (CA1, CA3), raphe nuclei
5-HT <sub>3</sub>	Amygdala (baso-lateral nuclei), entorhinal cortex, hippocampus
5-HT <sub>3A/3B</sub>	Amygdala, hippocampus
5-HT <sub>4</sub>	Hippocampus (CA1), caudate, amygdala, colliculi (superior layer)
5-HT <sub>5</sub>	Hippocampus, habenula
5-HT <sub>5B</sub>	Habenula, hippocampus (CA1), raphe nuclei
5-HT <sub>6</sub>	Piriform & prefrontal cortex, striatum, hippocampus (CA1–CA3, dentate gyrus), amygdala
5-HT <sub>7A</sub>	Hippocampus, amygdala, cortex, raphe nuclei
5-HTP	Hippocampus

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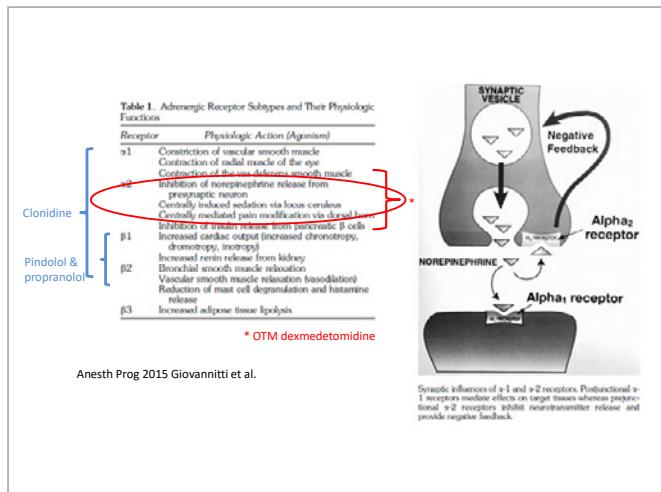
Parent compound	S-HT 1A	S-HT 1B	SHT1D	SHT2A	S-HT 2B	S-HT 2C	S-HT 3	$\alpha$ 1A	$\alpha$ 2A	$\alpha$ 1B	$\alpha$ 2B	D1	D2	H1	Ach
<b>TCAs</b>															
imipramine		++		++		++						++	++		
amitriptyline	+		++	++		++						++	++		
norimipramine	+		++	++		++						++	++		
clomipramine*IM=SSRI	IM		++	++	++	++						++	++		
<b>SSRIs</b>															
fluoxetine	++	+	+	+	+	++	+					+			
paroxetine	++														++
sertraline	++						++								
fluvoxamine*IM=SSRI	IM														
escitalopram/ citalopram							*** via SERT								
<b>SARIs</b>															
trazodone	++			++											
<b>Alpha-agonists</b>															
clonidine								+	++	++					
dexmedetomidine/Sileo								++	++	++					
<b>Beta blockers/antagonists</b>															
propranolol									++	++					
pindolol									++	++					

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## Alpha agonists



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## Alpha-2-agonists

- Clonidine
- \*Sileo® (Dexmedetomidine OTM)

\* Canine license

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Binding affinity (Ki in nM) alpha 1,2 agonists based on # alpha 2 ligands [the lower the Ki the smaller the amount of drug needed to bind 50% of the receptor sites even at low concentrations; Ki related to Kd - Ki helps determines efficacy and selectivity of drugs]

Drug	$\alpha$ 1A	$\alpha$ 1B	$\alpha$ 1D	$\alpha$ 2A	$\alpha$ 2B	$\alpha$ 2C
Clonidine	316.23	316.23	125.89	42.92	106.31	233.1
Dexmedetomidine	199.53	316.23	79.23	6.13	18.46	37.72

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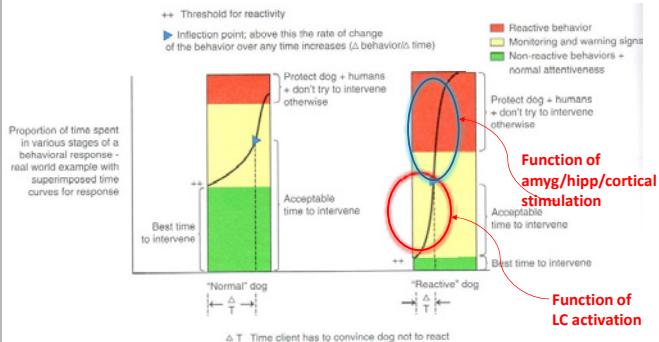
Parent compound	5-HT 1A	5-HT 5HT	5HT 5-HT	5-HT 5-HT	5-HT 3	$\alpha$ 1A	$\alpha$ 2A	$\alpha$ 1B	$\alpha$ 2B	D1	D2	H1	ACh
	1B	1D	2A	2B	2C								
<b>TCA</b>													
imipramine			**	**	**					***	***		
amitriptyline	*		***	***	***					***	***		
nortriptyline	*		***	***	***					***	***		
clomipramine (*IM=SSRI)			***	***	***	***				*	***	***	***
<b>SSRIs</b>													
fluoxetine	***	*	*	*	*	**	*			*			
paroxetine	**										*	***	
sertraline	***					***							
fluvoxamine* (*IM=SSRI)	IM												
escitalopram	***												
<b>5ARIs</b>													
trazodone	**		***	*		*					*		
<b>Alpha-agonists</b>													
clonidine						*	*	**	***				
dexmedetomidine/Sileo						***							
<b>Beta blockers/antagonists</b>													
propranolol							**	**					
pindolol							**	**					

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## Summary concept

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### When we treat, what do we seek to change?

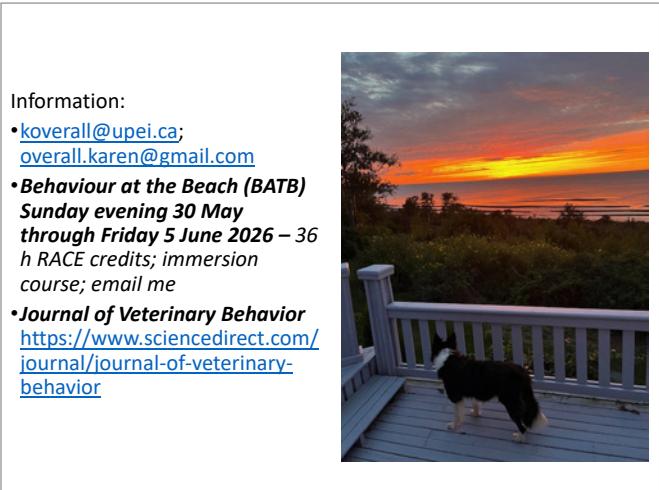


From Overall, 2013

61

The use of behavioral medication is about creating an effective, scientifically based, multi-focal, integrated and *humane* treatment program that respects everyone's needs. If we allow them to do so, our patients will change us for the better.

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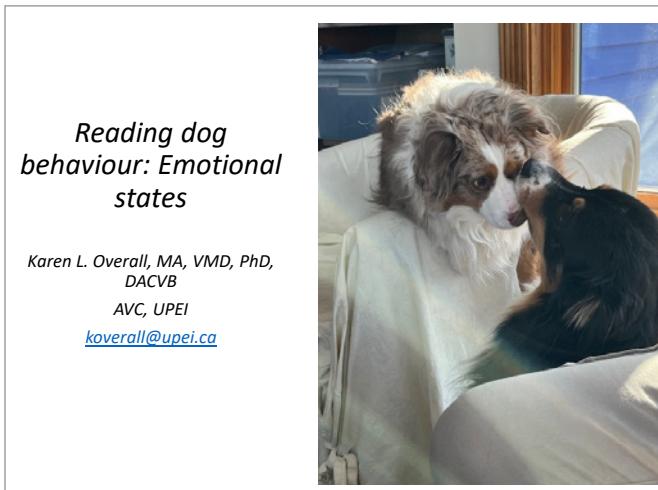


2002

# READING DOG BEHAVIOUR: EMOTIONAL STATES

SMALL ANIMAL PROGRAM | BEHAVIOUR

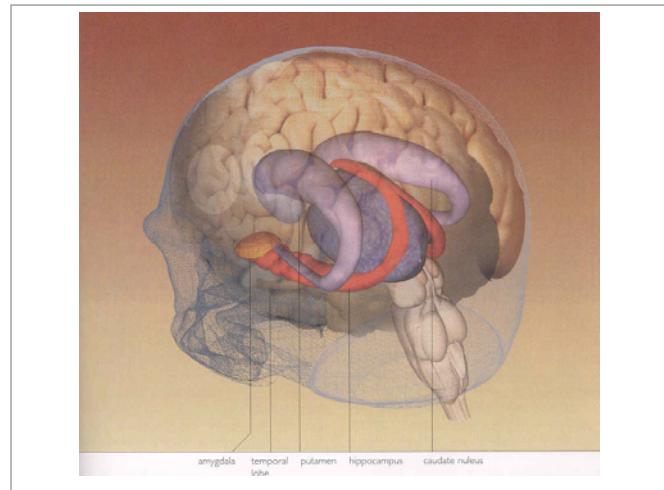
 Karen L. Overall, MA, VMD, PhD, DACVB



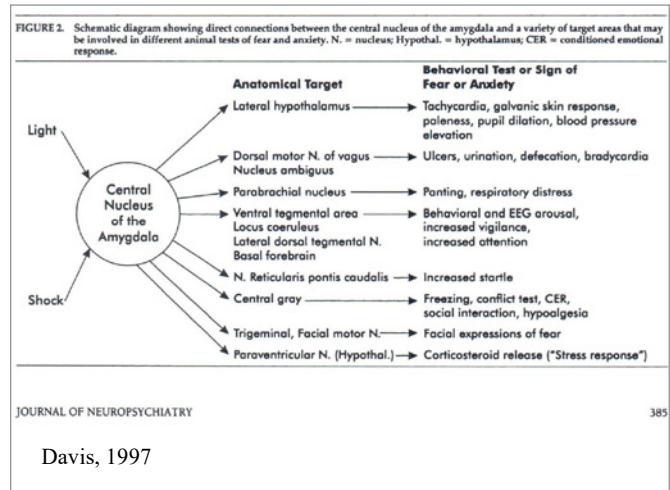
1

Roles for brain regions and non-specific signs of fear and anxiety

2



3



4

## What distinguishes fear and anxiety?

- Fear and anxiety have signs that overlap. Some non-specific signs like lowering of the back shaking and trembling can be characteristic of both fear and anxiety.
- The physiological signs of fear and anxiety **probably** differ at some very refined level, and the neurochemistries of each are probably very different in a way that is **not** addressed by the **relatively** non-specific medications we commonly use.

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• True fear is involves responses to stimuli (social or physical) that are characterized by withdrawal, and passive and active *avoidance behaviors* associated with the sympathetic branch of the autonomic nervous system, and in the absence of any aggressive behavior.

• Truly **fearful dogs** lower their entire bodies, necks, head, ears, and tails and appear to fold into their limbs (Galac and Knol, 1997). **Anxious dogs** do not do this and exhibit a wide range of stances.

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- True fear always involves avoidance, with an apparent intent to decrease the probability of social interaction. Dogs who are driven primarily by anxiety may put themselves into a social system, although it makes them uncomfortable and worried.
- Monitoring and vigilance are central to anxiety so removing yourself is not an option. Avoidance NOT first choice in anxiety

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## Summary – fear / anxiety comparison

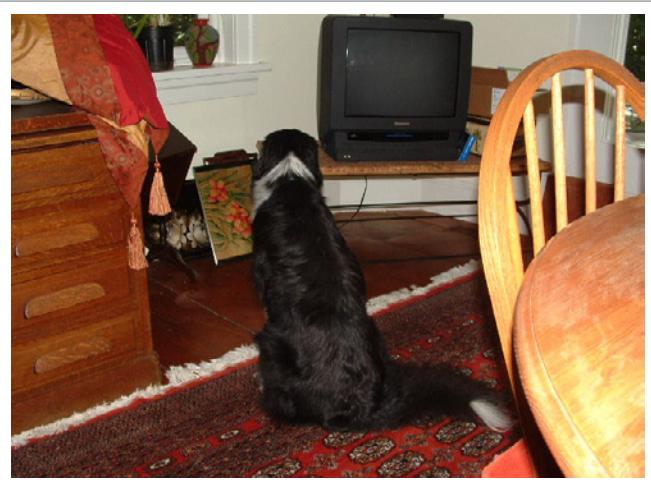
### Fear

- Response characterized by avoidance
- Response characterized by withdrawal
- Distance increasing
- May result in decreased social interaction
- Lower their entire bodies, necks, head, ears, and tails and appear to fold into their limbs – make small
- Sympathetic branch ANS – ‘fight or flight’
- Triggered by external stimulus, despite underlying liability

### Anxiety

- May be unable to avoid or withdraw
- Characterized by vigilance and scanning – monitoring is key and you must be present
- Avoidance not first choice
- May result in social contact
- Uncertainty – which may be endogenous – is key
- Enormous range and variability in body postures that may change with changes in interaction, distance and participants.
- May only be internally triggered

8



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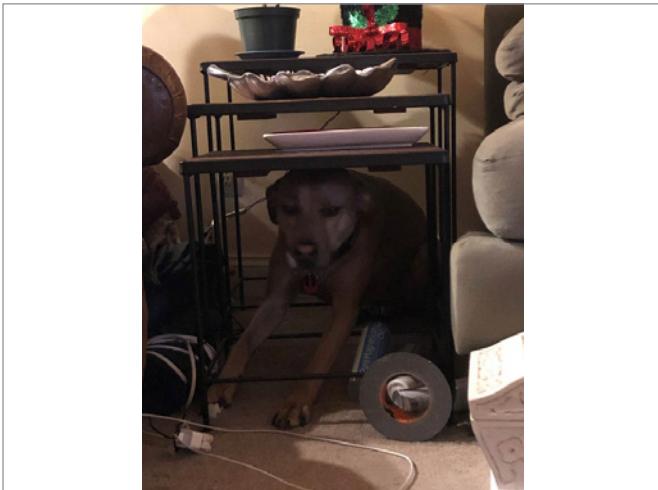


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## Phobia

- Profound, non-graded, extreme response to noise/storms with intense avoidance, escape, or anxiety as S ANS response. *This is a condition that is about arousal levels (NE in the LC)*. It also has a large cognitive component.
- Range of anxiety related behaviors from mania to catatonia, concomitant with decreased sensitivity to pain or social stimuli (meaning the threshold for response increases).

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## Anxiety - definition

- Consistent exhibition of increased autonomic hyperactivity and hyper-reactivity, increased motor activity, and increased vigilance and scanning that interferes with a normal range of social interaction.
- This behavior is due to the apprehension of risk or uncertainty.
- A feeling of apprehension about risk, characterized by physical autonomic nervous system signs, worsened by uncertainty.
- Can be part of very specific diagnoses (separation anxiety) or less specific ones (social anxiety).

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## Signs of anxiety

- Urination
- Defecation
- Anal sac expression
- Panting
- *Increased respiration and heart rates*
- *Trembling/shaking\**
- *Muscle rigidity (usually with tremors)*
- Lip licking
- Nose licking
- Grimace (retraction of lips)
- Head shaking
- Smacking or popping lips/jaws together
- *Salivation/hypersalivation*
- *Vocalization (excessive and/or out of context)*
- *Frequently repetitive sounds, including high pitched whines\*, like those associated with associated with isolation*

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- Yawning
- Immobility/freezing or profoundly decreased activity
- *Pacing and profoundly increased activity*
- Hiding or hiding attempts
- Escaping or escape attempts
- Body language of social disengagement (turning head or body away from signaler)
- Lowering of head and neck
- *Inability to meet a direct gaze*
- *Staring at some middle distance*
- Body posture lower (in fear, the body is extremely lowered and tail tucked)
- Ears lowered and possibly droopy because of changes in facial muscle tone
- Mydriasis
- Scanning
- *Hyper-vigilance/hyper-alertness (may only be noticed when touch or interrupt dog or cat – may hyper-react to stimuli that otherwise would not elicit this reaction)*

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- Shifting legs
- Lifting paw in an intention movement
- *Increased closeness/attentiveness to preferred associates*
- *Decrease closeness/attentiveness to preferred associates*
- Profound alterations in eating and drinking (acute stress is usually associated with decreases in appetite and thirst, chronic stress is often associated with increases)
- Increased grooming, possibly with self-mutilation
- Decreased grooming
- Possible appearance of ritualized or repetitive activities
- *Changes in other behaviors including increased reactivity and increased aggressiveness (may be non-specific)*

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Arousal v. reactivity – the experimental psychology definition – NOT the pop culture one

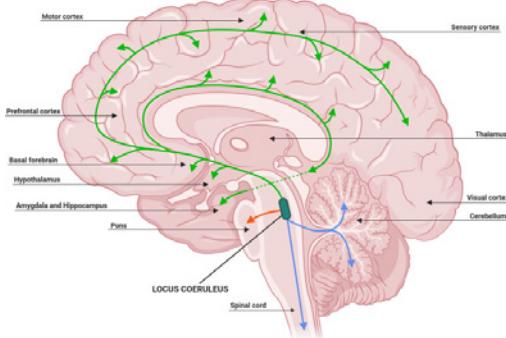
- **Arousal (the level to which you react)** and **reactivity (the number of behaviors you display and the number of situations across which you react)** is important in both fear and anxiety, although the specific patterns of behavior sort the groups.
- Part of the pathology may be in the arousal.

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Why do we want to avoid acute stress responses, fear and anxiety?

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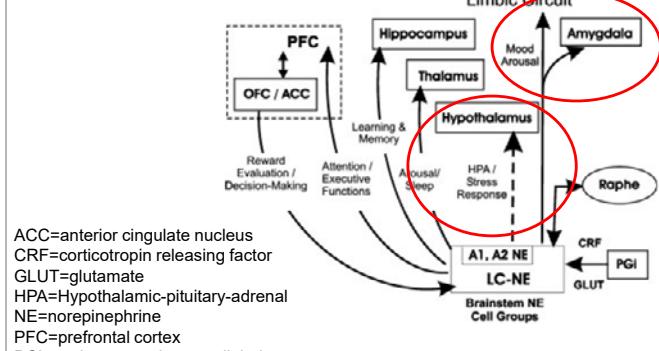
NE pathways/projections



Matchett et al. (2021) Acta Neuropathologica

20

Effects of LC stimulation

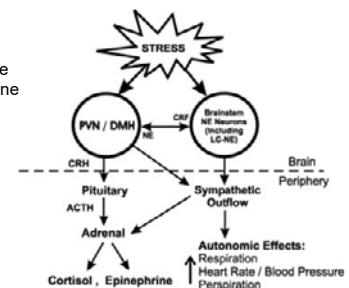


Goddard et al. (2010) Depression and Anxiety

21

Acute stress response: Increased NE activity stimulates corticotropin releasing hormone from the paraventricular nucleus of the hypothalamus, which then activates the hypothalamic-pituitary-adrenal-axis increasing sympathetic outflow to multiple peripheral organ systems

ACTH=Adrenocorticotrophic hormone  
CRH=corticotropin releasing hormone  
DMH=dorsal medial hypothalamus  
LC=locus ceruleus  
NE=norepinephrine  
PVN=paraventricular nu

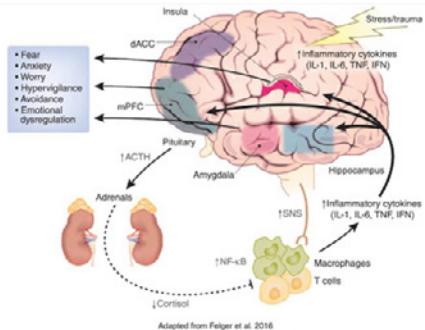


Goddard et al. (2010) Depression and Anxiety

22



### Neuroendocrine effects of fear/anxiety: inflammation

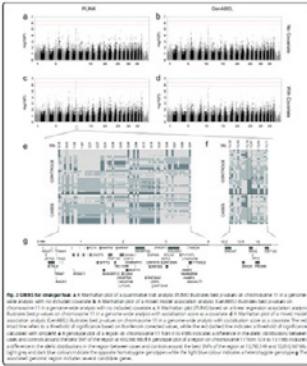


Michopoulos et al. (2017) *Neuropsychopharmacology Rev*

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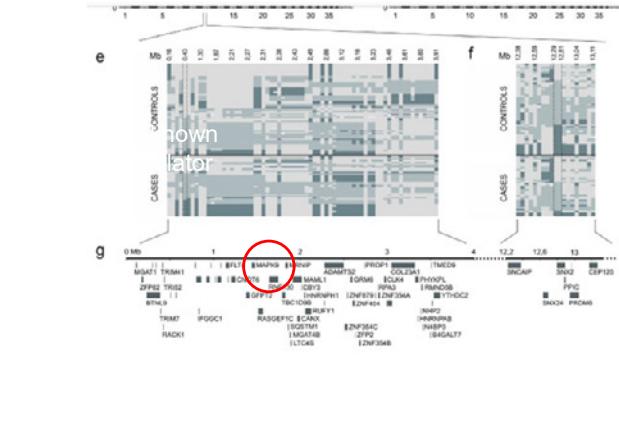
Are there genetic risk factors for fear?

Chr 11 and fearfulness in dogs



Sarviamo et al. (2020) *Trans Psych*

25



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Fear involves the amygdala (reactivity), the hippocampus (associative learning), and the frontal cortex (integration of information and planning of response). All of these regions are rich in **5HT1A** receptors.

The hypothalamus is likely involved in determining when the behavior shifts from passive to active threat. This involves **dopamine**.

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### Fear aggression

- **Out of context aggression** in situations where aggressor may have chosen escape from their fear, but lacks or acts as if they lack that choice.
- Passive escape and withdrawal may be attempted but are increasingly replaced by active aggression – proactive aggression – reflecting pathology within the amygdala/hypothalamus.
- Body postures include signals of avoidance and agonistic behavior: lowering of head and body, tucking of the tail, piloerection, ears moved back to wrinkled muzzles, horizontal and then vertical lip retraction, snarling, growling, barking.
- Affected dogs are often **hyper-vigilant** in contexts which may provoke their fear

28

- **Fear aggression** is an anxiety disorder where the anxiety prompts aggression in the contexts that elicit fear – passive escape and withdrawal are replaced by active aggression....likely this pattern reflects some pathology within the hypothalamus.
- Body postures include lowering of head and body, tucking of the tail, piloerection, ears moved back, wrinkled muzzles, horizontal and then vertical lip retraction, and snarling.

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- Vocal threats like growling, snarling, barking, generally occur while the dog is backing up with the tail tucked and back arched.
- Affected dogs are often *hyper-vigilant* in contexts which may provoke the fear: increased heart rate, increased respiration, shaking, trembling, salivation, mydriasis, lack of appetite, uncontrolled urination, uncontrolled diarrhea/loose stool, anal sac expression may accompany the increasing aggression that's a last resort.
- Be aware of the effects of duration of event and time penetrance (over time) on changing signs.
- Be aware of effects of changing distance on behaviours.

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- Fear aggression often occurs in the absence of an intended or overt threat.
- When intended or overt threats occur, neither fear nor fear aggression need be the choice. Signaling avoidance and disengagement conveys a lack of threat and can ramp down arousal levels.

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#### Generalized anxiety disorder - GAD

#### Generalized anxiety disorder (GAD)

- Consistent exhibition of increased autonomic hyper-reactivity (increased heart rate, increased respiratory rate, changes in gastrointestinal activity, and dilated/enlarged pupils), increased motor activity, and increased vigilance and scanning that interferes with a normal range of social interaction, **and** that occurs in the absolute absence of would be considered a truly provocative stimulus.
- These dogs/cats are reactive before any noticeable stimulus or trigger sets them off.
- Affected individuals do not easily habituate to or learn to ignore anything that arouses them.
- In novel situations these dogs cannot sit or lie down voluntarily and attend to others. In an office visit – if the dog is still exhibiting vigilance and scanning 20 minutes after entry, the GAD risk is great.

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#### Patterns of behavior

- These dogs CAN and DO relax and sleep when they are in secure circumstances (although they may not get **restorative sleep** – need movement measures). They are **not hyper-active**, where they cannot settle, run high heart rates and high temperatures, and become worse if restrained.
- These dogs **do not have situational anxiety**. There is no pattern to their monitoring (eg, strange humans, strange dogs, vehicles). Clients say that they react to everything.
- These dogs are **not under-exercised/over active**. Clients may avoid some activity because controlling the sequelae of the arousal exhausts them, but exercise makes a minimal difference.

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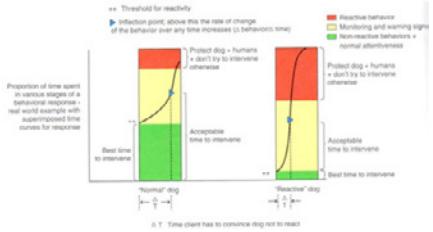
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- These dogs **may be hyper-reactive but need not have true hyper-reactivity** which is a neurodevelopmental diagnosis where – regardless of the stimulus – the dog innately cycles through calm and aroused cycles. These conditions could be co-morbid.
- These dogs arouse (= the level to which you react) so quickly that clients cannot interrupt early, and their arousal threshold appears to be quite low.

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## Roles for arousal

- These dogs arouse (= the level to which you react) so quickly that clients cannot interrupt early, and their arousal threshold appears to be quite low.



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## Roles for reactivity

- Reactivity is defined (in the experimental psychology literature on anxiety) as the number of situations in which you react and the number of reactions you demonstrate.
- GAD is an anxiety-based condition. It likely affects the LC, the hippocampus, the amygdala, the frontal cortex, and the hypothalamus.
- This is about how the cortex processes this reactive information; remember that high levels of the EAA, glutamate, are associated with the reactivity, and think about what is involved in social signaling and how complex the impairment can be:
  - Reading signals well,
  - Processing the information in the signals,
  - Using any information to formulate a response and/or
  - Signaling about that response.

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Given this....are you surprised that the most common client complaint in dogs diagnosis with GAD is aggressive behavior?



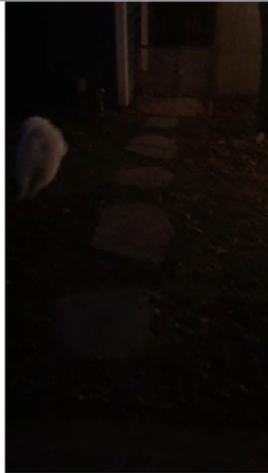
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- Screen for this early and often.
- We think that GAD may have a neurodevelopmental component and that increased reactivity of the amygdala may be caused by neonatal/maternal stress and that subsequent epigenetic effects that do not allow adequate glucocorticoid receptor responses to stressors.
- The result is chronic and easily provoked arousal.

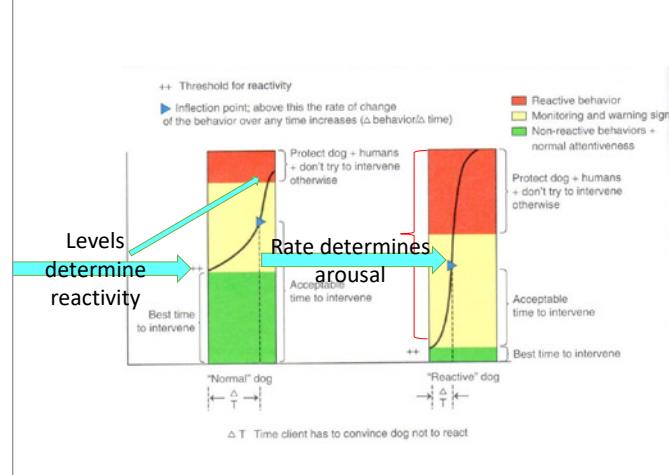
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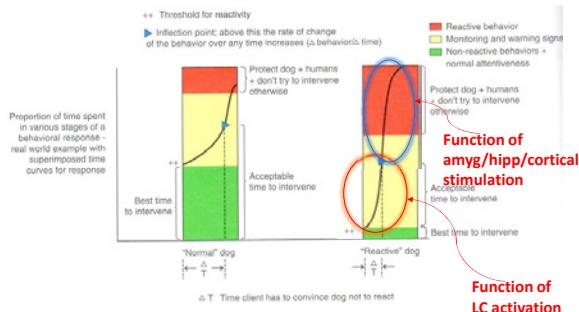
What if there is no real diagnosis? What if the emotional state is about likes and dislikes, and maybe some past history?

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## When we treat, what do we seek to change?



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## The solution?

- We treat both the fear or anxiety (SSRIs, TCAs, BZDs, gabapentinoids) and the arousal (alpha agonists – Sileo and clonidine).
- Protect
- Work to teach the dog to self calm.

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### Information:

- [koverall@upei.ca](mailto:koverall@upei.ca); [overall.karen@gmail.com](mailto:overall.karen@gmail.com)
- **Behaviour at the Beach (BATB) Sunday evening 30 May through Friday 5 June 2026 – 36 h RACE credits; immersion course; email me**
- **Journal of Veterinary Behavior**  
<https://www.sciencedirect.com/journal/journal-of-veterinary-behavior>



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2003

# NEW APPROACHES TO MINIMIZING STRESS & DISTRESS WHEN HANDLING DOGS & CATS VETERINARY VISITS

SMALL ANIMAL PROGRAM | BEHAVIOUR

Speaker Karen L. Overall, MA, VMD, PhD, DACVB

**New approaches to managing stress and distress when handling dogs and cats veterinary visits**

Karen L. Overall  
AVC UPEI; Charlottetown, PE, Canada  
[koverall@upei.ca](mailto:koverall@upei.ca)



Much of the information in this presentation is taken from Dr. Camille Squair's [my former resident/graduate student] MSc degree and publications.

1

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## Background:

### Behaviour at the Veterinary Clinic

- Within veterinary medicine, signs of distress have been normalized – and in many cases, expected – as part of routine care
- Contributing to this is that dogs who are fearful, withdrawn or frozen may be easier or quicker to examine.
- We need to alter our worldview to assess and understand the appearance of signs of distress (e.g. trembling, pacing, tucking their tail etc.) as a welfare concern



### Behaviour at the Veterinary Clinic

- Dogs have been shown to be distressed in waiting rooms, on scales, entering exam rooms and when examined<sup>1-3</sup>
- 78.5% of dogs showed fear on the examination table, and fewer than half the dogs entered the practice calmly<sup>4</sup>
- Each negative event an animal experiences at a vet clinic conditions them for future negative responses – subsequent visits then become more difficult and time consuming<sup>5</sup>
- Owners list stress for themselves and their pets as a reason to avoid/delay veterinary care<sup>6</sup>



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## Objective

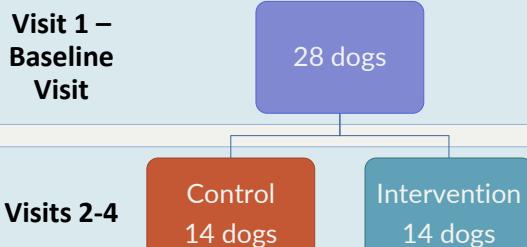
- Determine whether adaptive collaborative interventions differed from standard approaches to care in how they affect measurements of distress in canine patients during veterinary visits

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## Study Design

	30 dogs enrolled to achieve our target – 28 dogs completed	Questionnaire completed prior to enrollment
	4 visits across 8 weeks	Included physical exam, walking onto a scale, and blood draw
	Two treatment groups	Intervention and control

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## Control vs. Intervention - Scale

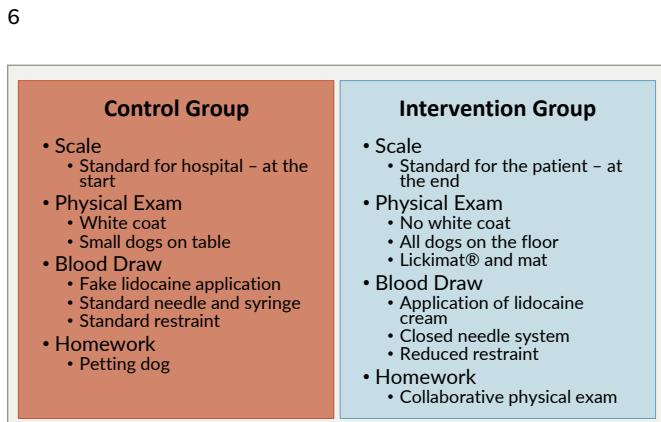
### Control Group

- Beginning of appointment
- No interventions

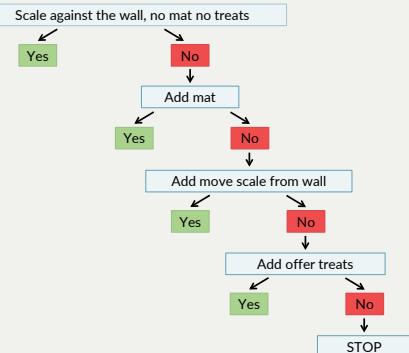


### Intervention Group

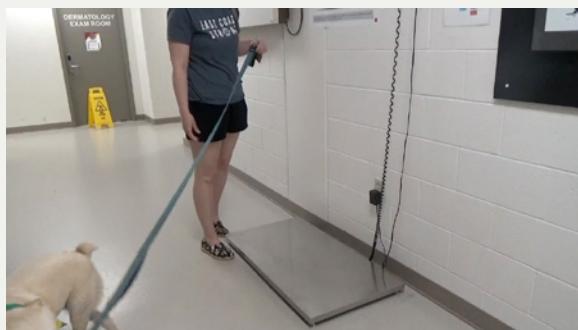
- End of appointment
- Blue mat, away from the wall, and with treats



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## Control vs. Intervention – Physical Exam

Same Standardized Physical Exam

### Control Group

- White coat worn
- Small dogs on the table



### Intervention Group

- No white coat
- All dogs examined on the floor
- Lickimat® and mat



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ONTARIO  
VETERINARY  
MEDICAL  
ASSOCIATION



### Control vs. Intervention – Blood draw

#### Control Group

- Fake lidocaine application
- Standard needle and syringe
- Standard restraint



#### Intervention Group

- Lidocaine cream (EMLA©)
- Closed butterfly needle system (vacutte)
- Reduced restraint



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### Control Group Blood Draw



14

### Intervention Group Blood Draw



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### Measuring Stress Responses

Used physiological and behavioural measures to assess distress within and between each group

#### Physiological measurements

- Heart rate (HR)
- Neutrophil lymphocyte ratio (NLR)
- Creatine Kinase (CK)
- Serum Cortisol (CORT)

#### Behavioural measurements

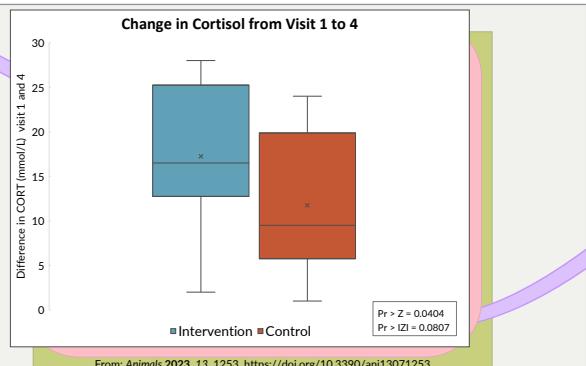
- Ordinal scale – in-person and video
- Ethogram
- Duration

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### Physiological measurements

Physiological marker	Stress measurement	Indication of adverse effect
Serum cortisol	Acute stress	Increased serum cortisol concentration <sup>7-10</sup>
	Chronic stress	Reduced serum cortisol concentration <sup>11-14</sup>
Neutrophil lymphocyte ratio	Acute stress	NLR ratio approx. 1:1 – neutrophilia and lymphocytosis <sup>15</sup>
	Chronic stress associated with inflammation	Increased NLR ratio – neutrophilia and lymphopenia <sup>16-21</sup>
Heart rate	Acute stress – immediate sympathetic response	Increased heart rate <sup>22-24</sup>
Creatine kinase	Muscle damage associated with panic	Increased CK concentration <sup>25,26</sup>

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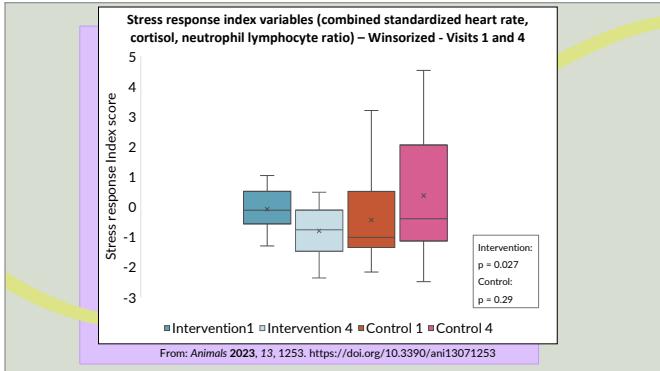
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### Statistical Analysis of Physiologic Parameters

- **Stress Response Index:** Because the effect on the individual is a combined measure of all response patterns – created a dimensionless composite index to assess the combined effect of the stress response indicators experienced
  - Summed standardized measures for HR, CORT and NLR
  - Winsorized to removed extreme outliers
  - Paired t-tests were used to test for differences between the first and fourth visit



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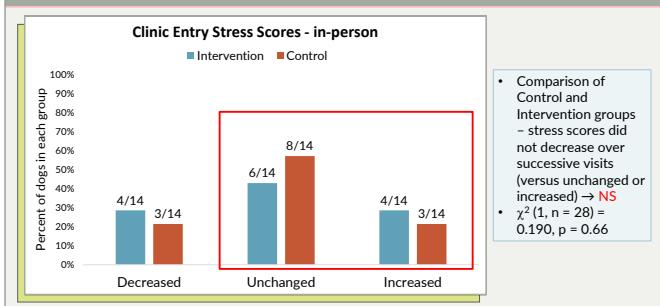
## Measuring Behaviour – Clinic Stress Scores

### Clinic Dog Stress Scale 4: The beginning and the end of the exam

Stress Level	Dog's Behaviour/Demeanour
0	Extremely friendly, outgoing, solicitous of attention
1	Calm, relaxed, seemingly unmoved
2	Alert, but calm and cooperative
3	Tense, but cooperative, panting slowly, not very relaxed but can still be easily manipulated for exam and cooperates with procedures
4	Very tense, anxious, may be shaking, whining or frozen, difficult to maneuver, tries to avoid exam, may hold onto table but not cooperate – more endures
5	Extremely stressed, barking/howling, tries to hide, needs to be lifted up or held to be examined or would bolt from table or room, difficult to control and may be tempted to bite, may not be able to complete exam.

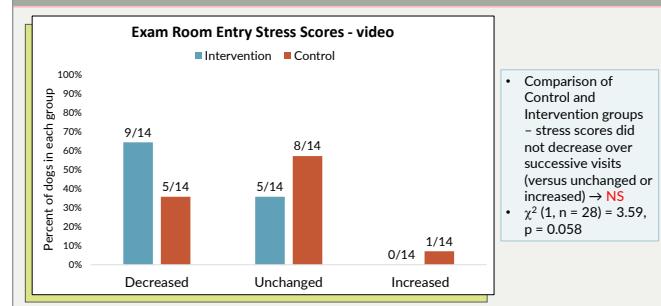
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## Clinic Stress Score – Clinic Entry



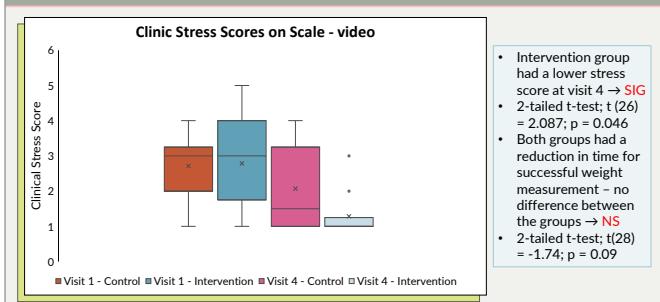
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## Clinic Stress Score – Exam Room Entry

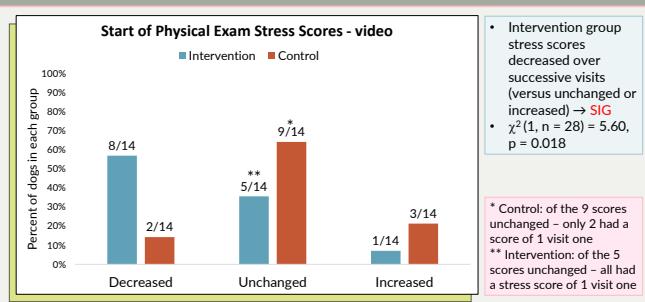


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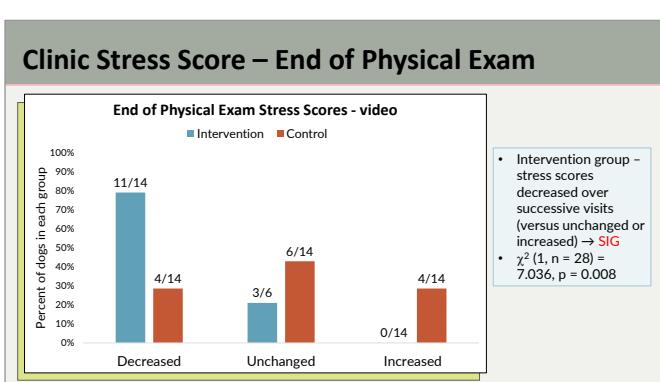
## Clinic Stress Score – Weight Scale



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## Ethogram Scale

- Components assessed
- Body posture
- Tail position
- Ear posture
- Gaze/eyes
- Mouth posture
- Activity
- Vocalization

Exam Steps
1. Pet
2. Pulse
3. Lidocaine
4. Heart
5. Lymph Nodes
6. Abdomen
7. Lift Paws
8. Lips
9. Ears
10. Temperature
11. Eyes
12. Venipuncture
13. Hand on back praise

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## Ethogram scale

SCORE	BODY POSTURE	TAIL POSITION
1	Relaxed, moves on own. Easily manipulated	Relaxed, normal position for the breed or held high.
2	Slightly tense Can be manipulated	Slightly lower than normal for the breed (but not completely down)
3	Stiff, possibly rigid. Body slightly lower	Completely down and low for the breed (but not tucked)
4	Can still be manipulated but less fluid (more rigid) Hunched (crouched) Low posture Difficult to maneuver	Tucked between legs
5	Curled Completely withdrawn – belly maximally tucked	Clamped hard up to the belly

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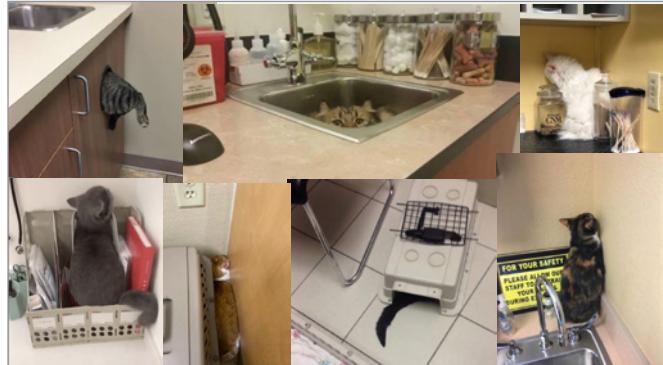
## Important takeaways - 1

- The goal was not to determine what specific intervention worked
- Rather, assessing the dog's complete experience from their perspective – what does the visit mean to them
- Determining the behavioural and physiological effects when simple and realistic low stress handling techniques are applied in a real-world setting
- Ultimately, reducing the level of distress experienced by veterinary patients can help to improve their welfare, care, health, and treatment outcomes

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*And cats??*

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## Ethogram Scores – Physical Exam

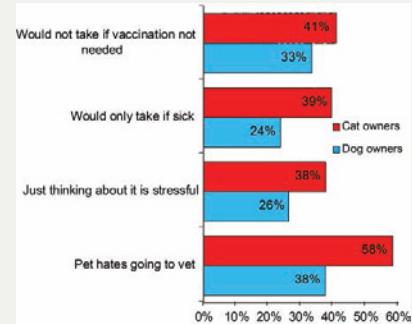
- Visit 4 – intervention group had significantly lower ethogram scores during:
  - Step 1: stroke the dog gently from head to base of tail ( $U = 46.0, p = 0.048$ )
  - Step 5: lymph node palpation ( $U = 35.0, p = 0.020$ )
  - Step 6: abdominal palpation ( $U = 40.0, p = 0.024$ )
  - Step 7: lifting each paw ( $U = 22.0, p = 0.002$ ),
  - Step 8: lifting upper lips ( $U = 29.0, p = 0.024$ ),
  - Step 11: examination of the eyes ( $U = 26.5, p = 0.018$ )
- Total ethogram score at visit 4 lower in intervention than control ( $U = 13.0, p = 0.030$ )
- Time to complete exam at visit 4 was greater for intervention group than control group (paired t-test, 2-tailed:  $t (15) = 2.72, p = 0.016$ )

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## Important takeaways - 2

- Remember – we used adult dogs who had had veterinary experience and most of whom were enrolled because they had some fear.
- These techniques MITIGATE fear in adult, fearful or worried dogs.
- These techniques should PREVENT fear in puppies and can prevent them from having scary experiences beyond their ability to control as they mature.

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JAVMA 2011 Volk et al., Executive summary of Bayer veterinary usage study

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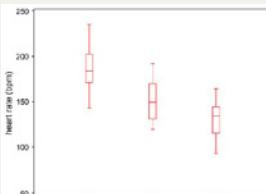
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**Table 1.** Recorded differences in physiologic parameters measured in cats in the home environment followed by the hospital environment. A statistically significant difference was detected between the home environment and the veterinary hospital environment for the parameters blood pressure ( $P=0.04$ ), heart rate ( $P<0.0001$ ), and respiratory rate ( $P=0.01$ ).

	Home environment		Hospital environment	
	Median	Range	Median	Range
Blood pressure (mmHg)	131	96–167	138	106–164
Rectal temperature (°F)	101.2	98.6–102.7	101.5	100.4–103.0
Heart rate (beats/min)	153	110–250	190	128–256
Respiratory rate (breaths/min)	50	24–84	58	18–192

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**Fig. 1.** Box and whisker plots of heart rate (beats/min) from 16 healthy cats recorded during manual restraint and during ambulatory electrocardiography. Heart rate was obtained first during manual restraint (Restraint) for edocardiography and then from ambulatory electrocardiographic recordings obtained with telemetry in the veterinary teaching hospital (VTH) and then again during a quiet period at home (Home). Heart rate is expressed in beats per minute (bpm). The box represents the interquartile range (IQR) and the line within, the median. The whiskers reflect the most extreme values that are less than 1.5 IQR beyond the upper or lower quartiles respectively.

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Journal of Veterinary Behavior 20(2015) 16–17



Contents lists available at ScienceDirect  
Journal of Veterinary Behavior



Feline Research  
2021 Journal of Veterinary Behavior Improving Pet Welfare Award  
Decrease in behaviors associated with pain during catheter placement using a topical anesthetic formulation in cats.<sup>a</sup>  
Carol Chávez<sup>b</sup>, María José Ubilla<sup>b</sup>, Mariela Goich<sup>b</sup>, H. Callejo-Jones<sup>b</sup>,  
María Paz Iturraga<sup>b,c</sup>  
<sup>a</sup> Sociedad de Investigación, Asociación de Ciencias Veterinarias y Agropecuarias, Facultad de Medicina Veterinaria y Agrícola, Universidad de San Martín de Pucón, Chile  
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**Keywords:**  
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Catheter placement  
Topical anesthetic cream  
Cat stress

In veterinary practice, peripheral catheter placement is very common. For some cats, this procedure can be associated with pain. The objective of this study was to evaluate the effect of a topical anesthetic cream related to catheter placement in healthy awake cats. 20 healthy cats, selected for one-lead electrocardiography, were randomized to either a lidocaine 2.5%/prilocaine 2.5% cream or a placebo cream. The cats were anesthetized with isoflurane and venipuncture was carried out using a 22-gauge catheter. The cat stress score system was used to evaluate the behavior of the cats during the procedure. The stress score was evaluated at the time of catheter placement and at the time of venipuncture. The behaviors associated with pain during venipuncture were evaluated in both groups. The stress score was evaluated at the time of catheter placement in both groups, with cats in the placebo group showing significantly fewer behaviors associated with pain than cats in the placebo group. The use of a topical anesthetic cream before peripheral catheter placement in non-emergency cases is feasible.

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What about medication?

Journal of Veterinary Medicine and Surgery (2005) 7, 195–202  
doi:10.1016/j.jvms.2004.12.003



## Heart rate and heart rate variability of healthy cats in home and hospital environments<sup>a,b</sup>

Jonathan A Abbott DVM, Dipl ACVIM (Cardiology)\*

Department of Small Animal Clinical Sciences—Marvin Regional College of Veterinary Medicine, Virginia Tech, Blacksburg, VA, USA

To investigate heart rate and its variability, a telemetry device was affixed to 16 healthy, young cats. Prior to inclusion in the study, cats were subject to a 4 month period of acclimation. The heart rate variability (HRV) was calculated for echocardiography (HRV) was calculated from 4–5 consecutive RR intervals obtained from a simultaneously recorded electrocardiogram. Heart rate variability data were collected by telemetry in a quiet room in the veterinary hospital (VTH) and later, in the owner's home (Home). The ambulatory data were digitally sampled and RR interval tachograms from a 4 month period subject to Fast Fourier Transform to yield measures of heart rate variability (HRV). The HRV data were collected in 16 healthy cats. Heart rate (bpm) expressed as mean ( $\pm$  SD) were HR<sub>rest</sub> 187 ( $\pm$  25); HR<sub>home</sub> 150 ( $\pm$  25); HR<sub>VTH</sub> 152 ( $\pm$  19); 15 of these rates was significantly different from the others. The HRV data were collected in 16 healthy cats. Heart rate variability was higher (and parasympathetic tone lower) when cats were in the hospital.

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## Venipuncture and other procedures that hurt



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**Table 2**  
Cat stress score in the treatment and placebo groups previous to venipuncture.

Score	Group	Treatment (n, %)	Placebo (n, %)
1—fully relaxed		0.6 $\pm$ 1.1 (7%)	1.0 $\pm$ 0.0 (10%)
2—weakly relaxed		2.0 $\pm$ 1.7 (20%)	2.0 $\pm$ 0.7 (20%)
3—moderately tense		2.0 $\pm$ 1.7 (20%)	2.0 $\pm$ 0.7 (20%)
4—very tense		2.6 $\pm$ 0.5 (28%)	1.6 $\pm$ 1.1 (17%)
5—fearful		0.6 $\pm$ 0.5 (7%)	0.6 $\pm$ 0.5 (7%)
6—very fearful		0.0 $\pm$ 0.0 (0%)	0.0 $\pm$ 0.0 (0%)
7—terrified		0.0 $\pm$ 0.0 (0%)	0.0 $\pm$ 0.0 (0%)
Score mean $\pm$ SD		2.9 $\pm$ 1.0	2.8 $\pm$ 1.0

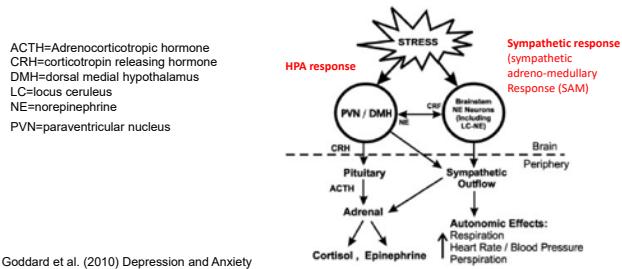
**Table 1**  
Scores of behaviors associated with pain during venipuncture in the treatment and placebo groups.

Score	Group	Treatment (n, %)	Placebo (n, %)
0—no reaction		8.0 $\pm$ 0.0 (80%)	2.0 $\pm$ 0.0 (20%)
1—slight movement of a limb, tensing of muscles		2.0 $\pm$ 0.0 (20%)	4.0 $\pm$ 0.0 (40%)
2—limb withdrawal, attempt to move away		0.0 $\pm$ 0.0 (0%)	2.0 $\pm$ 0.0 (20%)
3—marked attempts to escape, aggressive behavior, vocalization		0.0 $\pm$ 0.0 (0%)	2.0 $\pm$ 0.0 (20%)
Score mean $\pm$ SD		0.2 $\pm$ 0.4 <sup>a</sup>	1.4 $\pm$ 1.0 <sup>b</sup>

<sup>a</sup> and <sup>b</sup> represent statistical differences of  $P<0.001$ .

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**Acute stress response:** Sympathetic response stimulates NE neurons in the LC which has direct effects on central and peripheral components of the acute stress response. The peripheral component includes the standard sympathetic signs; the central component is responsible for the downstream response. Increased NE activity stimulates corticotrophin releasing hormone from the paraventricular nucleus of the hypothalamus, which then activates the hypothalamic-pituitary-adrenal-axis increasing sympathetic outflow to multiple peripheral organ systems.



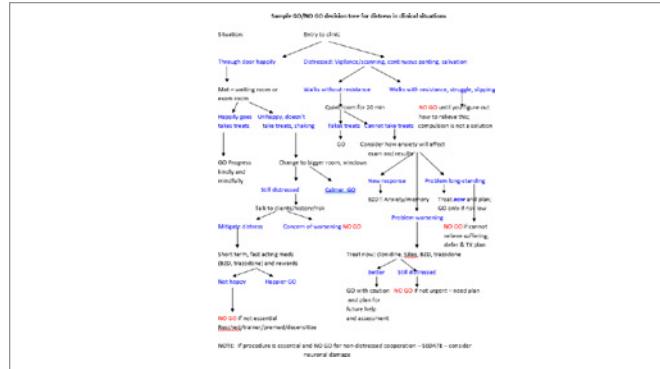
Goddard et al. (2010) Depression and Anxiety

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### 'Event' medications:

should be given at least 1 h before visit,  
may need to be repeated,  
may be best started 2-3 days prior to visit with  
q. 12 h dosing, and  
may be combined with other 'event'  
medications.

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### Alpha-2-agonists

- Clonidine
- Guafacine
- \*Dexmedetomidine OTM
- \*\*Tasipidimine liquid
- \* Canine license as Sileo
- \*\* Canine license as Tessie in Europe

Note: These are imidazole derivatives which stimulate  $\alpha$ 1-imidazoline brain/brain stem receptors which are thought to have a 'sympathoinhibitory' therapeutic response, themselves (J Med Tox 2014 Albertson et al.)

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### Medication choices of gabapentinoids as event meds

As event meds give at least 1 h before anticipated event

Medication	Dosage cats	Dosage dogs
Gabapentin	10-20 mg/kg	30-50 mg/kg
*Pregabalin	5 mg/kg	4-7 mg/kg

\* Blinded, placebo-controlled dose determination and efficacy studies for cats

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### Suggested medications

- Alpha agonists – Prazosin (alpha 1) and OTM dexmedetomidine (Sileo (alpha 2)
- Benzodiazepines
- Gabapentinoids

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### Medication choices of alpha-2 agonists as event meds - dogs

- Clonidine – 0.01-0.05 mg/kg – can be given up to q. 4 h (sedation!)
- Guafacine – nothing published in dogs but people are trying it – 0.5-2 mg/kg
- \*OTM dexmedetomidine - 125 mg/m<sup>2</sup> (4.65 µg/kg for 20 kg dog) up to 5x/24 h separated by 2 h
  - 1 ml~1 mg dexmedetomidine
  - 3 ml per syringe
  - 12 dots per syringe/1 dot=0.25 ml=0.25 mg dexmedetomidine
- \*Tasipidimine - 30 µg/kg
- \* Blinded, placebo-controlled dose determination and efficacy studies

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### Medication choices of benzodiazepines as event meds

1. BZDs are glucuronidated – cats do not do this well as an evolutionary adaptation to hypercarnivory.  
2. As event meds give at least 1 h before event, or split dose to 1-1.5 h and 30 minute before event – KNOW THAT PATIENTS DIFFER AND YOU MAY HAVE TO ADJUST THIS

Medication	Dosage dogs	Dosage cats	Duration
Alprazolam	0.02-0.04 mg/kg	0.0125-0.025 mg/kg	Short
Lorazepam	0.02-1.0 mg/kg	0.05 mg/kg	Moderate
Oxazepam	0.02-1.0 mg/kg	0.2-0.5 mg/kg	Moderate but slower peak
Clonazepam	0.02-0.04 to start	0.05 mg/kg	Long
Diazepam	0.5-2.2 mg/kg (3 meds: diazepam, N-desmethyl diazepam, oxazepam)	0.2 mg/kg	Very long

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Main take-home messages:

1. We need to do no harm.
2. We need to do better than we are doing now.
3. It is not sufficient that the examination was not a problem for us.
4. The examination must also not be a problem for the patient....true adaptive, collaborative care.

Forward.....

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Information:

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• **Behaviour at the Beach (BATB) Sunday evening 30 May through Friday 5 June 2026 – 36 h RACE credits; immersion course; email me**

• **Journal of Veterinary Behavior**

<https://www.sciencedirect.com/journal/journal-of-veterinary-behavior>



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VETERINARY  
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2026 OVMA Conference and Trade Show | 43

2004

# THE SUBTLE WORLD OF FELINE AGGRESSION

SMALL ANIMAL PROGRAM | BEHAVIOUR

Karen L. Overall, MA, VMD, PhD, DACVB

## *The subtle world of feline aggression*

Karen L. Overall, MA, VMD, PhD, Diplomate ACVB  
AVC UPEI  
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1

## Behavioural development in cats

It is critical to realize that cats do not have the domestication history that dogs do, and that *they are the quintessential sit-and-wait predator*. This latter behaviour has roles for how they play and react to scary or agonistic situations.

## Domestic cat development

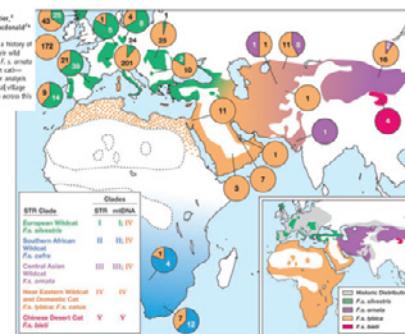
- Cats were not truly 'domesticated' as were dogs (who may have co-evolved with humans) and stock.
- Our relationship with cats is based on a symbiosis/commensalism: as humans became more agrarian, cats derived from the multiple groups of wildcats (*Felis lybica* ssp.) followed the migrating rodents out of Africa and across Asia and to the middle east ~9500 YBP (at least 5 founders –different variants of *Felis sylvestris*).
- They have been in Europe as 'domesticated cats' since 4200-2300 y BCE based on isotope analysis of bones (Karjcarz et al., 2020 PNAS) and can be considered 'opportunistic synanthropes' eating rodents that result from agriculture and fermented grains in manure.

3

## The Near Eastern Origin of Cat Domestication

Carles A. Driscoll,<sup>1,2</sup> Marilyn Menneti Raymond,<sup>3</sup> Alfred L. Rose,<sup>4</sup> Karleen Hope,<sup>5</sup> Marlene E. Donahue,<sup>6</sup> Eli Gallo,<sup>7</sup> Eric M. Marley,<sup>8</sup> Michael J. O'Connell,<sup>9</sup> Jennifer A. Johnson,<sup>10</sup> Andrew Kitchener,<sup>11</sup> Michael J. O'Brien,<sup>12</sup> David W. Macdonald<sup>13</sup>

Science Vol 317 27 JULY 2007



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• Selection for *physical characteristics* in cats is only ~200 years old in NA and Europe (selection for coat color, conformation, eye color is older in Asian breeds). This is a real contrast from dogs where selection was for work.

• Because the behaviours that we wanted from cats are those used to control rodents (and, hence disease, food theft and food soiling) are their standard, innate behaviors, humans exerted no selection on feline behaviour.

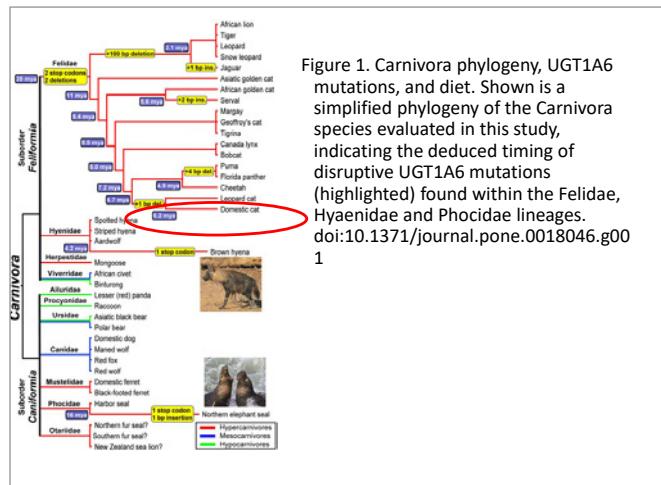
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• In fact, the blueprint of our lack of physical and behavioural selection can be found both two data sets: that from the neurodevelopment of cats and that from their physiological mutation history associated with hypercarnivory.

• *In neurodevelopment we see no selection for extended sensitive periods and quite short and early truncated periods for social and physical exposure. This is in contrast with dogs (but similar to wolves). Domestic dogs, like humans but not like wolves, have extended sensitive periods with very long tails for their social development.*

• *Dogs, like humans, can do well as omnivores/mesocarnivores and this pattern likely affected by, and is reflected in, the evolution of their relationship with humans.*

7



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• All intentional selection for feline *behaviour* has been recent ('the past 50-75 years). Otherwise, we have been content with the 'essential cat'....and that cat has a very interesting brain.

• Domestic cats have a ventral medial hypothalamus and a lateral hypothalamus that react in characteristic and rapid ways that recruit other neurons quickly...to the point that domestic cats are natural animal models for the 'kindling' behaviors used to study epilepsy.

• These regional responses to stimulation are adaptations for their foraging mode (sit and wait predation), but likely factor into aggressive pathologies.

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OPEN ACCESS Freely available online

PLOS ONE

## Evolution of a Major Drug Metabolizing Enzyme Defect in the Domestic Cat and Other Felidae: Phylogenetic Timing and the Role of Hypercarnivory

Binu Shrestha<sup>1,2</sup>, J. Michael Reed<sup>3</sup>, Philip T. Starks<sup>2</sup>, Gretchen E. Kaufman<sup>3</sup>, Jared V. Goldstone<sup>4</sup>, Melody E. Rouleau<sup>5</sup>, Stephen J. O'Brien<sup>6</sup>, Klaus-Peter Koepfli<sup>7</sup>, Laurence G. Frank<sup>8</sup>, Michael H. Court<sup>1,2</sup>

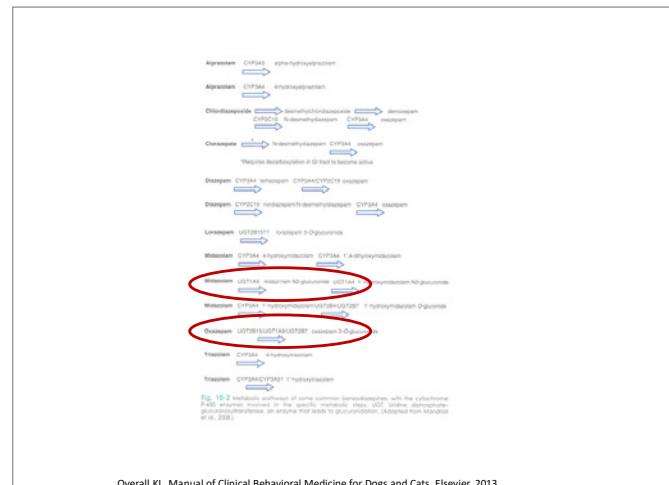
<sup>1</sup> Comparative and Molecular Pharmacogenomics Laboratory, Department of Molecular Physiology and Pharmacology, Tufts University School of Medicine, Boston, Massachusetts, United States of America, <sup>2</sup> Department of Biology, Tufts University, Medford, Massachusetts, United States of America, <sup>3</sup> Department of Environmental and Population Health, Tufts Cummings School of Veterinary Medicine, North Grafton, Massachusetts, United States of America, <sup>4</sup> Biology Department, Woods Hole Oceanographic Institution, Woods Hole, Massachusetts, United States of America, <sup>5</sup> Laboratory of Molecular Immunobiology, National Institute of Allergy and Infectious Diseases, National Institutes of Health, Bethesda, Maryland, United States of America, <sup>6</sup> Department of Cell Biology, National Cancer Institute at Frederick, Frederick, Maryland, United States of America, <sup>7</sup> Living with Lions Project (Kenya), Museum of Vertebrate Zoology, University of California, Berkeley, California, United States of America

### Abstract

The domestic cat (*Felis catus*) shows remarkable sensitivity to the adverse effects of phenolic drugs, including acetaminophen and aspirin, as well as structurally-related toxicants found in the diet and environment. This bioactivity results from pseudogenization of the gene encoding UDP-glucuronosyltransferase (UGT1A6), the major species-conserved phenol detoxification enzyme. Here, we established the phylogenetic timing of disruptive UGT1A6 mutations and explored the hypothesis that gene inactivation in cats was enabled by minimal exposure to plant-derived toxicants. Fixation of the UGT1A6 pseudogene was estimated to have occurred between 35 and 11 million years ago with all extant Felidae having dysfunctional UGT1A6. Our phylogenetic analysis also revealed that the only extant member of the subfamily *Paradoxynae* (brown hyena, *Parahyena brachyura* and northern elephant seal (*Mirounga angustirostris*)) showed inactivating UGT1A6 mutations. A comprehensive literature review of the natural diet of the sampled taxa indicated that all species with defective UGT1A6 were hypercarnivores (>70% dietary animal matter). Furthermore those species with UGT1A6 defects showed evidence for reduced amino acid constraint, increased dN/dS ratios approaching the neutral selection value of 1.0 as compared with species with functional UGT1A6. Our results were consistent with the evidence for reduced amino acid constraint in the same species with UGT1A1, the gene encoding the enzyme responsible for detoxification of endogenously generated bilirubin. Our results provide the first evidence suggesting that diet may have played a permissive role in the evolution of a mammalian drug metabolizing enzyme. Further work is needed to establish whether these preliminary findings can be generalized to all Carnivora.

Citation: Shrestha B, Reed JM, Starks PT, Kaufman GE, Goldstone JV, et al. (2011) Evolution of a Major Drug Metabolizing Enzyme Defect in the Domestic Cat and Other Felidae: Phylogenetic Timing and the Role of Hypercarnivory. PLoS ONE 6(6): e2046. doi:10.1371/journal.pone.0018046

8



Overall KL. Manual of Clinical Behavioral Medicine for Dogs and Cats, Elsevier, 2013.

10



## Considerations for how cats move and use space, given hypercarnivory

11

## Consider habitat and distance travelled per day free-ranging domestic cats

data from Macdonald and Apps, 1978; Turner and Bateson, 1988

- Average free-range “territory” • 0.1-0.45 ha
- Range for males • 0.4-990 ha
- Range for females • 0.02-170 ha

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### Home Range, Habitat Use, and Activity Patterns of Free-Roaming Domestic Cats

Author(s): Jeff A. Horn, Nehra Mateu-Pinilla, Richard E. Warner, and Edward J. Heske

Source: Journal of Wildlife Management, 75(5):1177-1185. 2011.

**Table 2.** Home range estimates (ha; 95% minimum convex polygon [MCP] and 95% kernel density estimator [KDE]) for owned and unowned free-ranging cats in Champaign-Urbana, Illinois, USA, 2007-2008. Data are means, standard error (SE), and sample size (n).

Ownership status and sex	95% MCP			95% KDE		
	Mean	SE	n	Mean	SE	n
Owned						
Male	1.83	1.42	3	5.16	4.89	3
Female	1.92	1.09	8	1.95	0.87	8
Unowned						
Male	157.01	89.44	6	103.37	73.52	6
Female	56.59	21.34	10	57.92	33.61	10

1180

The Journal of Wildlife Management • 75(5)



All photos courtesy of Anne Marie Dossche

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## Understanding normal

## Social structure

- Matrilineal
- Extended female family group (Crowell-Davis lab)
- Extended, extensive, and shared care of offspring
- “Bachelor” males
- Roles for resource environment
- Roles for induced ovulation and infanticide
- Heritability of ‘boldness’ via father

15

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## Roles for 'preferred associates'

### Influence of familiarity and relatedness on proximity and allogrooming in domestic cats (*Felis catus*)

Terry Marie Curtis, DVM, MS; Rebecca J. Knowles, MS; Shann L. Crossell-Davis, DVM, PhD

**Objective** To evaluate associations between familiarity and familiarity with the influence between relatedness and familiarity.

**Animals** 20 privately owned cats in 7 families.

**Procedure** Data representing 14 variables. End of one observed in 10 minutes. All variables were measured in 10 minutes of all observations of all grooming behavior were measured. At the end of the 10 minutes, the identity evaluation of all individuals was recorded.

**Results** Relatedness and familiarity were significantly ( $p < 0.05$ ) associated with the number of times a cat was groomed. The relatives and acquaintances that were usually favored were the most familiar. The familiarity of the relatives and acquaintances was significantly associated with all grooming behaviors. The familiarity of the relatives and acquaintances was significantly associated with all grooming behaviors.

**Conclusion** Familiarity and relatedness are significantly associated with all grooming behaviors.

**Keywords** Familiarity, Relatedness, Grooming, Domestic cat.

**Received December 11, 2002**

**Accepted January 15, 2003**

**From the Department of Anatomy and Radiology, College of Veterinary Medicine, Cornell University, Ithaca, NY; the Department of Animal Sciences, Cornell University, and the Department of Small Animal Clinical Sciences, College of Veterinary Medicine, Cornell University, Ithaca, NY; and the Department of Animal Sciences, Cornell University, Ithaca, NY**

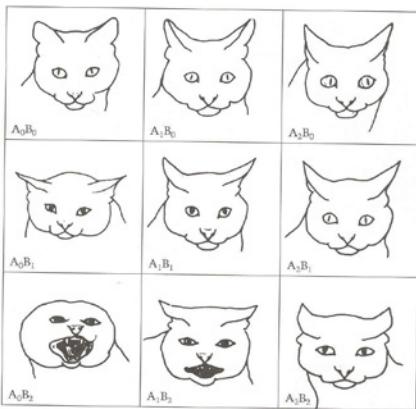
**Address reprint requests to Dr. Curtis**

**E-mail:** tmc21@cornell.edu

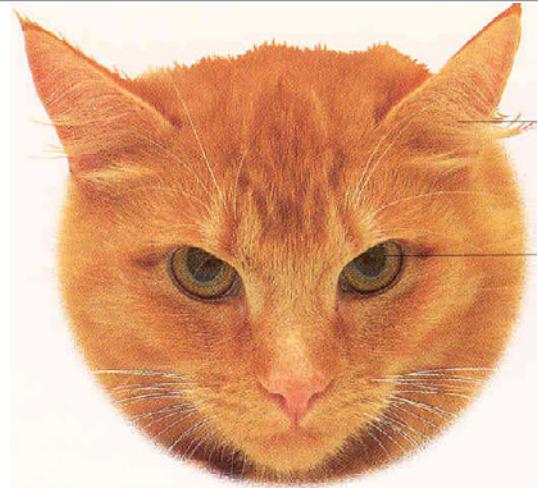
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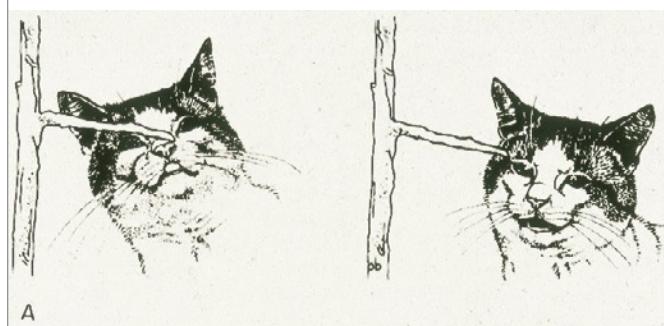
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Roles for our most impaired sense: olfaction

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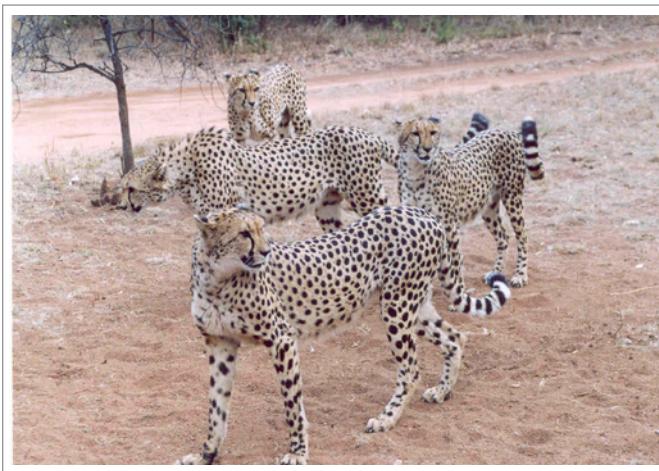


Feline flehmen (from Houpt)

26



27



28

## Feline marking behavior

29

When is marking normal behavior, and must it involve urine?

Roles for other scents and complex information

30

### What constitutes “marking behavior”?

- Marking behavior *involving elimination*:
  - Both spraying and non-spraying marking are types of marking behavior, and they may be ‘normal’
  - Non-spraying marking can involve either urine or feces or both
  - If non-spraying marking involves urine, the urine is left in 1 or many small puddles (helps to distinguish from a location preference)
- Marking behavior *NOT involving elimination*; these are ‘normal’ behaviors but changes in frequency, intensity and context tell you if the cat is distressed:
  - Bunting
  - Rubbing
  - Scratching

31



32



Photo courtesy of Anne Marie Dossche

33



Photo courtesy of Anne Marie Dossche

34



Photo courtesy of Anne Marie Dossche

35



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### Key concerns to evaluate in ANY problematic elimination or aggression in cats

- Social interactions and associations
  - Who grooms and sits with whom; who displaces whom?
- Olfactory environment
  - Are there areas of discolouration due rubbing some body part?
- Elimination behaviours
  - Are these driven by tactile considerations and early developmental preferences?
  - What is the litter box like: Does the cat have 'access' or are they denied it physically or socially? Can the cat be 'trapped'? Is the box at least 1.5 x the body length of the cat? Is the box 'clean' from the cat's perspective?

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### The intercat aggression story

38

### Necessary and sufficient conditions for intercat aggression

- Necessary: consistent, volitional, proactive aggression that is *not contextual* given the *social signal, threat circumstances, or response received*
- Sufficient: As above *in the absence of any directly provocative signal or interaction from the cat that is attacked, in the case of overt aggression, or that is the target, in the case of more covert aggression*
- *Remember the role for the ventromedial hypothalamus (VMH) and foraging strategy!*

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### Heuristic model for thinking about phenotypic patterns of feline aggression: Potential axes

- **overt v. covert aggression** – is the other cat there (overt) or are there no other cats present (covert) – this assessment is about the social environment
- **active v. passive aggression** – are you engaging the other cat (active) or are you signaling without direct engagement (passive) – this assessment is about the intent of the signal
- **offensive v. defensive aggression**: is this a response to the behavior of another (usually defensive) or is this a proactive aggression (offensive) – this assessment is about the quality of the behaviours and interaction and the role of distance (increasing: defensive; decreasing: offensive)

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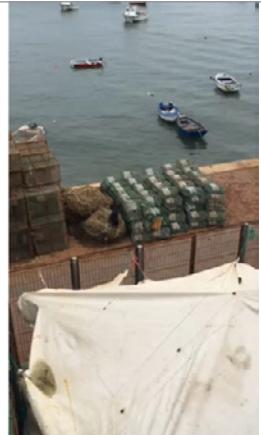
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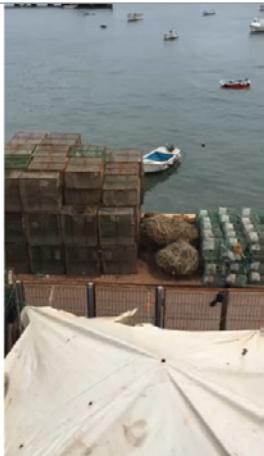
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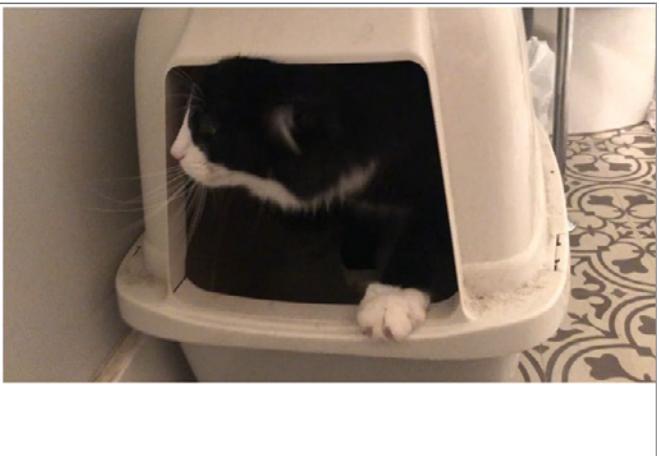
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48

The role of 'stress' and the feline environment

49

Stressors for laboratory cats (Carlstead et al., 1993)

- Irregular and unpredictable feeding times
- Irregular and unpredictable cleaning times
- Absence of stroking and petting by people
- Unpredictable and unfamiliar manipulations
- Changes in social environment

50

So what does this tell us about domestic, pet cats?

51

Stressors for pet cats

- Irregular and unpredictable feeding times
- Irregular and unpredictable cleaning times
- Absence of stroking and petting by people
- Unpredictable and unfamiliar manipulations
- Changes in social environment – think about “normal” social organization and the role for olfaction!

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## Association between aggression and changes in or problems with elimination behaviors

Preferences – Often secondary due to impaired social relationship.

- Location
- Substrates

Aversions – Often primary owing due to the impaired social relationship.

- Location
- Substrate

Marking – May be normal, may be covert, offensive aggression, may be associated with an outdoor cat, et cetera

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## Recommendations for decreasing stress, in general

- Regular schedules
- Stable social groupings
- Human interaction
- Relaxation [private time and space] and play periods
- 3-D space with hiding spots
- Predictability without rigidity

55

- Add anti-anxiety medication as needed – remember to treat the VMH induced arousal levels:
  - Gabapentin (Gabapentinoid) – 3-5 mg/kg q. 8-12 h to start to test for sedation (10-20 mg/kg q. 12 h is the target dose)
  - Pregabalin (Gabapentinoid) – 5 mg/kg q. 24 h
  - Fluoxetine (SSRI) – 0.5 mg/kg q. 24 h
  - Clomipramine (SSRI) – 0.5 mg/kg q. 24 h
  - Paroxetine (SSRI) – 0.5 mg/kg q. 24 h
  - Prazosin (alpha 1 antagonist) – 0.5-1.0 mg/kg po q. 8-12 h

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## Necessary and sufficient conditions for urine or fecal marking

- Necessary condition: urination or defecation that occurs in *frequencies and, or locations inconsistent only with evacuation of the bladder or bowel, but consistent with social and olfactory stimuli*
- Sufficient condition: repeated urination or defecation associated with *species typical behaviors or postures distinct from those occurring with simple elimination*, and as described above
- The **CONTEXT** determines whether this is normal behavior or a concern for the cat, regardless of whether the client dislikes it.

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## What can we do?

- Rule out medical conditions including pain(PE, CBC, UA +/- culture, fecal, chemistry panel)
- If elimination changes are involved: ID substrates and locations the cat likes and use those and use odor eliminators that bind odorants; clean well – club soda
- Recommendations for 'managing' the aggression:
  - Structure attention and access with the goals of rewarding appropriate behavior and decreasing anxiety (standard conditioning)
  - Go slowly - all behavior mod takes time and requires almost imperceptible steps
  - In the meantime, Venn diagrams can help you manage space and time
  - Bells
  - Protect the victim(s) and watch for what entraps them

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## Summary and word of caution

- Cats are tough because people do not know what 'normal' behaviour is in cats and do not understand the relative inflexibility of their neurobehavioural development and social structures. Here, genetics may really matter.
- Get people to start a video library asap.
- Assessment and treatment early is best.
- Tell people this at every visit, and screen for problems.

58

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- [koverall@upei.ca](mailto:koverall@upei.ca);  
[overall.karen@gmail.com](mailto:overall.karen@gmail.com)
- **Behaviour at the Beach (BATB)**  
**Sunday evening 30 May**  
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**h RACE credits; immersion**  
**course; email me**
- **Journal of Veterinary Behavior**  
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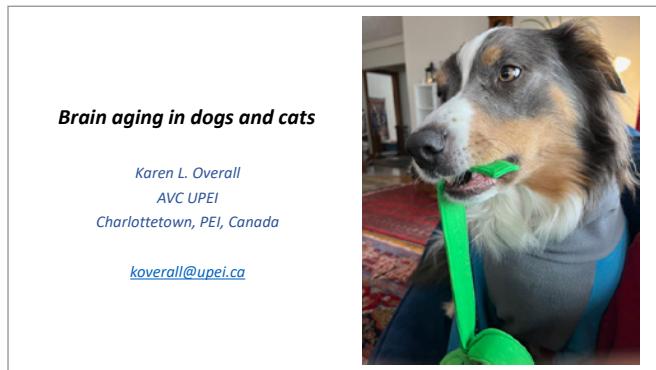


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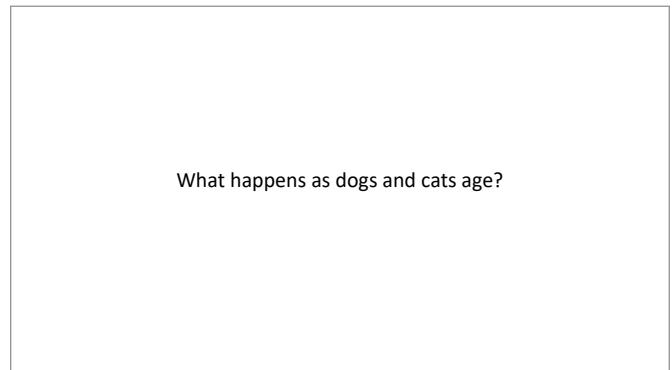
# BRAIN AGING IN DOGS & CATS

# SMALL ANIMAL PROGRAM | BEHAVIOUR

Karen L. Overall, MA, VMD, PhD, DACVB



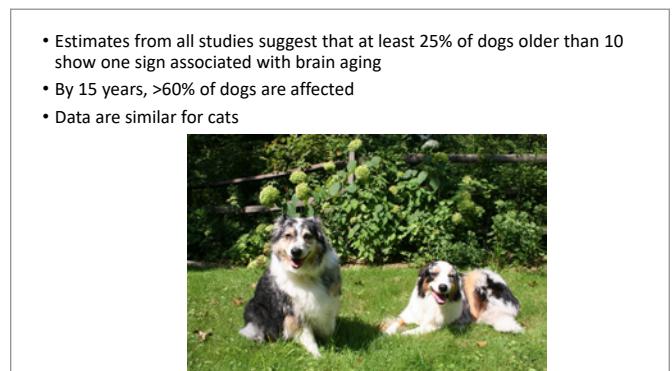
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## What happens as dogs and cats age?

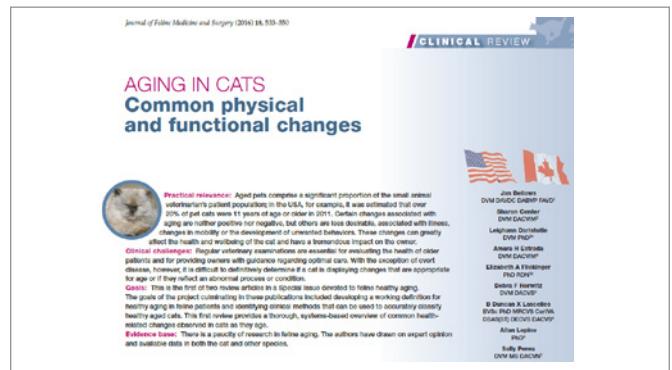


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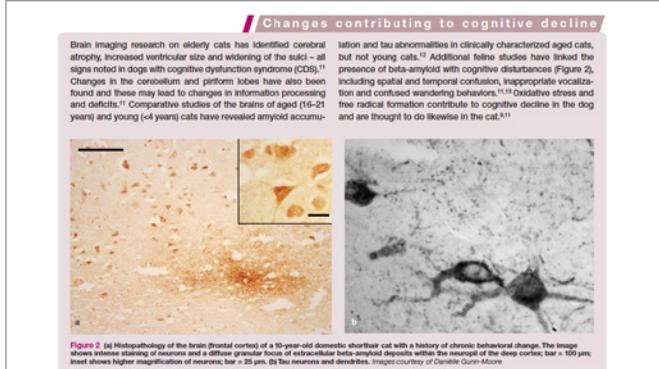
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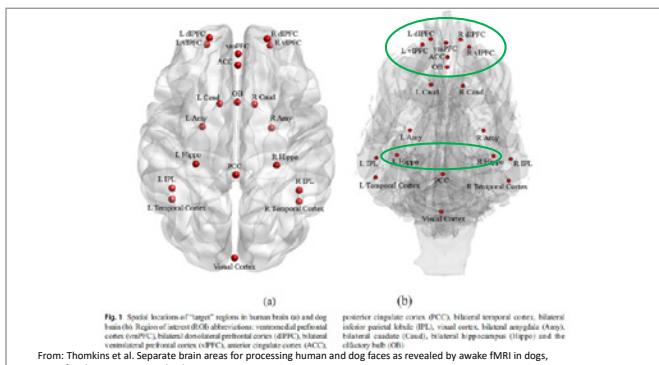




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Study	Age	Prevalence	Distribution of plaques	Type of plaques	Distribution of poorly circumscribed deposits	Type	Tau			
							Age	Prevalence	Distribution	Type
Nakamura et al., 1996	>10y	3/3	caudate cortex (medial lobe)	$\text{A}\beta_{1-40}$	caudate neuropil	$\text{A}\beta_{1-40}$	n/a	n/a	n/a	n/a
Breton et al., 2005	>10y	4/4	caudate layer of parental lobes	strong $\text{A}\beta_{1-40}$ and $\text{A}\beta_{1-42}$	caudate layer of the frontal and parietal lobes	strong $\text{A}\beta_{1-40}$ and weaker $\text{A}\beta_{1-42}$	n/a	n/a	n/a	n/a
Heal et al., 2005	>10y	4/5	prefrontal cortex, parahippocampal gyrus, parietal cortex, occipital cortex, anterior layer dentate gyrus	$\text{A}\beta_{1-40}$ and $\text{A}\beta_{1-42}$	prefrontal cortex, parahippocampal gyrus, parietal cortex, occipital cortex	$\text{A}\beta_{1-40}$ and $\text{A}\beta_{1-42}$ , frequent	>10y	5/5	hippocampus, CA1, subiculum, within dentate gyrus, hippocampal/parahippocampal gyrus, anterior layer dentate gyrus	AT8+ anti- $\text{A}\beta_{1-40}$ within dentate gyrus, AT8+, PEP1+, $\text{A}\beta_{1-40}$ within neurons ( $n=2/5$ )
Gunn-Moore et al., 2006	>10y	7/9	caudate layer (deep) of anterior mid-cerebrum	$\text{A}\beta_{1-40}$	n/a	n/a	>10y	2/5	caudate, hippocampus, ventral nucleus	AT8+ neurons
Takemoto et al., 2008	>10y	6/6	caudate cortex and hippocampus	$\text{A}\beta_{1-40}$	caudate cortex and hippocampus	$\text{A}\beta_{1-40}$	n/a	n/a	n/a	n/a
Claussens et al., 2015	>8y	14/15	caudate cortex	$\text{A}\beta_{1-40}$	interruption oligodendroglia	$\text{A}\beta_{1-40}$	>10y	9/14	medial cortex, hippocampus, cerebellar cortex, locus caeruleus	AT8 and AT100
Ponseti et al., 2019	>10y	9/11	temporal and frontal cortex	$\text{A}\beta_{1-40}$	interruption in hippocampus ( $n=1/9$ )	$\text{A}\beta_{1-40}$ and $\text{A}\beta_{1-42}$	>10y	4/11	hippocampus and adjacent subcortical structures	AT8 and PEP1
Ford et al., 2020	>10y	27/28	Cortical layers IV and VI most prominent <10y, V and VII most prominent >10y	4E10+	n/a	n/a	>10y	4/28	medial cortex – hippocampus, AT8+ and neurons	AT8+

9



From: Thomkins et al. Separate brain areas for processing human and dog faces as revealed by awake fMRI in dogs. *Canis familiaris*. Learning and Behavior 2018;46:561-573.

10

**REVIEW**

**Aging pet cats develop neuropathology similar to human Alzheimer's disease**

Jenna King<sup>a</sup>, Jessica M. Snyder<sup>a</sup>, Martin Darvas<sup>b</sup>, Denise M. Imai<sup>b</sup>, Molly Church<sup>b</sup>, Caitlin Latimer<sup>b</sup>, C. Dirk Keene<sup>b</sup>, Warren Ladiges<sup>b</sup>

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<sup>b</sup> Division of Neuropathology, Department of Laboratory Medicine and Pathology, School of Medicine, University of Washington, Seattle, WA, USA

<sup>c</sup> Comparative Pathology Laboratory, School of Veterinary Medicine, University of California, Davis, CA, USA

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**Abstract**

Aging pet cat can spontaneously develop  $\text{A}\beta$  deposition and tangle pathology (including neurofibrillary tangle formation with neuronal loss) in a similar distribution and with similar characteristics to Alzheimer's disease (AD) in humans. These three major pathologies that characterize AD rarely occur spontaneously in other nonhuman animals. In addition, cats develop cognitive impairment with increasing age, and some studies show an association with neuropathology. The aging pet cat may be a more valuable spontaneously occurring animal model for the study of, and therapeutic development of, AD compared to other domestic animals such as pet dogs. This review describes the unique translational potential of the domestic cat as a natural model of AD, with reference to other animal models of AD.

**Keywords:** Alzheimer's disease, pet cats, feline neuropathology, aging, feline cognitive dysfunction syndrome

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## Dogs

Ventricular enlargement	Ventricular volume was constant between 20 and 39 years of age, but increased dramatically after 40 years of age.	Rapid increase in the ventricle volume was observed in beagle dogs after age of 15.	[3–5,23]
Senile Plaques (SP)	The longer, more toxic $\text{A}\beta_{1-42}$ initially accumulated in the brain, followed by the shorter and more soluble $\text{A}\beta_{1-40}$ deposition in SF plaques. $\text{A}\beta$ plaques are widely present in the cortex and hippocampus of AD patients.	Dogs naturally develop SP of the diffuse (non- $\beta$ -sheet) subtype. $\text{A}\beta_{1-42}$ initially builds up in the brain, followed by $\text{A}\beta_{1-40}$ deposition in diffuse SP and blood vessels. $\text{A}\beta$ plaques are associated with CDS; three types of amyloid deposits were detected: diffuse, focal, and vascular deposits.	[1,3,7,8,42]
NFTs are the consequence of immunophilin aggregation of hyperphosphorylated tau protein (p-tau) in neurons and glial cells. NFTs were widespread in the cortex and hippocampus of AD patients.	NFTs have not been detected in dogs. However, p-tau was detected in neurons and astrocytes in dogs with CDS. The number of p-tau-positive cells in the brains were much higher in dogs with CDS than in the normal aged dogs. In addition, the levels of p-tau increased with the ages of dogs with CDS and were significantly higher with the $\text{A}\beta$ deposition in dogs with CDS.		[1,3,7,8]
Cerebral glucose metabolism	Cerebral glucose metabolism was reduced in old people, and was further compromised in AD patients compared with the age-matched controls.	Cerebral glucose metabolism was reduced in middle aged, and further reduced in senior dogs.	[15,47,49,50]

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Aging-Related Changes	Human Brains	Dog Brains	Reference
Cortical atrophy	Total brain volumes started to decline in the forties significantly and continued to decline through fifties and seventies. Cortical atrophy is not linear and uniform across the brain. Significant atrophy rates across all neocortical regions were observed in subjects with clinical signs of cognitive impairment. Medial temporal cortex had greater atrophy rates in subjects with early diseases while greater atrophy rates occurred in prefrontal, parietal, and temporal, and cingulate cortex in subjects at later stages of mild cognitive impairment and AD.	Significant decrease in total brain volume was observed only in subjects with dementia. Older frontal lobe atrophy developed in the old dogs aged 8–11 years. Hippocampal volume also decreased with age, but occipital lobe did not decline with age. The neuron density was significantly reduced in the brains of dogs with CDS compared with age-matched control dogs.	[1,4,8,21–24,113]
Cerebral amyloid angiopathy	Amyloid ( $\text{A}\beta_{1-40}$ ) deposits in blood vessel walls.	Amyloid ( $\text{A}\beta_{1-40}$ ) deposits in blood vessel walls.	[1,8]

From: Pan Y. Nutrients, cognitive function, and brain aging: What have we learned from dogs. *Med. Sci.* 2021, 9, 72.

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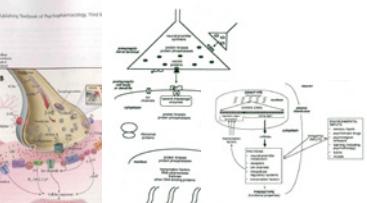
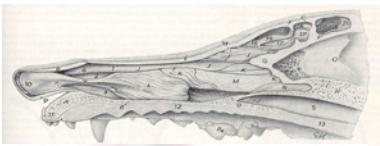
## Considerations for olfaction in older animals with a focus on dogs

Ventricular enlargement	Ventricular volume was constant between 20 and 39 years of age, but increased dramatically after 40 years of age.	Rapid increase in the ventricle volume was observed in beagle dogs after age of 15.	[3–5,23]
Senile Plaques (SP)	The longer, more toxic $\text{A}\beta_{1-42}$ initially accumulated in the brain, followed by the shorter and more soluble $\text{A}\beta_{1-40}$ deposition in SF plaques. $\text{A}\beta$ plaques are widely present in the cortex and hippocampus of AD patients.	Dogs naturally develop SP of the diffuse (non- $\beta$ -sheet) subtype. $\text{A}\beta_{1-42}$ initially builds up in the brain, followed by $\text{A}\beta_{1-40}$ deposition in diffuse SP and blood vessels. $\text{A}\beta$ plaques are associated with CDS; three types of amyloid deposits were detected: diffuse, focal, and vascular deposits.	[1,3,7,8,42]
NFTs are the consequence of immunophilin aggregation of hyperphosphorylated tau protein (p-tau) in neurons and glial cells. NFTs were widespread in the cortex and hippocampus of AD patients.	NFTs have not been detected in dogs. However, p-tau was detected in neurons and astrocytes in dogs with CDS. The number of p-tau-positive cells in the brains were much higher in dogs with CDS than in the normal aged dogs. In addition, the levels of p-tau increased with the ages of dogs with CDS and were significantly higher with the $\text{A}\beta$ deposition in dogs with CDS.		[1,3,7,8]
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13

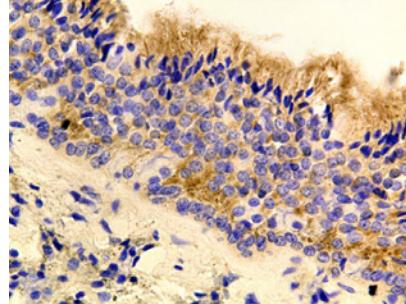
Aging-Related Changes	Human Brains	Dog Brains	Reference
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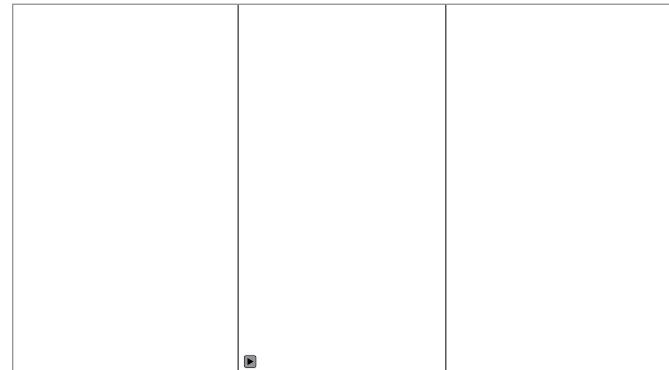
Olfactory Epithelium - Amyloid beta, counterstained with Cresyl violet



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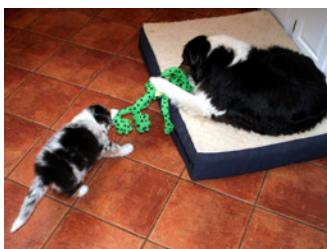


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### Boosting cognition – roles for supplements and diets

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### Potential role of diet and supplements



- Diet and supplements may play a significant role in managing cognitive decline
- Potential beneficial nutrients include antioxidants, omega-3 fatty acids and MCTs
- Preventing and treating effects of neuronal assaults is the goal
- All of these interventions will likely work best when coupled with brain and cognitive stimulation

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### Diets and supplements

- Anti-oxidant enriched diets and supplements (eg, Hill's B/D; Nestle Purina's EN and Bright Minds line; Neurocare, Nestle Purina; Omega-3 Pet, Nordic Naturals; Aktivait, VetPlus)
- Enhancers of cellular metabolism (Aktivait, VetPlus)
- Supplements that include precursors of neurochemicals (Aktivait, VetPlus)



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### Polyunsaturated fatty acids (PUFAs)

- Docosahexanoic acid (DHA) & eicosapentanoic acid (EPA)
- Long chain polyunsaturated fatty acids
- Essential for developing and maintaining the integrity of cells of the brain's membranes
- EPA (20:5, n-3) can be metabolized to DHA (22:6; n-3)
- Essential for early brain development – deficient diets especially affect the frontal cortex and reactivity

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## Omega-3 Pet (Nordic Naturals)

- 150 mg eicosapentanoic acid (EPA)
- 90 mg docosahexanoic acid (DHA)

### Guaranteed Analysis (Unflavored Soft Gels)

1 soft gel = 1500 mg
1 soft gel contains 1000 mg fish oil
Crude Fat (min.)
4.7%
Moisture (max.)
4%
Total Omega-3 Fatty Acids* (min.)
27%
Eicosapentaenoic Acid (EPA)* (min.)
17%
Docosahexanoic Acid (DHA)* (min.)
7%

1 Teaspoon = 6.0 mL (4600 mg)
1 Teaspoon contains:
Crude fat (min.)
50.4%
Moisture (max.)
0.1%
Total Omega-3 Fatty Acids* (min.)
37%
Eicosapentaenoic Acid (EPA)* (min.)
17%
Docosahexanoic Acid (DHA)* (min.)
10%



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### BDNF increases with behavioral enrichment and an antioxidant diet in aged dogs

Margaret Falzone<sup>a,\*</sup>, Monica Marchese<sup>a</sup>, Elizabeth Head<sup>b,c</sup>, Victoria Pop<sup>b,c</sup>, Bernadette Mikell<sup>b</sup>, William N. Milgram<sup>b</sup>, Carl W. Cotman<sup>b,c</sup>  
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<sup>b</sup> Institute for Brain Aging and Repair, University of California, Los Angeles, CA 90095-1600, USA  
<sup>c</sup> Department of Neurology, University of California, Los Angeles, CA 90095-1600, USA

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Received 4 May 2008; revised 20 August 2008; accepted 27 August 2008  
Available online 20 October 2008

Abstract  
The aged canine brain is at a greater risk to neurodegenerative disease. Cognitive impairment and memory loss are common in the elderly human and canine aging models. Behavioral enrichment and antioxidant diets have been shown to reduce cognitive decline in aged humans and dogs. We hypothesized that behavioral enrichment and an antioxidant diet would reduce cognitive decline in aged dogs. Although the mechanisms underlying these changes have not been established, we hypothesized that BDNF, a key neurotrophin involved in memory, would increase with behavioral enrichment and cognitive performance. We found that aged dogs that did not receive any treatment showed a significant decrease in BDNF mRNA levels in the hippocampus. Dogs receiving behavioral enrichment and an antioxidant diet showed increased levels of BDNF mRNA. However, dogs receiving both an antioxidant diet and an enriched environment showed the greatest increase in BDNF mRNA levels. BDNF mRNA levels were positively correlated with improved cognitive performance. Total mRNA levels did not differ between groups. BDNF mRNA levels were not positively correlated with improved cognitive performance. These results suggest that behavioral enrichment and an antioxidant diet may reduce cognitive decline in aged dogs. The results also suggest that the combination of behavioral enrichment and an antioxidant diet may be an effective treatment for cognitive decline in aged dogs. © 2008 Elsevier Inc. All rights reserved.

**Increases in BDNF function in combined enrichment + diet group compared to control diet + baseline enrichment.**

**Decreases in errors in a spatial memory task in combined enrichment + diet group compared to control diet + baseline enrichment.**

**Aged dogs were all laboratory beagles so they are an extreme group.**

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### Region specific neuron loss in the aged canine hippocampus is reduced by enrichment

Christina T. Siwak-Tapp<sup>a,\*</sup>, Elizabeth Head<sup>b,c</sup>, Bruce A. Muggenburg<sup>b</sup>,  
Norton W. Milgram<sup>b</sup>, Carl W. Cotman<sup>b,c</sup>

<sup>a</sup> Institute for Brain Aging and Repair, University of California, Los Angeles, CA 90095-1600, USA  
<sup>b</sup> Department of Neurology, University of California, Los Angeles, CA 90095-1600, USA  
<sup>c</sup> Department of Molecular, Cell, and Integrative Physiology, University of Colorado, Boulder, CO 80309-0345, USA

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Received 17 May 2008; revised 5 September 2008; accepted 17 September 2008  
Available online 2 November 2008

Abstract  
Neuron loss within the hippocampus and cerebral cortex occurs as a function of age in humans. We first tested the hypothesis that neuron loss occurs in the aged dog. The total neuronal number in the canine cerebral cortex and subregions of the hippocampus from the left hemisphere were estimated using the optical fractionator. The hippocampus showed a significant loss of neurons (~20%) in the aged dog brain compared to young (~3–4.5 years old) dogs. We hypothesized that behavioral enrichment and an antioxidant diet or behavioral enrichment would reduce the age-related loss of hilar neurons. Behaviorally enriched aged dogs had significantly more hilar neurons than control controls. These results suggest that the aged canine hippocampus in the left hemisphere shows selective neuron loss and that behavioral enrichment may reduce this loss.

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- The hilus of the hippocampus lost up to 30% of neurons in aged (13–15 years) compared with young (3–4.5 years) laboratory beagles. This was reduced with enrichment and enriched dogs had 18% more hilar neurons than similarly aged controls.

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- When comparing effects of behavioral enrichment and antioxidant-fortified diet on cognitive decline in old dogs
  - Both forms of intervention were effective
  - The greatest effect was seen in dogs treated with both enrichment and diet
  - These dogs had levels of nerve growth factors at levels similar to young dogs

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## Aktivait (VetPlus)

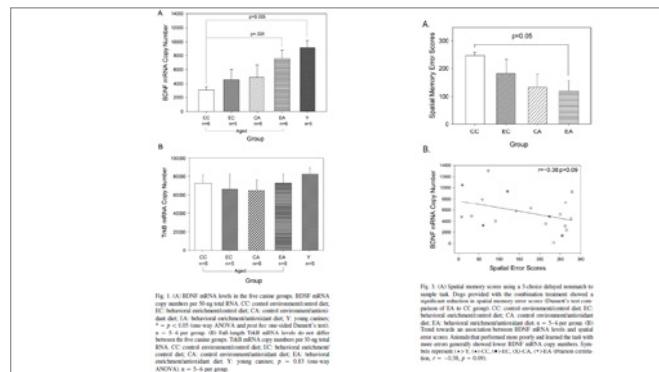
Table 3  
Nutrient groups included in Aktivait®

Constituents of Aktivait®	Brain strengthening	Signalling enhancer	Metabolic enhancer	Anti-oxidant
DHA/EPA	X			
Phosphatidylserine	X	X		X
Acetyl-L-carnitine		X		
L-Carnitine		X		
CoQ10		X		
Vitamin C				X
Vitamin E				X
Selenium				X
N-Acetyl cysteine				X
α-Lipoic acid				X

From Heath SE, et al., 2007. Nutritional supplementation in cases of canine cognitive dysfunction – A clinical trial. Appl. Anim. Behav. Sci. 105:284-296.



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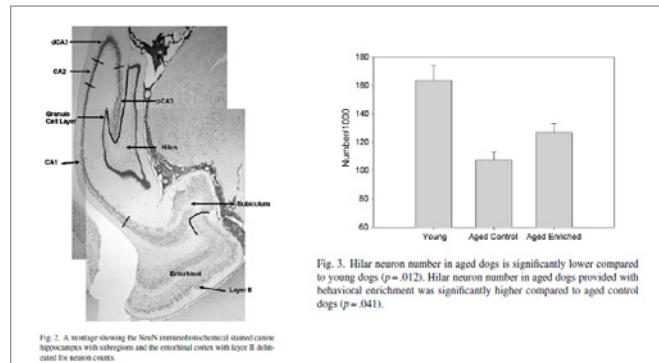


Fig. 3. (A) Spatial memory scores using a Y-maze delayed non-matching-to-sample task. Dogs provided with the combination treatment showed a significant increase in spatial memory scores compared to the control diet. DO, dog on diet; DC, dog on control diet; CA, control environment; EA, behavioral enrichment; Y, young control. \* = p < 0.05. (B) Total RNA levels per 10<sup>6</sup> RNA. DO, dog on diet; DC, dog on control diet; CA, control environment; EA, behavioral enrichment; Y, young control. Total RNA levels do not differ between groups. DO, dog on diet; DC, dog on control diet; CA, control environment; EA, behavioral enrichment; Y, young control. p = 0.93. One-way ANOVA. a = 3–4 per group.

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## Brain changes: essential points research summary



- Changes in gene expression
  - Increased expression of genes associated with stress and inflammation
- Neuronal loss in the section of the brain involved in learning associations (the hippocampus)
  - Behavioral enrichment may slow this loss
  - Decreased expression of genes involved in nerve's ability to send and receive signals is involved in cognitive change

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### Medium chain triglycerides (MCTs)

- Ketone bodies are produced from fat as an alternative energy source when brain glucose is low over time
- Sources of ketone bodies that can be given orally include medium chain triglycerides (MCTs)
- Fatty acids from MCTs cross the blood-brain barrier
- Ketone infusions (in humans) improve cognitive function
- Rodent models show that MCT sources of ketones may provide up to 20% of the energy used in normal brain tissue
- Dogs fed diets formulated with MCT supplements showed improved performance on cognitive tests when compared to control dogs\*
- In many Nestle Purina and other diets as  $\beta$ -OH-butyrate



\*Studzinski et al. Brain Res 2008;1226:209-217.  
\*Taha et al. Neurochem Res 2009; 34: 1619-1625.  
\* Pan et al. Br J Nutr 2010; 103:1746-1754.

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### Medical interventions that may help

- Selegiline (Anipryl; Pfizer) – MAO-I – cannot stop once on, earlier better than later
- Nicergoline - Europe
- Propentofylline - Europe
- Anti-anxiety medications (TCAs, SSRIs, gabapentin/pregabalin)
- Cognitive enhancers (memantine – NMDA antagonist - decreases glutamate and associated neurotoxicity)

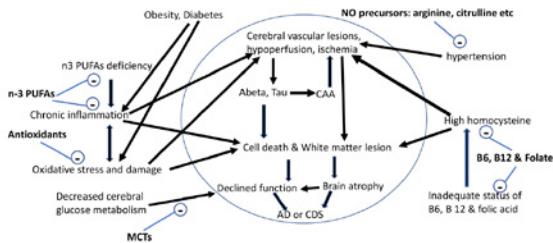
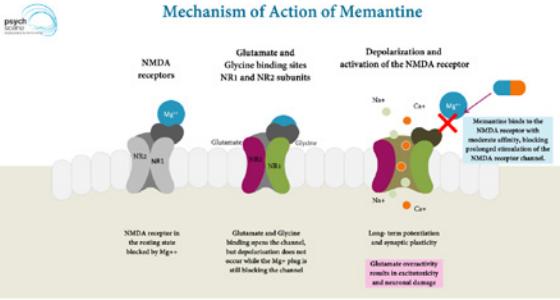


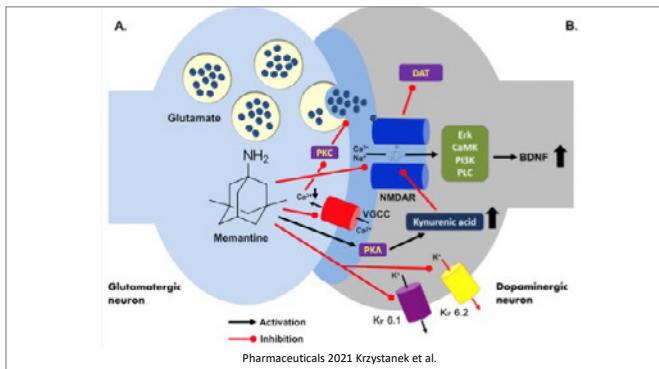
Figure 1. Nutritional management of the risk factors associated with brain aging and AD. Abeta:  $\beta$ -amyloid peptide; AD: Alzheimer's disease; CAA: cerebral amyloid angiopathy; CDS: cognitive dysfunction syndrome; n-3 PUFA: omega-3 polyunsaturated fatty acids; NO: nitric oxide; Tau: abnormal hyperphosphorylation of tau.

From: Pan Y. Nutrients, cognitive function, and brain aging: What have we learned from dogs. Med. Sci. 2021, 9, 72.

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### Summary

- Healthy, efficient neuron-to-neuron communication is important for maintaining the ability to learn and for cognitive plasticity – there is a role for lowering stress throughout life
- Neuroprotectants and anti-oxidants can improve this process by prohibiting neuronal degradation which adversely affects trophic factors
- Availability of brain E affects cognition any alternative source of energy may be neuroprotective and by stimulating neuronal metabolism will protect neurons from oxidative stress and neurotoxic assaults
- Diet and supplements that provide neuroprotective functions or that may provide precursors for neurochemicals or structural neuroanatomy may be able to play a huge role in how we prevent and treat effects of neuronal assaults, including neurocytotoxic damage associated with aging
- None of this happens in a vacuum – cognitive and physical stimulation matter

### In the pipeline

LOY-001 – injectable; LOY-003 – tablet

LOY-001/003 targeted to decrease IGF-1 (Insulin-like Growth Factor 1) levels in adult dogs (4 years giant; 7 years standard)

Rapamycin - Dog Aging Project (DAP) - mTOR (inhibits insulin signaling) inhibitor – immunomodulator/anti-inflammatory that prevents or stops insulin resistance in cells.

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Information:

- [koverall@upei.ca](mailto:koverall@upei.ca);  
[overall.karen@gmail.com](mailto:overall.karen@gmail.com)
- **Behaviour at the Beach (BATB)**  
Sunday evening 30 May  
through Friday 5 June 2026 – 36  
h RACE credits; immersion  
course; email me
- **Journal of Veterinary Behavior**  
[https://www.sciencedirect.com/  
journal/journal-of-veterinary-  
behavior](https://www.sciencedirect.com/journal/journal-of-veterinary-behavior)



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2005a

# AGING AND ASSOCIATED CHANGE ASSESSMENT

SMALL ANIMAL PROGRAM | BEHAVIOUR

## PLEASE COMPLETE THIS QUESTIONNAIRE ONLY IF THE DOG IS 8 YEARS AND OLDER.

This questionnaire is about behavior and physical changes associated with aging. This questionnaire was adapted from a series of veterinary medical questionnaires to assess changes in physical and behavioral states and includes modified questions from: Rofina, J.E., Van Ederen, A.M., Toussaint, M.J.M., Secreve, M., Van, D.S., Van, D.M., I, Van Eerdenburg, F.J.C.M., Gruijs, E., 2006. Cognitive disturbances in old dogs suffering from the canine counterpart of Alzheimer's disease. *Brain Res.* 1069, 216-226. Salvin, H.E., McGreevy, P.D., Sachdev, P.S., Valenzuela, M.J., 2011. The canine cognitive dysfunction rating scale (CCDR): A data-driven and ecologically relevant assessment tool. *Vet. J.* 188, 331-336. Overall, K.L. *Manual of Clinical Behavioral Medicine for Dogs and Cats*. Elsevier, 2013.

## BEHAVIOUR SCREEN FOR AGE ASSOCIATED CHANGES (SCORES ARE AS FOLLOW; 99 INDICATES TO REMOVE THIS DOG FOR THIS DATA POINT IN ANY ANALYSIS):

### 1. Locomotory/ambulatory assessment (tick only 1)

- No alterations or debilities noted (1)
- Modest slowness associated with aging from youth to adult (2)
- Moderate slowness associated with aging (3)
- Moderate slowness associated with aging plus alteration or debility in gait (e.g., limps, occasionally trips) (4)
- Moderate slowness associated with aging plus some loss of function (e.g., cannot climb stairs) (5)
- Severe slowness associated with extreme loss of function, particularly on slick surfaces (may need to be carried or need a support harness) (6)
- Severe slowness, extreme loss of function, and decreased willingness or interest in locomoting (spends most of time in bed) (7)
- Paralyzed or refuses to move (8)

### 2. Appetite assessment (may tick more than 1)

- No alterations in appetite (1)
- Change in ability to physically handle food (2)
- Change in ability to retain food (vomits or regurgitates) (3)

- Change in ability to find food when offered, dropped or in dish (4)
- Change in interest in food (may be olfactory, having to do with the ability to smell) (5)
- Change in rate of eating (6)
- Change in timing of eating (7)
- Change in preferred textures (8)
- Change in completion of eating (9)

### **3. Assessment of elimination function (tick only 1 in each category)**

Changes in frequencies and "accidents"

- No change in frequency and no "accidents" (0)
- Increased frequency, no "accidents" (1)
- Decreased frequency, no "accidents" (2)
- No change in frequency, with "accidents" (3)
- Increased frequency with "accidents" (4)
- Decreased frequency with "accidents" (5)

Bladder control

- No change in urination control or behaviour (0)
- No leakage or uncontrolled urination, but urinates in inappropriate or undesirable location (1)
- Leaks urine when asleep, only (2)
- Leaks urine when awake, only (3)
- Leaks urine when awake or asleep (4)
- Full-stream, uncontrolled urination when asleep, only (5)
- Full-stream, uncontrolled urination when awake, only (6)
- Full-stream, uncontrolled urination when awake or asleep (7)

Bowel control – please select the appropriate answer for the description you choose

- No change in bowel control (0)
- Defecates when awake and aware of action, but in inappropriate or undesirable locations (1)
- Formed stool      Diarrhea      Mixed
- Defecates when asleep (2)
- Formed stool      Diarrhea      Mixed
- Defecates without apparent awareness (3)
- Formed stool      Diarrhea      Mixed

**4. Visual acuity - how well does the client think the dog sees? (tick only 1)**

- No apparent change in auditory acuity (0)
- Some change in acuity dependent on ambient light condition (1)
- Some change in acuity not dependent on ambient light condition (2)
- Extreme change in acuity dependent on ambient light conditions (3)
- Extreme change in acuity not dependent on ambient light condition (4)
- Blind (5)

**5. Auditory acuity – how well does the client think the dog hears (tick only 1)**

- No apparent change in auditory acuity (0)
- Some decrement in hearing – not responding to sounds to which the dog used to respond (1)
- Extreme decrement in hearing – have to make sure the dog is paying attention or repeat signals or go get the dog when called (2)
- Deaf – no response to sounds of any kind (3)

**6. Play interactions - if the dog plays with toys (other pets are addressed later), which situation best describes that play? (tick only 1)**

- No change in play with toys (0)
- Slightly decreased ability to play with toys, only (1)
- Slightly decreased interest in toys, only (2)
- Slightly decreased interest and ability to play with toys (3)
- Extreme decreased ability to play with toys, only (4)
- Extreme decreased interest in toys, only (5)
- Extreme decreased interest and ability to play with toys (6)
- This dog has never played with toys (99)

**7. Interactions with humans - which situation best describes that interaction? (tick only 1)**

- No change in interaction with people (0)
- Recognizes people but slightly decreased frequency of interaction (1)
- Recognizes people but greatly decreased frequency of interaction (2)
- Withdrawal but recognizes people (3)
- Does not recognize people (4)
- This dog has never really interacted with people (99)

**8. Interactions with other pets - which situation best describes that interaction? (tick only 1)**

- No change in interaction with other pets (0)
- Recognizes other pets but slightly decreased frequency of interaction (1)
- Recognizes other pets but greatly decreased frequency of interaction (2)
- Withdrawal but recognizes other pets (3)
- Does not recognize other pets (4)
- No other pets or animal companions in house or social environment (5)
- This dog has never really interacted with other dogs or cats (99)

**9. Changes in sleep / wake cycle (tick only 1)**

- No changes in sleep patterns (0)
- Sleeps more in day, only (1)
- Some change - awakens at night and sleeps more in day (2)
- Much change - profoundly erratic nighttime and daytime sleep patterns with lots of nighttime activity or restlessness (3)
- Sleeps virtually all day, awake occasionally at night (4)
- Sleeps almost around the clock (5)

**10. How often does your dog pace up and down, walk in circles and/or wander with no direction or purpose?**

- Never (0)
- Once a month (1)
- Once a week (2)
- Once a day (3)
- More than once a day (4)

**11. How often does your dog stare blankly at the walls or floor?**

- Never (0)
- Once a month (1)
- Once a week (2)
- Once a day (3)
- More than once a day (4)



**12. How often does your dog get stuck behind objects and is unable to get around?**

- Never (0)
- Once a month (1)
- Once a week (2)
- Once a day (3)
- More than once a day (4)

**13. How often does your dog fail to recognize familiar people or pets?**

- Never (0)
- Once a month (1)
- Once a week (2)
- Once a day (3)
- More than once a day (4)

**14. How often does your dog walk into walls or doors?**

- Never (0)
- Once a month (1)
- Once a week (2)
- Once a day (3)
- More than once a day (4)

**15. How often does your dog walk away from or avoid being petted or other loving attention that they have been known to enjoy?**

- Never (0)
- Once a month (1)
- Once a week (2)
- Once a day (3)
- More than once a day (4)

**16. Has your dog changed in the way he/she behaves with humans, dogs or cats?**

No      Yes

Please tell us about any changes. Things in which we are especially interested are a change (increase or decrease – please tell us which) in avoidance, aggression, fear, withdrawal, approach or any other component of social interaction.

**17. Is there anything else you think we should know? If you think you have observed something interesting – even if you don't understand it – please tell us.**

3001

# THE COLLAPSING PATIENT: TIPS FOR APPROACH AND MANAGEMENT

SMALL ANIMAL PROGRAM | CARDIOLOGY

 Lynne O'Sullivan, DVM, DVSc, DACVIM (Cardiology)

The patient with a history of a collapse episode can be a diagnostic challenge to the small animal clinician for several reasons. They are often emotionally charged consultations presenting on an urgent or emergent basis with distressed and anxious owners. The clinician is tasked with determining the nature of an event they did not witness themselves - syncope, or seizure, or some other form of collapse event (musculoskeletal, neuromuscular, metabolic, etc.)? Causes of syncope can range from very benign conditions to life-threatening ones, which may cause clinicians to place significant pressure on themselves to definitively determine the cause. And yet in many cases of transient episodes, patients may appear normal by the time of examination, in which case identifying the cause can be challenging and frustrating. But with a thorough understanding of the pathophysiology of syncope and a thorough history and physical examination, an educated list of differential diagnoses can be determined and owner fears and anxieties hopefully eased.

## DEFINITION AND PATHOPHYSIOLOGY OF SYNCOPE

Syncope, the medical term for fainting, is defined as sudden and transient loss of consciousness (LOC) with loss of postural tone that is followed by spontaneous recovery. Presyncope or episodic weakness may have the same pathophysiologic basis and represent a less severe degree of syncope without the LOC. The underlying pathophysiology for all forms of syncope is sudden and transient cerebral hypoperfusion. The brain is highly dependent on continuous blood flow for oxygen and glucose delivery due to its limited stores, and cessation

of blood flow to the reticular activating system in the brainstem for 6-8 seconds may lead to LOC. Recognize that reduction in delivery of the absolute amount of oxygen or glucose due to profound hypoxia (e.g. respiratory disease, right-to-left shunt) or hypoglycemia (e.g. insulinoma, relative insulin overdose in diabetic, sepsis), independent of blood flow, can cause syncope though is more rare. The cause of loss of cerebral perfusion is often loss of systemic vascular resistance (SVR), reduction of cardiac output (CO), or both, causing transient systemic hypotension. Recall that blood pressure (BP) = SVR x CO, therefore it becomes clear that a reduction in either SVR or CO without a sufficient compensatory increase in the other can lead to a reduction in BP. Furthermore, recall that CO = stroke volume (SV) x heart rate (HR), therefore reduction in CO can come about by reduction in SV or extremes of HR (very low HR, or very fast HR such that SV falls due to lack of diastolic filling time).

### Causes of syncope

#### Loss of Vascular Resistance

##### Reflex-mediated syncope

- Central (vasovagal or neurally-mediated)

- Emotional distress or fear, pain
- Orthostatic stress

- Situational

- Hyperexcitability, hyperventilation
- Coughing
- Vomiting
- Defecation
- Urination
- Swallowing or choking

- Reflex-mediated syncope (continued)
  - Positional
  - Orthostatic hypotension (humans)
  - Carotid sinus hypersensitivity (humans)
- Drug therapy
  - Vasodilators like ACE-inhibitors, amlodipine, hydralazine, nitrates
  - Calcium channel blockers
  - Beta blockers
  - Phenothiazines
- Autonomic nervous system disease (humans)
  - Primary
  - Secondary
    - Diabetes mellitus
    - Paraneoplastic
    - Chronic renal disease
    - Immune-mediated disease

#### Reduction in Cardiac Output

- Obstruction to blood flow
  - Congenital stenosis of any of the four valves or inflow/outflow tracts
  - Cardiac tumor or thrombus
  - Obstructive hypertrophic cardiomyopathy (HOCM)
  - Restrictive cardiomyopathy (RCM)
  - Pulmonary thromboembolism (PTE)
  - Pulmonary hypertension (PH)
- Arrhythmia
  - Tachyarrhythmia
  - Atrial fibrillation
  - Atrial tachycardia
  - AV reentrant tachycardia
  - Ventricular tachycardia
  - Bradyarrhythmia
  - Third degree AV block
  - Intermittent high grade AV block with ventricular asystole
  - Sick sinus syndrome
  - Atrial standstill
  - Sinus bradycardia or AV block secondary to drug therapy with calcium channel or beta blockers

- Reduced preload
  - Pericardial effusion and tamponade
  - Caval thrombosis
  - Tumor obstructing venous return
  - Severe volume depletion (hemorrhage or dehydration)
  - Diuretic therapy

#### DIAGNOSTIC APPROACH TO THE SYNCOPAL PATIENT

While syncope is a challenging presenting complaint, it is imperative to recognize that a thorough history and physical examination are the most important and informative investigative steps in the diagnostic process. Much can be learned from careful questioning of witnesses to the event(s), and physical examination serves to focus the differentials and next steps in the diagnostic process, if further steps are necessary. In humans, the cause of syncope can be determined in more than 25% of cases on the basis of history and physical examination alone. We are at a disadvantage in veterinary medicine in that our patients cannot act as firsthand witnesses for us.

One of the challenges faced by the clinician is first identifying whether the event described is a presyncopal/syncopal event; or other event associated with impaired or LOC such as seizure, narcolepsy or cataplexy; or other event not associated with LOC such as musculoskeletal, neuromuscular, or metabolic weakness or collapse. Clients will frequently use the term collapse to refer to a variety of types of events, and frequently describe syncopal events as "seizures". Further complicating matters is the fact that behaviors associated with a syncopal event and a seizure event can be quite similar, though some distinct differences may be identified and are summarized below.

## DIFFERENTIATING FEATURES OF SYNCOPES VS SEIZURE.

	Syncope	Seizure
<b>Precipitating events</b>	May include exertion, excitement, stress, coughing, vomiting, urination, defecation	Often none May occur during sleep hours
<b>Prodromal signs</b>	Very brief (seconds) Weakness, stumbling/staggering, vocalization, signs of autonomic stimulation before reflex-mediated (mydriasis, pallor, nausea, hypersalivation)	Longer duration (minutes to days) Atypical behaviors
<b>Aura</b>	None	Present seconds to minutes before partial seizures Attention seeking or withdrawal, agitation or depression
<b>During the event</b>	No facial involvement Flaccid or rigid limbs Brief tonic-clonic motion may occur Opisthotonus may be noted Duration often transient (< 1 minute)	Chomping, hypersalivation, facial twitching Tonic-clonic limb motion with generalized seizures More focal motor involvement with partial seizure Duration longer (> 5 minutes supports seizure)
<b>Recovery</b>	Rapid recovery of consciousness Oriented and able to walk within minutes  In reflex-mediated syncope, more prolonged recovery with fatigue may occur	Slower return to consciousness Disoriented often for minutes after



## HISTORY

With the above in mind, a careful and thorough history must be sought, with particular attention to the following:

- Signalment
- Age at first event, number of events observed, time between events
- What the patient was doing at the time of or immediately preceding the event
- Relation to time of day, feeding, medications, exercise
- Whether prodromal signs were observed
- A step-by-step description of the event and what was observed, including whether the owner noted pale mucous membranes (often present in vasovagal syncope). Note that owners will often interpret the struggling to get up as the tonic-clonic motions of seizure, so frame questions carefully.
- A timeline of the event, including duration and time to return to normal (owners frequently overestimate the duration of events)
- Behaviors after the event (often quickly behave as if nothing happened after syncope, with the exception of some reflex-mediated events)
- Whether the patient is normal between episodes (as is often the case with syncope)
- Current medications
- Access to medications that may predispose to syncope
- Other systemic signs of cardiac disease, respiratory disease, neurologic disease, metabolic disease, etc.
- Other past medical history or diagnoses

## PHYSICAL EXAMINATION

Physical examination after or between events may be normal in the case of reflex-mediated syncope or intermittent arrhythmias particularly if they are not associated with underlying structural heart disease. A focus on whether there are signs of cardiovascular disease should clearly be a priority (jugular distension, auscultable murmur, arrhythmia, or gallop, bradycardia or tachycardia, muffled heart sounds, abnormal lung sounds, ascites, weak pulses, etc.). Blood pressure measurement,

though indicated, will typically be normal and is minimally informative when not during an event. If seizure is also a possibility, then a thorough neurologic exam is also indicated.

## LABORATORY BLOODWORK

A CBC and biochemistry profile may be indicated in cases where metabolic causes of weakness and collapse should be ruled out, such as anemia, hypoglycemia, or electrolyte disturbances; when evidence of a right-to-left shunt is sought (polycythemia secondary to chronic hypoxemia); when electrolyte disturbances need to be ruled out as the cause of brady- or tachyarrhythmias (e.g. hyper or hypokalemia); or when hemorrhage or profound dehydration is suspected. Recognize that routine labwork will be normal in many patients with a history of syncope. The role of cardiac biomarkers in the diagnosis of or differentiation of causes of syncope is largely unknown. One study in dogs found that cardiac troponin-I was not sufficiently discriminatory in differentiating syncope due to cardiac disease from either seizures or vasovagal syncope.

## ELECTROCARDIOGRAPHY (ECG)

If an abnormal heart rate or rhythm is auscultated during physical exam, then an in-house ECG is indicated for rhythm diagnosis. If heart rate and rhythm are normal, it is still prudent to document normal sinus rhythm. Furthermore, an ECG may reveal important diagnostic findings not appreciated on auscultation such as first or second degree heart block or bundle branch block which may suggest conduction system disease, premature beats or other intermittent arrhythmias, or evidence of myocardial ischemia. Ambulatory ECG monitoring devices such as Holter monitors, event recorders, and implantable loop recorders can be very useful to assess for intermittent arrhythmias and can be diagnostic, particularly should an event occur while wearing the device. Each type of device offers its own advantages and disadvantages. A Holter monitor records continuously for most commonly 24 hours with certainty of capturing the rhythm should an event occur during this time, however it provides short-term monitoring only with most units capable of recording for 72 hours maximum. A Holter recording may still yield suggestive information if a syncopal event is not experienced while wearing the device (e.g. arrhythmias present but insufficient to

cause collapse). It is also important to recognize that if syncope does not occur during wear, a normal recording cannot definitively rule out arrhythmia as the cause. An event recorder may be worn for longer periods of time, however it must be remotely activated by the owner to store the rhythm recording at the time of an event (it does not record continuously). Implantable loop recorders are small devices that are implanted subcutaneously, often with only sedation and a local block, and may be worn for many months, rendering them useful for very infrequent episodes. There is also a description of their successful use transcutaneously in dogs.

## DIAGNOSTIC IMAGING

If structural cardiac disease is suspected based on history and physical exam, then echocardiography is indicated. Thoracic radiography may also yield important diagnostic information, particularly if there are concurrent respiratory signs. Examples of structural cardiac diseases commonly associated with syncope are listed in Table 1.

## VIDEO RECORDING

Owners should be encouraged to video record any future episodes and to keep a log of frequency and circumstances surrounding the episodes. Observing a video of the event can sometimes yield very useful and diagnostic information that perhaps hasn't been communicated in the owner's description of the events.

An investigation focusing first on a thorough history and physical examination (including cardiac and neurologic physical examination), followed by targeted diagnostics including ECG and echocardiography and aided by video recording, can be reasonably productive in yielding a diagnosis. That being said, there are a percentage of syncope cases that will remain undiagnosed. Thankfully, at least one study suggests that deterioration and death are uncommon outcomes for dogs other than Boxers with collapse, syncope, or exercise intolerance of unknown cause. This may at least help to alleviate clients' fears when faced with the lack of a definitive diagnosis.

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3002

# CONGESTIVE HEART FAILURE: MYTHS AND TRUTHS

SMALL ANIMAL PROGRAM | CARDIOLOGY

 Lynne O'Sullivan, DVM, DVSc, DACVIM (Cardiology)

**Myth: Coughing in geriatric small breed dogs with myxomatous mitral valve disease (MMVD) is an indicator of congestive heart failure (CHF) and grounds for diuretic treatment**

While dogs with pulmonary edema from left-sided heart failure may experience cough, both clinical experience and the literature would support that coughing is not an indicator of CHF in dogs with MMVD and more likely reflects airway disease or severe left atrial dilation. In a recent study examining probability of coughing in dogs with MMVD by analyzing previously published data sets, the investigators found no effect of CHF on probability of coughing (Rishniw 2025).

Dogs with severe left atrial dilation had increased probability of coughing relative to those with lesser degree of left atrial dilation or no left atrial dilation, but this was independent of presence of CHF. And in the group of dogs with CHF, very rarely was coughing reported for the first time at the onset of CHF. This supports that coughing should not be a diagnostic criterion for CHF in dogs with MMVD, and small breed dogs with mitral regurgitation murmurs presenting for cough are not necessarily candidates for diuretic therapy. The non-diuretic effects of loop diuretics, which include anti-inflammatory and bronchodilatory effects, can further confound the perceived association; that is, a therapeutic "response" to diuretic frequently leads to the erroneous conclusion that cough = CHF. Unnecessary diuretic use can have unwanted side effects, renal effects, and feeds diuretic resistance (see below). Cough without tachypnea or dyspnea in dogs with MMVD should not be regarded as a sign of CHF and should not be treated as such.

**Myth or Truth? Left atrial dilation causes bronchial compression and cough in dogs with MMVD**

This has long been a controversial topic in veterinary medicine without clear resolution. In people, left atrial dilation and elevated pulmonary artery pressure reportedly cause left mainstem bronchial compression and cough, however evidence in dogs had been largely anecdotal. The study alluded to above (Rishniw 2025) would suggest that coughing in dogs with MMVD is more probable with severe left atrial dilation in comparison to lesser degrees of left atrial dilation, independent of presence of CHF, perhaps supporting a direct effect of left atrial dilation on mechanism and induction of cough. Conversely, an earlier study (Singh 2012) of bronchoscopy in coughing dogs failed to show an association between left atrial dilation and left bronchus compression. Coughing dogs with and without left atrial dilation were both found to commonly have collapse of cranial and caudal segments of the left bronchus as well as evidence of airway inflammation, hyperemia, and mucus accumulation. These results support the notion that coughing in dogs with MMVD may largely be due to concurrent airway disease (bronchomalacia and inflammation). Additional practical findings of this study included the following limitations of radiography: 1) radiographs were inaccurate in identifying cardiomegaly compared to echocardiography, 2) airway collapse on static radiography did not correlate with collapsing segments on fluoroscopy and bronchoscopy, and 3) radiographs were insensitive in detecting chronic bronchitis. The connection between left atrial dilation and bronchial compression, if any, is still unclear in dogs.

## **Truth: Increased home resting respiratory rate is one of the earliest indicators of CHF in dogs with MMVD**

The EPIC trial was a prospective multicenter, blinded, randomized, placebo-controlled clinical trial of the efficacy of pimobendan in delaying the onset of congestive heart failure in dogs with stage B2 MMVD. Of 208 dogs with multiple examinations and adequate follow-up time, 135 experienced CHF and 73 did not, allowing comparison of clinical and radiographic characteristics between the two groups (CHF and no CHF). Previous studies have reported change and rate of change in radiographic heart size (VHS) and echocardiographic variables leading up to CHF. Longitudinal analysis of home resting respiratory rate data demonstrated a dramatic increase from average 23 breaths per minute to average 41 breaths per minute during the 4 months preceding presentation for CHF. In clinic respiratory rate and heart rate were also noted to rise in the 4 and 10-months preceding CHF, respectively. Home resting respiratory rate monitoring has the potential to identify CHF early, before the onset of distress and severe signs, enabling early intervention.

## **Myth: Coughing in cats is a sign of CHF**

Many of us would agree with this myth as we've been repeatedly taught and have read in textbooks that coughing is not a sign of CHF in cats, and coughing in cats should prompt evaluation for primary respiratory disease. But what is the evidence? In a prospective study of 101 cats presenting for dyspnea in primary (first opinion) practice, 25% of cats determined to have a cardiac cause for dyspnea in fact had a history of cough (Dickson 2018). This was compared to 47% of those with respiratory disease and 40% of those with neoplastic disease. Still, 25% was surprising and suggests the presence of cough should not be used to rule out CHF! What is not clear is whether these cats may have had concurrent respiratory disease despite cardiac dyspnea, so cause and effect are still not being suggested here. A physical exam finding that was rather specific for cardiac dyspnea but not sensitive was the presence of a gallop (23% of cardiac dyspnea had, whereas 0% of other causes had). And cats with cardiac dyspnea had lower temperatures and higher respiratory rates compared to other causes.

Conversely, previous studies have supported the myth by reporting low frequency of coughing in cats with CHF

compared to respiratory disease (6% vs 63%, respectively, Smith 2011). The difference in findings may relate to the inclusion criteria for the cats under study (different presenting complaints – dyspnea vs cardiorespiratory evaluation).

## **Myth: Furosemide is effective with once daily administration**

When it comes to convenience and compliance, we'd all like to reduce dosing frequency to once daily for our clients. Furosemide's rapid onset of action following both oral and IV administration are ideal especially for emergency use, but its relatively short duration of action does not lend itself well to once daily administration, and doing so may leave the patient unprotected from avid sodium and water reabsorption for much of the day. It is more justified to divide the total daily dosage among 2-3 administrations. For a patient 'doing well' on once daily administration, it begs the question as to whether the patient needs diuretic at all. Consider the following pharmacokinetics in dogs and cats when making dosing frequency decisions:

IV (IM similar): onset 5-15 min, peak 30 min, duration 2-3 hrs (90% eliminated by 2 hrs, 100% by 3 hrs)

PO (SC similar): onset 20-30 min, peak 1-2 hr, duration 6 hrs

Transdermal application in cats produces no significant absorption!

## **Truth: Patients can develop resistance to loop diuretics like furosemide in both acute and chronic use settings**

Increasing awareness of diuretic resistance and the negative impact it may have on patient management and outcomes has led to increased research attention and thought to treatment strategies.

In dogs, diuretic resistance is typically defined as the need for  $> 8$  mg furosemide/kg/day (along with standard heart failure medications) to control clinical signs of CHF. Mechanisms of diuretic resistance can include:

- Underdosing or non-compliance
- Decreased absorption

- Impaired delivery to the kidney or secretion into the tubule
- Enhanced renal sodium absorption (hypertrophy of distal tubule segments)
- Rebound sodium and water retention
- Excessive sodium or water intake
- Excessive neurohormonal activation (RAAS, vasopressin)

Identification of contributory factors can inform management strategies, including:

- Changing dosage, frequency, or route of administration
- Added use of neurohormonal blockade (ACE-inhibitors, mineralocorticoid receptor blockers)
- Sequential nephron blockade (synergistic diuretics acting elsewhere in the nephron)
- Dietary sodium restriction
- Cautious water restriction
- Inotropic support

#### **Myth: Cardiogenic pulmonary edema (from CHF) should be radiographically bilaterally symmetric in terms of lung distribution**

Cardiogenic pulmonary edema absolutely can and does lateralize! In dogs it is not uncommon to see it worse in the right dorsocaudal lobe compared to the left, and in cats it is frequently asymmetric and patchy without the classic caudodorsal distribution. In fact, in some cats, it may be worse ventrally. Asymmetry of distribution should not be used to rule CHF out.

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3003

# DIAGNOSTIC IMAGING IN THE CARDIAC PATIENT

SMALL ANIMAL PROGRAM | CARDIOLOGY

 Lynne O'Sullivan, DVM, DVSc, DACVIM (Cardiology)

## LEARNING OBJECTIVES

1. Compare and contrast the diagnostic information (including strengths and pitfalls) that can be gained from thoracic radiography, point-of-care thoracic ultrasound, and echocardiography for known or suspected cardiac problems
2. Identify key radiographic anatomy and findings in cardiac patients
3. Understand indications and limitations of point-of-care ultrasound in cardiac patients
4. Understand indications for echocardiography and common components of an echo exam or report
5. Prioritize imaging techniques according to presenting complaints and disease processes

## THORACIC RADIOGRAPHY

Strengths: cardiac chamber dilation; airways, pulmonary parenchyma, and vasculature; pleural space; familiarity/skill

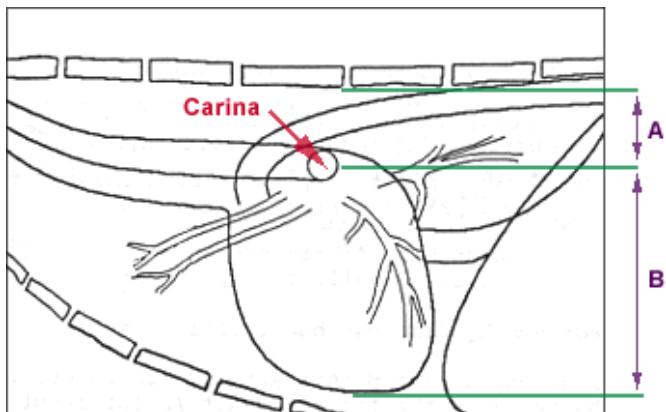
Weaknesses: cardiac hypertrophy, specific cardiac anatomy, cardiac masses, unstable patient

- Baseline in patients with preclinical cardiac disease and concurrent chronic respiratory conditions
- E.g. feline asthma and preclinical HCM, chronic bronchitis and MMVD
- Imaging of mediastinal / pleural space

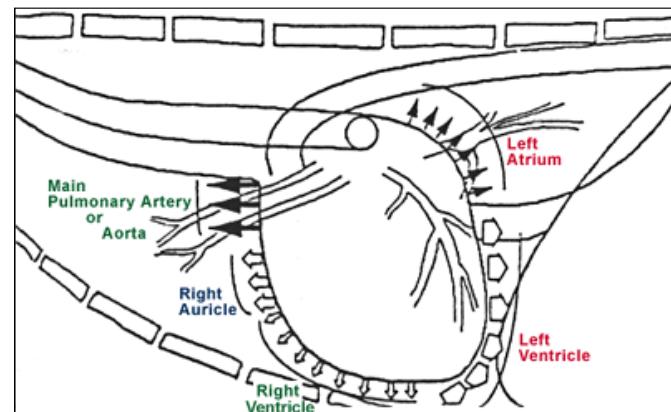
## RADIOGRAPHIC CARDIAC ANATOMY

Heart size may be assessed in general by measuring the height and width of the cardiac silhouette relative to the thorax (cardiothoracic ratio) in lateral and DV views (see rules below), respectively, and localized cardiac enlargement may be appreciated using the anatomic schematics below. In cats, the cardiothoracic ratio in the DV/VD view is more commonly used than the lateral view, whereas both are used commonly in dogs.

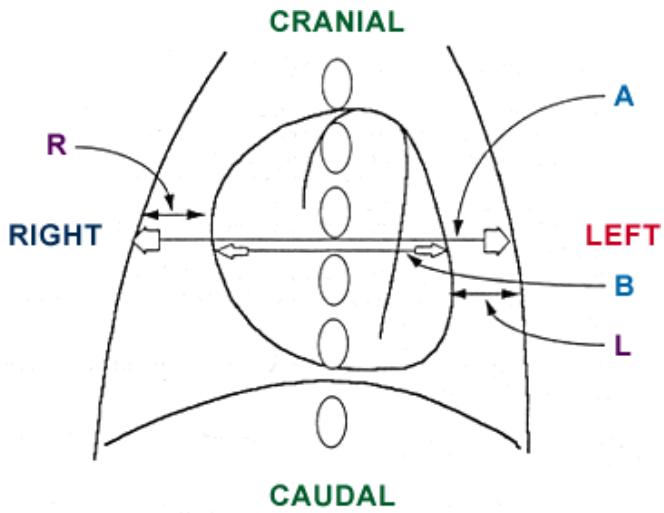
**Lateral view: Normal B  $\leq$  2/3 to 3/4 of A+B**



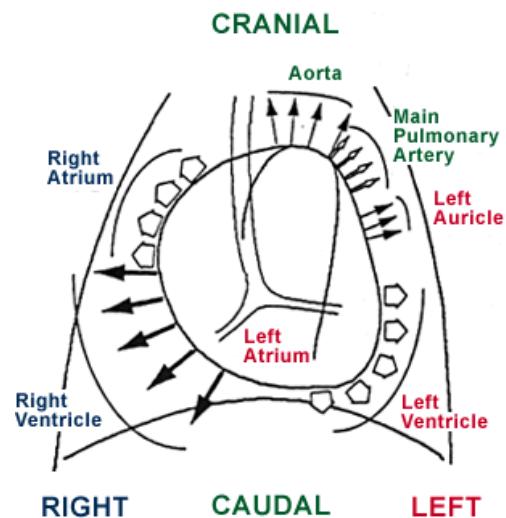
**Localized cardiac enlargement**



**DV view: Normal B  $\leq$  2/3 A**



**Localized cardiac enlargement**



DV view better for:

- Consistent cardiac silhouette
- Pulmonary lobar vessels
- Caudal dorsal lung
- Trachea and mainstem bronchi
- Left atrium

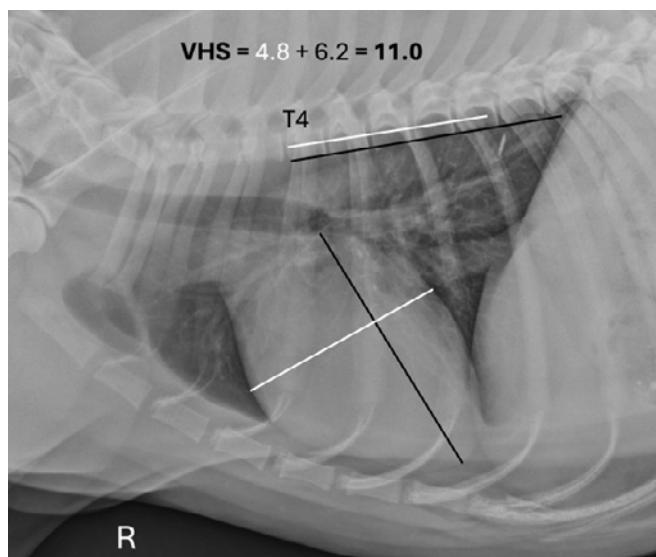
VD view better for:

- Cranial and caudal mediastinum
- Caudal vena cava
- Accessory lung lobe
- Pleural effusion

## OBJECTIVE RADIOGRAPHIC CARDIAC INDICES

The reported mean +/- SD (range) vertebral heart size (VHS) in 100 normal dogs of various conformations was 9.7 +/- 0.5 (8.5-10.6) in the original publication by Buchanan (Buchanan 1995). However, breed specific ranges should be used where available, as larger normal values are reported in several breeds including pugs, Pomeranians, Boston terriers, English bulldogs, Spitz, CKCS, Labradors, boxers, beagles, and miniature schnauzers. The reported mean +/- SD (range) in normal cats is 7.5 +/- 0.3 (6.7-8.1) in the lateral view (Litster 2000) and is generally unaffected by breed.

### Example calculation of vertebral heart size:



Current MMVD Consensus guidelines suggest that in small breed geriatric dogs with left apical systolic heart murmurs of 3/6 and greater, a VHS >11.5 may support the presence of stage B2 MMVD, a stage with therapeutic implications. This approach has limitations and additional radiographic indices have been investigated to increase the specificity of radiographic use in stage assessment (see below). A retrospective study of 90 dogs with coughing and clinical evidence of MMVD found that a VHS < 11.4 was supportive of a non-cardiac cause of cough, while VHS > 11.4 was indeterminate (may have had cardiac or respiratory cough, or both) (Guglielmini 2009). Another clinically relevant use of VHS is in serial monitoring of MMVD patients – studies have shown patterns of abrupt increase in VHS in the months leading up to congestive heart failure, thus change

in VHS may be a useful parameter in prediction of CHF onset (Boswood 2020; Lord 2011).

VHS has limitations in cats for ability to distinguish normal cats from cats with cardiac disease. A VHS cut-off of 8.2 on the lateral view was shown to be only moderately sensitive for detection of cardiac disease in cats (Guglielmini 2014). This is perhaps not surprising considering hypertrophic cardiomyopathy is the most common feline acquired heart disease and leads to concentric hypertrophy which may not produce overt enlargement of the cardiac silhouette to the same degree that chamber dilation does. VHS in cats has also been evaluated for utility in distinguishing CHF from other causes of respiratory distress. VHS values less than 8.0 likely rule out CHF while those >9.3 are highly specific for CHF cause of acute respiratory distress in cats according to one retrospective study (Guglielmini 2015).

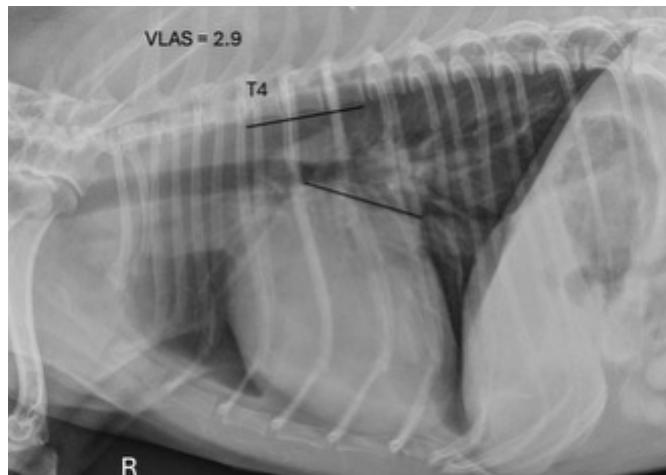
Several imposters for cardiomegaly may need to be considered when an enlarged cardiac silhouette is identified radiographically. Examples of conditions that may mimic true cardiomegaly include pericardial effusion, intrapericardial masses or fat, and pericardioperitoneal diaphragmatic hernia.

Additional radiographic indices targeted at detecting left atrial dilation include vertebral left atrial size (VLAS) in dogs and left atrial VHS (LA-VHS) in cats. Clinically, these indices may have value in staging MMVD, assessing cats for candidacy for anti-thrombotic therapy, and helping to support a diagnosis of congestive heart failure in dogs and cats with respiratory signs. VLAS is measured by extending a line from the mid-point of the ventral carina to the caudal aspect of the left atrium where it intersects with dorsal border of the caudal vena cava (line in canine radiograph below, showing left atrial dilation). A line of the same length is then extended from the cranial aspect of T4 parallel to the spine and the number of vertebrae counted to a tenth of a vertebra (in the case below, 2.9). Reported median in 15 smaller breed dogs was 2.1 (25th-75th percentiles: 1.8-2.3) whereas values >2.5 were consistent with LA dilation with 67% sensitivity and 84% specificity in 88 dogs with MMVD (Malcolm 2018).

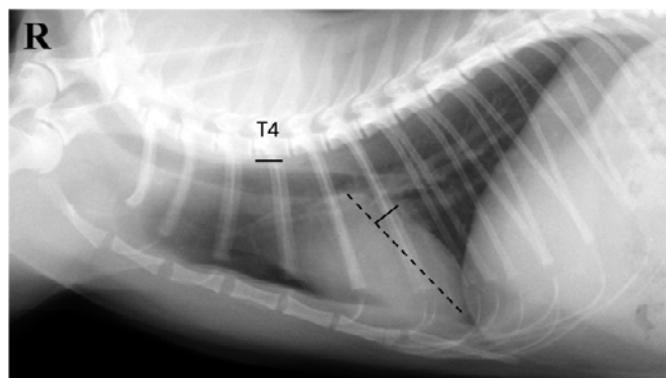
VHS-LA is measured in cats by constructing a long-axis dimension on a lateral view from the ventral aspect of the carina to the left apex of the heart (dashed line below), then placing a line perpendicular to that anchored at the caudal aspect of the left atrium where it intersects with the dorsal

border of the caudal vena cava (solid line below). The length of this line is then re-created along the vertebrae starting at the cranial aspect of T4 (as for VHS) and the measure reported to a tenth of a vertebra (in the case below, 0.9). Reported median normal values for VHS-LA in cats range between 0.87 to 1.0 vertebrae (Guglielmini 2015).

### VLAS in Dogs



### VHS-LA in Cats



### IDENTIFICATION OF LEFT-SIDED CONGESTIVE HEART FAILURE

The increase in hydrostatic pressure that precedes the development of CHF can be useful diagnostically as it leads to a constellation of radiographic findings, particularly when respiratory signs are secondary to chronically progressive left-sided cardiac disease. That sequence

includes increased left atrial pressure, increased pulmonary venous pressure, increased pulmonary capillary pressure, followed by pulmonary edema. From this, it can be appreciated that the presence of left atrial dilation and pulmonary venous distension should typically accompany the pulmonary opacities attributable to CHF. Exceptions to this include acute processes such as mitral valve chordal rupture, infective endocarditis, iatrogenic fluid overload, and in some cats. Cats with left-sided CHF are reportedly less likely to show detectable left atrial dilation and pulmonary vein enlargement than dogs, and more likely to have concurrent pleural effusion, which may make these classic changes difficult to detect.

The distribution of radiographic pulmonary changes should also be considered in the differentiation of CHF from primary respiratory disease, at least in dogs. In dogs, CHF is often worse caudodorsally though it may be diffuse when severe. Other processes can also be primarily distributed caudodorsally such as non-cardiogenic edema. It is not uncommon for cardiogenic edema to lateralize or be worse on one side (more commonly the right) on a DV or VD view, so CHF should not be excluded based on lateralization. Pulmonary changes that are predominantly cranoventral or lobar are more likely to be non-cardiac in origin. Cats, however, once again like to break the rules and are more likely to have non-uniform, regional, and ventral opacities when they have cardiogenic edema compared to dogs.

Particularly challenging cases include very small breed dogs such as Yorkies and Chihuahuas in whom normal hearts can often have the impression of being radiographically enlarged, geriatric small breed dogs who may have concurrent cardiac and respiratory disease, and cats with acute CHF or with concurrent cardiac and respiratory disease.

### POINT-OF-CARE ULTRASOUND (POCUS)

Utility in rapid assessment for: 1. Pleural effusion, 2. Pericardial effusion, 3. Wet vs dry lung, 4. Left atrial size, 5. Systolic function

Indications: tachypnea, dyspnea, trauma, collapse, syncope, tachyarrhythmia to aortic diameter ratio, subjective assessment of ventricular systolic function, presence of pericardial effusion, and caudal vena cava size.

Focused thoracic space and lung windows allow for evaluation of pleural effusion, and wet vs dry lung. Wet lung is identified by the presence of B-lines or “rockets” which are non-attenuating, hyperechoic, vertical lines that extend from the pleura all the way through to the distal far field of the sector and are an artifact created by interstitial fluid. B-lines are not specific for heart failure and may also be caused by non-cardiogenic processes such as non-cardiogenic pulmonary edema, ARDS, contusions, inflammation, or hemorrhage. Scoring systems exist to systematically evaluate cranial, middle, perihilar, and caudodorsal locations bilaterally for number of B-lines and determination of likelihood and distribution of wet lung. This coupled with physical examination and other POCUS indices can assist in ranking differential diagnoses and determining best next steps.

Cautions with POCUS include satisfaction of search bias and confirmation bias. Satisfaction of search can cause a premature stopping of searching for additional findings after identifying a first one, leading to errors of omission. Confirmation bias is the tendency to give greater weight to data that support a preconceived or preliminary diagnosis while failing to seek or dismissing contradictory evidence. Both types of bias that can lead to erroneous conclusions and diagnoses.

## ECHOCARDIOGRAPHY

What information does it yield?

- Morphologic diagnosis
- Hemodynamic data
- Severity
- Treatment options
- Treatment response
- Monitoring disease evolution
- Detection of complications

Common Indications:

- Investigation of new murmurs, arrhythmias
- Investigations of murmurs in puppies and kittens
- Screening high risk breeds for known predisposed cardiac diseases
- Screening breeding animals
- Assessment of patients with suspect CHF patients with:
  - Respiratory signs
  - Pleural effusion
  - Abdominal effusion
  - Investigation of syncope
  - Suspect pericardial effusion
  - Suspect pulmonary hypertension

The details of what should be included in a thorough echocardiographic report will be discussed.

## REFERENCES AVAILABLE UPON REQUEST.

3004

# FELINE HEART DISEASE: LATEST UPDATES

SMALL ANIMAL PROGRAM | CARDIOLOGY

Speaker: Lynne O'Sullivan, DVM, DVSc, DACVIM (Cardiology)

## ACVIM CONSENSUS STATEMENT GUIDELINES FOR THE CLASSIFICATION, DIAGNOSIS, AND MANAGEMENT OF CARDIOMYOPATHIES IN CATS. JOURNAL OF VETERINARY INTERNAL MEDICINE 2020.

These guidelines are the product of a rigorous process of literature review on the most important issues related to cardiomyopathy in cats, seeking evidence-based medicine wherever possible, carried out by a panel of experts (Diplomates in Cardiology) and openly reviewed by the ACVIM Cardiology membership.

Cardiomyopathies are the most identified cardiac conditions in cats, and they represent a group of heart muscle disorders characterized by various phenotypes and prognoses. Hypertrophic cardiomyopathy (HCM) remains the most common phenotype and is the focus of the guidelines. The open-access resource clarifies the classification of cardiomyopathies specifically in cats, provides practical and ranked guidelines on how to diagnose and manage these conditions, and has been a springboard for subsequent research given the limited evidence-based literature in feline heart disease.

### Practice-impacting highlights include:

- Staging of feline cardiomyopathies (stages A-D) serves a practical purpose built around risk, and specifically risk of congestive heart failure (CHF) and arterial thromboembolism (ATE)
- Left atrial size is one of the most important prognostic markers. Left atrial systolic function and extreme hypertrophy are also prognostically important.

- Prevalence of HCM increases with age and reported as high as 29% in the general population of older cats. Most cats with HCM are non-pedigree.
- Subclinical HCM has important clinical consequences, with almost 30% of cats experiencing a cardiac outcome (CHF, ATE) or cardiac mortality in the long term.
- Cats that experience CHF as a result of stressors or iatrogenic factors like IV fluids, general anesthesia, or long-acting corticosteroids have longer survival times than those experiencing CHF in the absence of those factors.
- Heart murmur intensity does not correlate with severity of disease or stage.
- Thoracic radiography and ECG have low sensitivity for detection of disease and are therefore poor screening tools.
- In cats presenting with respiratory distress, NT-proBNP and point-of-care ultrasound (POCUS, with appropriate training and practice), can be helpful in determining if CHF is the cause or not. A negative NT-proBNP is highly suggestive of a primary respiratory condition, and severe left atrial dilation with B-lines or effusion on POCUS is highly suggestive of CHF.
- Treatment is typically not recommended for cats with subclinical stage B1 disease, but a monitoring program is highly recommended.
- Thromboprophylaxis and home respiratory rate monitoring are recommended for cats with subclinical stage B2 disease.

- ACE-inhibitors and spironolactone have not been shown to have benefit in subclinical B2 disease. Atenolol (beta-blocker), while sometimes used in cats with severe left ventricular outflow tract obstruction, has not been shown to impact outcome.
- Decisions on diuretic and antithrombotic use can be made for cats with high suspicion of CHF without characterization of cardiomyopathy phenotype (i.e. without an echocardiogram). The added benefit of additional therapies may depend on phenotype.

## CLINICAL RECOGNITION OF TRANSIENT MYOCARDIAL THICKENING, AN HCM MIMIC

Transient myocardial thickening (TMT) mimicking HCM has been recently described associated with CHF. Unlike in HCM, left ventricular hypertrophy and left atrial dilation have resolved over time in this condition, leading to an excellent and very different prognosis than HCM. The etiology is suspected to be acute and transient myocarditis or myocardial edema, resulting in a hypertrophied phenotype that resolves with time.

Two independent multi-center case series have reported this condition in nearly 50 cats. The cats in these reports have been younger (average 2-3 years old) and often have a history of an antecedent stressful event like general anesthesia, trauma, or other illness. A minority have had histories of infectious diseases. Despite presentation for fairly severe clinical signs (some with shock), their hearts normalized on echocardiography over a period of months, and treatment for CHF was able to be discontinued in the majority.

This condition represents an important differential for cats presenting with CHF and an HCM phenotype, particularly those that are younger and with an antecedent stressful event.

Awareness of this condition has important implications for veterinarians facing treatment decisions and client communication, considering the impact it could have on prognosis and premature euthanasia.

## EMERGING THERAPIES FOR FELINE CARDIOMYOPATHY

Apart from antithrombotics for the secondary prevention of ATE, there are still no effective therapies in subclinical HCM that prolong survival time or reduce risk of adverse

outcomes. This demands investigation of disease mechanisms and novel therapies to improve the lives of cats with this disease.

Two such therapies that are currently under investigation in cats with HCM include sirolimus (rapamycin) and cardiac myosin inhibitors. With delayed-release sirolimus already conditionally approved by the FDA in the United States for treatment of subclinical feline HCM and the resultant potential for client inquiries, it is important for veterinarians to be aware of the present state of knowledge and potential limitations with these treatments.

### Sirolimus (Rapamycin)

Sirolimus (also known as rapamycin) is a macrolide produced by a species of *Streptomyces* bacteria that inhibits a protein kinase (mTOR), which when targeted appropriately, results in reduced protein synthesis and upregulated autophagy. In a recent small clinical trial of 43 cats with subclinical non-obstructive HCM, rapamycin administered once weekly reduced progressive left ventricular hypertrophy compared to placebo over a 6-month period. A greater number of adverse events were reported in the rapamycin group (though not statistically significant), including one death from diabetic ketoacidosis, an important adverse event given the known potential effect of glucose intolerance with rapamycin in some species.

A multicenter, randomized, blinded, placebo-controlled clinical trial (the HALT study) is currently underway in the U.S. to investigate the effects of delayed-release sirolimus (Felycin®-CA1, TriviumVet) in cats with stage B1 or B2 HCM over a 12-month period. The primary outcome is a composite of echocardiographic progression or clinical progression, and interim results (after 50% enrolment) are expected in mid 2026. Felycin®-CA1 received conditional approval by the USFDA in March 2025 for the treatment of subclinical HCM in cats, allowing 5 years to provide efficacy data to seek full approval. Felycin®-CA1 is not currently available in Canada.

### Cardiac Myosin Inhibitors

Small molecule inhibitors targeting cardiac myosin are an emerging group of drugs shown to have benefit in humans with obstructive HCM in several recent phase III clinical trials, and at least one of these products (mavacamten) is currently approved for use in humans with symptomatic obstructive HCM. The next-in-class drug, aficamten,

was shown to reduce systolic function and reduce left ventricular outflow tract pressure gradient following a single oral dose in a group of 8 purpose-bred Maine Coon cats with obstructive HCM. While not currently available in veterinary medicine, further study of the effects of this class of medication is warranted and expected in the future for feline HCM.

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3005

# CANINE DEGENERATIVE MITRAL VALVE DISEASE: LATEST UPDATES

SMALL ANIMAL PROGRAM | CARDIOLOGY

 Lynne O'Sullivan, DVM, DVSc, DACVIM (Cardiology)

## ACVIM CONSENSUS GUIDELINES FOR THE DIAGNOSIS AND TREATMENT OF MYXOMATOUS MITRAL VALVE DISEASE IN DOGS. JOURNAL OF VETERINARY INTERNAL MEDICINE 2019.

These guidelines represent an update to the previous guidelines published in 2009. Similar to the guidelines for feline cardiomyopathies, these guidelines were the product of a rigorous process of literature review of evidence-based medicine wherever possible, carried out by a panel of experts (Diplomates in Cardiology) and openly reviewed by the ACVIM Cardiology membership. Myxomatous mitral valve disease (MMVD) continues to be the most common heart disease in dogs in most parts of the world including North America, thereby having a high impact in many aspects of general practice.

### Practice-impacting highlights and important changes from the previous guidelines include:

- In small breed dogs, the disease is typically slowly progressive but can sometimes be unpredictably more rapidly progressive in individuals. Larger breed dogs with MMVD often have faster progression with accompanying systolic dysfunction.
- Depending on the age of onset, rate of progression, and co-morbidities, subclinical MMVD may or may not affect an individual's quality or length of life.
- With advanced subclinical disease, factors associated with risk of heart failure include age, progressive left atrial and ventricular enlargement,

evidence of increased filling pressures on echocardiography, increased NT-proBNP, and increases in resting heart rate.

- Staging of MMVD (stages A-D) serves a practical purpose in terms of screening, monitoring, and implementing interventions at the appropriate time points to improve outcomes.
- Recommendations for patients with stage B1 MMVD – subclinical with no cardiac enlargement or only mild left-sided enlargement – have not changed. No drug or dietary treatment is indicated. Monitoring by echocardiography (or radiography if not available) is indicated every 6-12 months.
- Asymptomatic dogs with more advanced MMVD resulting in substantive left atrial and left ventricular enlargement (stage B2), can benefit from treatment with pimobendan (benefit = delay of onset of heart failure). This stage is best defined by specific echocardiographic criteria. Where unavailable, it may be possible to capture this subset of dogs with reasonable likelihood based on radiographic indices including vertebral heart size (VHS) and vertebral left atrial size (VLAS), understanding the limitations.
- Coughing alone in the absence of tachypnea or increased breathing effort is rarely representative of congestive heart failure (CHF); rather, coughing in isolation is much more likely to represent primary respiratory disease.
- Increased/increasing home resting respiratory rate (RRR) can be a very sensitive marker for the onset or worsening of CHF, and clients should be educated

on how to monitor RRR and when to alert their veterinary caregiver.

- Therapeutic recommendations for chronic stage C MMVD (chronic CHF) include furosemide, pimobendan, ACE-inhibitor, and spironolactone, with the understanding that staged initiation for the latter two medications is preferred by some clinicians, particularly following an acute episode of CHF.
- Surgical interventional options for patients with stage B2 and stage C MMVD are becoming increasingly available and may be of substantive benefit in the right candidates. See below.

## SURGICAL INTERVENTIONAL OPTIONS FOR MYXOMATOUS MITRAL VALVE DISEASE

Since the publication of the above guidelines 6 years ago, surgical intervention for MMVD has become a rapidly evolving field. While still limited by geography and expertise, expansion of centres offering interventional options and encouraging data on refinement of techniques and positive patient outcomes suggest incredible potential for the future of MMVD management.

Open-heart surgical annuloplasty with artificial chord repair performed with cardiopulmonary bypass is available at few centers globally but now includes a North American center at the University of Florida with the reinstatement of their cardiac surgery program. <https://smallanimalvethospital.ufl.edu/clinical-services/surgical-repair-of-the-mitral-valve/>. Experienced centers report surgical success in >95% of patients with stage B2 and stage C disease with marked reductions in heart size and ability to discontinue cardiac medications in the long term in the majority of patients. Admittedly the cost and accessibility of this procedure continue to limit its applicability.

Transcatheter edge-to-edge repair (TEER) using the canine V-clamp device is an exciting new technique involving the placement of a device to bring the mitral valve leaflets closer together, thereby reducing the regurgitant orifice and valvular regurgitation. The device is deployed through a catheter advanced through a small incision in the thorax and cardiac apex of the beating heart. Recent publications and conference presentations have shared feasibility, operative success, and positive outcomes, with 1-year survival rate of 80% and 2-year survival rate of 75% at the most experienced institution in the US (Colorado State University). Multiple other centers

are now also offering this procedure, including the University of Illinois, Tufts, North Carolina State University, and others. The ongoing expansion of interventional centers offering this procedure has the potential to revolutionize options for MMVD on a wider scale.

Overall cost is substantively lower than open heart valve repair techniques.

A New Hope For Dogs With Mitral Valve Disease (MVD): TEER V-Clamp | Twin Trees Vet Talk (<https://www.youtube.com/watch?v=9FiFg-7-rZ4>)

Left atrial decompression is a minimally-invasive catheter-based technique offering effective palliation for patients with advanced MMVD and CHF that are refractory to medical therapy (stage D) and that are not candidates for the above surgical measures. An atrial septal defect is created via transtempal puncture and balloon catheter dilation, essentially "decompressing" or offloading the left atrium and resulting in significant reduction in left atrial pressure and amelioration of CHF signs. The veterinary cardiologists at VCA West L.A. have been the pioneers of this technique in veterinary medicine and have demonstrated feasibility, safety, and efficacy in reducing left atrial pressure and improving clinical signs, with a median survival time just over one year post-operatively. Risk of right heart overload and right heart failure exists, among other complications, but can be minimized through patient selection.

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4001

# SETUP FOR SUCCESS: HOW TO GET THROUGH YOUR PROCEDURES WITH MORE EASE

SMALL ANIMAL PROGRAM | DENTISTRY

 Jane Pegg, DVM, MS, DAVDC

The magic of a great dental procedure begins before the first contact and ends after the pet is completely recovered, and includes planning, equipment setup, patient positioning, instrumentation, and communication.

## PROCEDURAL

### Consultation phase

- As a vet, this is usually your first touch point
  - Setting realistic expectations
    - Best estimate of what will be needed versus 'we don't know until we're in there'
  - Presenting an accurate cost-estimate based on your physical exam
  - Lumping rather than splitting charges

### Pre-op Phase

- Setup for surgery versus actually setup for surgery

### Intra-op Phase

- Client update
  - Every client every time
- Patient positioning – the benefits of dorsolateral in a trough versus lateral
  - Heat loss prevention and warming
  - Radiograph acquisition
  - Surgical visualization
  - No need to 'flip' to the second side
- Helpful devices/hacks
  - Mouth prop (syringe case, syringe barrel, catheter cap, needle cap (cats))
  - Tongue retractor
  - Stay sutures
  - Other devices

- Human help
  - Dedicated RVT on anesthesia
  - Surgical assistant/surgical technician
  - A human dedicated to helping with surgery will improve efficiency, ease, quality and safety

### Discharge Phase

- Written instructions from templates
- Video instructions

## OTHER BIG PICTURE POINTS

### Ergonomics

- Comfortable working height (neck/back/shoulder comfort)
- Seated position, Bum on seat, open hip and knee angle, feet on the floor
- Hand position for comfort, control and strength
- Body position relative to patient

### Hospital and Dental suite design for flow

- Location of procedures, location of radiographs
- Dental acrobatics versus dental comfort
- Enough space?

### Helpful (major) Equipment

- Lift table
- Appropriate saddle or other stool
- Loupes and light

4002

# WHO DO YOU WANT TO BE WHEN YOU GROW UP? - DEFINING THE DENTISTRY CULTURE IN YOUR PRACTICE

SMALL ANIMAL PROGRAM | DENTISTRY

 Jane Pegg, DVM, MS, DAVDC

Making a conscious decision about what type of dental care your hospital aims to provide is something that most practitioners haven't thought of. It can be a very valuable exercise to consciously choose the type of care you aim to provide based on the resources available and the client base in your practice.

Moving beyond providing simple adjectives such as good and bad, and thinking about spectrum of care, we can break out a number of different continua on which practitioners can score themselves, which can help define the dentistry culture they want to build in their hospital.

## ENJOYMENT LEVEL OF DENTISTRY

- Do you (and your team) like doing dentistry?
- Do you have fun when you're doing dental cases?
- Does it impede your sleep the night before, or give you sweaty palms when you see it on the calendar?

## DVM TRAINING AND SKILL LEVEL

- Do you understand the pathology of the major disease of the mouth in the dog and cat?
- Do you have a solid command of how to perform safe, effective extractions in your patients?

## STAFF QUANTITY AND TRAINING

- Do you have lots of help available on the average procedure day?
- Is it you and one other person doing cases?
- Can a patient go under anesthesia and get the basics (radiographs, nerve blocks, cleaning) performed without any major frustrations?

## SPACE OPPORTUNITIES AND LIMITATIONS

- Where in the hospital is your dental space?
  - In a dedicated space or a mixed treatment room?

## TIME OPPORTUNITIES AND LIMITATIONS

- Do you have dedicated dentistry days or are they mixed with other things?
- Do you have a fixed time constraint such as afternoon appointments starting at a particular time?

## EQUIPMENT QUANTITY AND CAPABILITIES

- What type of equipment is available in your practice to work with?
- Dental radiographs?

- Dental machine?
- Dental instruments?
  - How many packs?

## VOLUME OF CASES YOU'D LIKE TO SEE (IN AN IDEAL WORLD)

- Are you happy doing the occasional rare dental procedure?
- Would you like to do more than that?
- Doing more than 1-2 dental cases per day is very difficult to do unless they are routine cleanings on healthy pets receiving annual/frequent dental care
  - Heavily dependent on solid infrastructure and staffing to go beyond 1-2 cases

## EXTENT/DEPTH OF THE TYPE OF CARE YOU'D LIKE TO PROVIDE

- Are you open to long challenging extraction cases?
- What about difficult anesthesia on old pets with comorbid conditions?
- Would you prefer to refer those?
- Are more straightforward cases more appealing to you?

## COST THAT MOST CLIENTS WILL SAY 'YES' TO

- In your demographic area, you may have a good idea for how much clients are generally able to spend for a non-emergent procedure
- Severity and urgency sometimes elevate the available budget, but many dental procedures don't seem severe or urgent to clients
- Areas or hospitals with a client base of more modest means may need to develop different strategies and cultures than areas where clients find it easy to say yes to several thousand-dollar treatment plans.

When summing up all these variables including your enjoyment, abilities, staff, physical and temporal resources, depth of desired procedures, and typical client budgets, you can begin to develop a dentistry 'personality profile' for your practice.

If a gap exists between your reality and your aspirations (there usually is) you can identify opportunities for growth that will help move your practice towards the practice you want to be in the future.

References available from the author on request



4003

# LOOK SMARTER NOT HARDER – DIAGNOSTICS IN VETERINARY DENTISTRY

SMALL ANIMAL PROGRAM | DENTISTRY

 Jane Pegg, DVM, MS, DAVDC

When presented with a straightforward case, such as a fractured canine tooth requiring extraction in an otherwise healthy patient (and mouth), a good approach is often to perform a single stage anesthetized procedure with a combination of an anesthetized oral exam, dental radiographs and immediate surgery. Many cases however fall outside of these narrow parameters and better investigation may be needed to come up with an appropriate treatment plan.

In dentistry it's easy to fall into a trap of all or nothing thinking, where we either 'do a dental' or we do nothing at all. It can be hard to commit to 'doing a dental' for a patient when we don't know what we're getting ourselves into, especially if we have an all or nothing mindset around dentistry.

What if we thought about dental diagnostics as a tiered set of tools, similar to the way we do for other conditions? Typically we perform cardiac auscultation as a screening tool. If we detect a murmur, we don't jump from there to a thoracotomy and figure it out along the way. If we detect a heart murmur, then we can follow it up with thoracic radiographs and possibly an echocardiogram to further characterize the disease before making a diagnosis and treatment plan.

We can use dental diagnostics in the same way. An awake oral exam is a helpful screening tool, and very useful to rule in severe disease, major conditions and obvious pathology, but it is less useful as a firm rule out, or to characterize the full extent of disease present. A sedated oral exam allows better visualization of the oral structures including tongue, sublingual region, palate, pharynx and tonsils, and of course a more detailed look

at the teeth themselves. Sedation similar to sedation used for hand free radiograph techniques, is very effective for facilitating oral examination in many patients. Sedated oral exams should include photographic documentation of findings in the medical record.

An anesthetized exam with periodontal probing and charting is the most detailed oral evaluation. It is often used to determine a final treatment plan before proceeding to treatment, but doesn't have to be combined with same day treatment in every instance.

Dental radiographs are a necessary part of a thorough oral evaluation in an anesthetized patient, and they allow visualisation of a wide variety of pathology that can't be detected on surface examination alone.

Computed tomography (CT) and Cone Beam CT (CBCT) are emerging as an additional aid for detection of pathology and are more sensitive for a wide variety of pathological entities but can't replace a traditional full mouth series of radiographs. Additionally, pre- and post-treatment radiographs still need to be taken following every extraction.

Biopsy is an additional tool to further characterize a wide variety of pathological entities that can affect the structures of the mouth.

By understanding what can be gained from each diagnostic modality available, practitioners can select from an armamentarium of tools to better characterize what disease processes they are encountering before committing to a particular treatment plan.

References available from the author on request

4004

# LITTLE TEETH, BIG PROBLEMS – PEDIATRIC DENTISTRY & DENTAL ISSUES IN THE FIRST YEAR OF LIFE

SMALL ANIMAL PROGRAM | DENTISTRY

 Jane Pegg, DVM, MS, DAVDC

Many dental issues can present in the first year of life in the dog and cat. Knowing what to look for allows you to give your clients the best advice on treatment and prevention of major dental issues that can be identified early.

## FRACTURED DECIDUOUS TOOTH

- Consequences: damage to permanent tooth, possible facial cellulitis
- Treatment: dental extraction

## NORMAL OCCLUSION VERSUS MALOCCLUSION

- Class 0 (neutroclusion)
- Class I – Single tooth malocclusion with normal maxillomandibular relationship
- Class II – Abnormal maxillomandibular relationship with either maxilla relatively too long, or mandible relatively too short (greyhounds, collies, dachshunds, doodles)
- Class III – Abnormal maxillomandibular relationship with either maxilla relatively too short, or mandible relatively too long (Boxers, bulldogs, shih-tsus)
- Class IV – Abnormal side to side relationship where the left/right symmetry of either the maxilla or mandible is abnormal
- Traumatic occlusion versus atraumatic occlusion

## DECIDUOUS TOOTH MALOCCLUSION

- Class I or Class II can require treatment.
- Mandibular canine tooth abnormally contacting the palate is an indication for treatment
- Consequences: Failure of the mandible to grow to genetic potential, pain
- Treatment: Extraction, adult occlusion can be normal or abnormal

## ADULT TOOTH MALOCCLUSION

- Similar to deciduous but with adult tooth
- Consequences: Oronasal fistula, failure of the mandible to grow to genetic potential, pain, non-vital maxillary teeth
- Treatment options: Extraction, crown reduction and vital pulp therapy, orthodontic movement

## UNERUPTED FIRST PREMOLARS (*and other teeth*)

- Most common in brachycephalics (boxers, frenchies)
- Tooth fails to erupt normally (full inventory of teeth should be present at 6 months of age in most pets)
- Consequences: Can form a dentigerous cyst
- Treatment: Surgical extraction (if superficial, relatively straightforward, if deeply embedded, can be more difficult)

## TEETH THAT FAIL TO SHED

- Persistent deciduous teeth – rule of 1 tooth per ‘position’
- Canines are common in small dogs (chihuahuas, poodles)
- Sometimes multiple canines, incisors, premolars result in ‘shark mouth’ where there appear to be multiple rows of teeth
- Consequences: predispose adult tooth to periodontal disease, can contribute to malocclusion (canines)
- Treatment: Deciduous teeth should be extracted except when no adult tooth develops in a particular position in the arcade, and the deciduous is in good health (common for maxillary second premolars).

## FELINE JUVENILE GINGIVITIS/PERIODONTITIS

- Poorly described disease syndrome – very little published
- Around time of eruption of adult teeth, cats develop profound gingivitis (not mucositis) and often proliferation of excess gingiva.
- Can also develop periodontal disease of affected teeth.
- Though inflammation is severe, this is not feline chronic gingivostomatitis.
  - No inflammation in the palatoglossal arch region
- Common in Maine Coon cats
- Consequences: Untreated teeth can develop progressive periodontitis necessitating many extractions at a young age
- Treatment: Professional dental cleaning +/- gingivectomy if proliferative gingiva, followed by at home oral care (very important). Some cats need a repeat cleaning and gingivectomy after 3-6 months.
- Prognosis: Most cats resolve completely with 1-2 treatments

## JUVENILE AGGRESSIVE PERIODONTITIS

- Some pets, particularly very small toy breed dogs (poodles, chihuahuas, yorkies) present with what appears to be a very aggressive form of periodontal disease at a very young age
- Pathology is similar to commonly encountered chronic periodontal disease, but happens rapidly to young patients
- Consequences: Severe destruction of alveolar bone, mobile teeth, multiple extractions usually needed
- Treatment: Dental cleaning and extraction, patients often wind up edentulous by 3-4 years of age

## MANDIBULAR CARNASSIAL MALFORMATION

- Malformation most commonly seen in the mandibular first molar
  - Historically term *dens invaginatus* was used, but that isn't an accurate term for the true pathology occurring (this condition is very different from *dens invaginatus* in humans)
- Crown is mildly abnormal, especially in the furcation, convergent roots, abnormally formed pulp chamber on rads
- Abnormal microscopic furcation structure with multiple micropores leading to the pulp chamber, become easily infected by bacteria and then become non-vital
- Consequences: Non-vital tooth with periapical disease, can present as a chronic draining tract on the ventral mandible, face or neck
- Treatment: Dental extraction

References available from the author on request

4005

# FOLLOWING A STEPWISE APPROACH TO DENTAL EXTRACTIONS

SMALL ANIMAL PROGRAM | DENTISTRY

 Jane Pegg, DVM, MS, DAVDC

- Like every procedure there can be a stepwise, systematic way to approach dental extractions
- Treating dental extractions like a familiar 'recipe' usually leads to the greatest success and ease, and the least stress
- Skipping steps should be avoided, once proficient, doing every step will on average speed up your procedure, not slow it down.

## STEP 1: SURGICAL PLAN

- Combine dental diagnostics to commit to a written plan, NOT a mental-only plan
- It should be posted within sight of the operator

## STEP 2: OPEN YOUR SURGERY SITE

- Use a scalpel to create initial flap incisions and releasing incisions
- Use a periosteal elevator to lift your soft tissue flap from the underlying bone

## STEP 3: ALVEOLAR BONE REMOVAL

- Use a carbide cutting bur in a high-speed handpiece to remove bone overlying tooth roots to mechanically break down attachment
- Meticulous, tidy bone removal gives the best visualization of the tooth to be extracted

## STEP 4: SECTION TEETH AS NECESSARY

- Multi-rooted teeth should be cut into single rooted segments.
- The maxillary fourth premolar (Carnassial) is a 3-rooted tooth, the canine is single rooted
- Anatomic landmarks for sectioning are well defined

## STEP 4: TROUGH MAKING TO OUTLINE TOOTH ROOTS

- A very small (size 1/2) round carbide cutting bur is used to outline the periodontal ligament space
- This gives a place for your luxator or elevator to fit snugly between the tooth and the bone

## STEP 5: LUXATE AND ELEVATE

- Luxators are used with slight pressure and the tip is moved around the tooth, between the tooth and the bone to cut the periodontal ligament fibres
- Elevators are used with a rotational force along their axis and the wings of the elevator are used to apply leverage to the tooth to tear the periodontal ligament fibres

## STEP 6: TOOTH ROOT REMOVAL

- Extraction forceps are used to grip the tooth root, and with rotation and avulsion force, deliver the root from its alveolus

## STEP 7: CEAN AND DEBRIDE

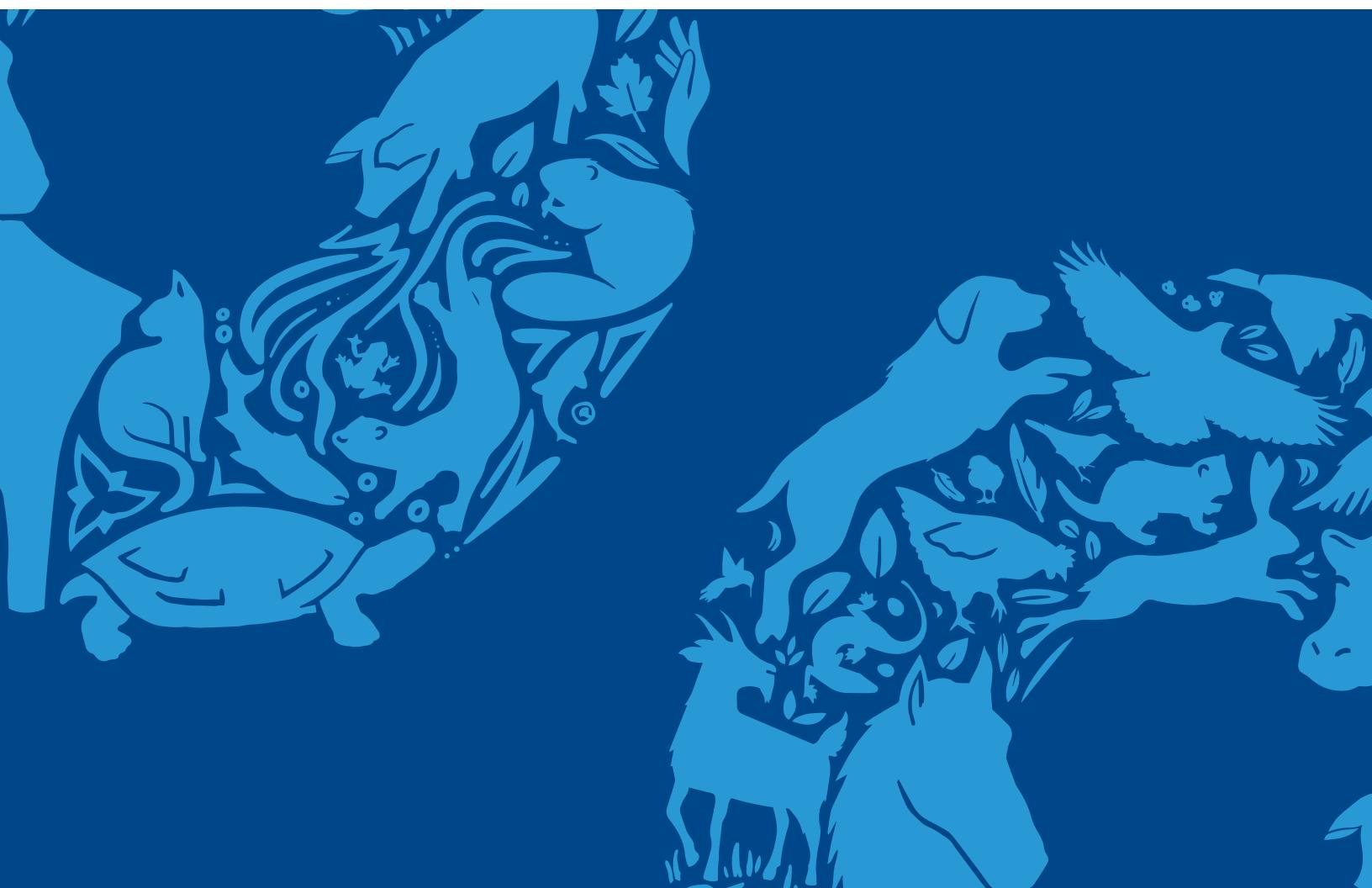
- A combination of curettage and a diamond bur is used to clean the alveolus
- A final flush with saline should be performed to clear any debris, blood clots, calculus etc.

By following this set of steps for every procedure and every extraction site within a procedure, practitioners can begin to systematize their thinking, which leads to greater efficiency and better surgical outcomes.

References available from the author on request

## STEP 8: CLOSURE

- Periosteal release is performed to ensure the flap will close without tension
- Final closure is performed using size 4-0 or 5-0 monofilament suture and any suture pattern (interrupted, continuous, cruciate etc.)



5001

# IT'S ALL ABOUT THE SAMPLE! CYTOLOGY AND BIOPSY IN VETERINARY DERMATOLOGY

SMALL ANIMAL PROGRAM | DERMATOLOGY

 Charlie Pye, DVM, DVSc, DACVD

Cutaneous cytology is a quick, inexpensive and non-invasive diagnostic test to determine whether patients have secondary infections. Knowing how to take samples, and being confident interpreting these samples, is critical to an animal's disease management and health. For these reasons, cytology should be part of the minimum database for any patient being seen for a "skin problem". Identification of a bacterial pyoderma or Malassezia dermatitis is important for treatment planning and resolution of clinical signs as well as prevention of antimicrobial resistance. There are many different ways to obtain accurate results from cytology and we will review these methods during this session. Biopsy is also an important diagnostic step in determining the cause of cutaneous disease. It requires careful site selection and biopsy technique. During this lecture we will review how to select biopsy sites, technique and some extra tips to make sure we get the best results from our skin biopsies.

## CYTOTOLOGY

Cutaneous cytology is extremely useful for identifying bacteria, yeast, inflammatory cells, neoplastic cells, and other abnormal cells such as acantholytic keratinocytes. It is also a quick, inexpensive, in-house test that can provide valuable information for treatment planning. There are many methods for performing cytology and different samples may be needed depending on the lesions you are sampling.

**Cotton swab:** If swabbing ears, you first take a cotton tipped applicator, insert into the ear canal until you reach the junction of the vertical and horizontal canal (there will be resistance here). Rotate the applicator a few times and

remove from the ear canal. It can be helpful to rub the other ear during sample collection. If sampling skin lesions, simply rub the cotton tipped applicator firmly onto the area of skin to be sampled. I think about it like a child colouring, keep swabbing until you get a colour change on the swab. Take the sample and then roll onto a slide. Multiple samples can be rolled onto the same slide with space between them. Don't forget to label which sample is which! Samples should then be heat fixed prior to staining. My preference is a lighter or barbecue lighter for heat fixing. A few seconds of heat fixing is more than enough until you see the ring of condensation reach the outer edge of the slide. If, after fixing, you have char on the bottom of the slide, this can be rubbed off but does indicate that the length of fixing time was too long or the flame was too close to the glass slide. If epidermal collarettes are present on the skin, the applicator can be rubbed under the scaly ring surrounding the collarette for the best sample. Cotton swabs are most useful for moist or exudative lesions and for ears.

**Tape prep:** Acetate tape preparations are most useful for dry lesions or if you are looking for Malassezia. The tape-strip preparation has been demonstrated to be more sensitive than other cytological methods in the detection of Malassezia organisms.<sup>1</sup> Clear tape should be used so you can visualize your sample. Take a small piece of tape, with one end designated as the finger-print end. Stick the tape onto the skin to be sampled and gently remove. Stick back down a few more times to get a good sample and then stain.

**Impression smear:** These types of samples are great for moist or exudative lesions. Take your slide and gently press onto the skin. You can also use the corner of the slide to

gently lift the corner of a crust and then do an impression smear right underneath the crust itself. Remember not to press too firmly onto the lesion or the slide can break in two. If a pustule is being sampled, the top of the pustule can be gently removed using a 25G sterile needle. An impression smear can be taken from the purulent exudate from the pustule.

Two newer cytological techniques appear to also be effective for the diagnosis of Malassezia and bacterial infections. Lo et al describe the toothpick technique for clawfolds.<sup>2</sup> This technique can be used for patients with paronychia where a toothpick is gently inserted into the claw fold, material is scraped from the claw, rolled onto the slide and stained accordingly. In the study they found this technique had a higher yield of Malassezia and cocci versus other techniques when samples were obtained from allergic and non-allergic dogs. A second study looked at a “slurry” technique where debris was collected from the skin, macerated in warm water, and then dried on the slide.<sup>3</sup> The slurry preparation did not differ significantly in detecting Malassezia but did appear to be more sensitive in detecting bacteria with a mean difference of 12.7 more bacteria per sample when compared with an impression smear.

When staining cytology samples, the most common stain utilized is Diff-Quik. If staining a non-acetate tape sample, dip the slide into the fixative 10-15 times and then the pink (eosinophilic) stain about 10-15 times. Then dip into the purple (basophilic) stain about 20-30 times. Yeast takes more time to “take up” the purple stain. Then rinse using tap water and a gentle stream. Rinse until the water runs clear and then dry the back using a paper towel or blot the whole slide dry using bibulous paper. There should not be any stain on the paper or towel. If there is, this indicates the slide was not rinsed thoroughly enough.

There are two different methods for staining tape. You can dip the tape into the pink and purple stain of the Diff-Quik and then rinse with tap water until the water runs clear (ie. excess stain has been removed). I like to stick one end of my tape onto a glass slide to hold the tape. After staining, put tape sticky side down onto the slide to blot dry. I prefer this method as the tape is clearer to read. Another way to stain tape is to take a drop of the purple stain and place this onto the slide. The tape can then be pressed onto the slide over the stain.

## VIEWING CYTOLOGY

Slides should first be scanned on low power (4X) for aggregates of inflammatory cells, nuclear streaming or acantholytic keratinocytes. Any of these identified cells are then examined on oil immersion. If there are no acantholytic or inflammatory cells noted, the objective can be switched to oil immersion and the slide scanned at this magnification. Multiple fields on the slide should be viewed.

## NORMAL FINDINGS ON CYTOLOGY

It is important to be familiar with what constitutes a “normal” finding on cytology, such as keratinocytes and melanin granules (which can often appear similar in shape and size to bacteria). Conchiformibius organisms can commonly be found on cutaneous cytology samples obtained from around the oral region or any location where a patient has been licking. This bacterial species has been isolated from the oral cavity of certain species.

## INTERPRETING CYTOLOGY

Classification of a semi-quantitative method for assessing cytology has shown good intra-observer and inter-observer reproducibility.<sup>4</sup> The Canadian Academy of Veterinary Dermatology has one of these types of scales available on their website. My personal preference is to grade cytology using an estimate of how many organisms are noted e.g. 0-2 yeast/OIF.

In my opinion, I do not have a rule as to “how many organisms are too many and require treatment”. I always pair my cytology findings with my patient’s clinical signs. However, documentation of an inflammatory reaction and intracellular bacteria provides confirmation of pyoderma.<sup>5</sup> Minimal data exists documenting normal yeast numbers on feline skin so the presence of any yeast on cutaneous cytology taken from a cat should be considered abnormal. When viewing otic cytology, rods are not commensals of the ear canal in dogs or cats, so the presence of any rods should be considered abnormal.

## SKIN BIOPSY AND HISTOPATHOLOGY

Histopathology can be helpful in diagnosing many immune mediated or neoplastic conditions. It can also provide extra information in cases of treatment failure or a patient’s

health status is declining. Below are times I reach for my biopsy punch.

1. If I am suspecting neoplasia
2. If ulcers, vesicles, or aggressive and serious disease are present
3. If a patient is not responding to therapy and I need more information
4. To confirm a disease process prior to starting therapy

Ideally biopsy samples should be taken sooner rather than later. However, if your patient has a disease with a waxing and waning progression and lesions come and go; it is best to wait until lesions are present prior to biopsy. If your patient presents with concurrent skin infection, this should be treated prior to biopsy to prevent misinterpretation of the histopathology results.<sup>6</sup>

If an individual is on glucocorticoid therapy and a biopsy is scheduled, the medication should be discontinued for 3 weeks prior to the procedure. If discontinuing glucocorticoids is not possible then the dose should be decreased, and biopsy samples taken after a few weeks on the decreased dosage.

When selecting where to biopsy on a patient with dermatologic disease, one should aim to select primary lesions such as pustules, papules, nodules etc. If an ulcer is present, care should be taken to avoid sampling the ulcer itself and instead biopsy the edge of the ulcer. If there are multiple lesions or a large region of dermatologic disease, then ideally 3-4 samples should be taken and submitted to maximize your yield.

When selecting the biopsy punch size, if the area to be biopsied is large (e.g. alopecia on the flank of a dog), then the largest biopsy punch should be used. With most dogs this could be a 6mm or 8mm punch. This provides more tissue for assessment by the histopathologist. Samples should be centered over abnormal tissue. If a specific lesion is to be sampled, then the punch should just fit over the lesion itself with minimal space around the edges. The junction of normal and abnormal skin does not need to be biopsied. Punches are cut in half in the direction of hair growth so if normal tissue is included it is possible for the diagnostic area to be in this region and a

diagnosis to not be obtained.<sup>10</sup> Once a lesion or region has been identified, the area does not need to be steriley prepared. A punch biopsy can be used by pressing firmly down onto the skin and rotating the punch. Rotation should occur in one direction only. Once all three layers of the skin have been incised by the biopsy punch, the punch can then be removed, and forceps and scissors can be used to collect the sample. The site should be closed with appropriate non absorbable suture. If, during the procedure, crust overlying the skin falls off the sample, this crust should also be placed into a formalin jar to be assessed. A note should be made on the submission that the crust is separate from the skin sample.

When submitting your samples, it is recommended to provide a brief summary of the history, photos if possible and your differential diagnosis list. All of this information helps histopathologists to correctly interpret their findings and provide the most information to allow a diagnosis to be made.

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5002

# WHEN THE IMMUNE SYSTEM DOESN'T PLAY NICE!

SMALL ANIMAL PROGRAM | DERMATOLOGY

 Charlie Pye, DVM, DVSc, DACVD

Immune mediated skin disease can be uncomfortable and painful for affected patients. Treatment revolves around the use of immunosuppressive medications with a plethora of potential side effects. Pemphigus foliaceus requires prompt diagnosis as well as chronic management. During this lecture we will discuss diagnosis as well as management options. We will also cover some important, but lesser known, immune mediated dermatologic diseases with some tips for diagnosis and treatment.

## PEMPHIGUS FOLIACEUS

Pemphigus foliaceus (PF), is the most common autoimmune dermatosis in dogs and cats.<sup>1,2</sup> Primary lesions of PF are large, fragile pustules which can easily rupture, so often individuals present with crusted lesions.<sup>1-4</sup> Implicated in the pathogenesis of PF are autoantibodies targeting antigens likely associated with desmosomes or other intercellular linker proteins. Impairment of these intercellular adhesions results in acantholysis (intercellular separation).<sup>4-8</sup> When acantholysis occurs in the upper epidermis, intracorneal and subcorneal pustules arise.<sup>6,8</sup> The major target antigen identified in dogs is desmocolin-1 but the target antigen in cats has yet to be identified.<sup>2,4,8</sup> While most cases of PF are considered idiopathic, potential triggers for PF have been identified including medications, vaccines and concurrent disease.<sup>1,2</sup>

## CLINICAL PRESENTATION

PF commonly affects middle-aged individuals.<sup>1,2,4</sup> In dogs, Akitas and Chows are predisposed breeds but there are no breed predispositions in cats.<sup>4,6</sup> PF does not exhibit a sex predisposition in either species.<sup>1,2,4,6</sup>

Lesions associated with PF include pustules, crusts, alopecia, erythema and erosions.<sup>1,2,4</sup> Pustules often span multiple hair follicles.<sup>9</sup> Dogs with PF commonly present with lesions involving the nasal planum, dorsal muzzle, periocular region, pinnae and paw pads.<sup>2,4</sup> In cats, the face, pinnae and nasal planum are often affected with the periareolar region and claw folds also exhibiting clinical signs.<sup>1,3</sup> Skin lesions are often bilaterally symmetrical.<sup>1,2,4</sup> Pruritus in cases of PF is variable.<sup>1-4</sup> Systemic signs such as fever, lethargy and anorexia may also be reported.<sup>1-4</sup> Systemic clinical signs appear to be more frequent in cats than in dogs, in which systemic signs most often occur in cases of severe and widespread disease.<sup>1</sup>

## DIAGNOSIS

Diagnosis of PF is based on history, clinical signs, exclusion of other differential diagnoses and microscopic evaluation of cytology and histopathology.<sup>1-4</sup> Cytology should be obtained from either an intact pustule or an impression smear from underneath a crust.<sup>4</sup> Cytology may show nondegenerate neutrophils, acantholytic cells or microorganisms if infection is present.<sup>1-4</sup> Histopathology will show pustules located in the superficial epidermis (intra or subcorneal). These pustules will be composed of neutrophils and acantholytic cells, which can present as individual cells or rafts of multiple cells, although eosinophils may also be noted. Dermal inflammation is predominantly neutrophilic but mast cells and plasma cells can be seen. The epidermis is hyperplastic with the stratum corneum exhibiting orthokeratotic hyperkeratosis.<sup>10</sup>

## TREATMENT

Prior to treatment, full bloodwork, including FeLV/FIV testing for cats, is warranted. Changes on bloodwork tend to be nonspecific with leukocytosis and neutrophilia the most common. Monocytosis, lymphopenia, eosinophilia, or anemia may also be present.<sup>7,9</sup>

With the disease having an immune mediated basis, owners should be informed that treatment will be lifelong and may have adverse side effects. Immunosuppression is the mainstay of treatment with glucocorticoid monotherapy being the most common initial therapy with complete remission achieved in most cases.<sup>1,2,4,7,11</sup> Prednisone or prednisolone are the most commonly used glucocorticoids. High-dose oral glucocorticoid pulse therapy can be considered, but the interval to disease remission and the cumulative dose of glucocorticoid does not vary greatly from standard treatment protocols.<sup>1</sup> Previously recommended doses for treatment were as high as 6.6 mg/kg per day in cats, but lower doses (2 mg/kg per day) are equally effective at inducing remission.<sup>1,9,11</sup> The most common adverse effects of glucocorticoids include polyuria, polydipsia, and polyphagia; the most serious adverse effects are diabetes mellitus, upper respiratory infections, urinary tract infections, and steroid hepatopathy.<sup>1,2,7</sup> Non-glucocorticoid options for treatment may need to be considered in animals not responding to glucocorticoids, in those with comorbidities that preclude the use of glucocorticoids, in those with severe adverse effects secondary to glucocorticoids, and in those in which glucocorticoids cannot be tapered to a dose considered safe for long-term use.<sup>7</sup>

Non-glucocorticoid treatments used as monotherapy or in combination with steroids in dogs include azathioprine, cyclosporine, mycophenolate, chlorambucil, tetracycline and niacinamide and gold salts.<sup>4,7,12</sup> Some of these therapies have also been used in cats; with the exception of azathioprine that should not be used in cats. Any therapies utilized should be tapered to the lowest effective dose once response is noted. Bloodwork every few weeks during initiation of therapy is important to look for side effects such as myelosuppression, liver or kidney enzyme elevations. Topical glucocorticoids can be used for localized disease.<sup>7</sup> Other treatments such as oclacitinib, topical tacrolimus and pentoxyfylline have been used in occasional cases with varying response; further studies are needed to recommend the use of these therapies.<sup>4,12,13</sup>

## PROGNOSIS

PF has a fair to good prognosis depending on the species.<sup>1,2,4</sup> In dogs with PF, remission often takes longer than cats and relapse can occur as part of the waxing and waning disease nature.<sup>1,2,9</sup> Client education regarding financial cost, time commitment, and emotional burden is important.<sup>9</sup> Euthanasia or death is often associated directly with PF or adverse effects of treatments.<sup>1,2,4</sup>

## SEBACEOUS ADENITIS

Sebaceous adenitis is an uncommon skin disease seen in the dog and rarely in cats, rabbits, horses and humans.<sup>4</sup> It is an inflammatory disease focusing on the sebaceous glands eventually leading to their destruction.<sup>4,10</sup> The exact pathogenesis of the destruction is unknown. Theories include a possible developmental and inherited defect, abnormalities in lipid storage and metabolism and a keratinization abnormality leading to obstruction of the sebaceous ducts.<sup>4,14,15</sup> It is also believed that glandular destruction is due to a cell-mediated immunologic reaction. Immunohistologic examination of samples from affected individuals show dendritic antigen presenting cells and T cells focused on the middle part of the follicle and extending into the sebaceous duct; suggesting an immune-mediated pathogenesis.<sup>14</sup>

One study showed that 43% dogs with sebaceous adenitis had a concurrent chronic disease such as hypothyroidism. In this study euthyroid sick syndrome is discussed and was not ruled out in every case.<sup>16</sup> However, these findings may indicate a link between different disease processes or underlying dysfunction of the immune system.

## CLINICAL PRESENTATION

Affected dogs are generally young to middle-aged with no sex predilection documented.<sup>4</sup> Breed predispositions are well known with Japanese akitas and standard poodles having an autosomal recessive mode of inheritance.<sup>15,17</sup> Other affected breeds reported include German shepherd dogs, samoyeds and vizslas. Other studies have also suggested that Havanese, Ihasa apsos, chow-chows and springer spaniels may be predisposed.<sup>16,18</sup>

Clinical signs include alopecia, hyperkeratosis and seborrhea with follicular casts as a distinctive feature of this disease.<sup>4</sup> Pruritus can be variable but is worsened

by secondary infection. Early lesions can include both scaling and erythema. Clinical signs differ slightly between individuals with long and short haircoats. Long coated breeds will present with darkening or lightening of coat colour and hair changing from curled or wavy to straight. Hair will then become dull and brittle and both scale and follicular casts will be noted. Alopecia will progress over time as the haircoat thins and then hairs are lost. Otitis externa can be seen. A secondary staphylococcal folliculitis is present in approximately 40% of cases.<sup>4,18</sup> Dogs with short hair coats will present with annular areas of scale and alopecia. These regions will coalesce to form larger regions of hair loss. The scales are often white and fine and do not adhere to the skin. These individuals may also present with nodular lesions and will more rarely have secondary pyoderma.<sup>4,14</sup> Lesions most often start on the head or cervical region along with the pinnae. Follicular casts are commonly seen along the lateral margins of the ear pinnae. Lesions will then spread to generalize over the dorsum and begin to involve the tail, trunk and legs.<sup>14</sup> Owners may complain of an "odour" which is due to the abnormal lipid layer and the presence of secondary infection. Certain breeds have been noted to exhibit more severe clinical signs. In one study, springer spaniels had more severe alopecia, pyoderma and seborrhea. In this study 57% of springer spaniels had otitis externa compared to 21% of the standard poodles and none of the akitas.<sup>16</sup>

## DIAGNOSIS

A diagnosis of sebaceous adenitis can be suspected based on history, signalment and physical examination.<sup>4,14</sup> To definitively diagnose sebaceous adenitis, skin biopsies should be performed. Histopathologic changes vary depending on the chronicity. In many cases a mild to moderate acanthosis along with moderate to severe hyperkeratosis with follicular plugging will be noted. Keratin will be found surrounding hair shafts as they emerge.<sup>10</sup> Inflammation is variable and, if present, it will be found surrounding a sebaceous gland or in the region of a previous gland. In early lesions there will be nodular granulomatous to pyogranulomatous reactions sometimes with sebocytes visible within the granulomas. The cellular infiltrate is comprised of histiocytes, neutrophils, lymphocytes and plasma cells. In chronic cases there will be complete destruction of the sebaceous glands with minimal inflammation.<sup>14</sup> In short-coated individuals nodular

inflammation centered on the isthmus (middle section) of hair follicles can be found.<sup>14</sup>

## TREATMENT

Treatment of sebaceous adenitis revolves around removing scale and follicular casts from the skin and coat, improving the quality of the coat and hair regrowth.<sup>4,14</sup> Owners should be counselled on the need for lifelong therapy. There is not one treatment effective in 100% of cases so multiple treatment options exist. Dogs can also be prone to recurrences even when receiving medication and studies have noted flares in clinical signs during treatment.<sup>18</sup> Any secondary infections with bacteria or *Malassezia* should be treated appropriately.

There are both topical and oral treatments available for. Topical treatments include shampoos, humectants and oil soaks. Shampoos containing both sulfur and salicylic acid can be used 2-3 times per week. A conditioner can be applied after bathing or a 50-75% dilution of propylene glycol as a spray or rinse can follow bathing. Baby oil soaks have historically been used to treat sebaceous adenitis. The oil or a 1:1 dilution with water is massaged into the coat and then left to sit for 1-6 hours. Bathing then follows to remove the excess oil. These soaks would be recommended every 7-30 days.<sup>4,14</sup> Anecdotal reports support the use of spot-ons or sprays and improvement in scaling and alopecia has been noted when using these therapies.

Oral treatments include omega fatty acids, systemic retinoids, cyclosporine, vitamin A and tetracycline and niacinamide. The disease is commonly unresponsive to glucocorticoids.<sup>4,14,19,20</sup>

When using both topical therapy and cyclosporine, there was evidence of a synergistic effect on scaling and alopecia and inflammation of the sebaceous glands is also reduced when a combination of these therapies is used for treatment.<sup>21</sup>

## PROGNOSIS

Sebaceous adenitis generally carries a good prognosis, however, in the study by Tevell *et al* in Sweden, 14 out of 44 dogs were euthanized because of clinical signs.<sup>16</sup> Owners should be made aware that this disease requires lifelong treatment and that flare ups can occur.

## SYMMETRIC LUPOIDONYCHODYSTROPHY (SLO)

Claw disease is an uncommon presenting complaint at veterinary hospitals. "Onychodystrophy" refers to abnormal claw formation, including changes to shape, texture, and growth. In SLO, changes to the claws are secondary to inflammation of the claw bed, leading to abnormal claw formation.<sup>10</sup> This poorly understood disease is likely a reaction pattern that has a multifactorial pathogenesis including immune-mediated and hereditary components.<sup>4,10,22</sup>

## CLINICAL PRESENTATION

Symmetric lupoid onychodystrophy most commonly affects dogs between the age of 2 and 6 yrs.<sup>4</sup> The disease mainly affects large-breed dogs such as the German shepherd dog, Gordon setter, rottweiler, among others.<sup>4,6</sup> Dogs present to the clinician for licking/chewing at their feet, lameness or loss of toenails. Physical exam can reveal sloughing of the claws, splitting of the claws, claw separation from the claw bed, and roughened texture of the claws. A single claw may be affected at first but the disease will progress to affect multiple nails. When claws regrow they may be misshapen, dry and brittle or grow at an abnormal angle. Dogs with SLO generally do not display signs of systemic disease. In one study, 17% of dogs with SLO were diagnosed with hypothyroidism; therefore signs of this disease may be noted in some individuals.<sup>23</sup>

## DIAGNOSIS

Cytology should always be obtained to check for secondary infection. Other differential diagnoses include trauma, parasites, leishmaniosis, and immune-mediated diseases.<sup>4,24</sup> However, most of these diseases would present with concurrent systemic signs.<sup>4</sup> Definitive diagnosis requires a biopsy of affected claws, involving amputation of the third phalanx (P3).<sup>4</sup> Amputation of an affected dew claw is always recommended, to avoid weight-bearing complications. An alternative approach for biopsy has been described to avoid amputation but frequently yields nondiagnostic results so is not commonly recommended.<sup>25</sup> Histopathologic findings include interface inflammation composed of lymphocytes and macrophages, with fewer neutrophils and plasma cells at the junction of the claw bed epithelium and the dermis, with or without a lichenoid band.<sup>10</sup> Many dermatologists

make a presumptive diagnosis of SLO based on clinical findings and history.

## TREATMENT

One of the most common treatment options for SLO is tetracycline (or doxycycline) and niacinamide. This combination has immunomodulatory properties, although the exact mechanism is poorly understood. In 1 study of 22 dogs treated with this combination there was an excellent response in 7 and a partial response in another 7 dogs.<sup>26</sup> Cyclosporine has also been utilized as a treatment for SLO.<sup>4,26,27</sup> Corticosteroids have been shown to be efficacious in treating this disease but due to their side effects it is recommended to reserve their use for refractory cases. Oral essential fatty acids, may be helpful as adjunct or maintenance therapies and have shown partial to complete response in studies when used as the sole agent or in combination with other therapies.<sup>4,27</sup>

## PROGNOSIS

Although response to treatment is usually slow (due to nail growth rates), prognosis is good, with most dogs achieving a good quality of life with treatment. Assessment of new nail growth should be done every 6-8 weeks. Upon examination the nail base should be strong with no inflammation present and no pain for the patient.

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5003

# IT'S ONLY SKIN DEEP! TOPICAL THERAPY IN DERMATOLOGY

SMALL ANIMAL PROGRAM | DERMATOLOGY

 Charlie Pye, DVM, DVSc, DACVD

Topical therapy is often underutilized in managing veterinary dermatology patients. However, topical therapy can be an essential part of a multimodal treatment plan, aiding in the repair of the skin barrier, which is so often disrupted in patients with skin disease. During this lecture we will review barrier dysfunction in allergic patients; some of the barriers to implementing topical therapy; the benefits of topical therapy for dermatology patients including infection management.

## BENEFITS OF TOPICAL THERAPY

Topical therapy in veterinary dermatology is often underutilized; most likely due to a perception that topical therapy may be more difficult for clients and concerns about client compliance. When I am approaching a patient with dermatologic disease and considering topical therapy, I have an open discussion with my clients about what they are able to do or what their pet will tolerate. If they can do topical therapy such as bathing, spot-ons etc, I commonly include this as part of my treatment regimen. Topical therapy has so many benefits that can be explained to the clients. Side effects are minimal, if present at all. In a patient with comorbidities, a sensitive gastrointestinal tract or an individual on other medications, topical therapy can provide the safest approach to treatment with the fewest adverse effects. A decreased microbial burden, mechanical removal of debris from the skin and decreased allergen penetration are just a few of the benefits topical therapy can provide.

## TREATMENT OF SECONDARY INFECTIONS

Patients with dermatologic disease are prone to secondary infections so cytology should always be performed

in any patient presenting with pruritic skin disease. Current consensus statements on treatment of bacterial pyoderma recommend topical therapy for any surface or superficial pyoderma, if the clients can be expected to be compliant.<sup>1</sup> For treatment of such infections, this author often recommends twice weekly bathing with a 3-4% chlorhexidine based shampoo combined with daily application of a 3-4% chlorhexidine based spray or mousse. A previous study has shown that a topical regimen of twice weekly bathing with 4% chlorhexidine shampoo and daily chlorhexidine spray was as effective as oral amoxicillin-clavulanate in treating pyoderma and pruritus in affected individuals. Both methicillin susceptible and methicillin resistant *Staphylococcus pseudintermedius* were documented to respond to topical therapy in this study.<sup>2</sup> *Malassezia* dermatitis can also be effectively treated topically, using agents with 3-4% chlorhexidine or topical antifungals such as miconazole.

If patients are intolerant to bathing; sprays, mousses and wipes can be used to topically treat any infections or microbial overgrowths. Some species or individuals will dislike application of spray because of the sensation, so mousses and wipes can be an excellent alternative. In one study by Gatellet *et al*, wipes/pads containing ophytrium and chlorhexidine were used once daily for 14 days to treat superficial pyoderma.<sup>3</sup> At the end of the study, mean microbial counts decreased and pruritus improved in every dog.

## WHAT IS SO DIFFERENT ABOUT ATOPIC SKIN?

Skin acts as a barrier between the body and the environment. Within the stratum corneum is the lipid matrix. This matrix acts to prevent excessive water

loss across the skin as well as to prevent percutaneous allergen exposure. Three lipid classes make up this matrix: cholesterol, free fatty acids and ceramides. Together they adopt a structure containing densely packed lipid layers called lipid lamellae. In atopic individuals we see many changes in the lipid composition of the skin (decreased ceramides), organization of these lipid lamellae and moisture retention. These changes facilitate allergen penetration through this abnormal barrier. Decreased moisture retention, decreased levels of ceramides as well as changes in the keratinocytes and immune reaction all contribute to disease progression. Topical agents can greatly help to improve this barrier function.

## BATHING FOR ATOPIC DERMATITIS

Current guidelines note that once weekly bathing with a mild shampoo and lukewarm water can benefit individuals with atopic dermatitis.<sup>4</sup> Bathing will mechanically remove allergens from the skin as well as reduce bacterial colonization. If allergens cannot enter the body no allergic reaction can occur! Veterinary shampoos should be recommended to clients as these are specifically formulated for animal skin. When bathing, clients should use lukewarm water as opposed to hot water to help keep the skin hydrated (I generally tell clients the best temperature is when they put their hand in and cannot appreciate a temperature difference).

In my opinion, shampoos that lather well and have a pleasant smell are best tolerated by clients. When discussing bathing, clients should also be told that a 10-15 minute contact time is recommended. Clients should be told to wet their pet down and then begin shampooing the most affected areas of the body. A timer can be taken into the bathroom so clients monitor this specific contact time. By starting with areas of the body most affected, these regions will always have the longest contact time. Time in the tub can be used for training, pets can be given treats if allowed – anything to keep them calm and comfortable during the bathing. During the warmer weather, clients can also do these baths outside and allow their dog to run around while the shampoo is “sitting” on their skin prior to rinsing with lukewarm/cool water. Pets should then be towel dried. Using a hair dryer will lead to moisture evaporation across the skin. Oatmeal based shampoos can be soothing for patients with skin disease. Alternatively using a product like Virbac’s Allergroom® with spherulite technology ensures extended release of active ingredients.

Glycotechnology within this shampoo also helps to reduce adhesion of micro-organisms to the stratum corneum. Ophytrium, an ingredient in Douxo products, can block inflammatory pathways, help maintain barrier function and reduces microorganism adherence and biofilm formation.<sup>5</sup>

Another tip I provide for clients is to wipe their pet down with a wet washcloth to remove allergens after being outside.

## TOPICAL GLUCOCORTICOIDS FOR ATOPIC DERMATITIS

Consensus guidelines also recommend the use of topical glucocorticoids or tacrolimus during atopic flares.<sup>4</sup> The efficacy of topical glucocorticoids for treatment of atopic dermatitis has been confirmed through studies and controlled trials. There is evidence that using a 0.0584% hydrocortisone aceponate spray (Cortavance®, Virbac) initially once or twice daily and then tapered, can aid in preventing flares of atopic dermatitis.<sup>6</sup> Hydrocortisone aceponate (HCA) is lipophilic and penetrates quickly to achieve a high level in the epidermis. It has low plasma bioavailability and a low rate of percutaneous absorption, meaning that systemic levels are low; a general concern when using corticosteroid medications. This product is labelled for 7 day use using 2 sprays per 10 x 10 cm area (to not more than one third of the body). HCA can also be used proactively. Studies show that when used daily to remission of clinical signs and then tapered to two consecutive days a week, the time to relapse of the atopic dermatitis was increased to 115 days (versus 33 days in placebo group). No adverse events were noted in this study.<sup>6</sup> As a dermatologist I also use this product off label for hot spots, acral lick granulomas, flare ups of pruritus and longer term sporadic use for milder cases of atopic dermatitis. Daily use long term should always be minimized to prevent any thinning of the skin. Although we often think of topical therapy as an “adjunct” to systemic therapy, using solely topical therapy is an excellent option for specific atopic individuals. One study showed that dogs receiving cyclosporine versus dogs receiving HCA, all achieved a greater than 50% reduction in their pruritus and clinical scores after 28 days. At 84 days, every other day or twice weekly therapy was achieved in 13/24 HCA and 12/21 cyclosporine treated dogs. There were no significant differences in scores for efficacy, tolerance or ease of administration.<sup>7</sup> Often topical application of glucocorticoid is best suited for focal lesions over short durations.

## BARRIER REPAIR FOR ATOPIC DERMATITIS

There are two available topical products that aid in restoring barrier function/preventing infection. Improvement in clinical signs of atopic dermatitis was documented when a spot-on formulation, containing polyunsaturated fatty acids and essential oils, was applied once weekly for 8 weeks. Significantly more improvement was noted in the treatment group in-regards-to clinical scores and pruritus.<sup>8</sup> Other studies noted that after 3 weeks of application of barrier repair products, there were increased ceramides in the skin, cholesterol was more evenly distributed between layers and glucosylceramides were barely detectable (these substances are detectable in large amounts in untreated atopic skin).<sup>9</sup> Another study looked specifically at the lipid lamellae of atopic individuals. In 3-day intervals, for 6 treatments, a spot-on treatment was applied to the thorax on non-lesional atopic skin (other side left untreated as a control). On the treated skin the lipid lamellae increased in size and filled up more of the intercellular space. This was compared to the untreated side, where intercellular spaces were almost empty and the lamellae were sparse.<sup>10</sup>

Epidermal barrier repair products should be considered for chronic allergic cases as they are unlikely to provide fast relief during acute flares. Studies suggest that improvements noted with these types of therapy may take longer to be achieved.

## MY APPROACH TO DEALING WITH ATOPIC DERMATITIS

Many patients I see have severe and chronic disease so often I am reaching for systemic therapies or allergy testing and allergy shots as the main portion of my treatment. With every allergic patient I continue to recommend at least weekly maintenance bathing.

Even well controlled patients can have acute flares, based on allergen levels within the environment. With acute flares I often recommend increasing or modifying the patient's bathing regimen. If focal areas of the body are affected, I also recommend topical HCA spray or topical steroid containing ointments. At the same time, I also review their treatment plan and adjust or add in other medications to prevent flares in the future. Any time an allergic patient becomes more pruritic, cytology must be performed to document presence of infection. Infections can be treated topically using medicated shampoo, sprays, mousses and wipes.

For chronic disease I also discuss with my client options including: dietary management of the allergies (diets for atopic individuals) as well as barrier repair to try and prevent future flare ups of disease.

## BIOFILMS

Microorganisms such as bacteria and yeast are known to create biofilms when they irreversibly attach to a surface. Studies show that high numbers of *Pseudomonas*, *Staphylococcus pseudintermedius* and *Malassezia* can form biofilms.<sup>11-13</sup> Mechanical disruption of the biofilm is one of the best ways to break down a biofilm and this can be achieved with bathing. Antimicrobial treatment can then be utilized to treat the infection.

For biofilms within the ear, mechanical disruption is hard due to anatomy. Previous studies document that minimum inhibitory concentrations (MIC's) for antimicrobials are higher for bacteria within a biofilm.<sup>11</sup> Certain compounds such as Triz-EDTA and Ophytrium are known to "break through" a biofilm to allow penetration of antimicrobials and therefore lowering the MIC required from such antimicrobials.<sup>14,15</sup>

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# PODODERMATITIS

SMALL ANIMAL PROGRAM | DERMATOLOGY

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Pododermatitis, also known as pedal folliculitis and furunculosis, is a multifactorial inflammatory disease process affecting the feet. More commonly pododermatitis is noted in canines as opposed to felines. It is not a diagnosis but more a clinical presentation indicative of many different underlying diseases. Clinical signs related to the paws may appear the same with any of the underlying conditions. Tissue affected may include interdigital webbing, nail folds, nails and footpads. Lesions can wax and wane and also spontaneously resolve in certain cases. Due to the complexity of the disease and the changing nature of lesions, pododermatitis can be frustrating to diagnose and treat. Many factors should be evaluated including age, breed, coat length, conformation, presence of other clinical signs and the number of paws affected. This information will help guide the diagnostic approach to the case as well as treatment plan.

## CLINICAL SIGNS

All four paws can be involved but the front are more often affected. This is thought to be due to weight bearing in canines.<sup>1</sup> In certain disease processes, only one foot may be affected. Patients may present with pruritus of the paws, erythema, edema, paronychia, alopecia, erosion, ulceration, draining tracts. Paw pad involvement may present as hyperkeratosis and erosion. Due to the magnitude of inflammation the feet often appear swollen. Dogs may exhibit pain through lameness or licking and chewing at their feet.

Lesions can be further exacerbated by licking, trauma to the feet from normal daily activity or trauma to the feet due to an orthopedic abnormality. Friction along the bottom of the paws combined with inflammation, can lead to plugged follicles and comedone formation. Follicles dilate and then rupture so hair and bacteria are released into the dermis. This incites a further inflammatory reaction and furunculosis can then develop.

## CONFORMATION

One report suggests the flatness of the feet and “scoop-shaped” interdigital web of breeds such as Pekingese and some terriers may predispose to folliculitis and pedal dermatitis.<sup>4</sup> Another study suggested that Labradors had wide-based paws with greater distance between pads, compared with other breeds, predisposing them to paw disease.<sup>5</sup> This suggests that weight bearing might be distributed more to haired interdigital skin leading to plantar paw trauma and irritation. I feel that dogs with short coats are overrepresented in cases of pedal furunculosis.

## DIFFERENTIAL DIAGNOSES

Below are the differential diagnoses to consider when examining a patient with pododermatitis (list may change depending on what parts of the paws are affected).

<b>Traumatic</b>	Sterile interdigital pyogranulomatous pododermatitis Foreign body
<b>Age related</b>	Osteoarthritis
<b>Genetic</b>	Lethal acrodermatitis of bull terriers Familial paw pad hyperkeratosis
<b>Nutritional</b>	Zinc-responsive dermatitis
<b>Metabolic</b>	Superficial necrolytic dermatitis
<b>Immune mediated</b>	Symmetric lupoid onychodystrophy Pemphigus foliaceus Systemic lupus erythematosus Vasculitis Adverse drug reaction Lymphocytic plasmacytic pododermatitis
<b>Neoplastic</b>	Nail bed tumour (melanoma, squamous cell carcinoma) Cutaneous T cell epitheliotropic lymphoma
<b>Endocrine</b>	Hypothyroidism Hyperadrenocorticism
<b>Allergic</b>	Atopic Dermatitis Cutaneous adverse food reaction Contact dermatitis Flea allergy dermatitis
<b>Infection</b>	Deep pyoderma and furunculosis (Nocardia, Actinomyces) Superficial bacterial pyoderma Superficial fungal infection (Malassezia, dermatophytosis) Deep fungal (blastomycosis, sporotrichosis, phaeohyphomycosis) Parasitic (demodicosis, hookworm dermatitis, Pelodera, ticks) Viral (distemper)



Amongst these differentials, allergic skin disease is one of the most common reasons for pododermatitis ranging from interdigital erythema to pedal furunculosis due to secondary infection. Both the underlying allergic skin disease and infection must be managed/treated to allow full resolution of the disease. Parasitic pododermatitis is also of concern. This presentation is perhaps less common with the use of isoxazolines for flea/tick prevention. In chronically inflamed lesions, skin biopsy may be required for diagnosis. Demodectic pododermatitis can present on the feet solely without any other lesions on the body.<sup>1</sup> Based on previous studies, demodicosis of the feet is one of the most commonly misdiagnosed skin diseases in dogs older than four years of age.<sup>5</sup>

## DIAGNOSTIC APPROACH

Pododermatitis can often be self-perpetuating, multi-factorial, and resistant to empirical therapy. Such factors can make this a frustrating condition to deal with. Lesions heal with scarring, which makes the paw more susceptible to future infections. As this condition has the potential to cause chronic changes, substantial effort should be made to diagnose the underlying primary disease or predisposing factor.<sup>5</sup> Patients should be treated aggressively early while pursuing diagnostics. A systematic approach towards ruling out differential diagnoses coupled with follow-up examinations and client education will often lead to a diagnosis being made.<sup>1</sup>

As with any dermatologic case, it is important to always collect a thorough history from your clients. In cases of pododermatitis, historical clues may allow identification of the underlying primary cause for inflammation.<sup>6</sup> If the patient concurrently has other systemic signs alongside the pododermatitis, this may provide evidence or a focus of diagnostic testing. Physical examination may identify any other problem areas on the body that, again, could help narrow the differential list. A minimum database should then be pursued including cytology and skin scrapings or hair plucks.<sup>7</sup> Cytology verifies the presence or absence of neutrophils, other inflammatory cells, and bacteria, yeast or fungal hyphae.<sup>6</sup> Skin scrapings and hair plucks are important tests used to diagnose parasitic pododermatitis.<sup>7</sup> If a bacterial infection is noted on cytology then a bacterial culture and susceptibility should be obtained and submitted to guide antimicrobial therapy. These cultures can be obtained by either swabbing areas with purulent

exudate or by obtaining aspirates from tissue. In certain circumstance a skin biopsy submitted for macerated tissue culture maybe required to diagnose a deeper infection. Secondary infections in cases of pododermatitis are often due to a deep pyoderma (if furunculosis is present). Systemic antimicrobial therapy is warranted in these cases. Empirical therapy should not be started without a culture due to increasing numbers of resistant bacterial species. If antimicrobial therapy is pursued, rechecks and repeat cytology every few weeks will allow the clinician to monitor response to therapy. Systemic antimicrobials may be required for an extended period (4-8 weeks). A fungal culture or skin biopsy may be performed next based on the differential list for each individual patient.

If one or two paws are affected in an older individual, radiographs of the paws and legs should be assessed to check for any boney changes, signs of osteoarthritis or osteomyelitis. Routine bloodwork as well as testing for endocrine disease via a thyroid profile may also be required in older patients with systemic signs of disease compatible with this differential.

In patients with persistent pododermatitis of one or multiple paws, histopathology may be required to demonstrate foreign bodies, deep bacterial infection, parasites, fungi, and neoplasia. Special stains are often used during histopathologic evaluation. In general, the histologic response shows perifolliculitis, folliculitis, or furunculosis; with nodular to diffuse pyogranulomatous inflammation being the most common.<sup>1,8</sup>

## TREATMENT

Treatment of the primary cause of inflammation should always be instituted as early as possible. If a foreign body is identified, then surgical exploration/removal is warranted. If osteoarthritis is observed, a treatment plan to decrease inflammation and decrease pain is important. Ectoparasiticid medications, thyroid medication, allergy work-up, digit amputation etc would also be potential treatment options based on diagnostic testing results.

If a patient is licking, chewing or biting at their feet and the clinician feels this is due to pruritus, then anti-pruritic/anti-inflammatory therapy should be instituted. I feel that oral glucocorticoids provide the quickest and fastest relief in cases of pododermatitis and I often use dexamethasone

at a dose of 0.05 mg/kg given every 24 hours and then tapered.

In the literature there are reports of surgical ablation with a carbon dioxide laser being used for the treatment of pedal furunculosis.<sup>1,9</sup> Success rate is 70%, dependent on the skill level of the user. Skin is left to heal by second intention and patients are treated for infection as well as given NSAIDs and frequent bandage changes.

To decrease swelling and clinical signs, treatment will be multi-modal. Any infections present should be treated both systemically and topically. Prolonged antibiotic treatment based on bacterial culture and susceptibility, is needed in cases of deep bacterial pododermatitis.<sup>1,2</sup> A dramatic improvement in the first 2 to 4 weeks may be noted but it is essential that antibiotic therapy not be discontinued too abruptly. For chronic or draining lesions, topical washes and foot soaks are also beneficial. Some sources recommend Epsom soak foot soaks, others antimicrobial shampoos and solutions. Fluorescent biomodulation or fluorescent light energy therapy (Phovia™, Vetoquinol) is a treatment option for pedal furunculosis as an adjunct to systemic antibiotics. Previous studies have shown that when this treatment is combined with systemic antibiotics, time to clinical resolution is accelerated.<sup>10</sup>

When large interdigital lesions are noted, the patient's activity may need to be restricted or they may need to wear protective booties when on rough surfaces. Ideally, they would be limited to smooth surface during treatment to prevent further inflammatory reactions of the skin.

Unfortunately, with any treatment, recurrence is common if the underlying cause of the pododermatitis is not addressed.

## CAT FEET

So as not to leave our feline friends out, pododermatitis, such as described in dogs is not seen in cats. Cats can, however develop secondary infections in the interdigital region or around the nail bases. If cats are noted to be licking or chewing at their feet then cytological samples should be obtained to diagnose any infection that needs to be addressed. Pemphigus foliaceus can also lead to a sterile paronychia in cats.

A disease of feline paw pads is plasma cell pododermatitis (pillow foot). Cats will present with swollen pads that can

then fissure and ulcerate and induce pain. In most cases, more than one footpad is affected. The metacarpal and metatarsal footpads are those primarily affected. Digital footpads may also be affected, but the lesions generally tend to be less severe. This disease is believed to have an immune mediated basis. Plasma cell pododermatitis is characterized by infiltration of plasma cells into the paw pad tissue. This infiltration causes the pads to swell. Some patients can present with seasonal relapses suggesting a seasonal allergy may be a trigger factor. Some authors also suggest an infectious etiology, specifically feline immunodeficiency virus (FIV). In fact, published case reports suggest FIV positivity rates of 44 to 63%.<sup>11,12</sup> Diagnosis is via histopathology as well as FIV/FelV testing. Cats with this disease generally respond well to steroids, cyclosporine or doxycycline.<sup>11</sup>

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# DERMATOLOGY COMMUNICATION TIPS & TRICKS

SMALL ANIMAL PROGRAM | DERMATOLOGY

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Pets with dermatologic problems provide a substantial caseload in primary care veterinary practice. It can be difficult to identify the underlying cause of a pet's dermatologic condition, so it can be challenging to efficiently treat these patients. These cases can be further complicated by clients' financial, emotional, physical, or time constraints. If clients do not understand why their pet is being treated and what they are being treated for, this serves to decrease compliance. The chronicity as well as the waxing and waning nature of some dermatologic diseases can lead to increased frustration in your clients. Communication in veterinary dermatology is key to prevent this and set realistic expectations. During this lecture we will discuss some tips for effective communication with clients about their pet's disease, treatment and outcomes.

## COMMUNICATION AND FRUSTRATION

With most dermatologic diseases, our aim is management of the disease as opposed to cure. Setting up these realistic expectations for clients helps decrease frustration. We all appreciate how frustrating skin disease can be. With allergic skin disease and immune mediated disease there can be many ups and downs. Often our treatment plans are multi-modal involving many different treatments. This can impact our clients relationship with their pet. A study by Spitnagel *et al*, showed that when owners of dogs with skin conditions took their pet to a dermatologist, their caregiver burden was already higher than that of owners of healthy dogs.<sup>1</sup> The frustration your clients are exhibiting should be acknowledged and we should always try to be empathetic with our clients. If clients feel heard and understood they are more likely to listen and follow through with our treatment plans. I always make sure to ask my clients if they have

questions about their pet's skin disease so that these clear lines of communication set us up for success. The same study, also highlighted that when a pet's skin disease was well managed, the caregiver burden was the same as that for owners of healthy dogs.<sup>1</sup> To help mitigate frustration, we can plant the seed early for patients that present with signs of allergic skin disease. Clients can be educated that we can make their pet more comfortable but that it may take time for us to "get to the bottom of" why their pet is itchy and that it may not be "an easy fix". Being up front with clients sooner rather than later helps decrease frustration.

When new treatments are started, it is ideal to provide a timeline for when you expect to see response to treatment. If these timelines are not outlined, this can lead to frustration when a treatment doesn't work straight away.

As dermatology diseases are chronic in nature, follow up is of upmost importance. Call backs or emails every 2-4 weeks during initiation of therapy is ideal to keep track of a patient's progression. I often send home calendars for them to fill in. The calendars have space for to input medications and how itchy the pet is that day. They can then send me these calendars for assessment and I can look for trends in pruritus. An example of a calendar can be found on the Canadian Academy of Veterinary Dermatology website ([www.cavd.ca](http://www.cavd.ca)). Along with the calendar I send them the dog and cat itch scale (also available through the CAVD website). This allows for more objective evaluation of their pet's pruritus.

## EDUCATE YOUR CLIENTS

Rechecks are so important with dermatology cases but clients sometimes will not come back for rechecks as

their pet is doing well. With all my clients I take a minute to explain to them why the recheck is important. "If we don't repeat cytology to verify the infection has resolved then if Fluffy develops another infection in 2 months, we won't know whether it is new or still the same one". Repeating cytology is so important as a first step to making our patients comfortable as well as knowing whether our therapy for the underlying disease process is effective.

I make a point of always being honest with my clients in terms of success, time to resolution and diagnosis. Clients should be aware of the fact that for most dermatologic cases, we are not going to see improvement overnight and it could be a few months before we know exactly what is going on with their pet. Again, setting those realistic expectations up front sets us up for success and decreases frustration. Words matter in this context. Telling a client that their pet will need long-term treatment is not the same as life-long treatment. Some clients perceive long term as being a year. For allergy cases, if treatment is stopped, clinical signs generally recur.

If there are two treatment options I review both with pros and cons and possible adverse effects and let the client decide which will work best for them and their family. In return I ask clients to be honest with me. Before prescribing a shampoo I ask "so how is Fluffy when you give her a bath?". If I get a laugh and eye roll, then I tell the client we need to change our plan and maybe consider a different topical for their pet. I also ask them how Fluffy does with medications by mouth or injections. Depending on their answer this may lead me to recommend a different approach. The great thing with dermatologic diseases like atopic dermatitis....we have so many treatment tools that we can tailor our plans to each individual.

It is also important to let clients know that, with allergic skin disease there can always be flares of pruritus depending on allergen levels within the environment. So although our treatment goals are to keep their pet comfortable, other goals include minimizing flares and preventing them from being as severe as they were previously. Our aim is not to eliminate flares altogether as we cannot control mother nature! Along the same line, in a pruritic patient our aim is not to eliminate pruritus. If we do this then we are overmedicating the individual. All animals and humans scratch. We want our clients to understand that, ideally, we want to be proactive to prevent flares as opposed to

reacting when a flare happens. I equate it to a forest fire. It is far simpler to use a fire extinguisher to put out a small fire than to call in help from across the country when a full forest is on fire!

During a dermatology appointment there is a lot of information provided to clients. Clients can become easily overwhelmed. Sometimes it is beneficial to take a break and schedule another appointment to discuss the rest of the case. For example, during the first appointment you talk about resolving infection and keeping the pet comfortable and itch free. Maybe at this appointment the client goes home with oclacitinib and topical therapy for a pyoderma. You see them back in 2-4 weeks to a) check the patient's progress and b) Discuss how to manage the disease longer term including how to truly diagnose allergies in a pet via restricted food trial and then treatment for atopic dermatitis. Repetition is key! Even in my practice I have clients come back for a second or third visit and when I discuss allergies with them, they react as if I am providing brand new information (when in fact we have discussed allergies at every visit). Sending home written instructions is always beneficial for clients. This takes time to do for every appointment so having instructions written up ahead of time for common scenarios such as otitis, food trials, allergy management etc that can then be printed or emailed to the client is very valuable. Utilizing sources already available to provide educational content for clients prevents misinformation from unreliable sources. Sources such as the CAVD, websites by industry partners or scientific websites can be written down and sent to clients for further reading. The Canadian Academy of Veterinary Dermatology has many handouts available to members that can be printed and given to clients to read at home.

Wording of prescriptions is also an area where clients can become confused easily (especially with the number of medications they might be leaving with). I advise my students to say "by mouth" instead of "oral" and to say "once every 24 hours" instead of "daily". I also tell my students to let clients know that once a day medications should be given at the same time every day, not 6am one day and noon the next.

When clients return for rechecks with their pets and there has been an improvement, make sure to acknowledge the work they have been doing at home and praise them for this. Anyone who has lived with an itchy pet knows it

is challenging and having to do multiple treatments takes time and effort and therefore clients should be rewarded for this. Over the years I have found that clients of pets with skin disease can become discouraged so being their cheerleader can go a long way in keeping them engaged and on board with diagnostics and treatment. It is also important to be empathetic with clients. We often do not know how much their pet's illness is affecting them and their family.

## BE CLEAR AND LISTEN

Be clear about goals of treatment with your clients. First goal is to bring relief as soon as possible and clearing secondary infections. Make sure clients are aware of costs associated with diagnostics and treatment. I find clients often nervous to ask me for estimates. I assure them that knowing costs associated with treatment for their pet is an important part of our conversation. Altering strategies does not mean substandard care, it means we are being realistic in what we can achieve.

Truly listening to your clients and addressing their main concerns can be challenging with limited time in general practice but does serve to build valuable client relationships. Dysart *et al* studied solicitation of client concerns during an appointment. In this study they found that client responses to solicitation were interrupted 55% of the time by the attending veterinarian. When client concerns were not solicited at the beginning of an appointment, this increased the odds of a concern arising during the final minutes of the appointment which required extending the appointment, booking a second appointment or ignoring the concern, none of which are ideal.<sup>2</sup> Allowing a client to relay their major concerns and addressing these, often leads to clients being more open to other suggestions you may have. Listening to your clients may also provide extra information important when developing treatment plans. For example, Mrs Smith tells you her major concern today is Fluffy's feet. You ask her what is going on with Fluffy's feet and she tells you that Fluffy's feet are very itchy and that Fluffy gets very aggressive when she tries to touch her feet. This likely precludes the recommendation of topical therapy for Fluffy's feet.

## OFFERING REFERRAL

Veterinarians and students often ask me "When is the best time to refer?". Anytime you feel the pet would

benefit from referral is the best time to offer it! With a very dedicated client whose Frenchie you have seen twice for bad ear infections - you may offer referral at the second or third appointment. For a German shepherd who previously had two ear infections a summer that resolved with treatment but now has otitis that cannot be resolved and has been ongoing for 4 months - you might offer referral 2.5 years after the first episode of otitis. If referral is an option for your clients, I would recommend discussing with your clients sooner rather than later. The American College of Veterinary Dermatology performed a client-based survey of 288 clients who visited a board-certified dermatologist after seeing their family veterinarian. The survey found that clients reached a tipping point of frustration with their family veterinarian after their third visit or a total of \$925 spent on the problem. 73% had already reached this tipping point before seeing the dermatologist and, at that time, only 62% said they would return to their family veterinarian for more than basic care.<sup>3</sup> The same survey also found that clients would save approximately 25% if a referral was made before the tipping point. Discussing referral with clients can be challenging based on the client and their expectations as well as geographical location. The ACVD survey did, however, find that 58% of clients felt better about their primary veterinarian for recommending a board-certified dermatologist.<sup>3</sup>

Another retrospective study looking at 65 cases of chronic otitis externa compared outcomes of dogs treated by their family veterinarian before referral and after seeking collaboration with a board-certified veterinary dermatologist. Dogs with chronic otitis had better long-term outcomes when collaboration with a dermatologist was pursued within 6 months of treatment with dogs under dermatologist care having fewer otitis recurrences, longer median times to recurrence as well as increased improvement with proliferative changes in the ear.<sup>4</sup>

## INCREASING EFFICIENCY IN PRACTICE

In academia I have the luxury of spending two hours with each client and patient...and sometimes that isn't even enough to discuss everything about their pet's case. Having limited time in general practice can lead to frustration when dealing with dermatological cases. Dermatology cases come with a lot of "baggage" in the form of detailed histories, different treatments and changing clinical signs. Following are a few things to

consider to make your dermatology appointment more efficient and productive.

Technicians play an integral role in increasing efficiency during appointments. They can take the history from the client and relay to the veterinarian. Technicians are capable of reading cytology which allows veterinarians to use appointment time for education and treatment planning. When cytology has been read, technicians can update the veterinarian who can then make a plan for infection control. I also recommend having your front desk staff and client liaisons teaching and educating your clients.

Let's face it, doing a good job with dermatology takes time. So how can you give yourself more time in clinic. If you have the ability to do so, skin cases should be booked for at least 45-minute appointments as opposed to a standard 20 minutes. If you do not have the ability to provide these longer appointments, consider a drop-off appointment for diagnostics. During the actual appointment spend the time speaking with your client and educating them. Have them drop their pet off the next day to perform diagnostics. These diagnostics can be fit in during the day with the help of other members of your veterinary team. Clients can be informed that they will receive an email or phone call the following day with results. I do this with my clients for follow up cytology rechecks. These drop off appointments can also be considered for rechecks. I have them come in during the day, my technician or intern takes the samples and has a brief conversation with the client to verify how the pet is doing and what medications or therapies they are on. We then take a look at the samples whenever time permits and then email the client with the results and our recommendations. If a pet is not doing well these, cytology drop offs are not ideal, a full appointment would be warranted.

Dermatology questionnaires are available through various sources online or can be made simply within a word document. These questionnaires can be sent to clients prior to their appointment to be brought on the day of the appointment. Clients can also be asked to bring photos of food their pet is on or photos of medication bottles/topical bottles. Alternatively, clients could be asked to arrive early for their appointment and fill in the questionnaire in the waiting room.

## THE DREADED FOOD TRIAL CONVERSATION

One conversation that most veterinarians hate is the restricted food trial conversation. We anticipate clients will not want to do the trial or will object to not being able to give treats. Below are a few tips to make this conversation go more smoothly.

I always emphasize to clients that the food trial is not forever and we can have flexibility longer term. If diet does not control their pet's clinical signs, then we can go back to giving treats. If the diet does resolve their skin disease then a) This is wonderful as their pet won't need life-long medications and b) We can still add things back into the diet we just have to be methodical with these additions. There is a big difference in saying to a client "You can only feed this diet and nothing else" versus "You can only feed this diet and nothing else for 8 weeks".

I explain to clients that a restriction diet trial is basically a diagnostic test, same as when we would recommend bloodwork and urinalysis for kidney disease as a first step.

Pitfalls of diet trials include pets receiving food items not allowed during the trial. I try to discuss with my clients these possible pitfalls during the appointment.

## REFERENCES

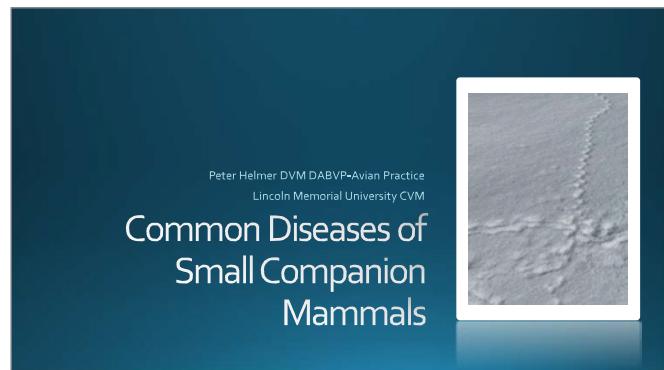
1. Spitznagel MB, Solc M, Chapman KR, et al. Caregiver burden in the veterinary dermatology client: comparison to healthy controls and relationship to quality of life. *Vet Dermatol.* 2019 Feb;30(1):3-e2
2. Dysart LM, Coe JB, Adams CL. Analysis of solicitation of client concerns in companion animal practice. *J Am Vet Med Assoc.* 2011 Jun 15;238(12):1609-15.
3. Hanna B. Earlier dermatology referral rewards primary care practice (part 1): easing client expenses and enhancing patient care. *DVM360* 2022. Available at: <https://www.dvm360.com/view/earlier-dermatology-referral-rewards-primary-care-practice-part-1-easing-client-expenses-and-enhancing-patient-care>. Accessed May 24, 2023.
4. Logas D, Maxwell EA. Collaborative care improves treatment outcomes for dogs with chronic otitis externa: A collaborative care coalition study. *J Am Anim Hosp Assoc.* 2021;57(5):212-216.

6001

# COMMON DISEASES OF SMALL COMPANION MAMMALS

SMALL ANIMAL PROGRAM | EXOTICS

Peter Helmer, DVM, DABVP-Avian Practice

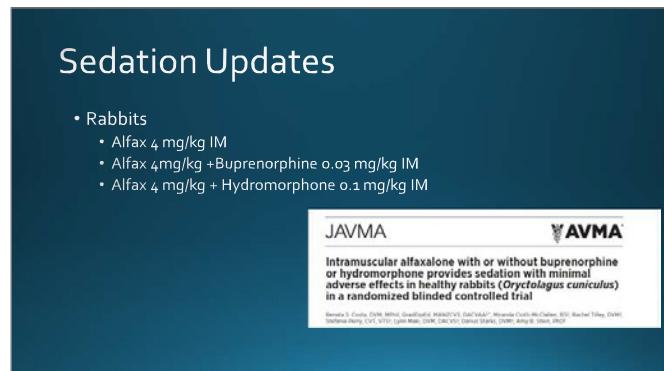


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## Outline

- Sedation updates
- RHDV
- Skinny guinea pigs
- Exophthalmia in rabbits
- Hedgehog facial masses and analgesia
- "Hematuria"
- ECUN
- Pot-pourri of small mammal stuff

2



3

## Sedation Updates

- Ferrets
  - Alfaxalone at 10-12.5 mg/kg SQ



4



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## RHDV

- Rabbit Hemorrhagic Disease Virus
- Calicivirus that causes hepatic failure and resulting coagulopathy
- High morbidity and mortality
- Shed in bodily fluids and viable at room temp for months on fomites



6





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## RHDV vaccination

- 2 European vaccines
  - Eravac and Filavac
  - No longer allowing importation
- US vaccine
  - Medgene
    - Highly effective
    - 2 doses SQ 21 days apart with annual booster
    - Subunit (no viral shedding)



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## The shrinking guinea pig

### The shrinking guinea pig

- Not this kind of skinny pig....
- Middle-aged guinea pigs that lose weight



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- Not this kind of skinny pig....
- Middle-aged guinea pigs that lose weight

- DDx
  - Dental/middle ear disease
  - Cardiac/thoracic disease
  - Renal/urinary disease
  - Endocrine disease



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## Thoracic disease

### Otitis media-Guinea pig

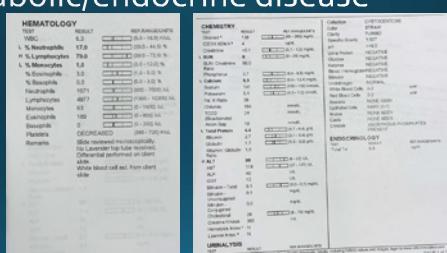


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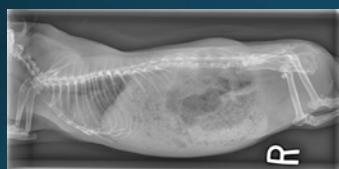
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## Metabolic/endocrine disease



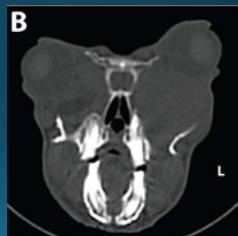
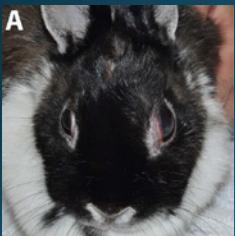
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## Bladder stones



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## Rabbits with unilateral exophthalmia



Journal of the American Veterinary Medical Association 261, 10; 10.2460/javma.23.05.0226

15

## Rabbits with unilateral exophthalmia

- Long-term systemic antibiotics and surgical treatment can be an effective treatment option for retrobulbar abscesses in rabbits (*Oryctolagus cuniculus*): 21 cases (2011–2022) | Levy and C Mans. <https://doi.org/10.2460/javma.23.05.0226>; Volume 261: Issue 10
- 15/21 associated with odontogenic infection
- Strep and *Fusobacterium* most common isolates
- Rx and antibiotics 9/21 cases
  - Extraction or flush/pack
  - Resolution for >6mth in 7/9
- Euthanasia 8/21 cases
- Antibiotics alone 4/21 cases
  - ¾ resolved

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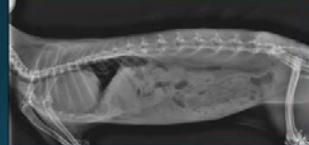
## Rabbits with bilateral exophthalmia

- Middle aged to older rabbits with exophthalmia +/- dyspnea



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## Anything wrong?



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## Thymoma treatment

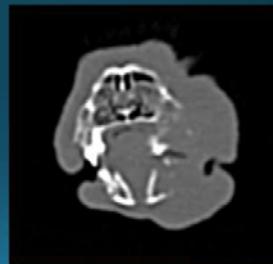
- Surgery
- Radiation
- Medical
  - Prednisolone
    - 1 mg/kg q24h

• Outcomes and survival times of client-owned rabbits diagnosed with thymoma and treated with either prednisolone or radiotherapy, or left untreated | Exotic Pet Med. July 2023;38(3):35-43. Alyssa Palmer, Chih Chun Wu, Yasutugu Miwa, Michelle Turek, Kurt K Sladky 2

• Retrospective study examined the clinical outcomes and survival times associated with two non-surgical treatment options, intensity-modulated radiotherapy (n = 10) and prednisolone therapy alone (n = 18). A cohort of rabbits that received no treatment (n = 10) was also evaluated and used for comparison. This study found prednisolone alone may significantly extend survival time and improve quality of life in rabbits with thymoma and provides owners with an additional low-cost, effective option for treating this neoplasm with minimal side effects shown in the population.

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## Hedgehog facial swellings



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## Hedgehog analgesia

- Buprenorphine
  - 0.03 mg/kg SQ (into the mantle) q36h or 0.05 mg/kg SQ q48h
- Methadone
  - 0.5 mg/kg SQ-no effect
  - 1.0-1.5 mg/kg SQ: <2hrs
- Hydromorphone
  - 0.15 mg/kg SQ q4hrs
  - 0.3 mg/kg SQ q 6h
- Both M and H caused transient sedation, nausea, vocalization, ataxia



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## "Hematuria"

- Guinea pigs/hhogs/rabbits
- Cystitis relatively uncommon
- Rabbits
  - Porphyrinuria
- Hhogs and GP
  - Bladder stones/sludge
  - Uterine disease



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## ECUN

- Microsporidian parasite closely related to fungus
- Transmission: Vertical or horizontal in rabbits
  - Vertical in first trimester and localize in lens (resulting in cataracts/glaucoma)
  - Horizontal: following ingestion spores replicate in intestinal epithelium then systemic spread including CNS
- Clinical signs:
  - CNS (vestibular, seizures), renal (azotemia), ocular (uveitis, cataracts, glaucoma)
- Diagnosis:
  - Histo, Ab titres only indicate exposure
  - "Clinical signs are not necessarily correlated with a high antibody titer, and only a negative result can rule out encephalitozoanosis"
- Tx
  - FBZ 20 mg/kg PO q24h x 30 d
  - Guarded prognosis

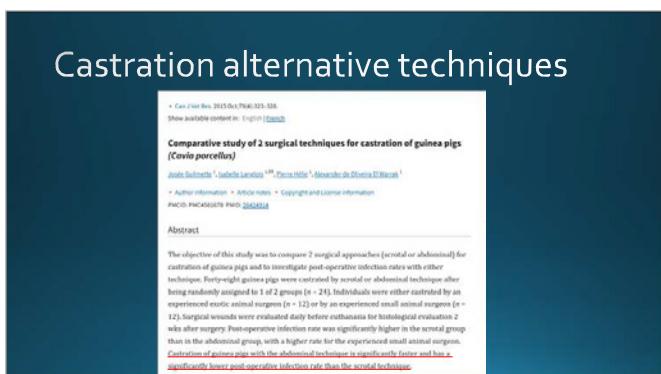
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## Routine surgical considerations

- Open inguinal rings
- Dirty incisions post-op



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## OVH

- Very dorsally adherent ovaries
- Pesky caecum in the way



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## Ovariectomy



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## Common Diseases by species

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### Rats

- Harderian secretions
- Respiratory disease
- Mammary neoplasia
- Pododermatitis



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### Rats

- Harderian secretions
- **Respiratory disease**
  - Combination of
    - Virus
    - Mycoplasma
    - COPD
- Mammary neoplasia
- Pododermatitis



<http://www.ratfarmclub.org/species.html>

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### Rats

- Harderian secretions
- Respiratory disease
- Mammary neoplasia
- Pododermatitis



35

### Rats

- Harderian secretions
- **Respiratory disease**
  - Combination of
    - Virus
    - Mycoplasma
    - COPD
- Mammary neoplasia
- Pododermatitis



<http://www.ratfarmclub.org/species.html>

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### Mice

- Mange
  - *Myobia musculi*
- Mammary neoplasia



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### Mice

- Mange
- Mammary neoplasia
  - Usually adenocarcinoma which is aggressive and metastatic



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## Hamsters

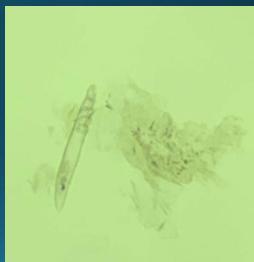
- Adrenocortical disease
  - Adrenal tumors secreting cortisol
- Mange
- Wet tail
- Proptosis
- Cheek pouch disease



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## Hamsters

- Adrenocortical disease
- Mange
  - *Demodex aurati*
  - Fluralaner safe and effective tx
- Wet tail
- Proptosis
- Cheek pouch disease



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## Hamsters

- Adrenocortical disease
- Mange
- "Wet tail"
  - Multifactorial
    - Parasitic
    - Stress
    - Dietary
    - *Lawsonia* (proliferative bowel disease)
- Proptosis
- Cheek pouch disease



Pathophysiology of the Exotic Companion Mammal Gastrointestinal System  
May 2014, Vol 12, No 5 • Journal of the American Animal Hospital Association  
DOI: 10.5326/JAAHA-05212  
Source: PubMed

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## Hamsters

- Adrenocortical disease
- Mange
- Wet tail
- Proptosis
- Cheek pouch disease



https://www.revistaclinicaaveterinaria.com.br/noticias/especialidades/oftalmologia/hamster-de-caixa

42

## Hamsters

- Adrenocortical disease
- Mange
- Wet tail
- Proptosis
- Cheek pouch disease
  - Prolapse
  - Infection
  - Neoplasia



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## Gerbils

- Squamous cell carcinoma of scent gland
- Tail slip

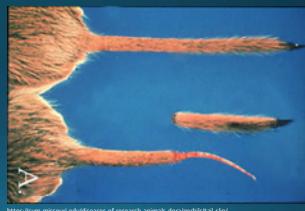


https://cm.missouri.edu/diseases-of-research-animals-dora/gerbils/squamous-cell-carcinoma

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## Gerbils

- Squamous cell carcinoma of scent gland
- Tail slip



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## Prairie dogs

- Dental disease and odontoma
- Obesity



https://iv.iarpumalk.org/content/jo165

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## Prairie dogs

- Dental disease and odontoma
- Obesity



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## Squirrels

- Dental disease
  - Extraction vs life-long periodic trimming



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## Guinea Pigs

- Scurvy
  - Hypovitaminosis C
- Pneumonia
- Dental disease
- Mites and lice
- Ovarian cysts
- Cystoliths
- Pododermatitis
- Dystocia



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## Guinea Pigs

- Scurvy
- Pneumonia
  - Bordetella sp.
  - Rabbits are carriers
- Dental disease
- Mites and lice
- Ovarian cysts
- Cystoliths
- Pododermatitis
- Dystocia



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## Guinea Pigs

- Scurvy
- Pneumonia
- Dental disease
  - Tongue entrapment
- Mites and lice
- Ovarian cysts
- Cystoliths
- Pododermatitis
- Dystocia



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## Guinea Pigs

- Scurvy
- Pneumonia
- Dental disease
- Mites and lice
  - *Trixacacis caviae*
  - *Giracola porcelli*
- Ovarian cysts
- Cystoliths
- Pododermatitis
- Dystocia



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## Guinea Pigs

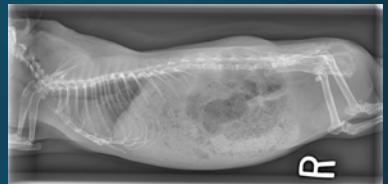
- Scurvy
- Pneumonia
- Dental disease
- Mites and lice
- Ovarian cysts
  - Symmetrical alopecia
  - AUS or even palpable
  - Drain vs remove
- Cystoliths
- Pododermatitis
- Dystocia



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## Guinea Pigs

- Scurvy
- Pneumonia
- Dental disease
- Mites and lice
- Ovarian cysts
- Cystoliths
  - Common cause of hematuria
  - Must remove surgically
  - Commonly recur
- Pododermatitis
- Dystocia



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## Guinea Pigs

- Scurvy
- Pneumonia
- Dental disease
- Mites and lice
- Ovarian cysts
- Cystoliths
- **Dystocia**
  - Must breed prior to 6 months
  - Large feti



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## Chinchillas

- Heat stress
- Ringworm
- Fur-ring
- Fur slip
- Dental disease
- Trauma/fractures
- Cardiac disease



<http://animals.sandiegozoo.org/animal/a/chinchilla>

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## Chinchillas

- Heat stress
- **Ringworm**
  - Usually Trichophyton
  - Does not fluoresce
  - DTM and PCR for dx
- Fur-ring
- Fur slip
- Dental disease
- Trauma/fractures
- Cardiac disease



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## Chinchillas

- Heat stress
- Ringworm
- **Fur-ring**
- Fur slip
- Dental disease
- Trauma/fractures
- Cardiac disease



58

## Chinchillas

- Heat stress
- Ringworm
- Fur-ring
- **Fur slip**
- Dental disease
- Trauma/fractures
- Cardiac disease



<https://www.foreverfeistychinchilla.org/picking-up-and-holding.html>

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## Chinchillas

- Heat stress
- Ringworm
- Fur-ring
- Fur slip
- **Dental disease**
- Trauma/fractures
- Cardiac disease



Sulk, Małgorzata & Scholcowska, Ewa & Seremka, Beata & Eyz Chmielewska, Halina & Ficzałk, B. (2007). Radiological evaluation of chinchilla mastication organs. Bulletin of the Veterinary Institute in Pulawy, 53, 225-234.

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## Degus

- Diabetes mellitus
- **Cataracts**
- Dental disease



[https://www.reddit.com/r/Degus/comments/1cjjphq/degu\\_cataracts/](https://www.reddit.com/r/Degus/comments/1cjjphq/degu_cataracts/)

61

## The end



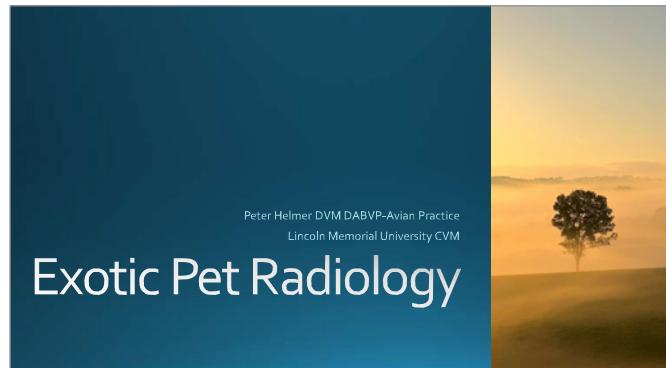
62

6002

# EXOTIC PET RADIOLOGY

SMALL ANIMAL PROGRAM | EXOTICS

Speaker: Peter Helmer, DVM, DABVP-Avian Practice



1

## Outline

- Sedation
- Skull imaging
- Respiratory tract imaging
- GI tract imaging
- Abdominal imaging
- Repro/urinary tract imaging

2



3

## Sedation

- Midazolam (0.5-1 mg/kg) + butorphanol (0.5-1 mg/kg) IV/IM/SQ
- Dexmedetomidine
- Alfaxalone
- Iso/Sevoflurane



4

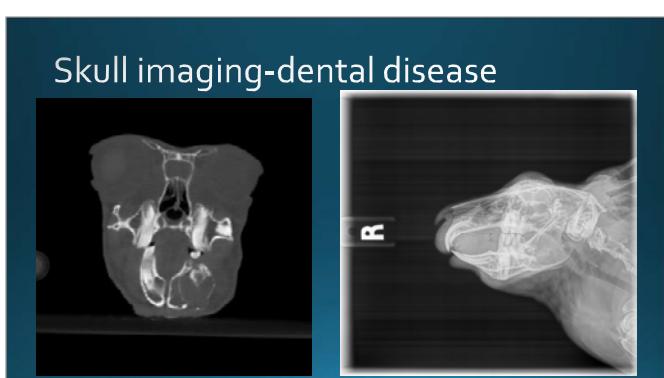
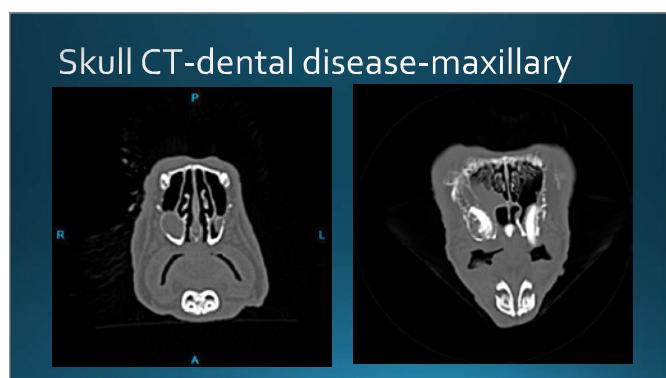


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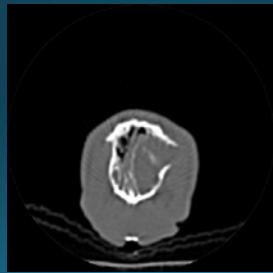
## Skull rads-otitis



6



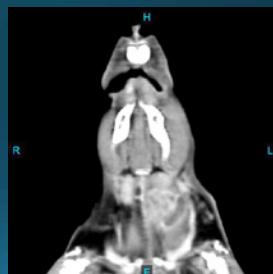
### Skull CT-Nasal disease GP



15

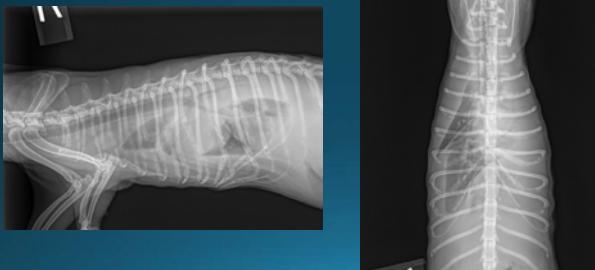
### Cervical mass imaging

- 10 year old squirrel with cervical mass



16

### Respiratory tract imaging: pleural effusion



17

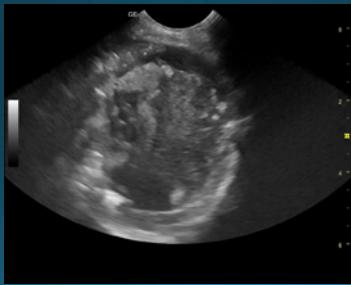
### Respiratory tract imaging

- 2 year old FS ferret
- Presented for evaluation of dyspnea and decreased appetite



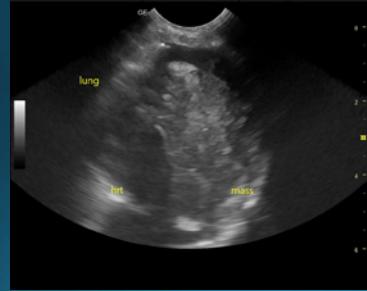
18

### Thoracic ultrasound



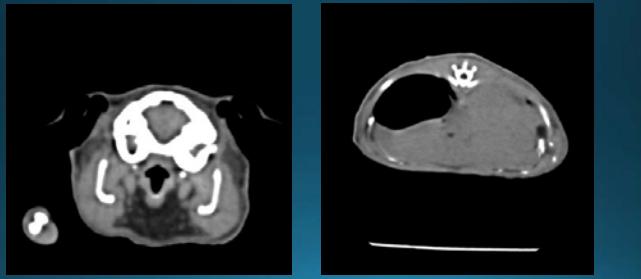
19

### Thoracic ultrasound



20

### Dyspneic GP



21

### Dyspneic rabbit



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## Dyspneic rabbit post-lasix



23

## GI tract imaging-Rabbits

- Rads are generally more useful than AUS for GI



24

## GI tract imaging

- Ferrets
  - Remember that foreign bodies are very common
  - Don't rule out based on lack of vomiting



25

## GI tract imaging-Ferret FB



26

## GI tract imaging-Ferret LN



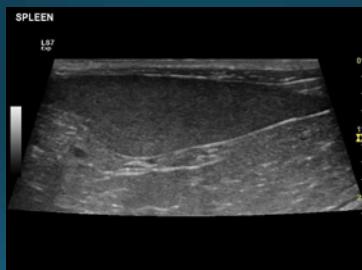
27

## Splenomegaly-ferrets



28

## Splenomegaly-ferrets



29

## Ferret adrenal disease

- Common symptoms are
  - Hair loss, particularly on tail
  - Vulvar/prostatic enlargement
  - Increased aggression
- Also reported in rabbits



30



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## Ferret normal adrenal glands

- Ultrasonography Of Adrenal Glands In Normal Ferrets
- Vet Radiol Ultrasound. 1996;37(6):445-448. 33 Refs

R O'Brien 1, J Paul-Murphy, R Dubielzig

The adrenal glands of 20 normal ferrets were imaged with ultrasound. Of the ferret glands, only 4 (three right and one left) could not be clearly identified. Mean (+/- standard deviation) dimensions of the right (7.6 +/- 1.8 mm length by 4.2 +/- 0.8 mm width) and left (7.0 +/- 1.1 mm length by 2.8 +/- 0.5 mm width) glands were similar. The glands had a hypoechogenic outer zone and hyperechogenic central region, were elongated and ovoid in shape and located medial and ventral to the level of the cranial pole of the ipsilateral kidney.

Approx 8mm x 3mm is normal



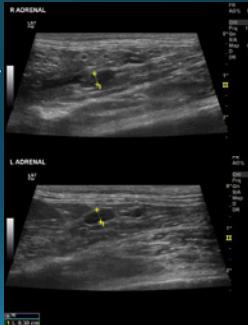
31

## Ferret adrenal

- Ultrasonographic visualization of the adrenal glands of healthy ferrets and ferrets with hyperadrenocorticism
- J Am Anim Hosp Assoc. 2007 Mar-Apr;43(2):78-84.

A Kuijten 1, N Schoemaker, Voorhout

A protocol was developed to compare the ultrasonographic characteristics of the adrenal glands of 21 healthy ferrets and 37 ferrets with hyperadrenocorticism. Based on the findings of this study, adrenal glands may be classified as abnormal when they have an irregular appearance, increased size of the cranial/caudal pole (thickness >3.9 mm), a heterogeneous structure, increased echogenicity, and/or signs of mineralization.



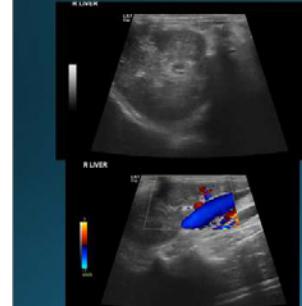
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## Rat adrenal



33

## Rabbit hepatic imaging



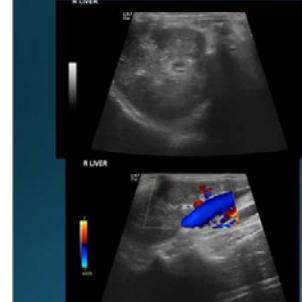
34

## Rat adrenal



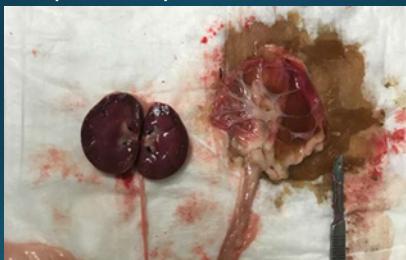
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## Rabbit hepatic imaging



36

## Rabbit hydronephrosis



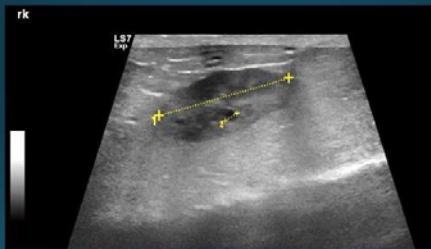
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## Urinary tract imaging-ferret kidney



38

## Urinary tract imaging-ferret kidney



39

## Urinary tract imaging

- Ferrets
  - Consider stones (usually cysteine) vs prostatitis vs other



40

## Urinary Tract Imaging-Rabbits

- Calciuria/sludge makes US imaging challenging



41

## Workup of hematuria

- Guinea pigs
  - Urinary:
    - Stones
    - Cystitis
  - Reproductive
    - Neoplasia
    - Metritis



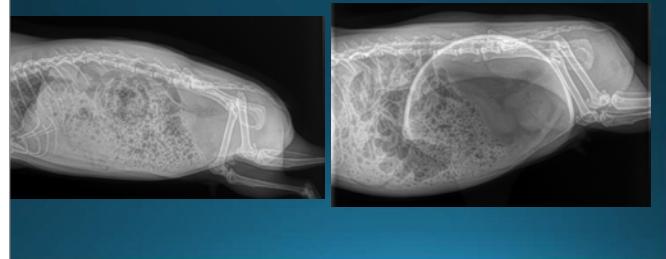
42

## Bladder stones



43

## Compression study



44

## Workup of hematuria-Guinea pig

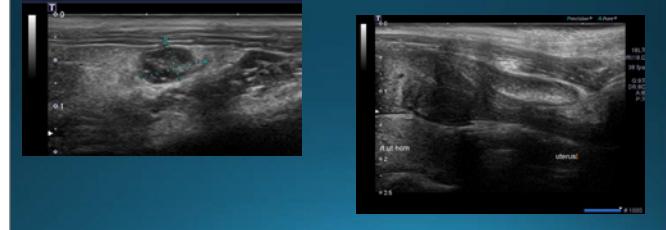
- Bladder/kidney



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## Workup of hematuria-Guinea pig

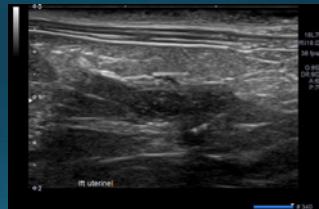
- Repro tract-ovary



46

## Workup of hematuria

- Rats
  - Pigmenturia
  - Urinary
    - Stones
    - Cystitis
  - Reproductive
    - Neoplasia
    - Metritis



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# THE INAPPETENT RABBIT

SMALL ANIMAL PROGRAM | EXOTICS

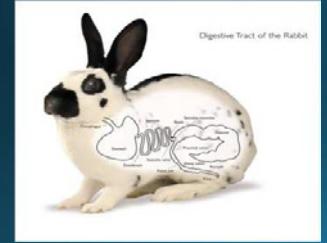
Speaker: Peter Helmer, DVM, DABVP-Avian Practice

Peter Helmer DVM DABVP (Avian Practice)  
Lincoln Memorial University

## The Inappetent Rabbit

1

### GI tracts with legs and ears



- Decreased appetite and fecal production is a sign of disease, not a disease in and of itself

2

### Diagnostic approach History

- Recurrent or first-time offender
- Duration of problem
- Diet
- Stressors



3

### Diagnostic approach Physical exam-TPR



- Rabbits with hypothermia at admission had a risk of death before or within 1 week after hospital discharge 3 times that of rabbits without hypothermia
- For each 1°C (1.8°F) decrease in admission rectal temperature, the odds of death were doubled
- 117 rabbits with RGIS
  - 15 died, 18 euth, 84 discharged
  - T<98.5°F 5x more likely to die or be euth

4

### Diagnostic approach Oral exam




5

### Diagnostic approach

- Physical exam
- Often normal
- Now what??



6



## Radiographs



7

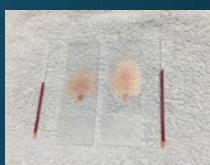
## Bloodwork



8

## Bloodwork

- Rabbit blood in general
  - PCV lower than dogs/cats with normal range 30-40%
  - Heterophils replace neutrophils
  - Hypercalcemia is common
  - Lactate is not particularly useful in rabbits



9

## So now what?

- Outpatient vs inpatient therapy
  - Inpatient allows monitoring of temperature, food intake, fecal production
  - Always a guarded prognosis

10

## Non-specific therapy

- Fluids
  - IV (inpatient) vs SQ (outpatient)
  - Maintenance is 100-120 mL/kg/day



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## Non-specific therapy

- Feeding
  - 30 mL/kg q6h



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## Non-specific therapy

- Analgesia
  - Buprenorphine
    - 0.03 mg/kg IM/IV q8-12h
    - No evidence of TM absorption, Poor bioavailability
    - Similar to no effect on GI motil
    - Consider as single injection for outpatient than oral meloxicam
    - High conc buprenorphine (Ex: Simbadoll) substantial variability in pharmacokinetics
  - Meloxicam
    - 0.5-1 mg/kg PO/SQ q24h
    - Caution with GI disease, hypovolemia, renal, hepatic dysfunction



13

## Non-specific therapy

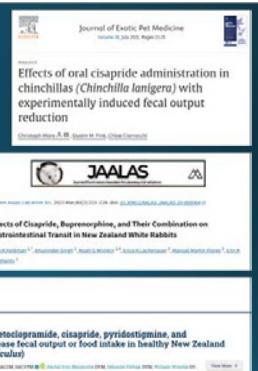
- Appetite stimulants
  - TD mirtaz (Mirataz) 0.5 mg/kg and 1.0 mg/kg q24h was more effective at increasing feed intake and fecal output
  - Capromorelin (Entyce, Elura) 4-8 mg/kg PO q12-24h also effective but less than mirtaz



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## Non-specific therapy

- Promotility drugs that don't help
  - Metoclopramide
    - No effect
  - Cisapride?
    - No effect on GI motility in chinchillas or rabbits
  - Simethicone?



15

## Rabbits-lidocaine CRI



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## Case 1

- T 98.1 F
- Firm cranial abdominal mass
- BW normal except for BG of 641 mg/dL



18

## Case 1

- Normal physical exam
- Normal bloodwork, normothermic
- Treatment plan?



18

## Hyperglycemia

- Clinical value of blood glucose measurement in pet rabbits. *Vet Rec. June 2012;270(6):67x. F M Harcourt-Brown.*
- Severe hyperglycemia (≥ 60 mg/dL) was associated with conditions with a poor prognosis. Rabbits with confirmed intestinal obstruction had a mean blood glucose of 445 mg/dL (n=38). This was significantly higher than the rabbits with confirmed gut stasis, which had a mean value of 153 mg/dL (n=51).



20

## Treatment

- Intense supportive care
- Fluid support/monitor BP if possible
- Thermal support if needed
- Oro-gastric tube for decompression?
- Surgery?



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## Case #3

- PE NSF
- Normothermic, non-painful abdomen
- Owner approves radiographs



22

### Case #3



23

### Case #3

- CBC

- WBC 9.16 k
- 52% heterophils
- 41% lymphs
- 7% monos

• PCV 22%

- Chem

- Gluc 236
- Creati.3 BUN 24
- ALT 607 ALP 61
- Ca 10.9

24

### Case #3



25

### Liver lobe torsion

- Liver lobe torsion in rabbits: 16 cases (2007 to 2012) J Exotic Pet Med. July 2014;23(3):258–265. Graham JE

- Exploratory laparotomy and liver lobectomy were performed in 9 rabbits, and all of them survived. Supportive care alone was provided for 7 rabbits, 3 of which survived



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6004

# ANALGESIA IN AVIAN EXOTIC ANIMAL PRACTICE

SMALL ANIMAL PROGRAM | EXOTICS

Peter Helmer, DVM, DABVP-Avian Practice



1

### Overview

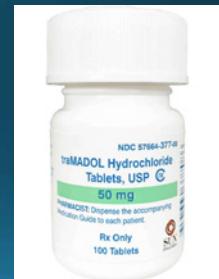
- Classes of drugs
- Analgesia in:
  - Ferrets
  - Small herbivores
  - Birds
  - Reptiles



2

### Synthetic opioids

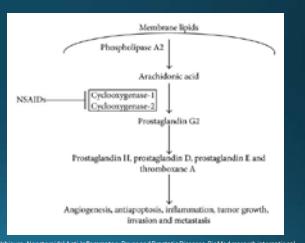
- Tramadol
  - Synthetic mu agonist and serotonin uptake inhibitor
  - Parent molecule as well as M1 metabolite are active
  - Huge variety in response, even within species



4

### NSAIDs

- Inhibition of cyclooxygenase in arachidonic acid degradation
- Meloxicam
- Carprofen



5

### Local anesthetics

- Sodium channel blockers that prevent pain signal from damaged afferent neurons
  - Lidocaine, bupivacaine
  - Some debate about mixing or using on their own



6

## Gabapentin

- Exact mechanism of action unknown
- Appears to work through calcium channel blockade
- Also has anti-seizure activity



7

## Ferrets

- In general respond to drugs like dogs and cats
- Often become more sedated with opioids than expected
- Signs of pain
  - Bruxism
  - Lack of movement
  - Splinting abdomen



9

## Ferret surgical anesthesia

- Premed midaz/mu opioid
  - Buprenorphine 0.01-0.05 mg/kg SC/IM/IV
  - Hydromorphone 0.1 mg/kg SC/IV/IM
- Induce with propofol, alfax, ket/midaz
- Mask difficult due to jaw tone



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## Ferret analgesia

- Meloxicam
- Buprenorphine
  - TM appears to be effective
- No good data on tramadol or amantadine
- No analgesia with alfax/tramadol



Journal of Veterinary Pharmacology and Therapeutics  
Volume 37, Issue 4, 2014  
Anesthetic effects in the ferret of buprenorphine alone and in combination with medetomidine or tramadol: a pilot study  
M. Gaita, G. C. Garcia-Gomez, L. A. and A. Gomez de Segura  
First published online June 17, 2014  
Journal homepage: <http://onlinelibrary.wiley.com/journal/17500287/14/4>

13

## Nerve growth factor inhibitors

- Monoclonal antibodies are species-specific
- No expectation of them working in non-target species



8

## Ferret sedation

- Ultrasound, IV cath placement, phlebotomy
- Midazolam 0.2 mg/kg + butorphanol 0.2-0.4 mg/kg IM
- Monitor temp and pulse ox
- Artefactual drop in PCV in sedated/anesthetized ferrets



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## Ferret surgical analgesia

- Local blocks
  - Dental blocks
  - Ring blocks for tail/digit amputations
- CRF
  - HLK as for other mammals
  - Fentanyl
- Epidurals
  - Buprenorphine, morphine
- NSAIDS
  - Meloxicam most commonly used
    - 0.2mg/kg SQ achieved plasma concentrations that are therapeutic in horses



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## Rabbits

- Signs of pain
  - Teeth grinding
  - Hunched posture
  - Lack of movement
- Sedation for procedures (rads, IV cath, CT scan)
  - Midaz 0.5 mg/kg + butorphanol 0.5-1 mg/kg IV
  - Butorphanol more appropriate as sedative than analgesic



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## Rabbits

- Buprenorphine
  - 0.01-0.05 mg/kg IM/IV q6
  - Good initial analgesic in GI stasis, oral disease while rehydrating
  - Poor bioavail given SQ
  - Significant variability in PK with high-conc products (ex: Simbadol)
  - No effect on GI motility in single dose study at 0.1 mg/kg



15

## Rabbits and maropitant

- Failed to reduce pain at 2 mg/kg or 10 mg/kg SQ
- Did (insignificantly) increase food intake and fecal production



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## Gabapentin in Rabbits

- 25 mg/kg PO
- Decreased reactivity which peaked at 2 hr, no negative effects
- Analgesia not assessed



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## Other small fuzzies

- Meloxicam
  - Guinea pigs
    - 0.1-0.3 mg/kg PO/SQ
  - Rats
    - 1.0 mg/kg PO/SQ
  - Mice
    - 1-5 mg/kg PO/SQ
- Tramadol
  - Chinchillas
    - No analgesic effect at 40 mg/kg (PD)
    - Others???
- Gabapentin
  - Mice
    - 10-30 mg/kg PO (PD)



21

## Rabbits-lidocaine CRI



16

## Rabbits

- TM buprenorphine is unknown...keratinized epithelium
- Meloxicam
  - 1 mg/kg PO q24h
    - (PK data with 29-day study)
- Tramadol?
  - 4-4.4 mg/kg IV
    - No isoflurane sparing activity
  - 11 mg/kg PO
    - Did not reach adequate plasma levels



18

## Other small fuzzies

- Buprenorphine
  - Rats
    - 0.05 mg/kg SC/M q12h (PD)
  - Mice
    - 0.1 mg/kg insufficient analgesia for laparotomy
  - Chinchillas
    - 0.2 mg/kg SQ q4-6h (PD)
  - Guinea pigs
    - 0.2 mg/kg TM q5h (PD)
    - 0.2 mg/kg IV q7h (PD)



20

## Hedgehogs

- Buprenorphine
  - 0.03 mg/kg SQ (into the mantle) q36h or 0.05 mg/kg SQ q48h
- Methadone
  - 0.5 mg/kg SQ-no effect
  - 1.0-1.5 mg/kg SQ: <2hrs
- Hydromorphone
  - 0.15 mg/kg SQ q4hrs
  - 0.3 mg/kg SQ q 6h
- Both M and H caused transient sedation, nausea, vocalization, ataxia



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## Birds-Psittacines

- Opioids
  - Kappa receptors predominate over mu
  - Buprenorphine, hydromorphone, etc are ineffective analgesics
  - Butorphanol is most effective opioid in parrots
  - 1-5 mg/kg IM q2-3h
    - Usual premed is midaz 0.5 mg/kg and butorphanol 1-2 mg/kg IM



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## Birds

- Tramadol
  - Amazon parrots
    - 30 mg/kg PO q6h (PK/PD)
  - Red tail hawks
    - 8-11 mg/kg PO q12h
  - Bald eagles/kestrels
    - 5 mg/kg PO q12h



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## NSAIDS in Poultry



- The median lethal dose (LD<sub>50</sub>) was 156.5 mg/kg intraperitoneally. The median effective analgesic dose (ED<sub>50</sub>) of meloxicam in chicks was 8.25 mg/kg intraperitoneally.
- 5 mg/kg decreased lameness in broilers

27

## Maropitant in Psittacines



- Did not assess analgesic properties
- No apparent contraindications
- Short half-life and low plasma concentrations

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## Birds-Falconiformes

- Kestrels
  - Butorphanol is ineffective, buprenorphine provides analgesia
  - 0.1-0.6 mg/kg IM q8h
  - Not effective in red tail hawks...
  - Effective in pigeons for 2-5 hours at
    - 0.25 mg/kg IM



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## NSAIDS in Psittacines



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## Birds

- Gabapentin
  - Amazon parrot
    - 15mg/kg PO q8h (PK)
  - GHO
    - 11 mg/kg PO q8h (PK)



28

## Maropitant in Poultry



- 1-2mg/kg SQ q12-24h maintains concentrations similar to dogs
- Analgesic properties unknown

30

## Birds

- Local anesthesia
  - Lidocaine
    - <4 mg/kg peripherally
    - No adverse effects at 6mg/kg IV in broilers
    - Onset of action approx. 1.5 min and duration 18 min at 2 mg/kg
  - Bupivacaine
    - 2 mg/kg peripherally
    - Onset of action approx 9 min and duration approx. 1hr at 0.5 mg/kg

Research Paper  
Cardiovascular tolerance of intravenous lidocaine in broiler chickens (*Gallus gallus domesticus*) anesthetized with isoflurane  
Jutta Brändström<sup>1</sup>, Anderson F de Góis<sup>2</sup>, R. B. Bruno Piyadasa<sup>3</sup>, Brett Stott<sup>4</sup>, Javier Navarro<sup>5</sup>, Thomas N. Tully Jr.<sup>6</sup>  
Original Study  
Comparison of Anesthetic Efficacy of Lidocaine and Bupivacaine in Spinal Anesthesia in Chickens  
Ali Khansariabadi, DVM, Somayeh Karimi-Darvishi, DVM, DPKC, and Ghasem Akbari, DVM, DVSc

31

## Reptiles

- All over the map
- Opioids
  - Butorphanol not useful
  - Buprenorphine not useful
  - Hydromorphone
    - 0.5-1 mg/kg in RES
  - Morphine
    - 1-5 mg/kg
      - Good in RES/BD
  - Tramadol
    - 5-10 mg/kg q48-72h
      - Chelonians



33

## Reptiles

- All over the map
- Opioids
  - Butorphanol not useful
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  - Hydromorphone
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      - Good in RES/BD
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## Take home points

- Ferrets are like dogs and cats
- Rabbits like buprenorphine, lidocaine, and meloxicam doses are high
- Parrots like butorphanol, lots of meloxicam, and lots of tramadol
- Reptiles are weird
- Locals can work

## Reptiles

- Signs of pain
  - ???



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## Reptiles

- NSAIDS
  - No evidence of efficacy in any group
- Local anesthetics
  - Lidocaine
    - 2 mg/kg (anecdotal)
    - Topical EMLA cream at 1g/10cm<sup>2</sup> for penile amputation
  - Bupivacaine
    - 1 mg/kg (anecdotal)

ACTAVET BRNO 2015; 80: 71-75. doi:10.2784/vetbr.2015.8010073

Combination of lidocaine/bupivacaine with tramadol for short time anaesthesia analgesia in chelonians: 18 cases

Filippo Spadola<sup>1</sup>, Manuel Morici<sup>2</sup>, Zdeněk Krostek<sup>2</sup>

<sup>1</sup>University of Messina, Department of Veterinary Sciences, Messina, Italy

<sup>2</sup>University of Veterinary and Pharmaceutical Sciences Brno, Faculty of Veterinary Medicine, Brno and Faculty of Veterinary Chiropractic, Brno, Czech Republic

Received July 3, 2014  
Accepted October 27, 2014  
Abstract

34

## Reptiles

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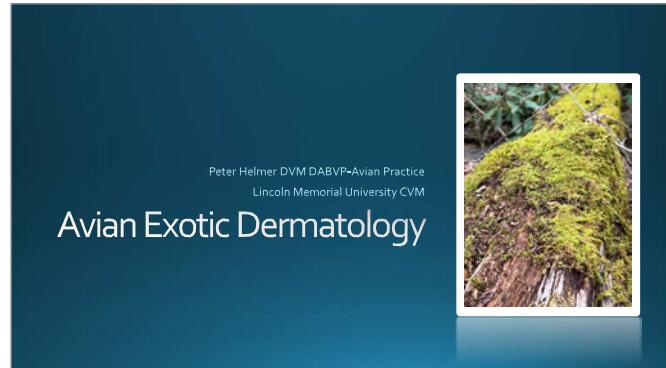
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6005

# AVIAN EXOTIC DERMATOLOGY

SMALL ANIMAL PROGRAM | EXOTICS

Speaker: Peter Helmer, DVM, DABVP-Avian Practice



1

## Objectives

- Alopecia
- Bugs and Bacteria
- Cancer
- Dermatophytes
- Erythema
- Fungal

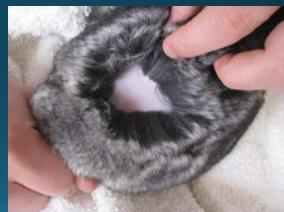
2



3

## Alopecia-Chinchilla

- Traumatic fur slip
- Dx:
  - History
  - Non-inflamed, non-crusting
- Tx:
  - None



<https://www.foreverloveyourchinchilla.org/picking-up-and-holding.html>

2

## Alopecia-Rabbit

- Ventral neck
- Dewlap for nesting
  - Dx:
    - Intact?
  - Tx:
    - OVH
- Also consider dental disease
  - Dx:
    - Oral exam/imaging
  - Tx:
    - As needed



<https://www.rabbitsonline.net/threads>

4

## Alopecia-Ferret

- Adrenal gland disease
  - May be pruritic
  - Overproduction of androgens and estrogens
- Initially tail and sacral area
- Dx:
  - Blood sex hormone profile
- Tx:
  - Deslorelin (Suprelorin implant)



5

## Alopecia-Guinea Pig

- Ovarian cysts
  - Non-pruritic
  - Serous vs folliculogenic
  - Flanks, bilaterally symmetrical
- Dx:
  - Palpation, US
- Tx:
  - OVH or percutaneous drainage
  - HCG 100 ug SQ q 7d x 3 tx
    - Only folliculogenic cysts



6



## Alopecia-Guinea pig



7

## Alopecia-Birds

- Feather Damaging Behavior
- Dx:
  - History, PE, +/- medical workup
- Tx:
  - Frustrating, multi-modal



9

## Bugs-Chickens

- Scaley leg
  - Dx:
    - Appearance
    - Scrape
  - Tx:
    - Petroleum jelly qd x 1 week
    - Ivermectin (off label)
    - Elector (Spinosad) off-label but no withdrawal for fowl mites or red mites
    - Contact FARAD



<https://www.bhvet.org/bhvet-health/health-problems/scaley-leg/>

11

## Bugs-Ferrets

- Ear mites
  - Very common
  - Brown aural exudate
  - Dx:
    - Ear cytology
  - Tx:
    - Ivermectin
    - Revolution
    - Bravecto
    - Treat all in contact animals



13

## Alopecia-Hamster

- Cushing's
  - Usually older hamster
  - PUPD
- Dx:
  - Challenging
  - Do skin scrape (see Bugs)
- Tx:
  - Quality of life



8

## Bugs-Budgies

- Scaley face or scaley leg mites
- Dx:
  - Appearance
  - Scrape
- Tx:
  - Ivermectin
  - Mineral oil?



10

## Bugs-Ferrets

- Fleas
  - Dx:
    - Visualization of fleas or feces
    - Pruritus
  - Tx:
    - Advantage Multi (on label)
    - Revolution (off label)
    - Bravecto (off label)



12

## Bugs-Rabbits

- Fleas
  - Dx:
    - Visualization of fleas or feces
    - Pruritus
  - Tx:
    - Advantage Multi (on label)
    - Revolution (off label)
    - Very short duration in rabbits, reapply qd
    - Bravecto (off label)
    - Capstar
    - Frontline is TOXIC to rabbits



14

## Bugs-Rabbits

- Ear mites
  - *Psoroptes cuniculi*
- Dx:
  - PE
  - Ear cyto
  - Very painful
- Tx:
  - Ivermectin
  - Revolution
  - Bravecto
  - +/- analgesic
  - Do not need to clean ears



15

## Bugs-Hamsters

- *Demodex aurati*
  - Often in conjunction with Cushings disease
  - Pruritic
  - Dx:
    - Tape prep, skin scrape
  - Tx
    - Bravecto 20 mg/kg topical



The Canadian Veterinary Journal / La Revue vétérinaire canadienne  
Vol 49 No 2 March 2008  
Oral fluralaner as a treatment for *Demodex aurati* and *Demodex criceti* in a golden (Sylvan) hamster (*Mesocricetus auratus*)  
G. Léveillé, B. Léveillé, L. Léveillé

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## Bugs-Hedgehogs

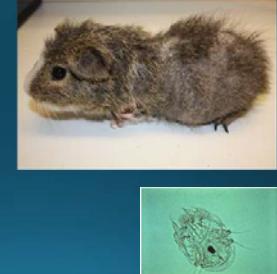
- Skin/quill mites
- Quill loss, hyperkeratosis
- "dust" on face
- Dx:
  - Visualize mite
- Rx:
  - Ivermectin
  - Revolution
  - Bravecto



17

## Bugs-Guinea Pigs

- Skin mites
  - *Trixacarus caviae*
  - Very pruritic
  - Sometimes described as "seizures"
  - Dx:
    - Skin scrape
  - Tx:
    - Ivermectin
    - Revolution
    - Bravecto



https://cm.missouri.edu/diseases-of-research-animals/diseases/guinea-pig/skin/strange-mites-in-guinea-pig/

18

## Bugs-Guinea Pigs and Rats

- Lice
  - Often incidental
  - Sacral area, white nits at hair base
- Dx:
  - Tape prep
- Tx:
  - Bravecto
  - Revolution



19

## Bacteria-Rabbits

- Syphilis
  - *Treponema cuniculi*
- Rabbits with nasal/labial and genital lesions
- Dx:
  - PE
  - Serology/biopsy
  - Response to therapy
- Tx:
  - Benzathine penicillin injections (60 000 IU/kg SQ q 7d x 2-3 tx)
  - Azithromycin 15 mg/kg PO q24h x 25d



Comparative Study: Azithromycin Chemother. 1990 Jan;25 Suppl A:91-9.  
doi:10.1007/BF02251931.  
Efficacy of azithromycin for therapy of active syphilis in the rabbit model  
C. A. Lukens Jr., T. M. Fehl, S. A. Baker-Zande

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## Cancer-Psittacines

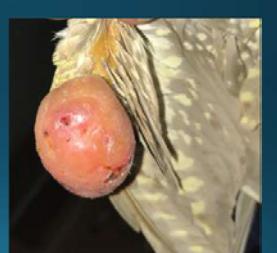
- Squamous Cell Carcinoma
  - Uropygial gland and oral
  - Dx:
    - Deep biopsy
  - Tx:
    - Excision with wide margins
    - Radiation tx
      - Strontium vs external beam
    - Intralosomal carboplatin



21

## Cancer-Psittacines

- Xanthoma
  - Benign neoplasm
  - Often on wing tips, particularly cockatiels
  - Dx:
    - FNA (tend to bleed....)
  - Tx:
    - Excision



22

## Dermatophyte-Guinea pig/chinchilla

- Usually *Trichophyton mentagrophytes*
  - Carriage rates for spores very high
  - Dx:
    - DTM +/- PCR (don't rely on PCR alone due to species variability)
    - Will not fluoresce with Wood's lamp
  - Tx:
    - Topical azoles/terbinafine
    - Systemic terbinafine (20 mg/kg PO q24h in GP, 20-40 mg/kg PO q24h in chinchillas)
    - Itraconazole (5-10 mg/kg PO q24h)



23

## Dermatophyte-Rabbit



24



25

## Fungal testing

**MICROBIOLOGY**

**MOLECULAR DIAGNOSTICS**

	TEST	RESULT
Microsporum canis	RealPCR	NEGATIVE
Microsporum canis RealPCR	RealPCR	NEGATIVE
Trichophyton spp. RealPCR	RealPCR	NEGATIVE

**NOTES**

Received 08/01/2017

A note: Effective Day 1, 2017, test code 9888 now includes a fungal test for *Microsporum canis* and *Trichophyton spp.* on a selective dermatophyte media regardless of PCR results.

A positive *Trichophyton* RealPCR Panel result indicates that DNA of *Trichophyton mentagrophytes* spp. was detected in the samples submitted. In a patient with dermatophytosis, this may indicate a primary infection or a secondary infection in a non-infected patient or a carrier state. Dermotest potential results.

A negative *Trichophyton* RealPCR Panel result indicates that DNA of *Trichophyton mentagrophytes* spp. was not detected in the samples submitted. However, a negative PCR result may be caused by the sample being submitted too early in the disease process, incomplete PCR amplification, or incomplete sample state, or the occurrence of a new strain variation.

**DESCRIPTION**

Examined are superficial suppurative crusts and keratin flakes submitted by numerous fungal organisms (dermatophytes).

**MICROSCOPIC FINDINGS:** ULCERATIVE CRUSTING DERMATITIS WITH INFILTRATE DERMATOPHYTES

**COMMENTS:**

Dermotest potential is fairly common in rabbits. *Trichophyton mentagrophytes* is most commonly isolated in pet rabbits.

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## Treatment

- Itraconazole
  - 5-10 mg/kg PO q24h
- Topical therapy
  - Terbinafine
  - Ketoconazole, climbazole, miconazole



<https://vet-us.virbac.com/itrafungol>

27

## Erythema-snakes

- Consider burns vs other trauma vs septic bluish
- Dx:
  - CBC
  - +/- Culture of blister fluid
- Tx:
  - Topical and systemic antibiotics
  - Betadine soaks?



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## Fungal-Bearded Dragons

- CANV (*Chrysosporium anamorph of Nannizziopsis vriesii*)
  - Dx:
    - Biopsy
    - PCR
  - Tx:
    - Voriconazole
    - Topical terbinafine



29

7001

# DECIPHERING PET FOOD LABELS

SMALL ANIMAL PROGRAM | NUTRITION

Dr. Jenna Manacki, DVM, Residency-Trained in Clinical Nutrition

## OBJECTIVE EVALUATION OF COMMERCIAL PET DIETS

Most veterinary nutritionists analyze commercial diets using the World Small Animal Veterinary Association (WSAVA) Global Nutrition Committee Recommendations for Selecting a Pet Food. This is a free handout that is available in the online WSAVA Toolkit (WSAVA.org). Critical assessment of a commercial diet includes:

1. The qualifications of the diet formulators (ideally an individual with a PhD in companion animal nutrition or a board-certified veterinary nutritionist).
2. Whether the diet was formulated to be complete and balanced, or if it has undergone a clinical feeding trial.
3. The proper use of AAFCO statements to discern what species and life stage the diet is intended for.
4. The location of manufacturing facilities, and whether the manufacturing is done by a third-party.
5. The availability of a typical nutrient analysis upon request.
6. The caloric value of the food by weight (g or kg) and by common measure (cup, can, pouch, box etc.).
7. Whether peer-reviewed product research has been conducted.

Of the criteria listed above, only numbers 2, 3, 4, and 6 are required on a pet food label.

## PET FOOD REGULATION

In Canada, the Canadian Food Inspection Agency (CFIA) regulates the import and export of animal products but does not oversee domestic pet food manufacturing. Most Canadian companies voluntarily adhere to AAFCO (U.S.) or FEDIAF (Europe) guidelines. It is important to note that AAFCO and FEDIAF do not approve or certify pet foods. Products manufactured in Canada that are not exported have no regulatory oversight.

## LABEL INTERPRETATION: NAMING RULES

- 95% Rule: A named ingredient with no descriptor is at minimum 95% of the total weight of the product (e.g., Beef Dog Food). This type of food is uncommon in North America.
- 25% Rule: The named ingredient(s) in front of a descriptor (meal, dinner, entrée etc.) are  $\geq 25\%$  of the total weight of the food (e.g., Chicken and Rice Dinner).
- 3% or "With" Rule: The ingredient stated after "with" in a name is at least  $\geq 3\%$  of the total weight of the food (e.g., "Cat Food Stew with Tuna").
- "Flavour" Rule: The flavour mentioned in the name must be detectable in the food, but can be  $<1\%$  of the total weight of the food (e.g., Filet Mignon Flavour Dog Food).
- "All" Rule: The food is comprised of only one ingredient. This is intended for diet toppers and treats only (e.g. All Beef Liver Treats).

## LEGALLY DEFINED TERMS

The terms premium, super-premium, and holistic are not regulated and serve as marketing devices.

### Legally protected terminology includes:

- “Natural”: There are no chemically synthesized ingredients included in the food, unless there is an added statement of “with” or “including” vitamins and minerals.
- “Human Grade”: The company meets all FDA standards for human food handling and safety.
- “Organic”: The company is certified according to USDA or the equivalent CFIA human food standards.
- Light/Lite/Low Calorie: The food must meet AAFCO-defined energy density thresholds for a lower calorie density.

## NUTRITIONAL ADEQUACY STATEMENTS

Every label must include an AAFCO adequacy statement, identifying whether the food is formulated with software or tested through a feeding trial. The gold standard for pet food formulation is to complete a feeding trial, but this can be cost prohibitive.

The adequacy statement must also specify the intended life stage for the food (growth, maintenance, late gestation and lactation, or all life stages). “All life stage” diets are formulated to meet the energy demands for pets in late gestation and lactation as this is the most energy demanding life stage.

Therapeutic diets, diet toppers, and treats can state that they are meant for intermittent or supplemental feeding only, as they are not intended to be complete and balanced foods.

Therapeutic diets must state that they are intended to be used under the supervision of a veterinarian.

## GUARANTEED ANALYSIS AND NUTRIENT COMPARISONS

The guaranteed analysis lists minimum and maximum nutrient levels for specific macronutrients. The guaranteed

analysis does not account for moisture, and therefore cannot be used to compare diets. The guaranteed analysis must include crude protein, crude fat, crude fibre, and moisture content. Other ingredients and diet components, such as ash, nutraceuticals, and probiotic cultures may be included with a statement that they are not recognized as an essential nutrient by AAFCO.

The guaranteed analysis is not an accurate measurement of protein content and quality, fat content, or the fibre content of a diet.

Accurate comparison between diets requires conversion to a dry matter or metabolizable energy (ME) basis. Therapeutic diet nutrient information can be found in product guides. Some commercial food companies will post nutrient profiles on a dry matter or ME basis on their websites.

## INGREDIENT LISTS

Ingredients are listed by descending weight, including moisture. Therefore, a dried ingredient such as “chicken meal” may provide more protein than a whole food ingredient with more moisture, such as “chicken breast”, despite appearing later on the list.

All ingredients must be described using names approved by and listed in the AAFCO guidelines.

## FUTURE AAFCO LABEL UPDATES

Planned updates to pet food labels are mandated to be in place by 2030. These updates include a nutrition fact box similar to human food products. This box will include the calories per serving, macronutrient distribution, and more accurate macronutrient measurements. New ingredient definitions will be introduced that are intended to add clarity to the ingredient list for consumers.

The new labels will also include a more descriptive intended-use statement (e.g., “Complete Cat Food for Adults” or “Veterinary Diet for Dogs”). Optional product handling graphics will make pet food labels more consumer friendly and more descriptive for pet owners.

7002

# ALTERNATIVE DIET TRENDS

SMALL ANIMAL PROGRAM | NUTRITION

Microphone icon **Caitlin Grant, DVM, DVSc, Dip ECVN**

**CAVN**  
Canadian Academy of Veterinary Nutrition  
Académie Canadienne de Nutrition Vétérinaire

## Alternative Diets Trends

Dr. Caitlin Grant, DVM, DVSc, Dip ECVN  
OVMA 2026

1

### What is an Alternative Diet?

**CAVN**  
Canadian Academy of Veterinary Nutrition  
Académie Canadienne de Nutrition Vétérinaire

- ▷ Opposite of 'conventional'?
  - Commercial manufactured kibble or canned diet
  - Many varieties of foods in this category
- ▷ Alternative could be:
  - Homemade
  - Raw – commercial or homemade
  - Plant based – commercial or homemade
  - Fresh
  - Others?



2

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### Homemade Diets

- ▷ Can be individualized for the pet
- ▷ Can be more palatable
- ▷ Pet owners have more control
- ▷ Likely not complete and balanced without consulting a veterinary nutritionist



3

### Raw Diets

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- ▷ Uncooked food (meat, bones, vegetables?)
- ▷ Commercial or homemade
- ▷ Likely not complete and balanced
- ▷ Risk for bacterial contamination



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4

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### Plant Based

- ▷ Lacking any animal products
- ▷ May be more aligned with pet owner preferences
- ▷ Could be commercial or homemade
- ▷ Concern for nutrient availability
- ▷ Possibly not complete and balanced



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### Fresh Diets

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- ▷ Commercially available but resemble homemade
- ▷ Less time consuming compared to homemade
- ▷ Can be complete and balanced, but not all are



6



**Who is Feeding Alternative Diets?**

2018 Survey (US, Canada and Australia)

- ▷ 63% dogs and **62% cats** fed commercial + homemade
- ▷ 7% dogs and **4% cats** fed homemade diet exclusively

Dodd et al., 2018




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**Can Alternative Diets be a Good Choice?**

- ▷ Not all alternative diets are equal
- ▷ Need to assess individual animal and diet



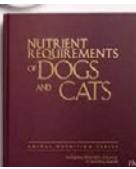

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**Feeding Dogs and Cats**

- ▷ Consider life stage requirements
  - Gestation/lactation
  - Growth (large breed)
  - Adult maintenance



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**Feeding Dogs and Cats**

- ▷ Feeding Management
  - Free vs meal feeding
  - Meal frequency
  - Mental stimulation






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**Motivations**

- ▷ Control over what is fed
- ▷ Feeding dogs and cats like wild ancestors
- ▷ Mistrust of commercial pet foods
- ▷ Instructed by breeder or other paraprofessional
- ▷ Improvement in one or more clinical signs





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**Feeding Dogs and Cats**

- ▷ Nutrient requirements
  - Amino acids
  - Fatty acids
  - Vitamins
  - Minerals
- ▷ Unique requirements for cats
  - Taurine
  - Arginine
  - Arachidonic acid
  - Preformed vitamin A
  - Niacin




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**Feeding Dogs and Cats**

- ▷ Pets with medical conditions
  - Many health conditions are responsive to diet
  - Identify key nutrients of concern
  - Feeding an appropriate food can then
    - Prevent, treat, support these medical conditions




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**Research - Raw**

- ▷ Minimal research on benefits
- ▷ Risk of bacterial contamination
  - Spread in the environment – feces and saliva
  - Risk for other pets and family members
- ▷ Nutritional adequacy




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14

## Evaluating Nutritional Adequacy of Commercial Raw Food Diets

- ▷ How do we assess commercial diets to determine whether they are nutritionally adequate?
  - Information reported by the company – pet food label
  - Information reported online
  - Contacting company
  - 3<sup>rd</sup> party analysis



15

## Reading Pet Food Labels



Nutrition   Per 100 g	
Calories	202
Protein (Min)	16.6%
Fat (Min)	13.8%
Moisture (Max)	64.4%
Fibre (Max)	0.6%
Ash	2.5%
Calcium	0.34%
Phosphorus	0.36%

Diets differ in their ME due to content of fat, protein and carbohydrates

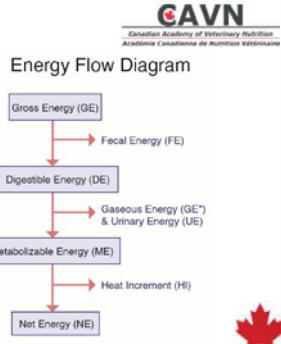


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## ME Equations

- Manufacturers have different methods of calculating ME<sup>4</sup>
  - Traditional Atwater equation
  - Modified Atwater equation
  - National Research Council (NRC) predictive equation
  - Many more!

NRC 2006



## Reading Pet Food Labels



Nutrition   Per 100 g	
Calories	202
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Moisture (Max)	64.4%
Fibre (Max)	0.6%
Ash	2.5%
Calcium	0.34%
Phosphorus	0.36%

Modified Atwater equation: ME (kcal/kg) = 10 X (3.5 x Crude Protein % + 8.5 X Crude Fat % + 3.5 X Nitrogen Free Extract%)

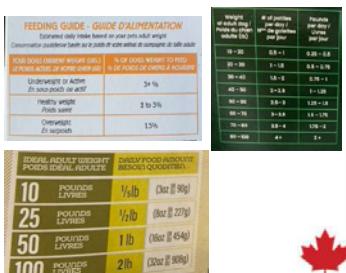


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## Reading Pet Food Labels



Feeding directions vary between companies and differ between weight categories



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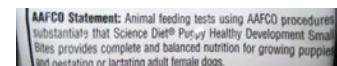
## Knowledge Gaps

- ▷ No standard method of calculating ME
  - How do CRFD companies calculate ME? Is it comparable to standard equations?
- ▷ Are Canadian made raw food canine diets complete and balanced?



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## Reading Pet Food Labels



- ▷ Nutritional adequacy statement
- ▷ Assures diet meets minimum nutrient requirements for a given life stage and species
- ▷ 'Complete & balanced'
- ▷ Not always present (in Canada)



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## Objectives

- ▷ **Objective 1:** Investigate differences in the ME reported on the label in comparison to the laboratory analyzed ME.
- ▷ **Objective 2:** Assess the essential nutrient content of select diets



## Diet Selection



### Research

Compiled list of CRFDs available in Guelph pet stores



### Inclusion Criteria

- Narrowed list based on inclusion criteria
- No nutritional adequacy statement
- Fed as a whole diet
- Manufactured in Canada
- Intended for dogs



### Random Selection

Randomly selected 2 diets from 5 companies. Each diet was purchased at 2 locations.

$$5 \times 2 \times 2 = 20 \text{ samples}$$



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## Impact of Findings: Objective 1

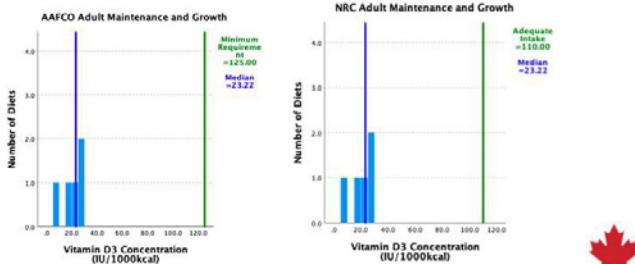


- ME reported on label is lower than analyzed ME
  - ME on product may not be accurate → underestimate calorie intake → obesity/overconsumption
- Potential batch variation for same diet purchased at different locations



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## Objective 2: Nutrient Analysis



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## Impact of Findings: Objective 2

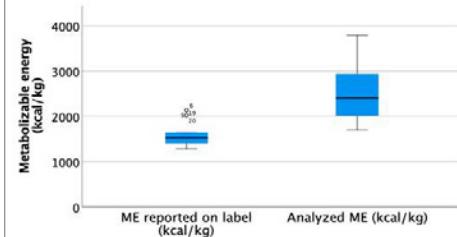


- Several other cases regarding micronutrient deficiencies found in raw diets documented in the literature
- Vitamin E – antioxidant that protects cells against oxidative damage
- Vitamin E deficiency can lead to muscle weakness and neurological conditions
- Pet owners should exercise caution when selecting CRFDs without a nutritional adequacy statement**



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## Objective 1: Label vs Analyzed ME



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## Objective 2: Nutrient Analysis



- ANALYSES:** Energy, crude protein, crude fat, crude fibre, crude ash, minerals, and vitamins A, D3 and E.

Nutrient	Units per 1000 kcal ME	Median [Interquartile Range]	Adult Maintenance		Growth and Reproduction
			AAFCO MR	NRC AI	
Phosphorus	g	2.23 [1.38-2.77]	1	0.75	2.5
Potassium	g	0.769* [0.65-0.96]	1.5	1	1.5
Sodium	g	0.41 [0.34-0.48]	0.3	0.3	0.35
Chloride	g	0.21* [0.15-0.33]	0.3	0.3	0.72
Iron	mg	13.02 [12.28-15.17]	10	7.5	22
Magnesium	mg	0.11* [0.09-0.13]	0.15	0.045*	0.1
Copper	mg	1.1 [0.8-1.2]	1.2	1.5	3.1
Manganese	mg	0.61 [0.49-2.91]	1.25	1.2	1.8
Zinc	mg	10.45* [8.46-14.67]	20	15	25
Vitamin D3	IU	23.77** [15.92-25.01]	125	110	110
Vitamin E	IU	N/A	12.5	6	12.5

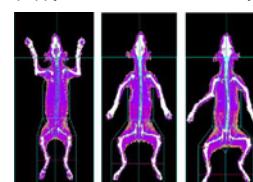


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## Impact of Findings: Objective 2



- Vitamin D3 deficiency can cause reduced absorption of calcium and phosphorus – can lead to skeletal abnormalities
- Case report – 4 month old puppy with nutritional secondary hyperparathyroidism



Dodd et al. (2019) J Anim Physiol Anim Nutr. 00:1-8



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## Research – Raw Diets



Polish Journal of Veterinary Sciences Vol. 27, No. 1 (2024): 151-159

DOI 10.24425/pjvs.2024.149344

Review paper

### Raw diets for dogs and cats: Potential health benefits and threats

D. Główny<sup>1</sup>, N. Sowińska<sup>1,2</sup>, A. Cieślak<sup>1</sup>, M. Gogulski<sup>1,2</sup>, K. Konieczny<sup>1</sup>, M. Szumacher-Strzelb<sup>1</sup>

- Benefits**
  - Natural
  - Digestibility
  - Less mutagenic risk
  - Immunity
  - EPI treatment



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## Assessing Alternative Diets

CAT NUTRITION & DIET

### 6 Delicious Homemade Cat Food Recipes (Vet Approved)

By KATE BARRINGTON | 0 Comments | Last updated Feb 10, 2023

Medically reviewed by Jelena Paskaraca, DVM

Share

#### ▷ Ingredients:

- 5 pounds chicken thighs with bone
- 7 oz. raw chicken liver
- 14 oz. raw chicken heart
- 8 oz. bottled spring water
- 4 raw egg yolks
- 2,000mg taurine
- 4,000mg wild salmon oil
- 200mg vitamin B complex
- 200 IU vitamin E
- 1 ½ tsp lite iodized salt
- 4 tsp psyllium husk powder (optional)

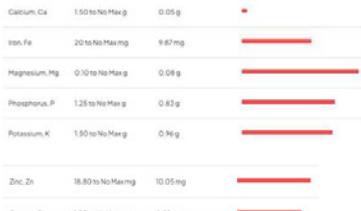


39

## Assessing Alternative Diets



## Assessing Alternative Diets



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## Assessing Alternative Diets

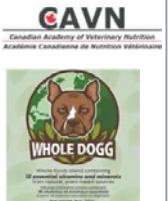
- ▷ Assume its not complete and balanced if:
  - The 4 key items from the quick assessment are not present
  - The recipe was obtained online from an unknown source
  - The creator of the recipe does not have any appropriate nutrition or veterinary credentials
  - A recipe is not being followed



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## Assessing Alternative Diets

- ▷ Supplements
- ▷ OTC options vs specific HMD supplements
- ▷ Specific HMD Supplements
  - Highly concentrated
  - Majority made in the US – shipping
  - Do not have a set dosing
  - Should be introduced slowly
  - Are not interchangeable



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## Communication Tips

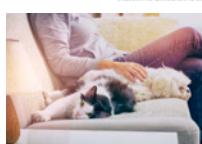
- ▷ Seek to understand motivations
- ▷ Learn exactly how they are feeding the alternative diet
- ▷ Ask permission for discussion
- ▷ Enquire about experience and knowledge
- ▷ Ask permission for information sharing
- ▷ Consider baby-steps
- ▷ Be willing to compromise



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## Resources for Homemade Diets

- ▷ For healthy pets:
  - BalanceIt.com
  - Hilary's Blend
- ▷ Consider referral
  - Pets with medical conditions
  - Pet owners that need more support/want a more individualized approach
- ▷ Options for referral to veterinary nutritionists in Ontario!
  - Telehealth and in-person
  - Direct with pet owners and vet-vet



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## Case Examples - Rusty

- Background Info
- ▷ 12 yr old MN DSH
- ▷ On a duck based HMD from Hilary's Blend cookbook
- ▷ Pursued nutrition consult after diagnosis of CKD IRIS Stage II



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## Case Examples - Rusty

### Nutrition Consult

- ▷ Assessed recipe – complete and balanced for adult cat
- ▷ Changed to lower P supplement



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## Case Examples - Lola

### Nutrition Consult

- ▷ Asked owner to complete food diary
- ▷ Owner then attempted to reduce calories from treats
- ▷ Needed to restrict calories for weight loss – unsafe with current raw diet
- ▷ Successfully transitioned to therapeutic weight loss food



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## Case Examples - Sylvester

### Nutrition Consult

- ▷ Assessed recipe
- ▷ Replaced supplement and recommended cooking



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## Resources

CVMA Position Statement



CAVN Raw Feeding Handout



WSAVA Handout



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## Case Examples - Lola

### Background

- ▷ 3 yr Pomeranian
- ▷ Picky eater – will only eat raw food
- ▷ BCS 8/9



48

## Case Examples - Sylvester

### Background

- ▷ 18 yr old MN DSH
- ▷ On a raw chicken based HMD from a recipe online
- ▷ Small intestinal neoplasia, CKD IRIS Stage IV, underweight, muscle wasting



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## Ways to Compromise

- ▷ If control is important or there is mistrust in pet food companies
  - Could a homemade cooked diet be an option?
- ▷ If feeding raw (or other alternative type) is important
  - Could a commercial food diet from a more trusted company be an option?



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## Summary

- ▷ Many types of 'alternative' diets – important to know what exactly is being fed – benefits and risks of each
- ▷ Research to support pathogen risk
- ▷ Some mixed research on nutritional adequacy
- ▷ Resources available to help cat owners feeding alternative diets
- ▷ Important to keep open lines of communication with pet owners – building trust



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## Nutrition Referrals

- ▷ Dr. Caitlin Grant – Animal Hospital of Cambridge
- ▷ Dr. Jenna Manacki – VEC Toronto
- ▷ Dr. Sarah Dodd – Dodd Veterinary Services
- ▷ OVC Nutrition Service



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**THANK YOU!**  
Questions?



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7003

# HOMEMADE DIETS – THE ART OF ASSESSMENT & FORMULATION

SMALL ANIMAL PROGRAM | NUTRITION

Speaker: Caitlin Grant, DVM, DVSc, Dip ECVN  
 Jenna Manacki, DVM, Residency Trained in Clinical Nutrition

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## Homemade Diets: The Art of Assessment and Formulation

Dr. Caitlin Grant, DVM, DVSc, Dip ECVN  
 Dr. Jenna Manacki, DVM, Residency Trained in Clinical Nutrition

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## Companion Animal Diets – The Basics

### ANIMALS NEED NUTRIENTS, NOT INGREDIENTS



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## Companion Animal Diets: The Basics

- ▷ The pet nutrition industry advocates for a “complete and balanced” diet plan...what does that mean?
- ▷ “Complete”
  - The diet meets all MINIMUM levels of daily required nutrients
- ▷ “Balanced”
  - The nutrients in the diet are in the appropriate ratios and proportions

3

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## ‘Complete’ and Balanced Diets

<b>Essential Nutrients for DOGS</b> <ul style="list-style-type: none"> <li>▷ Ten amino acids</li> <li>▷ Three(ish) fatty acids</li> <li>▷ Twelve minerals</li> <li>▷ Twelve vitamins</li> <li>▷ Water</li> <li>▷ Energy</li> </ul>	<b>Essential Nutrients for CATS</b> <ul style="list-style-type: none"> <li>▷ Eleven amino acids</li> <li>▷ Four(ish) fatty acids</li> <li>▷ Twelve minerals</li> <li>▷ Thirteen vitamins</li> <li>▷ Water</li> <li>▷ Energy</li> </ul>
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4

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## Complete and ‘Balanced’ Diets

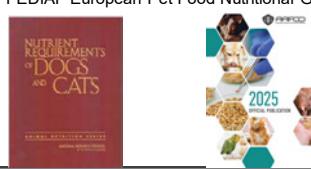
- ▷ Certain nutrients require specific ratios and proportions in order to have optimal metabolic function
  - Some amino acids can be “spared” by other amino acids
    - Methionine/cysteine, Phenylalanine/tyrosine
- Omega-6 to Omega-3 essential fatty acids
  - Proportionally pets need more omega-6 fatty acids
- Calcium to Phosphorus ratio

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## Guidelines for Nutrient Requirements

- ▷ National Research Council (NRC) Nutrient Requirements of Dogs and Cats (2006)
- ▷ Association of American Feed Control Officials (AAFCO) official publication (updated annually)
- ▷ FEDIAF European Pet Food Nutritional Guidelines (updated annually)



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## Why Do Owners Choose to Home Cook?

- ▷ General distrust of commercial pet foods
  - Fear of contamination or recalls
  - Use of ingredients the owners deem as "fillers", "unnatural", or "preservatives"
  - Ethical objection to certain ingredients
- ▷ Cost of commercial or therapeutic diets
- ▷ Palatability
- ▷ Lack of appropriate commercial options
  - Living in a remote region



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## Risk: Poor Hygiene



- ▷ Meats must be cooked to an optimal internal temperature to ensure all food-borne pathogens are killed
- ▷ Cooking utensils and containers must be washed appropriately
- ▷ Owners need to have good self-hygiene
- ▷ Food must be stored appropriately before and after cooking to prevent spoilage



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## Risk: Unbalanced Diets

- ▷ Multiple studies have found that recipes available from textbooks, cookbooks, and websites are unbalanced
  - Stockman et al. 2013 (Maintenance diets for dogs - 200 recipes)
    - 95% of recipes did not meet NRC or AAFCO nutrient profile minimums
  - Heinze et al. 2012 (Diets for dogs with cancer - 27 recipes)
    - 0% of recipes met NRC or AAFCO nutrient profile minimums
  - Larsen et al. 2012 (Diets for dogs and cats with kidney disease - 67 recipes)
    - 0% of recipes met NRC minimum requirements
    - 46% of recipes had an inappropriate calcium-to-phosphorus ratio



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## Diet Assessment: Is the Recipe Balanced?

- ▷ If the diet allows substitutions, it is unbalanced
  - Every ingredient has a unique nutrient profile
  - There are very few instances where one ingredient can be directly substituted for another without changing the nutrient composition of the diet

INGREDIENTS
• 7 lbs 90% lean ground beef or lean ground turkey or chicken
• 1/2 cup hempseeds or 1/3 cup hempseed oil
• 16 oz canned sardines in water or 4 tsp cod liver oil
• 4 tsp ground ginger
• 4 tsp kelp powder
• 8 eggs (without yolks)

90% Lean Ground Beef	Lean Ground Chicken	93% Lean Ground Turkey
230 kcal/ 100 g	189 kcal/ 100 g	213 kcal/ 100 g
138 g protein/ Mcal	123 g protein/ Mcal	127 g protein/ Mcal
45 g fat/ Mcal	58 g fat/ Mcal	55 g fat/ Mcal
57 mg Ca/ Mcal	42 mg Ca/ Mcal	146 mg Ca/ Mcal
15 mg Iron/ Mcal	5 mg Iron/ Mcal	7 mg Iron/ Mcal



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## Homemade Diets: Risks and Limitations

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## Risk: Diet Drift

- ▷ Diet Drift: The tendency of owners to substitute, add, or remove ingredients or instructions in a home prepared recipe
  - Improper measurement of ingredients
  - Use of one ingredient for another
  - Removing an ingredient entirely
  - Adding a new ingredient to the recipe
  - Johnson et al. 2016 - Only 13% of dog owners cooking a home prepared diet formulated by a veterinary teaching hospital were adhering to the recipe a few years later



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## Diet Assessment: Is the Recipe Balanced?

- ▷ Where is the recipe coming from?
  - Online recipes and cookbooks are likely unbalanced
- ▷ Who formulated the diet? What are their credentials?
  - "Nutritionist" is not a protected term
  - There are many groups who will give lay people "credentials" after paying for an online course
  - If a veterinarian formulated the diet, have they undergone a formal residency?
- ▷ When was the diet formulated?
  - Food nutrient databases and nutrient requirements are constantly updated



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## Diet Assessment: Is the Recipe Balanced?

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  - There are very few instances where one ingredient can be directly substituted for another without changing the nutrient composition of the diet

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• 7 lbs 90% lean ground beef or lean ground turkey or chicken
• 1/2 cup hempseeds or 1/3 cup hempseed oil
• 16 oz canned sardines in water or 4 tsp cod liver oil
• 4 tsp ground ginger
• 4 tsp kelp powder
• 8 eggs (without yolks)

16 oz Canned Sardines	4 tsp Cod Liver Oil
944 kcal	162 kcal
4.45 g EPA + DHA	19 g EPA + DHA
22 mcg Vitamin D	45 mcg Vitamin D
50% ME protein	0% ME protein
50% ME fat	100% ME fat



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## Example #2

(This makes about 3 weeks of only homemade food, or 5-6 weeks if I added kibble.)

2 lbs uncooked brown rice

6 eggs

Sunflower oil (About half a cup to 1 cup)

Salt

2 cans of string beans

1 pound of carrots - washed and trimmed, leave whole

6 pounds of boneless, skinless chicken breast

1 to 2 pounds of yams - washed and trimmed, leave whole

and/or 1-2 cans of pumpkin

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1) Cook the rice (I use a rice cooker - set and forget)  
2) In a really big pot (use a stock pot, if you have one), add the chicken first and all fresh veggies (carrots, yams) after. Then add enough water to cover everything. Cover the pot and cook on a medium heat on the stove. When the water is boiling, lower the heat to a simmer. Cook for about 90 minutes, or until everything is very soft.  
3) While the rice and chicken cook, make the eggs. The easiest way to cook the eggs is to break them into a microwave safe bowl and microwave them for a minute at a time. Stir in between. When the eggs are firm, they're done. It won't take long.  
Now this is the hard part. This gets to be a huge amount of food. You can try combining everything in the largest bowl you have or use the stock pot. Or, what I do, is mix everything in two batches. Whatever works for you.  
3) Starting with the rice, oil and salt, thoroughly mix in one ingredient at a time. Make sure to break up the eggs. Stir in the chicken and veggies last. The veggies will fall apart as you stir. Stir in as much of the liquid from the stock pot as you can with out making everything soupy.

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## Benefits of a Homemade Diet

- ▷ Formulated for the individual
- ▷ Consider all factors and incorporate that into the formula
- ▷ Ingredient selection
- ▷ Palatable
- ▷ Highly digestible



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## Candidates for Homemade Diets

- ▷ Pets with multiple medical conditions – no commercial option
- ▷ Pet prefers human foods
- ▷ Owner preference
- ▷ Pet is done growing\*



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## Other Requirements

- ▷ Full nutritional assessment including:
  - Detailed medical history
  - Current and previous diets fed including snacks and treats
- ▷ Owner preferences/expectations



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## Despite the Risks...

2018 Survey (US, Canada and Australia)

- ▷ 63% dogs and 62% cats fed commercial + homemade
- ▷ 7% dogs and 4% cats fed homemade diet exclusively

Dodd et al., 2018



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## A Nutritionist's Approach to Homemade Diets

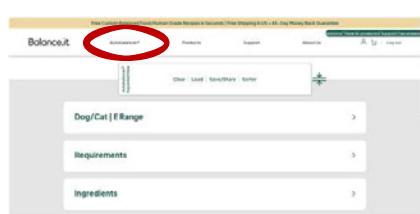
- ▷ When will we agree to formulate a homemade diet\*
- ▷ Tools/resources needed
- ▷ Art of formulating
- ▷ Communicating with owners
- ▷ Follow up



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## Formulation Tools

- ▷ Access to formulation software
  - Ingredient databases
  - Ability to adjust nutrient targets



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## Art of Formulating

- ▷ Calorie target
- ▷ Set nutrient profile or conditions
- ▷ Select ingredients
- ▷ Add in supplements
- ▷ Adjust as needed – some trial and error



30

## Art of Formulating

- ▷ Calorie target
- ▷ Set nutrient profile or conditions
- ▷ Select ingredients
- ▷ Add in supplements
- ▷ Adjust as needed – some trial and error

31

## Art of Formulating

- ▷ Calorie target
- ▷ Set nutrient profile or conditions
- ▷ Select ingredients
- ▷ Add in supplements
- ▷ Adjust as needed – some trial and error

32

## Art of Formulating

### Ingredients

- ▷ Calorie target
- ▷ Set nutrient profile or conditions
- ▷ Select ingredients
- ▷ Add in supplements
- ▷ Adjust as needed – some trial and error

33

## Art of Formulating

### Ingredients

- ▷ Calorie target
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34

## Art of Formulating

### Ingredients

- ▷ Calorie target
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35

## Art of Formulating

### Ingredients

- ▷ Calorie target
- ▷ Set nutrient profile or conditions
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36

## Art of Formulating

- ▷ Calorie target
- ▷ Set nutrient profile or conditions
- ▷ Select ingredients
- ▷ Add in supplements
- ▷ Adjust as needed – some trial and error

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## Art of Formulating

### Veggies

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**Art of Formulating**

Total calories fed: 479 kcal/day  
OR 95.70% of the calculated requirement

AAFCO-NRC	Bar Graph	Show all nutrient requirement w/ Supplement
(001) Protein	43.00 mg	43.00 mg
(004) Total Lipid	10.00 mg	34.38 mg
(005) Calcium, Ca	1.25 to 4.25 g	1.25 g
(011) Magnesium, Mg	1.00 to 4.00 mg	1.71 mg

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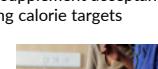
# Art of Formulating

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## Follow Up

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- ▷ Short term
  - During transition – ingredient and supplement acceptance
  - Assessing weight/BCS and adjusting calorie targets
- ▷ Long term
  - Recipe drift
  - Patient medical status



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**A Long Road to Recovery**

Date	Weight	BCS	Muscle Wasting	Body Condition Score (5 to 7)	Notes
Nov 2017	6.9 kg	5/9		5 to 6	Initial Consult, Hills z/d canine dry
Dec 2017	6.4 kg	4/9		5 to 6	Trial of Metamucil, Decreased appetite, Add in canned feline z/d
Jan 2018	6.35 kg	4/9	Mild	5 to 7	Switched to Purina HA
May 2018	5.5 kg	3/9	Mild		Decreased appetite, Enticements added (ground beef)

**Now what??**

45

# Art of Formulating

40

# Communicating with Owners

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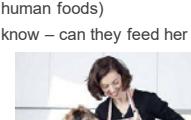


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## What to Recommend Next?

- ▷ Challenging to find a food that meets all criteria that Abby would actually eat
- ▷ Enticements being added to regular food to get her to eat (ground beef, rice, other human foods)
- ▷ Owners want to know – can they feed her more human food?





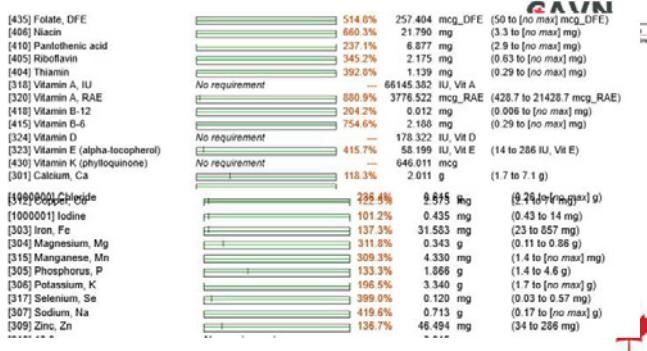
46

## A Recipe for Abby

[n11510] Sweet potato, cooked, boiled, without skin	250.000 g.	250 g	190.98 kcal
[n23561] Beef, 85% lean, 15% fat, crumbles, cooked, pan-browned	125.000 g.	125 g	350.00 kcal
[n20037] Rice, brown, long-grain, cooked	125.000 g.	125 g	138.66 kcal
estimated yield from 37.5 g (approx. 37.5/8 g) of raw/unprepared/dry Rice...			
[n11323] Peas and carrots, frozen, cooked, boiled, drained, without salt	100.000 g.	100 g	47.79 kcal
[n12240] Eggs, whole, cooked	50.000 g.	1 Large	77.54 kcal
[n11458] Spinach, cooked, boiled, drained, without salt	50.000 g.	50 g	11.41 kcal
[m7] Cheese, cottage, lowfat, 2% milk fat (BalanceIt.com)	30.050 g.	30 g	26.90 kcal
[b100] Oil, Canola	5.000 g.	5 g	39.70 kcal
[p4583] Oil, canola	4.500 g.	1 tsp	39.78 kcal
	Total: 768.875 g.		822.25 kcal



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## Homemade Diets in GP

- ▷ BalanceIt.com
- ▷ Hilary's Blend
- ▷ Can refer for:
  - Pets with medical conditions
  - Owner wants a customized recipe
  - A different supplement is preferred (e.g. recipe from Balance It but owner wants to use Whole Dogg)
  - Vet to vet consults an option!



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EXHIBIT HALL

THANK YOU!  
Questions?



53



7004

# STUBBORN FAT

SMALL ANIMAL PROGRAM | NUTRITION

Shoshana Verton-Shaw, RVT, VTS (Nutrition), RLAT

Pet obesity is a growing concern in veterinary medicine, with the majority of small animal patients presenting as overweight or obese. Global rates of pet obesity are on the rise, indicating that this entirely preventable health condition has reached endemic proportions. Misunderstood as a cosmetic concern, excess body fat wreaks havoc on all body systems, increasing inflammatory cytokines, adding to mechanical strain, and heightening the risk of anesthetic and surgical complications. Pets with an overweight or obese body condition have been demonstrated to be at an increased risk of developing disease sooner than their lean counterparts, with an overall reduced life span.

Veterinary teams are aware of the risks of obesity in pets, working to educate pet guardians on lifestyle and environmental modifications, appropriate treating and dietary recommendations. Some clients even start weight loss plans independently, with limited success. Even with veterinary guidance and close follow-up, success rates and long-term compliance tend to be low. So where is the disconnect?

The 2021 AAHA Nutrition and Weight Management Guidelines can be used as a solid foundation for successful weight loss plans. The busy practitioner may be reluctant or limited in their ability to fully implement the guidelines due to time and staffing constraints, minimizing or neglecting the weight loss plan in its entirety. Follow-up and compliance can be a challenge with nutrition, with feeding behavior being an emotional part of the client-patient relationship. Some team members may be uncomfortable discussing weight with clients if there is a pre-conceived concern about the client's own body mass. Clients may be reluctant to try a weight loss protocol due to past attempts at weight management, personal beliefs, and societal pressures.

A focus on communication skills and techniques, such as current research on assessment of the client's willingness to change can be utilized to efficiently communicate with clients. Taking a team-based approach to weight management can increase job satisfaction in employees, while increasing efficiency of nutrition and weight management plans. Development of a consistent, yet flexible protocol can help to ensure continuity of weight management focus throughout busy periods and time challenges.

All practitioners who have implemented weight loss plans in practice have encountered challenging patients, for whom even the best protocols and diets seem to have limited success. There is no simple solution to effective weight management in pets. Communication is key in the patient and diet assessment, implementation and follow-up. The practitioner may need to revisit the diet history for clues about the diet and hidden calories, whether in a written form, oral history, or a combination. The use of a food log or journal may prove advantageous to identify sources of excess calories despite energy restriction. Ensuring the feeding plan closely aligns with preferred feeding plans will minimize the emotional strain of the program on the client, such as maintaining the same number of treats while reducing caloric intake. The selected diet may need to be modified to an alternative weight loss technology.

Accurate diet calculations are essential in both the initial assessment phase of the weight loss plan while assessing dietary intake, and the implementation of the weight management plan. Calculation of the base metabolic rate based on ideal body weight will help to illustrate to the client the excessive dietary intake and need for intervention. Appropriate and accurate assessment of the patient's body condition score (BCS) is critical in the

initial patient assessment, implementation and monitoring of weight loss. Although a basic assessment tool, the BCS system is problematic due to the variety of scoring systems, visual aids and the subjective nature of this tool. A further challenge is the growing prevalence of the mega-obese patient, those who exceed the maximum range of the standard BCS system. An alternative system has been developed by Hill's Pet Nutrition using a body fat index (BFI). This can be assessed subjectively alongside the BCS system, or with the integration of morphometric measurements which may help to provide a more objective goal body weight and caloric requirement in mega-obese patients. In other patients, advanced imaging may be the best option, demonstrating body composition and body fat with a high degree of accuracy alongside an associated imaging demonstrating excess body fat on the individual.

Other forms of technology can be beneficial in challenging weight loss programs. For multi-pet homes, the use of various forms of feeding technology may be advantageous. Feeding stations available only to some pets due to smaller opening sizes in doors or access ports may be a lower-budget tool which can be implemented. For more complex homes, there are feeding stations equipped with microchip or tag readers which will allow access or distribute food only to the appropriate pet. Timed feeders and puzzle toys can help to reduce the incidence of begging, increase satiety and improve compliance. The use of a digital kitchen scale will provide increased accuracy and consistency in patient feeding, further reducing the risk of excess caloric intake. Smart scales for both pets and diet are available with app integration to help integrate the veterinary team into daily feeding management. Activity monitors can provide valuable insight into a client's ability to implement increased physical activity.

References available upon request

## ADDITIONAL RESOURCES:

### **Hill's Pet Weight Care Program, Ontario Veterinary College**

<https://pwcc.uoguelph.ca/>  
[ovc.petweight@uoguelph.ca](mailto:ovc.petweight@uoguelph.ca)

Accepting referrals for telehealth, in-person and hybrid weight management plans, supporting existing plans with family veterinary team, or entire plan and follow-up.

### **WSAVA Global Nutrition Committee**

<https://wsava.org/Global-Guidelines/Global-Nutrition-Guidelines/>

Unbranded resources, including BCS & MCS charts, diet history forms, guidelines, treat information and more.

### **Association for Pet Obesity Prevention**

<https://www.petobesityprevention.org/>

Unbranded veterinary and pet guardian resources, including charts, infographics and calculators. Biannual veterinary surveys contribute to ongoing data on pet obesity rates and trends.

### **2021 AAHA Nutrition & Weight Management Guidelines**

<https://www.aaha.org/resources/2021-aaha-nutrition-and-weight-management-guidelines/>

A comprehensive resource available to veterinary professionals developing or refining nutrition and weight management protocols. Communication strategies, diagnostic, treatment and follow-up tools are included.

7005

# ROUND TABLE DISCUSSION - TEAM-BASED NUTRITION AND COMMUNICATION IN VETERINARY PRACTICE

## SMALL ANIMAL PROGRAM | NUTRITION

Speaker: Caitlin Grant DVM, DVSc, DECVN  
*Jenna Manacki DVM, Residency Trained in Clinical Nutrition  
 Shoshana Verton-Shaw RVT, VTS (Nutrition)*

### OVERVIEW

Nutrition plays a pivotal role in patient health and wellness, yet effective communication about nutrition can be a challenge in many veterinary practices. This round table will bring together veterinary professionals with different backgrounds and qualifications to share experiences, strategies, and tools for improving nutrition discussions through a team-based model.

The conversation will emphasize how leveraging the unique skills of veterinarians, registered veterinary technicians (RVTs), and support staff can make nutrition recommendations more efficient, consistent, and client-friendly. Attendees will be encouraged to participate with questions and examples from their own practices.

Panelists will describe various models of teamwork used in clinical and referral settings. In general practice, an RVT passionate about nutrition may assist by managing referral communication, assessing diets, gathering diet histories, and maintaining client follow-up. In larger or specialty settings, communication liaisons may coordinate referrals and documentation, allowing clinicians to focus on consultations.

In all settings, success will depend on clear delegation, trust, and defined communication roles. Team members who share a commitment to consistent messaging will help create a unified experience for clients, increasing compliance and improving patient outcomes.

Ever a hot topic, panelists will discuss the growing number of clients interested in raw or home-prepared diets. Recommended strategies will include avoiding assumptions, understanding the owner's current knowledge and experience, seeking permission to educate, and focusing on balanced, respectful conversations. Emphasis will be placed on finding common ground and offering evidence-based alternatives rather than confrontation.

A collaborative approach to nutrition will help ensure consistent messaging to clients and provide multiple touchpoints for education and support. When all team members are confident discussing nutrition, the overall quality of communication improves.

Potential challenges may include differing personal beliefs about pet diets among team members. The panel will suggest approaching these moments as educational opportunities—clarifying the evidence, discussing professional responsibility, and ensuring that the clinic's unified message to clients remains consistent.

Common struggles may include reluctance to delegate and difficulty articulating personal or professional needs within the team. These challenges can be mitigated by fostering mutual trust, open communication, and a shared understanding that collaboration improves efficiency and care quality.

The discussion will also touch on the role of RVTs in providing nutrition advice within their scope of practice. While RVTs can play a central role in diet assessment and client education, cases involving complex medical conditions, home-cooked diets, feeding tubes, or rapid weight loss will often warrant referral to a board-certified veterinary nutritionist.

For veterinary teams interested in enhancing nutrition communication skills, several key resources will be highlighted:

- Applied Small Animal Nutrition, 2nd Edition (Delaney & Fascetti)
- Purina Veterinary Handbook of Canine and Feline Nutrition
- Communication-focused texts and continuing education materials specific to veterinary team dynamics.

The round table will underscore the importance of teamwork in veterinary nutrition communication. By empowering all members of the veterinary team to participate in these discussions, practices will be better equipped to enhance client relationships, improve adherence to recommendations, and ultimately deliver better patient care.



8001

# AVOID THE “PEEK AD SHRIEK” – THE SYSTEMATIC EXPLORATORY LAPAROTOMY!

SMALL ANIMAL PROGRAM | SURGERY

 *Lissie Henderson, BVSc (Hons), DipECVS*

## DEFINITIONS

Celiotomy: Incision into peritoneal (coelomic) cavity by any approach

Laparotomy: Flank incision into peritoneal cavity (common usage synonymous with celiotomy)

## DIAGNOSTIC

Ensure appropriate investigations and interpretation

Commonplace - TP/PCV, Urea/creatinine, Electrolytes, Abdominal radiographs and/or ultrasound,

Other - +/- Abdominocentesis, Coagulation profile, Thoracic radiographs, Contrast radiographs, Urinalysis, CT/MRI

## PLANNING

- Ensure adequate patient stabilisation and preparation
- Fasting times (4-6 hours), Antibiosis, Cardiovascular stabilisation
- Ensure appropriate client communication (inclusive of potential negative exploratory laparotomy)
- Ensure adequate knowledge of common surgical techniques
- Execution – perform therapeutic procedures, perform biopsy (avoid the ‘peek and shriek’)

## GENERAL PRINCIPLES/EQUIPMENT

### Exposure

- Wide aseptic preparation
- Lighting
- Removal of falciform fat
- Use of abdominal retractors
- Consideration of intestinal adhesions: previous surgery

Ensure complete exploration - systematic  
Compartmentalise; lift out, pack off  
Biopsy, Biopsy, Biopsy!  
Swab Count  
Suction/Lavage  
Glove and Instrument Change

## SYSTEMATIC EXPLORE

### Entry into the abdomen

Communication with chest – check for diaphragmatic concavity

Gastric dilation and volvulus, mesenteric torsion

Adhesions

Delay exploratory laparotomy - bleeding, large masses, contamination will occur

Check for free fluid- para-lumbar fossa: cytology/bacterial culture

Do I need to make more room?

## **Cranial Quadrant**

Diaphragm  
Terminal oesophagus  
Liver  
Gall bladder and portal vessels (duodenal manoeuvre)  
Gastric cardia, fundus and body, antrum and pylorus

General principles - consider haemostatic abnormalities, gentle handling with fingers, use absorbable suture material

Surgical Options – i) strangulating/guillotine suture technique ii) skin biopsy punch + haemostatic foam iii) partial lobectomy • transabdominal stapler

## **Right Quadrant**

Descending duodenum (duodenal manoeuvre)  
Right limb of pancreas  
Pancreas  
Right kidney/ureter  
Right adrenal gland  
Right ovary/uterus  
Vena cava  
Open omental bursa - Dorsal stomach, left limb of pancreas

## **Cholecystocentesis – apex of gall bladder, drain large volume**

Gastrointestinal viability - Subjective: Colour, Thickness, Vascular pulse, Peristalsis

Overestimate vs Underestimate - However, Wait and Reassess

Importance of controlling contamination

## **Left Quadrant**

Ascending, transverse and descending colon  
Left kidney/ureter (colonic manoeuvre)  
Left adrenal gland  
Left ovary/uterus

## **Gastric**

Indications - ulcers, gastritis, mucosal neoplasia – endoscopic biopsies suffice however if intrabdominal – obtain full thickness (two-layer) biopsy (increased concern for dehiscence if not excisional)

General Principles - “Pack off” abdomen, handle stomach with stay sutures/babcock forceps, biopsy from “avascular” area for diffuse disease

Surgical options - full thickness incision into lumen and excise section parallel to incision, tent stomach wall with a stay suture and incise, variety of closure techniques

## **Caudal Quadrant**

Vagina and uterus  
Prostate, Vas Deferens  
Bladder (lateral and ventral ligaments), Urethra  
Colon, Rectum  
Inguinal canal

## **Central**

Spleen  
Jejunum (from duodenal flexure), Ileum and caecum  
Mesenteric vessels  
Lymphatics, lymph nodes – throughout abdomen, inclusive of sublumbar  
Omentum, peritoneal surfaces

## **Intestinal**

Indications - clinical signs potentially attributable to SI disease, gross/palpable lesions

General principles – “Pack off”, biopsy duodenum, jejunum & ilium if suspect inflammatory bowel disease, ensure two-layer biopsy

Surgical options – full thickness via enterotomy, placement of initial suture knot, incision with scalpel and ellipse with Metzenbaum. Resection and anastomosis for focal lesions

## **BIOPSY – OBVIOUS ABNORMALITIES DETECTED, NO EXCUSE NOT TO BIOPSY**

## **Liver**

Indications - clinical signs attributable to hepatobiliary disease, laboratory support of hepatic disease, gross lesions detected at surgery

## **Pancreatic**

Indications – minimal, gross/palpable lesions

General principles – resultant pancreatitis

Surgical options – Guillotine technique/Cautery (tip), Dissection and ligation technique (body). Need to close mesenteric defect if present

### **Lymph node**

Indications – staging (regional drainage node e.g. splenic), gross/palpable lesions

Surgical options - Incisional wedge or excisional biopsy. Fine, curved mosquito to isolate. close any mesenteric defect. NB. Avoid nodes directly over cranial mesenteric artery/vein

### **POSTOPERATIVE ANALGESIA**

Caution with Non-Steroidal Anti-inflammatories

Analgesia is critical - welfare reasons, reduce peripheral vasoconstriction (so helps blood flow to healing tissues), increases appetite (protein source)

If ileus is going to occur, this will not be prevented by withholding opioid medications

### **POSTOPERATIVE FEEDING**

Eating as soon as recovered from anaesthesia is desirable  
- positive energy balance (anabolic), nutrition supplied to enterocytes, small amounts of easily digestible food, encourages intestinal motility which increases blood flow to healing areas

#### Feeding Tube

If dehiscence is going to occur, this will not be prevented by withholding food

### **GENERIC COMPLICATIONS**

Pancreatitis – anti-nausea medications, gastroprotectants

Oesophagitis – gastroprotectants, prokinetics

Gastrointestinal ileus – ambulation, feeding, prokinetics

Adhesions – care for repeat surgery

References available from author on request



8002

# THE MEGA INTESTINE – SURGICAL APPROACH TO THE LARGE INTESTINE

SMALL ANIMAL PROGRAM | SURGERY

 *Lissie Henderson, BVSc (Hons), DipECVS*

## ANATOMY

Caecum – short (cat), long/spiral (dog)

Cecocolic and ileocolic junction are closely located (<1cm in both species)

Colon – ascending, transverse, descending

Ascending colon is attached via the mesoduodenum (duodenocolic ligament) and mesocolon. Transverse colon passes from right to left. Descending colon

Layering (external-internal): serosa, muscularis, submucosa, mucosa

Mucosal cell types (no villi/aggregated lymph nodes): columnar epithelial, cuboidal epithelial, enterochromaffin.

### Arterial supply:

Cranial mesenteric artery – common colic – ileocolic and right colic, middle colic.

Caudal mesenteric artery – (distal half of descending) – left colic, cranial rectal artery

Branches of the above form the vasa recta, which supply both the subserous and mural (submucosal) arterial networks

### Venous supply:

Left colic, middle colic, right colic, ileocolic drain into the portal vein

Lymphatic drainage

Lacteals drain into the right, middle and left colic lymph nodes

Autonomic innervation:

Cranial and caudal mesenteric plexuses

## PHYSIOLOGY

- Faecal store
- Reservoir for microbial ecosystem
- Immune system - Epithelial barrier (innate), Microfold cells (adaptive)
- Maintaining fluid balance (conserve water)
- Maintaining electrolyte balance (conserve sodium/chloride and short chain fatty acids, secretion of potassium, bicarbonate, mucus)

## SURGICAL PRINCIPLES

Preserve segmental bloody supply

Preserve mural vascular network

Reduce anastomosis tension

### Suture:

Single layer, simple interrupted appositional pattern

Monofilament, absorbable

Round body, taper-cut point

### Stapling devices:

Gastrointestinal anastomosis

Triangular apposition – thoracoabdominal stapler

End-end anastomosis (inverting) – transcecal, enterotomy, transrectal

Biofragmentable Anastomosis Ring: polyglycolic acid and barium sulphate

Sutureless closure: laser, tissue glue, fibrin sealant

## DIAGNOSIS

**Clinical signs:** diarrhoea, dyschezia, tenesmus, haematochezia

Clinical examination and rectal examination

**Radiography:** Size and position, pelvic anatomy. Positive contrast/Negative contrast can be used to delineate mass lesions or identify leakage. Care: overdistension of colon. Contrast options can include barium (do not use if risk of colonic leakage)/Non-iodinated contrast.

**Ultrasonography:** Challenging due to gas content. Typically indicated if regional lymph node assessment or guided biopsies.

**CT/MRI:** Not well established. CT pneumocolonography has been described.

**Colonoscopy:** Preparation for procedure – fasting (48 hours), oral cleansing solution (24 hours), warm water enema (prior to procedure). Left lateral recumbency.

## SURGICAL PROCEDURES

### Preoperative Preparation:

Colonic cleansing: Clinician and procedure dependent. However, considered counterproductive because it turns faecal material into a liquid slurry within the colon, which is much more likely to leak and contaminate the abdominal cavity during surgery

Antibiotic prophylaxis: Addition of metronidazole can be considered however, despite the increased numbers of anaerobic bacteria; no documented comparative to other intravenous antibiotics.

### Caecal Resection: Typhlectomy

**Indications:** Impaction, perforation, neoplasia, inversion

**Technique:** Preserve the ileocolic junction. Transection of the ileocaecal fold that attaches the ileum to the proximal colon is transected. Resection of the caecum is performed, and the opening is closed with a parker-kerr, simple continuous suture pattern or thoracoabdominal stapler. Initial closure can be oversewn and inverted if necessary.

### Colectomy

**Indications:** Neoplasia, perforation, trauma, intussusception (non-reducible), megacolon

**Technique:** Resection of the colon at the level at the junction of the rectum and colon (2cm cranial to the pubic brim and/or 1cm caudal to the site of caudal mesenteric penetration of the serosa). Lumen disparity can be addressed by spatulating the intestine, partial closure of one lumen or suture placement. Side-side anastomosis is not often possible due to tension. Closure is carried out via a simple interrupted or simple continuous suture pattern. Colonic stenosis is a concern if performing simple continuous closure. The least accessible side closure is carried out first. Stapled anastomosis can also be performed. Use of a biofragmentable anastomosis ring is an alternative option.

### Colopexy

**Indications:** Fixation of the colon with the left abdominal wall. Rectal prolapse, perineal hernia (reduction of rectal sacculation), colonic volvulus,

**Technique:** Length of colon to be pexied – 3-4cm. i) simple appositional technique (manual irritation of the abdominal wall and colon with a swab) ii) longitudinal seromuscular incision in antimesenteric surface of the colon and in the peritoneum. Apposition is carried out via a simple continuous suture pattern. Epidural anaesthesia minimizes postoperative pain and tenesmus.

## OTHER

### Colostomy

**Indications:** Bypass of the distal gastrointestinal tract.

**Technique:** i) End on colostomy ii) Loop colostomy. Colon located to the flank in both techniques.

**Complications:** Faecal incontinence. +/- colostomy bag attachment

### Combined Abdominal Transanal Pull Through – Colorectal Amputation

**Indications:** Bypass of the distal gastrointestinal tract.

**Technique:** Laparotomy performed and colon resected cranially and caudally to lesion. Ends of colon are subsequently oversewn. Perineal approach performed and rectum incised; colonic ends are pulled caudally. The proximal colonic segment is subsequently anastomosed to the rectum.

**Complications:** Bleeding, tenesmus, faecal incontinence (typically resolves if 1-1.5cm of distal rectum is left in situ). Septic peritonitis is commonly reported.

### Colotomy

**Indications:** Rare; impaction/foreign body

**Technique:** Small antimesenteric biopsy taken. Closure: simple interrupted or simple continuous.

Postoperative Preparation:

Analgesia: Epidural anaesthesia – consideration when wishing to prevent straining/tenesmus. Non-steroidal anti-inflammatory drugs should be used with caution in colonic surgery. The NSAID caprofen has been shown to cause sloughing of cells and erosion of the colonic mucosa, thus compromising the integrity and barrier function of the colonic mucosa of dogs.

Diet: High residue (fibre), low fat diet – promotion of normal motility (and faecal consistency)

## SURGICAL CONDITIONS – CAECUM

### Caecal Inversion

**Pathophysiology:** Rare. Associated with intussusception/neoplasia

**Signalment:** < 4 years old

**Clinical signs:** Malaise, diarrhea, haematochezia, tenesmus, vomiting and weight loss. Clinical signs associated with gastrointestinal obstruction.

**Diagnosis:** Radiography may reveal caecal soft tissue density.

**Treatment:** Typhlectomy. Prior colotomy to evert the caecum may be required.

### Caecal Impaction

**Pathophysiology:** Impaction with faeces/foreign material

**Clinical signs:** Non-specific

**Diagnosis:** Imaging may reveal loss of gas-filled caecum.

**Treatment:** Typhlectomy. Gastrointestinal biopsies are also recommended to assess for disorders that could affect motility.

### Caecal Neoplasia

**Pathophysiology:** Gastrointestinal stromal tumour (GIST), leiomyosarcoma, adenocarcinoma, undifferentiated sarcoma

**Clinical signs:** Malaise, diarrhea, haematochezia, tenesmus, vomiting and weight loss. Clinical signs associated with gastrointestinal obstruction or septic peritonitis.

**Diagnosis:** Ultrasonography +/- regional lymph node fine needle aspiration.

**Treatment:** Mass removal with margins

**Prognosis:** GIST without metastatic disease – good (1-2 year disease free interval)

## SURGICAL CONDITIONS – COLON

### Megacolon

**Pathophysiology:** Constipation – abnormally delayed or infrequent passage of usually dry, hardened faeces. Severe constipation – obstipation (unable to pass faeces); resultant megacolon.

The cause of megacolon is typically idiopathic (primary), however it is suspected to be secondary to changes in smooth muscle function. Acquired causes of constipation that may lead to megacolon include i) mechanical causes and ii) functional causes.

Mechanical causes: Extraluminal compression (pelvic canal stenosis – narrowing >45%, strictures, postneutering, prostatomegaly), Intraluminal obstruction (foreign body, neoplasia, atresia ani), Metabolic (hypokalaemia, hypothyroidism), Neuromuscular abnormalities (sacral spinal cord deformity (manx), dysautonomia).

**Signalment:** Most commonly middle-aged male cats.

**Clinical signs:** Reduced, painful, or absent defecation. Tenesmus/discomfort may be noted. Dilated colon with very firm feces may be palpable on abdominal palpation. Cats may also become anorexic and dehydrated, vomit, or develop abdominal pain.

**Diagnosis:** Radiographs demonstrate dilation of colon or impaction of feces. Megacolon – 1.5 times the length

of the seventh lumbar vertebra. Pelvic and lumbar spine should be examined for abnormalities causing secondary constipation as well as observation for possible mass lesions causing obstruction. Neurological examination should be carried out. Laboratory results should be elevated from dehydration/metabolic causes.

### Medical Management:

Acute: Correction of dehydration (IVFT), manual enema (administer antibiotics due to risk of bacterial translocation).

Long-term: Diet management (low-residue, modified fibre diets), periodic enemas, stool softeners (lactulose, liquid paraffin – not recommended due to aspiration risk and passes around faeces versus softening) and prokinetic administration (cisapride – effect can be temporary).

**Surgical Management:** Sub-total colectomy +/- removal of ileocaecal junction; total colectomy (ICCJ). ICCJ importance is controversial. Removal can lead to reflux of bacteria and/or a potential increase in diarrhea. Preservation of the ICCJ does not result in increased likelihood of megacolon reoccurrence. Important to take into account the tension/laxity of gastrointestinal mobility when performing colectomy e.g. resection 2cm cranial to pelvic brim.

Complications: Loose stools are expected for 6-8 weeks (time taken for normal motility to be reestablished) – villi height/absorption potential alters. A covered litter tray is recommended. Cats where the ICCJ is removed are likely to have soft stools for 3 months however diarrhea is not typically permanent. Long-term faecal incontinence is low risk.

**Prognosis:** Variable depending on severity at time of diagnosis. Cats with milder signs may be adequately managed with medical therapy but the disease may progress. Reoccurrence rate: 45%. Stricture is a rare outcome.

## Colonic Neoplasia

### Pathophysiology

Dog: Adenocarcinoma, Lymphosarcoma, Gastrointestinal stromal tumours (GIST), Leiomyosarcomas

Cat: Lymphosarcoma, adenocarcinoma, mast cell tumour, neuroendocrine tumour

**Clinical signs:** Anorexia, vomiting, diarrhea, constipation, (haematochezia, tenesmus - more common with rectal mass lesions)

**Diagnosis:** Imaging, histopathology

**Treatment:** Surgical intervention is recommended for non-metastatic, nonlymphomatous lesions. Margins required: 5-8cm. Techniques: i) incision cranial to pubic brim ii) pubic osteotomy iii) transanal pull-through. Adjunctive therapies are often required. Alternative: colonic stent placement

### Prognosis:

Dog: Adenocarcinoma – mean survival time: 6-22 months. GIST – median survival time 7.8 months. Leiomyosarcomas – 37.4 months.

Cat: Adenocarcinoma – median survival time 4-5 months. Chemotherapy required. Lymphoma – variable.

## OTHER

Colonic and Caecocolic Volvulus

**Pathophysiology:** Compromised cranial mesenteric artery, dilatation, congestion and ischemia of the caecum.

**Signalment:** Young-middle aged. Medium-large breed.

**Location:** ICCJ

**Clinical signs:** Various

**Diagnosis:** Radiography reveals marked gaseous distension of caudal intestinal loops.

**Treatment:** Stabilisation. Derotation subsequent to decompression (aspiration of the colonic contents; typically air).

**Prognosis:** Favorable if intestinal viability confirmed (minimal ischaemia).

### Colonic Entrapment

**Pathophysiology:** Secondary to fibrosis/scar formation – typically following ovariohysterectomy. Also reported secondary to rupture of the duodenocolic ligament.

**Clinical signs:** Variable. Dependent on degree of obstruction and/or ischaemia.

**Diagnosis:** Radiography +/- positive contrast colonogram.

**Treatment:** Surgical resection

## Colonic Duplication

**Pathophysiology:** Rare.

Type I – colon/rectum. Type II – in conjunction with other congenital abnormalities

**Classification:** Spherical non-communicating, tubular non-communicating and tubular communicating.

**Clinical signs:** Asymptomatic, faecal retention, tenesmus, rectal prolapse

**Diagnosis:** Radiographs and positive contrast colonogram. Colonoscopy

**Treatment:** Surgery (even if asymptomatic due to malignant transformation). Division of the communicating septum, side – side anastomosis, excision of non-communicating portion.

References available from author on request



8003

# FELINE PERINEAL URETHROSTOMY: FIXING THE FLOW, ONE CAT AT A TIME!

SMALL ANIMAL PROGRAM | SURGERY

 *Lissie Henderson, BVSc (Hons), DipECVS*

## INDICATIONS

Lesion Location – Caudal to Bulbourethral Glands

Repeated episodes of urethral obstruction

- Rule of three episodes – higher recurrence rate with urethral plug
- Failure of medical management
- Financial constraints
- Client compliance: Environmental management
- Treatment of obstruction that cannot be eliminated by catheterization.
- Permanent partial obstruction e.g. stricture formation
- Urethral tear
- Perineal wound
- Perineal, preputial or urethral neoplasm

## PREOPERATIVE CONSIDERATIONS

- Blood Sample - Electrolyte and acid-base abnormalities, Ionized calcium
- Hydration
- Urinary diversion – i) urinary catheterisation ii) cystocentesis

**Top tips:** General anaesthesia, pre-placement bladder decompression (cystocentesis), sacro-coccygeal local anaesthesia, rectal massage/retrograde hydropulsion, lidocaine instillation, muscle relaxant, sterile lubrication, use of weasel wire (0.018" hydrophilic guidewire)

- Plain radiography – urogenital tract (upper and lower) - urolithiasis
- Ultrasound – urolithiasis
- Urinalysis – urolithiasis, infection
- Contrast cystourethrography – positive, retrograde. Normograde can be performed if you are not planning to perform perineal urethrostomy. Exclusion of proximal urethral pathology
- Timing – non-emergent

## SURGERY – GENERAL PRINCIPLES

Gentle tissue handling, Haemostasis (pressure, topical vasoconstrictor), Suture material (monofilament, absorbable versus non-absorbable; no significant difference), Suture pattern (simple continuous; decreased haemorrhage, simple interrupted), Suture size (1.0-1.5 Metric),

## POSITION

Sternal recumbency, hindquarters elevated, anal purse string

Semi-rigid urinary catheter

## SURGICAL PROCEDURE

1. Elliptical incision (prepuce and scrotum), blunt dissection
2. The ventral penile ligament, a fibrous connection between the penile body and pubis, is transected sharply and bluntly elevated.

3. Transect the ischiocavernosus and ischiourethralis muscles at their ischial attachments – palpate intrapelvic space following detachment. The penile body, which is attached to the pelvis by the ventral penile ligament and ischiourethralis and ischiocavernosus muscles, must be completely mobilized to prevent postoperative stricture. Dorsal and lateral dissection cranial to these muscles should be avoided to reduce the risk of fecal or urinary incontinence secondary to pelvic nerve damage.
4. The retractor penis muscle (or its remnant in castrated males) is identified on the dorsal aspect of the penis and excised.
5. Dissection continues cranially and proximally until the paired bulbourethral glands are identified. Adequate dissection should allow the urethra and bulbourethral glands to lie at the level of the skin incision without retracting into the pelvic canal.
6. Incise the dorsal prepuce (the side facing toward the surgeon and the anus) to expose the penile tip
7. The urethra is incised on its dorsal midline with either a scalpel blade or fine scissors, beginning distally and extending proximally to the level of the bulbourethral glands, where the urethral diameter is sufficiently wide – fibrous texture. Extend the skin incision proximally (toward the anus) as needed to widen the opening.
8. The diameter of the urethra is assessed by inserting a 5- to 8-Fr catheter or Halsted mosquito forceps into the opening; the new stoma should easily accommodate the hemostat to the level of the hinge.
9. Preplace the first two sutures from the urethral mucosa to the skin at the 10 and 2 o'clock positions. Take bites of mucosa that are less than one-third of the urethral diameter. To reduce the risk of mucosal tearing, take the mucosal and skin bites separately, pulling the needle through the tissue between bites. A horizontal mattress suture can be used.
10. Insert a hemostat within the urethra while placing the 12 o'clock suture. This improves visualization of the dorsal urethral wall and prevents accidental inclusion of the ventral urethral mucosa.
11. After tying the preplaced sutures, appose the

urethral mucosa to the skin on each side with a simple continuous pattern of rapidly absorbable suture, placing bites 1 to 2mm apart. Continue the appositional pattern until the urethra begins to narrow. In over-weight cats, elliptical pieces of skin and underlying subcutaneous fat can be removed lateral to the finished urethrostomy to evert the stomal edges.

12. Ligate and amputate the distal penile body with absorbable suture before completing the skin closure. The final drain board is usually 1 to 2 cm long.

## ALTERNATIVE POSITIONING

Dorsal recumbency – cystotomy can be performed concurrently, accurate identification of the dorsal surface of the urethra is the most critical

## ALTERNATIVE TECHNIQUE

Modified urethrostomy - anastomosis of the urethra to a remnant of the preputial mucosa. Altered cosmesis and potential reduction in iatrogenic trauma

## POSTOPERATIVE CARE

Diuresis - occurs in 46% of cats following relief of obstruction. Serum potassium concentrations must be monitored to prevent hypokalemia.

Elizabethan collar

Paper/Foam Litter

Petroleum jelly

## POSTOPERATIVE COMPLICATIONS

### Feline lower urinary tract disease (FLUTD) - clinical signs

The disease will still be present, and further medical treatment may be required although re-obstruction is less likely to occur. Cystitis occurs after perineal urethrostomy in 40% of cats with feline lower urinary tract disease; therefore, periodic urinalysis and culture are recommended. Clinical signs may reoccur in cats that form calculi or develop urinary tract infections.

### Haemorrhage

Hemorrhage is reduced by using a continuous pattern, including the mucosa in each suture bite, preventing

self-trauma, and keeping the cat sedated immediately after the procedure.

### Stricture formation

Strictures usually occur within 6 months after surgery and most commonly result from failure to free the penile body from its pelvic attachments, inadequate mucosa to skin apposition or incise the urethra to the level of the bulbourethral glands. They may also occur if urine leaks between the mucosa and skin edges. They can occur secondary to marked inflammation, granulation tissue, indwelling urinary catheters, suture tension, wound infection or self-trauma,

### Periurethral urine extravasation

The urethral mucosa retracts away from the incision edge; it is easy to miss during urethrocuteaneous apposition. Poor mucosal apposition and postoperative swelling may allow urine to travel through gaps in the suture line and into the subcutaneous tissues, increasing postoperative swelling and risk of subsequent stricture. Subcutaneous urine leakage may also occur with catheter-induced urethral lacerations or suture line inversion secondary to anastomotic tension (e.g., short urethra). In cats with a known predisposition, 5 French Foley catheter can be left in place for 2 to 3 days until the surgical site seals. Use of catheters is otherwise not routinely recommended.

### Stoma Dehiscence

Atypical without concurrent urinary/wound infection. In severe cases there may be tissue necrosis leading to sloughing of the perineal skin. Treatment in this case typically warrants a period of temporary urinary diversion and open wound management before definitive revision.

### Urinary incontinence

Many cats have permanent loss of striated urethral sphincter function after this procedure, although incontinence is rare. Urinary incontinence occurs from excessive dorsal dissection around the urethra or too short a length of urethra being retained. However, most cases due to dissection are temporary and resolve within a matter of days.

### Urinary tract infection

Recurring urinary tract infection (UTI) is often seen as a long-term postoperative complication. Perineal

urethrostomy per se does not predispose cats to bacterial UTI, but surgical alteration of the urethral meatus combined with an underlying uropathy may increase the prevalence of ascending bacterial UTI after surgery e.g. underlying FLUTD animals. In addition, the proximity of the stoma to the anus may predispose to a UTI.

### Urinary scald dermatitis

## STRICTURE – REVISION SURGERY

Primary stoma revision - Strictures are corrected by incising carefully around the urethrostomy and dissecting the remaining urethra up to the pelvis, where its attachments are transected. If the urethra cannot be found, a cystotomy is performed simultaneously and the urethra is catheterized antegrade. After urethral attachments are freed ventrally, the urethral opening is widened and sutured as described above. The resulting drain board will be much shorter than the original perineal urethrostomy; however, urine scald does not seem to be a problem in these cats.

Prepubic urethrostomy was initially proposed but is now considered a salvage procedure (used if other techniques have failed). This technique may result in urinary incontinence and urine scalding, likely to be caused by damage to the pelvic nerves when dissecting the pelvic urethra and damage to the internal urethral sphincter. In this technique, the proximal portion of the membranous urethra is directly anastomosed to the skin. The urethra is severed proximal to the pubic bone and tunnelled to the skin through a separate incision. It is important to spatulate the end of the urethra to minimize stricture/stenosis formation. A risk of subcutaneous urinary leakage can cause extensive problematic necrosis.

Subpubic urethrostomy was proposed to alleviate some of the complications associated with prepubic urethrostomy. The adductor muscle and gracilis muscle are reflected to expose the pubis, which is followed by a pubic osteotomy exposing the intrapelvic urethra. It differs from a prepubic urethrostomy as the urethra is exteriorized caudal to the pubic brim. By placing the urethrostomy site caudal to the pubic brim, it minimises damage to the internal urethral sphincter. This reduces the risk of urinary incontinence.

Transpelvic urethrostomy has showed a successful outcome in permanent urinary diversion. This technique

involves a small caudal partial pubic osteotomy. This technique is appropriate for caudal urethral injuries and proposed as a salvage procedure after failure of PU. It does not involve manipulation of the dorsal aspect of the urethra therefore decreases risk of innervation damage associated with perineal urethrostomy. There is a decreased risk of urinary incontinence as the periprostatic urethral

sphincter is less likely to be damaged. Temporary urinary incontinence has been reported in minimal patients. The incontinence was likely to be due to urethritis caused by passing a large urinary catheter during surgery.

References available from author on request



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8004

# LUMPECTOMY 101 – SKIN RECONSTRUCTION – MASS RESECTION

SMALL ANIMAL PROGRAM | SURGERY

 *Lissie Henderson, BVSc (Hons), DipECVS*

## PREOPERATIVE

Patient Factors - Clinical history

Concurrent medications that affect decision to perform surgery - length of surgery, medical conditions that increase risk of wound complications, current medications that increase risk of wound complications

Patient Factors - Location and size – is the mass resectable?

Skin tension, skin quality, mass size, margin requirement, clinical skillset

## Tumour Factors – Diagnosis

- Tumour - Fine needle aspiration (not always representative), Incisional/excisional biopsy
- Node (regional) - Fine needle aspiration, +/- excision at the time of mass removal
- Metastatic Disease (distant) – thoracic/abdominal imaging

## Client Factors

Would the owner elect for wide resection/major surgery if this resulted in a cure?

Would the owner elect for postoperative adjunctive treatment if surgery did not result in a cure? e.g. radiation therapy

**Example One:** Subcutaneous mast cell tumour adjacent to the anus?

**Example Two:** Subcutaneous mast cell tumour on the lateral thigh?

## SURGICAL EXCISION – TYPE

- Intracapsular technique – ‘shelling out’
- Marginal – just outside pseudocapsule
- Local excision – small radial margin (1cm), down to fascial plane
- Wide resection – large radial margin (2-3cm), resection of deep fascial plane
- Radical excision – amputation, injection site sarcoma

## SURGICAL EXCISION – APPROPRIATE MARGIN

### Radial margin

Deep margin - thick fascial planes e.g. muscle, periosteum

- What is the appropriate radial and deep margin for your type of neoplasm?
- Can you achieve BOTH appropriate margins - Do not mix wide radial marginal with local excision (lack of deep margin)
- Primary closure (tension relieving, graft, axial pattern flap) versus open wound management

## SKIN TENSION – ASSESSMENT

High level of Tension (limited skin elasticity) - eye, anus, extremities

Low level of tension - thorax and abdomen (skin plentiful)

- Consider tension lines – close wounds parallel to lines

- Reduction of tension via i) tension relieving suture patterns ii) reconstructive techniques (like flaps or free skin grafts)

## SURGICAL TECHNIQUE

1. Pick up the skin and assess tension lines
2. Clip the hair WIDELY
3. Apply your drapes to the edge of the clip
4. Towel clamps should be placed superficially
5. Separate your surgical kit
6. Draw out your radial margin – objectively measure (2.2cm versus 1.8cm)
7. Sequentially incise your radial margin, avoid 'coning in' and accidentally reducing your radial margin.
  - Sharp incision of the skin, sharp versus blunt dissection of the subcutaneous tissues, mark and incision of your deep margin.
8. Haemostasis
9. Consideration of drain placement – 'seeding of disease and increase of your neoplastic region'
10. Change gloves
11. Change instrumentation
12. Closure

## TENSION RELIEF

Undermining versus walking sutures – cruciate mattress

Reduction of tension when placing first 'holding sutures'  
 i) three twist/throw knot ii) use of towel clamps in muscle/  
 skin iii) surgical assistant iv) muscle relaxant

First sutures ideally placed in region of greatest tension,  
 pause and assess skin reconstruction/direction of 'pull' of  
 walking suture

Simple continuous suture pattern – reserved for superficial  
 layers where tension is minimal

Aim – no tension at skin

## HISTOPATHOLOGY

Provide case history, mark the margins (suture tags; two  
 areas to confirm orientation) +/- use of dye, circumferential  
 suture (apposition of superficial and deep layers)

Confirmation of diagnosis/tumour type

Grade of tumour

Confirmation of complete/incomplete resection

Objective measurement of margin achieved

## ALTERNATIVE PRIMARY CLOSURE TECHNIQUES

Relaxing Incisions

Pedicle Flaps (local) – advancement, rotation/transposition

Axial pattern flaps

Grafts



8005

# WOUND MANAGEMENT: A CASE-BASED LECTURE

SMALL ANIMAL PROGRAM | SURGERY

 *Lissie Henderson, BVSc (Hons), DipECVS*

## CLIENT COMMUNICATION

- Advise clients that the healing process is lengthy, expensive and requires both client commitment and patient cooperation
- Sedation/anaesthesia is required for if there is poor patient cooperation, which can increase expense
- Outpatient treatment – ideal – i) cost ii) nosocomial infections iii) hospital space

## WOUND CARE

1. Clip the hair around the wound after applying sterile lubrication gel to the open wound – WIDE. Repeat on future dressing changes as needed.
2. Clean the skin – aseptic preparation. Repeat at ALL dressing changes
3. Lavage the wound to remove the dirt and debris – Use a non-cytotoxic and isotonic flushing solution. Dilution is the solution to the pollution! Flush at every dressing change
4. +/- mechanical debridement (scalpel blade)
  - Remove foreign material, devitalized tissue, contaminated tissue
5. Ensure appropriate prevention of infection – ascending and initial contamination. Use topical antimicrobials where possible. Use broad spectrum antibiotics and/or antibiotics based on culture/sensitivity results.
6. Use a primary layer that is appropriate to the expected or observed discharge level – foam versus film (foam – high/unknown level of discharge, an alternative dressing - alginate), padding requirements (foam – high likelihood of pressure sores).

7. Dress the wound wherever possible (particularly within a hospital environment) - Ensure your dressing is secure – i) limb dressing ii) adherent dressing (iii) tie-over dressing (be mindful of the damaging surrounding flaps/good quality skin for closure)
8. Monitor – Photos, Objective measurements (ruler – mm/cm; largest width and length). Assess ongoing/clearance of infection – discharge level, cytology (in-house)
9. Frequency of dressing change – dependent on discharge level (strike through of primary layer), requirement of topical treatment (antimicrobial application) - not dependent on patient cooperation, cost, requirement for sedation, day of the week. Frequency of dressing change often can be reduced upon control of infection/degree of discharge.

## DEBRIDEMENT OPTIONS

Surgical Debridement - En bloc, Scrape with scalpel blade  
 Mechanical - Application of adherent dressing (wet-dry)  
 Chemical - Enzymatic/Topical

- Honey
- Hydrogel (care with use on discharging/infected wounds – lack of frequent dressing changes – increased moisture can lead to maceration)
- Hydrocolloid (excellent for high levels of discharge and reduced dressing change frequency)
- Biological – Maggots

## Do not underestimate the viability of skin – preserve wherever possible

- Antimicrobial Options - Antibiotic stewardship, protection of public health, client communication
- Honey – ointment or dressing. Care with maceration of the surrounding skin. Does the honey stay within the wound? Can you make your own impregnated dressing?
- Silver – expensive, cut to size, ensure activation: water for injection
- Hypochlorous acid (Vetericyn spray) – requires frequent use (skin versus open wound – ascending versus active wound infection)
- Gentamicin/Flamazine (compounded) – refrigeration, thin film application
- Antibiotics based on culture

## Common errors with limb dressings

- Application of tape circumferentially and soft tissue injuries (iatrogenic) – micropore, durapore, elastoplast
- Application of primary dressings with adhesive surface circumferentially
- Lack of padding around pressure points – metatarsals/metacarpals (distal), carpal/tarsal joint, calcaneus/olecranon
- Lack of padding/use of a foam dressing on calcaneus/olecranon

## TIMING OF WOUND CLOSURE

	WOUND CLASSIFICATION	TIME OF CLOSURE
Primary closure	Clean/clean contaminated (aseptic surgery)	Immediate
Delayed primary closure	Clean contaminated, contaminated	2-5 days - no granulation tissue
Secondary closure	Contaminated, Dirty	> 5 days - granulation tissue formed
Second intention healing	Unsuitable for closure	Left to heal

- Most wounds where primary closure or delayed primary closure occurs involve en bloc excision of affected tissue and/or clean lacerations.
- Second intention healing in large wounds – often can be managed as outpatients with minimal treatment however, large periods of time.



9001

# A (VERY BRIEF) DENTAL REVIEW FOR PEOPLE IN A HURRY

SMALL ANIMAL PROGRAM | PRACTICE PEARLS

Dr. Jane E. Pegg, DVM, MS, DAVDC, Board Certified Veterinary Dentist®

## REVIEW OF ANATOMICAL STRUCTURES OF THE HEAD, FACE AND MOUTH, AS WELL AS FUNCTIONAL CONSIDERATIONS

- Hard tissues - Maxilla, mandible, orbit, calvarial region, temporomandibular joint, teeth
- Soft Tissues – Gingiva, mucosa, tongue, hard and soft palate
- Tooth anatomy – Enamel, dentin, pulp
- Teeth in the dog and cat, tooth root numbers
- Periodontal anatomy – Gingiva, alveolar bone, periodontal ligament fibres, cementum

## MOST COMMON DENTAL PATHOLOGIES

### Periodontal disease

- Periodontal disease is a complex immunoinflammatory disease caused by bacteria directly and the body fighting those bacteria.
- Biofilm gingivitis attachment loss (recession and periodontal pocketing)
- Extraction becomes treatment of choice when return to health by cleaning only is impossible

### Tooth Fractures

- Fractures are traumatic injuries to teeth that may extend to different layers depending on severity
- Treatment depends on the depth of the fracture and what structures are affected. If pulp is affected, extraction and endodontic treatment are the only options.
- Proactive monitoring is only appropriate for uncomplicated fractures, and includes dental radiography.

### Tooth Resorption

- The animal's own body is breaking down tooth structure secondary to chemical signalling molecules
- Mediated by odontoclasts
- Can be more 'replacement' where tooth is being replaced by bone or more 'inflammatory' where tooth is being destroyed but not replaced
- Extraction or crown amputation is appropriate depending on the type of resorption encountered

### Feline Chronic Gingivostomatitis

- Immune mediated condition causing profound inflammation in the oral cavity of cats
- Hallmark lesion is inflammation in the palatoglossal arch region
- Can be extremely painful,
- Dental extractions (usually full mouth or near full mouth extractions) is the first line treatment.
- Immunomodulatory medications can be helpful as adjunctive treatment

### Contact Mucositis

- Severe immune mediated inflammation +/- ulceration of mucosa (in addition to gingiva)
- Usually adjacent to large teeth, where mucosa 'contacts' a tooth with plaque/calculus
- Following extraction of 'hopeless' teeth, meticulous hygiene is usually required to maintain comfort
- Sometime large scale or full mouth extractions are required for management

References available from the author on request

9003

# MEDICAL & SURGICAL MANAGEMENT OF CANINE HIP DYSPLASIA

SMALL ANIMAL PROGRAM | PRACTICE PEARLS

 Debbie Reynolds BVSc, BSc, DACVS-SA

Hip dysplasia is the most common congenital orthopedic condition of the dog, with cruciate ligament disease the most common cause of pelvic limb lameness. Hip dysplasia causes joint inflammation and secondary osteoarthritis, which ultimately results in variable degrees of clinical discomfort and disability. It is a disease of complex inheritance, meaning that multiple genes, combined with environmental factors, can influence the expression of the condition.

## Etiology and Pathogenesis

The true cause of canine hip dysplasia remains unclear; however, it has been accepted that the disease reflects the interaction of multiple genes with environmental influences. The manifestation of the disease phenotype occurs in genetically predisposed animals exposed to environmental (nongenetic) factors that enhance expression of the genetic weakness. Hip dysplasia is a disease that stems from a "varying degree of laxity of the hip joint, permitting subluxation during early life, giving rise to varying degrees of shallow acetabulum and flattening of the femoral head, finally inevitably leading to osteoarthritis."

## Hip Development

At birth, canine hip joints are normal and they are thought to continue normal development if complete congruity between the femoral head and the acetabulum is maintained. During development of the hip, the earliest dysplastic joint changes are observed at 30 days of age: an edematous ligament of the head of the femur with torn

fibers and capillary hemorrhage at the tearing sites. After the initial 2 weeks, the ligament slowly begins to lengthen, and it has been posited that in dysplastic dogs it is this excessive lengthening that permits lateral subluxation of the adult hip joint. The first radiographic signs of canine hip dysplasia, are subluxation of the femoral head and underdevelopment of the craniodorsal acetabular rim. When subluxation occurs, the articular cartilage is worn and roughened on the dorsal surface of the femoral head at its point of contact with the acetabular rim. Osteoarthritis is a progressive disease and with age will variably lead to clinical signs of pain and disability.

## Genetics

The scientific community has long recognized that canine hip dysplasia has a genetic cause. The slow progress in reducing the incidence of canine hip dysplasia via conventional hip-extended radiographic phenotypic testing has fueled research to develop a genomic test for the disease. Despite intense research efforts, however, progress in this area has been disappointingly slow. In the life span study, all Labrador Retrievers had hip joint laxity, based on the distraction index, indicating susceptibility to osteoarthritis of canine hip dysplasia, and, indeed, 98% of the study dogs developed radiographic or histopathologic evidence of osteoarthritis by the end of life.

## Joint Laxity

Joint laxity, as measured by distraction index, has been shown to be the primary risk factor for the development of hip joint osteoarthritis in all breeds studied. Passive

hip joint laxity, an estimation of functional hip joint laxity, permits subluxation of the femoral head during the gait cycle, resulting in abnormal force distribution across the joint. This leads to premature wear of the articular cartilage and microfractures in the subchondral bone and ultimately progresses to osteophyte formation and osteoarthritis. Laxity measurements by distraction index are predictive of osteoarthritis susceptibility. The risk for osteoarthritis increases as the distraction index increases beyond 0.30, and dogs with a distraction index below 0.30, are not susceptible to developing osteoarthritis, even later in life. Although laxity appears to be a common and well-recognized factor in the development of osteoarthritis, it is unclear what actually causes joint

laxity. Pelvic muscle mass, hormonal factors, weight and growth, nutrition, environmental factors have all been implicated in hip laxity.

## PHYSICAL EXAMINATION

A general physical examination should be performed, both to rule out other disease processes. Orthopedic and neurologic examinations are necessary to localize clinical signs to the hip joint and to eliminate other orthopedic or neurologic conditions that may have a similar presentation to hip dysplasia. Many breeds at high risk for hip dysplasia have a high incidence of other causes of pelvic limb lameness, such as panosteitis, osteochondrosis, or rarely, hypertrophic osteodystrophy in the juvenile dog, and cranial cruciate disease, lumbosacral disease, or neoplasia in the mature dog, it is imperative to accurately localize the clinical signs to avoid incorrect diagnosis.

Dogs with hip dysplasia can have a wide- or narrow-based stance, depending on the stage of disease. When the gait of a dysplastic dog is observed, a hip or spinal sway may be noticed, which is thought to be an attempt to decrease pain by decreasing hip joint excursion. Patients with hip dysplasia have a stiff, short-strided gait, along with shifting weight to the thoracic limbs by extending both the stifle and tarsocrural joints. A bunny-hopping gait, in which the dog uses both pelvic limbs together, particularly when running or when going up stairs, is sometimes seen in puppies with severe hip joint laxity. Range of motion of the hip joint may be decreased and is typically most limited during extension. As the disease advances, crepitus can be palpated during range of motion. In the young dog,

palpation maneuvers may be used to assess joint laxity, including the Bardens, Barlow, and Ortolani tests.

## RADIOGRAPHY

Radiography is the principal diagnostic modality used to detect canine hip dysplasia. Other diagnostic modalities, including ultrasonography, computed tomography (CT), and magnetic resonance imaging (MRI) although show promise, none have been shown to have improved diagnostic or prognostic utility.

### Hip-Extended Radiography (OFA)

The VD hip-extended radiograph has been used to evaluate the canine hip joint since the first case of canine hip dysplasia was reported, with the first hip-extended radiograph of the pelvis published in 1937. By convention, this hip-extended positioning has been accepted and used worldwide for nearly 50 years to screen for canine hip dysplasia.

### Neutral-Position Radiography

The University of Pennsylvania Hip Improvement (PennHIP) program was introduced in 1993 as a clinical hip dysplasia diagnostic tool. It is the most evidence-based hip screening available to date. The distraction radiograph permits quantification of the relative degree of femoral head displacement from the acetabulum by means of a distraction index. The PennHIP procedure can be performed with documented accuracy as early as 16 weeks of age compared with the 2 years minimum recommended age with OFA. Dogs having the tightest hip joints ( $DI < 0.30$ ) have a very low likelihood of developing osteoarthritis of canine hip dysplasia.

## NONSURGICAL MANAGEMENT OF CANINE HIP DYSPLASIA

Medical therapy for the treatment of hip osteoarthritis is largely considered palliative. Current goals of medical management include reducing the clinical signs of pain, improving limb function and potentially changing the joint environment to slow the degenerative process. A multimodal approach to the alleviation of chronic pain, is recommended. This approach can be divided into three

main categories: exercise modification/physical therapy, nutritional, and pharmacologic management.

### **Exercise:**

Encourage leash walks and low impact activities such as walking and swimming. Attempt to avoid high impact activities such as running, jumping, off leash activity and rough housing with other animals or children. Exercise should be done on a daily basis and at a level that does not cause significant pain or lameness and improve muscle mass. Studies have demonstrated that there is a benefit to therapeutic exercise and physical rehabilitation in treating osteoarthritis. If the patient is painful or lame either acutely or within 24 hours of activity the amount of activity should be decreased or the type of activity changed. Reported benefits of regular exercise include increased range of motion, increased cartilage metabolism, weight reduction, and muscle strengthening and pain reduction.

### **Diet:**

It is imperative to keep the patient lean to avoid any extra weight. Excessive weight will put increased stress on their bones and joints and hasten the progression of degenerative joint disease. A body condition score of 4/9 is recommended in patients with orthopaedic disease.

#### Medications:

During episodes of acute lameness and pain we recommend the use of NSAIDs for a period of 2 to 3 weeks to decrease joint inflammation and control the pain. NSAIDs are a relatively safe family of medications however there is the possibility of both short and long term side effects including gastric ulceration, and should be used with caution in kidney and liver disease. The expectation should be that intermittent NSAID use will be required for the remainder of the pets life.

Other analgesics that can be used for longer term management of osteoarthritis that have less side effects. Gabapentin (10 mg/kg every 8 to 12 hours) is used to help modulate pain and can be used chronically with minimal side effects. Amantidine (3-5 mg/kg SID or BID) is an NMDA antagonist that has also been used for longer term pain management with minimal side effects. The most common side effect seen with both of these medications is sedation.

Neutraceuticals: Omega 3 fatty acid supplementation (fish oils - 50 to 100 mg/kg daily) has evidence to suggest that Omega 3 fatty acids play an important role in management of osteoarthritis. Collagen II (Flexadin Advance) is the building block for cartilage and supplementation with UCII may improve cartilage repair and slow the progression of osteoarthritis. UCII can be given with Omega 3 fatty acids but should not be given with glucosamine supplements. Other supplements such as 4Cyte, glucosamine/chondroitin sulfate, green lip muscle extract also may have benefit in the treatment of dogs with osteoarthritis.

Pentosan polysulphite (Cartrophen) and glycosaminoglycans, may assist the body in rebuilding damaged joint cartilage and joint fluid providing anti-inflammatory properties that could be further beneficial for arthritis.

## **SURGICAL MANAGEMENT OF HIP DYSPLASIA**

Surgical management of canine hip dysplasia are prophylactic, salvage, and palliative procedures. Prophylactic procedures, such as juvenile pubic symphysiodesis and pelvic osteotomy, are performed in skeletally immature dogs that do not yet have secondary osteoarthritis. Salvage procedures, such as total hip arthroplasty and femoral head and neck excision, replace or eliminate the source of pain and, in the case of total hip arthroplasty, reliably restores function in a patient that is suffering the clinical consequences of osteoarthritis of the hip joint. Palliative procedures, including pectineal myotomy/myectomy and hip denervation, are relatively uncommon procedures. The options available for any particular dog depend on the age and current status of the hip joint and on the perceptions and the expectations of the owner.

References are available from the author on request.

9004

# KEEPING PATIENTS SAFE: FROM INDIVIDUAL BLAME TO SYSTEMS-BASED SOLUTIONS

SMALL ANIMAL PROGRAM | PRACTICE PEARLS

 Debbie Reynolds BVSc, BSc, DACVS-SA

## THE NEED FOR CULTURE CHANGE

Veterinary professionals take an oath to "do no harm," yet our field lacks structured approaches to error prevention and safety management. Prioritizing safety is fundamental to excellence in patient care. Unlike human healthcare, which has developed accredited programs and national safety networks, the discipline is absent in veterinary. Lessons from human healthcare and other industries that have prioritized safety can be applied to veterinary medicine. Key is recognizing that errors occur within a complex system, and reporting and monitoring are essential to learn where improvements are required.

### Defining Errors and Adverse Events

**Error:** A failure in execution or planning, whether or not harm occurs.

**Adverse Event:** Injury caused by medical management, not the underlying disease.

**Preventable Adverse Event:** Linked to substandard care and may constitute negligence.

**Near Miss:** An error that did not result in harm due to chance or early detection.

These definitions provide clarity in discussing errors. Adverse events are often conflated with "complications." Complications –adverse events that happen during or after a medical intervention - may or may not be preventable or due to negligence.

## CONSEQUENCES OF ADVERSE EVENTS

Adverse events have wide-reaching impacts:

- Patient health: The health and well-being of patients entrusted to our care
- Client trust and reputation: Loss of confidence, social media backlash, and increased scrutiny.
- Legal and regulatory risks: Complaints to regulatory bodies (e.g., CVO), potential litigation.
- Financial consequences: insurance payouts, time devoted to minimizing the impact that could be otherwise spent on generating income, out-of-pocket financial losses.
- Staff wellbeing: The "second victim" phenomenon is the emotional toll felt by clinicians and veterinary staff involved in errors, contributing to burnout, attrition and mental health problems.

## FROM BLAME TO SYSTEMS THINKING

Historically, errors were attributed to individual failings, leading to punitive responses. Modern safety science, however, emphasizes the systems approach, recognizing that fallible, imperfect humans interact with complex environments. The Swiss Cheese Model, developed by James Reason, outlines how multiple system failures can align to produce catastrophic outcomes. In this model, there are layers of defense, each with vulnerabilities that are envisioned as holes. When the holes align, an error passes through all defenses unchecked. The layers are safety protocols, training and skills, equipment design, facility design, etc.

# DEFENSES



Envisioning the cause of errors this way encourages us to examine the broader system instead of blaming the individual, who is a cog in the wheel of the big system. By identifying the layers intended to prevent the error and looking for the holes in the layers, we can make progress preventing similar events. As a simple example, a wrong medication may have been administered because of similar packaging (equipment design), similar medications are adjacent on a shelf (pharmacy design error), the staff did not perform the required double-check protocol because of lack of training (procedural error, training error) and they were fatigued working overtime hours (human factor).

## HUMAN FACTORS AND ERROR CLASSIFICATION

Errors can be classified as “active” and “latent” (James Reason). Active errors are committed by the individual at the point of contact with the patient. Latent errors are the accidents waiting to happen: failures of the organizational design that set up the individual to commit the active error.

Active errors are further classified as mistakes and slips. A slip occurs when the “autopilot” action fails. A slip is a lapse of concentration, often due to fatigue, stress or competing distraction in a busy and chaotic environment. A mistake is an incorrect choice, due to lack of experience, inadequate training or negligence.

Latent errors range from institutional factors (e.g., regulatory pressures) to work environmental factors (e.g., staffing issues) and team factors (e.g., safety culture). A root cause analysis can help uncover some of the latent errors that contribute to active errors. Slips are often mischaracterized as mistakes, resulting in inappropriate responses. More training, more protocols or punishment will not prevent slips caused by latent errors.

Humans are fallible and will inevitably make mistakes, especially when placed in a complex system. Striving for perfection is unrealistic and punishing individuals who err is ineffective. A systems approach anticipates that individuals will make errors and strives to catch them

before they turn into catastrophes. This is more useful than attempting to develop perfect employees. Recognizing that almost all employees come to work intending to do good and are functioning in a complex, imperfect system is a more realistic, humane and constructive mindset.

Human Factors Engineering (HFE) is the application of human factors knowledge to the design and construction of equipment, products, work systems, management systems and tasks. The objective is to provide equipment and systems that reduce the potential for human error and improve safety. Choosing equipment and hospital design with HFE will help minimize errors. Examples: fluid pumps that, once loaded with a line, will not open again until the tubing clamp is applied to the line, prevent inadvertent fluid administration to a patient. This by-passes the need for staff to remember to clamp the line. Equipment that is intuitive and simple to operate likely has been designed with HFE principles.

## STRATEGIES FOR ERROR PREVENTION

### Reduce slips and mistakes:

- Develop and implement clear protocols.
- Use checklists and standardize equipment and procedures.
- Apply Human Factors Engineering (HFE) in facility and equipment design.
- Minimize distractions and reduce hand-offs.
- Introduce constraints and forcing functions (e.g., nasogastric feed tubing that cannot be attached to IV tubing).
- Strengthen training and supervision.
- Differentiate between slips and mistakes to avoid inappropriate disciplinary action.
- Foster a learning environment that supports skill development.

### Build a Safety Culture

A safety culture reflects the institutional beliefs, values and norms that promote patient safety. The concept was developed outside of healthcare with the concept of a “high reliability organization”, which minimizes errors despite a complex or dangerous work environment.

It is a measure of the commitment of the organization/

hospital, from the CEO through to the animal care attendant, to prioritizing patient safety. A hospital with a high safety culture encourages error reporting without fear of punishment, engages all staff levels in safety initiatives, prioritizes frontline perspectives in error analysis and commits to continuous improvement in patient safety.

Leaders and managers have significant influence on the safety culture. When deciding how to act in any situation, staff balance their psychological safety (will I be punished, shamed or praised?), job security (will I be fired?) and moral intuition (what is the right thing to do?). Leaders can nudge behaviour towards a safety culture by rewarding staff for acknowledging and reporting mistakes (both witnessed and their own), providing a non-judgmental forum for discussing errors, and inviting frontline staff to join in the hospital-wide mission of minimizing adverse events in the hospital. To foster a true safety culture, leaders must work to dismantle hierarchical barriers that prevent junior staff from speaking up about patient safety concerns.

### Perform Root Cause Analysis

Root cause analysis is the process of investigating the ‘holes’ in the layers that would have otherwise prevented the error. Perform root cause analysis by gathering information about the event from all who participated. Review the records and invite individuals to share their perspectives. Scrutinize the physical space and environment in which the error occurred. Examine staffing levels, training and other contexts. Identify contributing factors: the individuals involved often have valuable insight. Develop a list of root causes and design appropriate changes to address them. Monitor for resolution.

The author’s hospital has implemented a range of initiatives to embed patient safety into daily practice:

- Communication and hand-off protocols: SBAR techniques
- Medication safety:
  - Call-back verification
  - Double witness system for high-alert drugs
  - Tall man lettering to help differentiate drugs with similar names
  - Electronic flowsheets with dosing alerts
  - Syringe labeling

- Hospital-wide standardization and training of common procedures
- Clinical guidelines for common emergency presentations
- Centralized, easy access to protocols
- TIMEOUT procedures in anesthesia and surgery
- Checklists
- Technology and equipment safety
- Web-based forms for easy staff reporting of errors, including “near miss” errors

## CONCLUSION

Veterinarians and their support staff are immensely caring and compassionate. Almost always errors occur because staff are fallible humans working in complex systems. When errors result in adverse events, the consequences can be devastating for patients, clients, hospital reputation, and the staff member who committed the error. By shifting from a culture of blame to one of systems awareness and continuous improvement, veterinary institutions can better protect patients, support staff, and enhance client trust.

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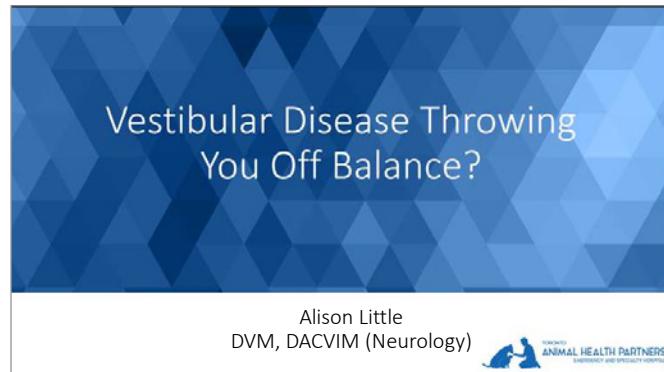


9005

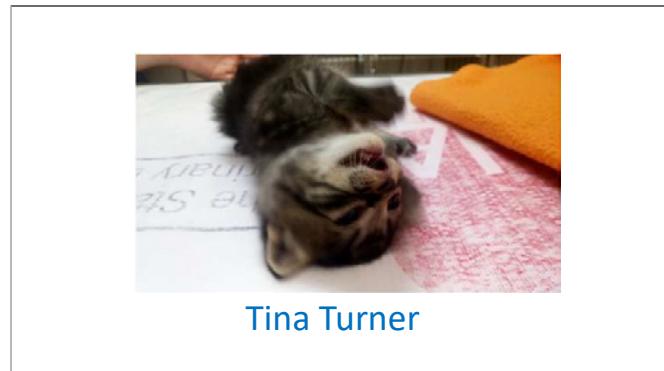
# VESTIBULAR DISEASE THROWING YOU OFF BALANCE?

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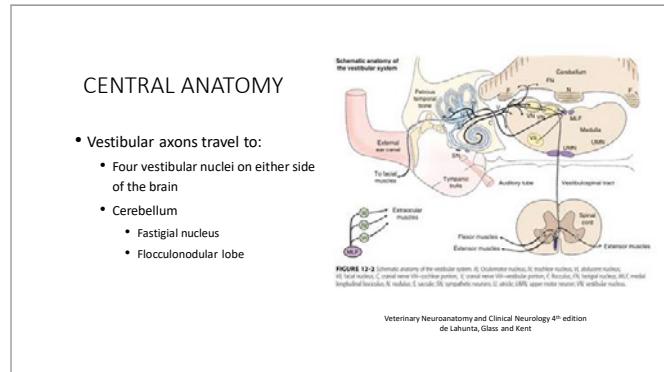
PALison Little, DVM, DACVIM (Neurology)



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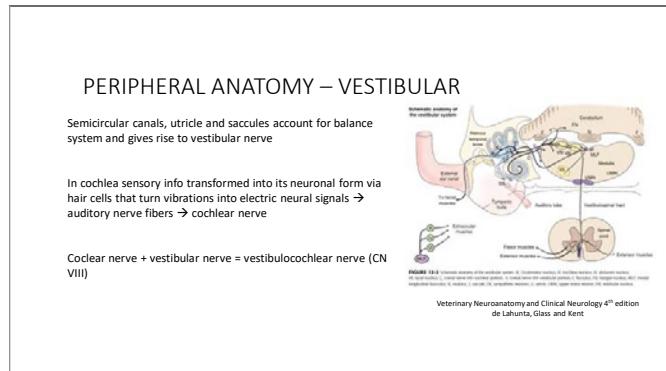
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## Outline

- Neuroanatomy (only two slides, I promise)
- Neurological Examination
- Localizing the Lesion
- Differentials
- Diagnostics

2



4

## NORMAL FUNCTION

- Maintains the animal's position in space
- Coordination of sensory input and reflex output is done subconsciously
- Input from both sides of the head go up to the forebrain for conscious understanding of position, to extraocular muscles and down the spinal cord to stimulate ipsilateral extensor tone

6



## Neurological Exam Findings

- Mentation
- Gait/Posture
- Cranial Nerves
- Postural Reactions
- Spinal Reflexes
- Hyperesthesia



7

## Mentation

- Alert, obtunded, stuporous, comatose
- Disoriented
- Vocal
- Nauseous (hypersalivating, vomiting, etc)
- Anxious



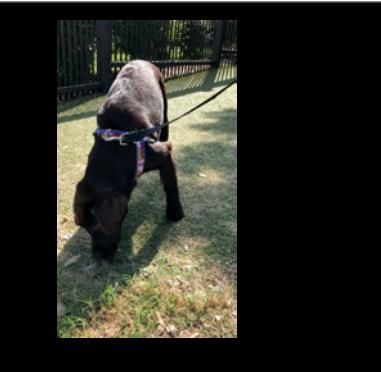
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## Gait/Posture

- Vestibular ataxia
- Head tilt
- Falling/rolling
- Circling?



9



10

## Cranial Nerves - Nystagmus

- Rhythmic, involuntary oscillation of the eyes
- Pendular vs. jerk
- Named by fast-phase direction
- Conjugate vs. disconjugate
- Resting/spontaneous vs. positional



11

## Disconjugate Nystagmus

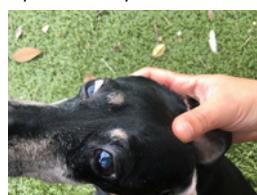
## Positional Nystagmus



13

## Cranial Nerves - Strabismus

- Abnormal position of the eye
- Generally deviation of the eye ventrally and laterally
- Usually on the ipsilateral side
- Positional vs. spontaneous



14

## Postural Reactions

- +/- delayed on the ipsilateral side
- Can be difficult to perform

15

## Spinal Reflexes and Hyperesthesia

- Within normal limits

16

## Localization of Vestibular Disease

- Peripheral
  - Unilateral
  - Bilateral
- Central
  - Ipsilateral
  - Paradoxical



17

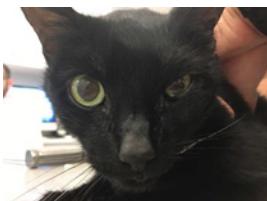
## Peripheral Vestibular Disease

- Mentation: Alert, nausea/vomiting 25%
- Gait/posture: Vestibular ataxia, NO paresis
  - Falling, rolling, head tilt, circling (*ipsilateral*)
- Cranial Nerves:
  - Head tilt (*ipsilateral*)
  - Nystagmus – Fast phase away from the lesion
  - Positional strabismus (*ipsilateral*)
    - +/- Facial nerve paresis/paralysis (*ipsilateral*)
    - +/- Horner's syndrome (*ipsilateral*)
- Postural Reactions: NO proprioceptive deficits
- Spinal Reflexes: Normal
- Hyperesthesia: None

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## Horner's Syndrome

- Sympathetic innervation to the eye travels through the tympanic bulla
- Characterized by:
  - Miosis
  - Ptosis
  - Enophthalmos
  - Prolapsed third eyelid



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## Facial Nerve Paresis/Paralysis



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## Central Vestibular Disease

- Mentation: **+/ - Altered**
- Gait/posture: Vestibular ataxia **+/ - paresis and postural deficits**
- Cranial Nerves:
  - Head tilt (*generally ipsilateral*)
  - Nystagmus – Fast phase usually away from the lesion
    - Vertical and shifting
    - Positional
  - Strabismus
  - Other CN deficits other than CN VII and VIII
- Postural reactions: **+/ - deficits (*ipsilateral*)**
- Spinal Reflexes: Normal
- Hyperesthesia: None

*Lack of centralizing signs on exam makes central disease less likely, but cannot completely rule it out!*

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## Bilateral Vestibular Disease

- Usually peripheral
- Often no nystagmus or head tilt
- Crouched stance, tentative gait
- Wide, side-to-side head excursions



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## Paradoxical Vestibular Disease



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## Paradoxical Vestibular Disease

### • What is it?

- Central lesion localized to the cerebellum causing vestibular signs

### • Clinical Signs

- Head tilts away from lesion
- Nystagmus – fast phase towards the lesion
- Postural deficits present

• **Lesion on the same side as postural deficits!!!!**

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## Diagnostic Tests

### • Otoscopic examination

- Blood work - CBC/chemistry/T4

### • Urinalysis

### • Skull radiographs

- CT or MRI +/- CSF

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## Differential Diagnosis

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## Peripheral Vestibular System

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## Inflammatory/Infectious

- Otitis media/interna is the most common cause of peripheral vestibular disease in dogs and cats (50%)
- Can occur due to:
  - extension of otitis externa across the tympanic membrane
  - extension from the nasopharynx via the Eustachian tube
  - hematogenous spread
- Caused by: allergies, parasites, foreign bodies and tumors
- Most common infectious agents: Staph, Strep, Pseudomonas, Proteus, and Malassezia



## Otitis Diagnosis

### • Physical examination

- Otitis externa
- Pain on palpation of the bulla or opening jaw

### • Otoscopic examination

- Bulging/ruptured tympanic membrane

### • Skull radiographs

- Look for increased opacity in the bulla

### • CT or MRI

- CT shows bony involvement
- MRI shows fluid, cochlear changes and invasion into brainstem

### • Myringotomy – Video otoscope versus by hand (Clinician's Brief step by step)



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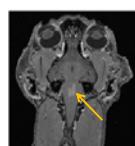
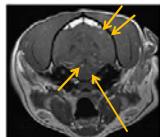


## Central Vestibular System

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### Inflammatory/infectious

- Meningoencephalitis of unknown origin (95% of cases)
- Infectious disease:
  - Canine distemper virus, feline infectious peritonitis, Rocky Mountain spotted fever, ehrlichiosis, Toxoplasmosis, Cryptococcus, Coccidioidomycosis, bacterial meningitis
- MRI +/- CSF
- Bacterial: blood, urine, CSF culture
- NeuroPCR
- 4DX
- Thoracic radiographs
- Treatment:
  - MUO
    - Immunosuppressive prednisone (2 mg/kg/d) tapered over 6-9 months
    - +/- secondary immunosuppressant
  - Infections
    - Doxycycline, clindamycin, fluconazole



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### Vascular disease

- Peracute onset
- May have paradoxical vestibular signs
- Definitive diagnosis via MRI
- Treatment:
  - Supportive care
  - Identify underlying cause of stroke (50%)
    - Abdominal ultrasound, thoracic radiographs, coagulability testing, echo, UPCR



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Tina Turner!!!

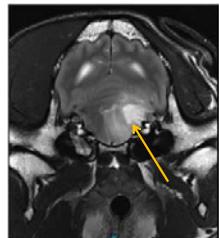


Questions???

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## Neoplasia

- meningioma, choroid plexus tumor, ependymal tumors, lymphoma etc
- Diagnosis: MRI
- Radiation therapy versus palliative therapy
- Palliative therapy with:
  - prednisone 1 mg/kg/day
  - Cerenia



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### Thiamine deficiency

#### • Thiamine deficiency

- Mostly cats
- Unconventional diets (eg, raw food diets, nutritionally incomplete or unbalanced commercial pet foods, or home-prepared diets)
- Clinical signs: Multifocal intracranial (central vestibular, seizures, dullness, ventroflexion)
- Supplement thiamine 100 mg per cat PO q 24



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### Metronidazole Toxicity

- Recent metronidazole (Flagyl ®) administration
  - 50 mg/kg PO BID
- Metronidazole toxicity
  - High dose (>60 mg/kg/day) **or** chronic administration of lower doses
  - Not always dose or duration dependent
- Exact mechanism unknown
  - Proposed: binds post-synaptic GABA receptors
- Treatment is supportive
- Discontinue metronidazole
  - Spontaneous recovery in 1-2 weeks
- Diazepam can hasten recovery
  - 0.25-0.5 mg/kg IV once, then PO for 3-5 days or until symptoms resolve
  - Competes for the GABA receptors

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