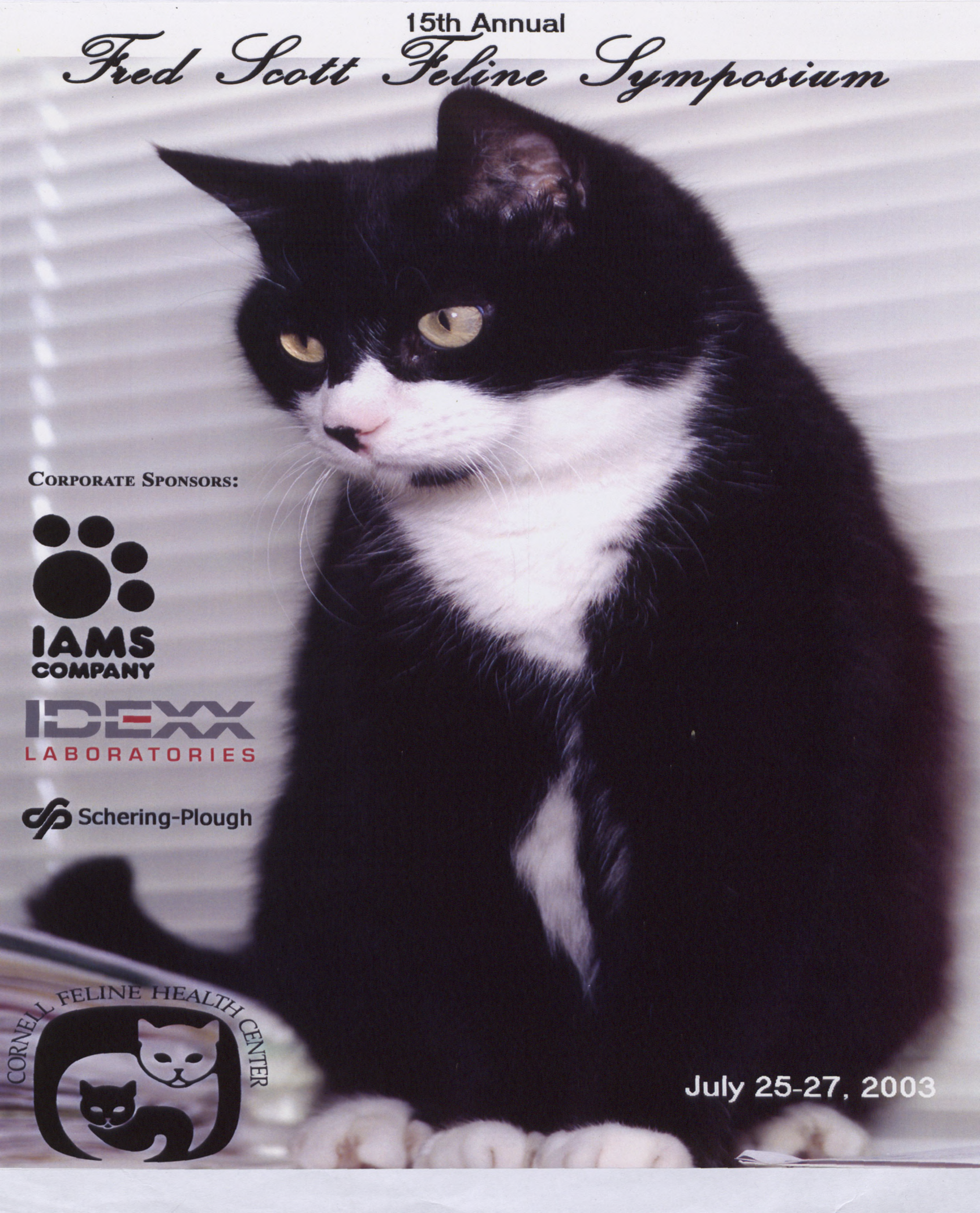
15th Annual

Fred Scott Feline Symposium



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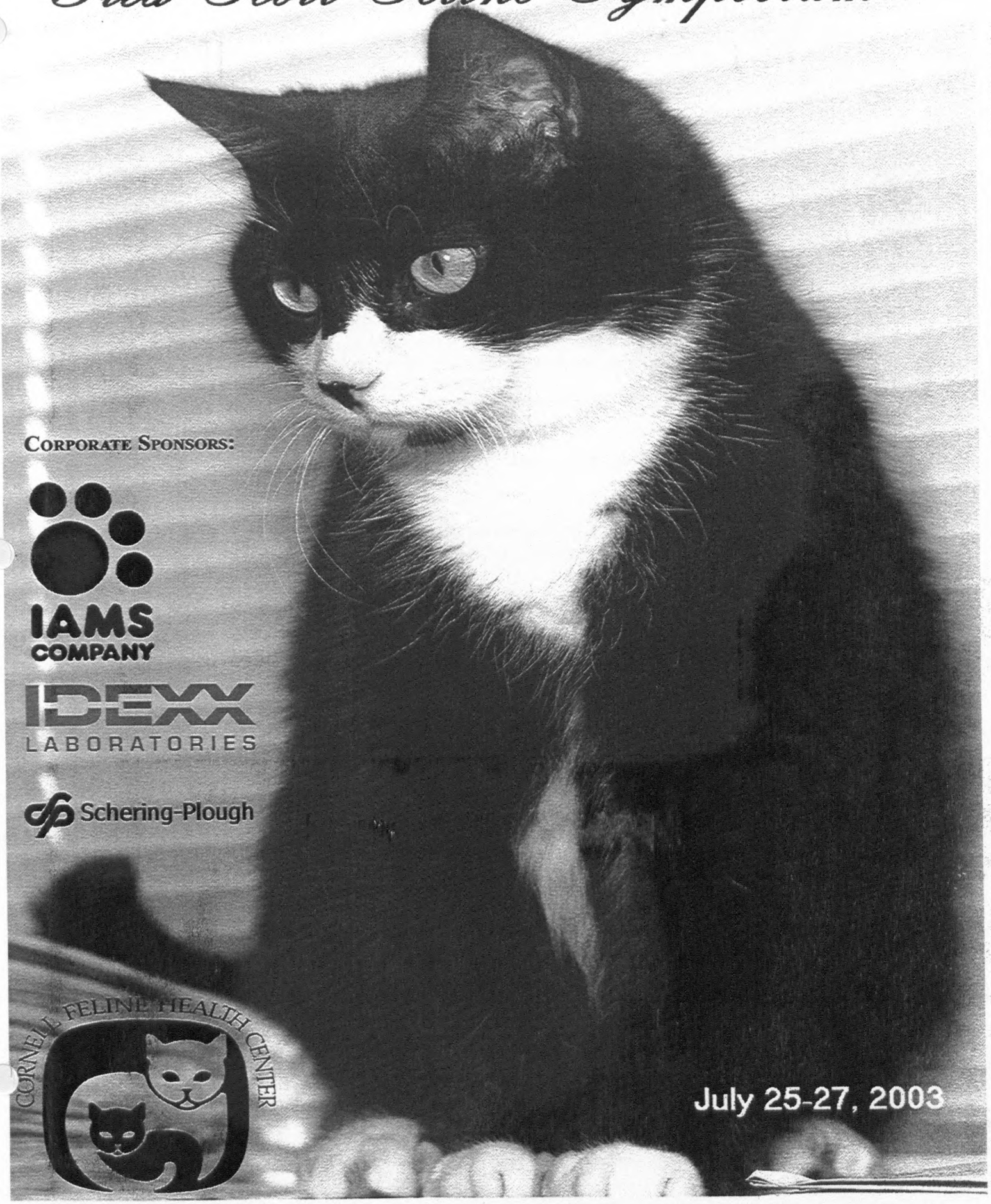
CORNELL FELINE HEALTH CENTER

July 25-27, 2003

15th Annual

Fred Scott Feline Symposium



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CORNELL FELINE HEALTH CENTER

July 25-27, 2003

Information about this conference and other continuing education programs offered by the College of Veterinary Medicine at Cornell University are available by contacting:

Office of Continuing Education
College of Veterinary Medicine
Box 52, T1 002 VRT
Cornell University
Ithaca, NY 14853-6401

Phone	607-253-3200
Fax	607-253-3198
Email	CVMERM@cornell.edu
Website	www.vet.cornell.edu/extension/conedu

Information about the Cornell Feline Health Center at the College of Veterinary Medicine at Cornell University contact:

Cornell Feline Health Center
College of Veterinary Medicine
Box 13, S3 111 Schurman Hall
Cornell University
Ithaca, NY 14853-6401

Phone	607-253-3414
Fax	607-253-3419
Website	www.vet.cornell.edu/public/fhc

General Information and Logistics

15th Annual Fred Scott Feline Symposium
July 25-27, 2003

Course Overview

This year's 15th Annual Fred Scott Feline Symposium will educate and update veterinarians in the latest developments in feline neurology, cardiology, infectious disease, shelter medicine, and the timely and controversial topic of feral cat management. Optional wet labs will give hands-on experience in the latest techniques in laser surgery and dentistry.

Accreditation and Continuing Education Credit

The College of Veterinary Medicine at Cornell University accredits this symposium for a maximum of 17.0 hours of continuing education credit. Each attendee should claim only those hours of credit that he/she actually spends in the educational lectures.
You are asked to sign-in at the registration desk on the first day so that there is evidence of your attendance.

For questions about accreditation and continuing education credit please contact:

Office of Continuing Education	Phone	607-253-3200
College of Veterinary Medicine	Fax	607-253-3198
Box 52, T1 002 VRT	Email	CVMERM@cornell.edu
Cornell University	Website	www.vet.cornell.edu/extension/conedu
Ithaca, NY 14853-6401		

Dental Laboratory and Laser Surgery Lecture and Wet Lab

If you pre-registered for the Dental Laboratory or the Laser Surgery Lecture and Wet Lab you will find, in the back of your nametag, your admission ticket to the appropriate lab.

Evaluation

It is important for the Cornell Office of Continuing Education, faculty, corporate sponsors, and exhibitors to receive your feedback. We ask that you complete the evaluation form and return it to the registration desk before you leave the symposium. The information you provide us is essential in the development of future educational programs. We welcome and encourage your comments on all aspects of this symposium.

Certificate of Participation

You will receive a certificate of participation, which will be available at the registration desk at the conclusion of the symposium. The certificate shows your attendance at the 15th Annual Fred Scott Feline Symposium.

Course Materials

The course materials that are distributed during this symposium are under the auspices of the Office of Continuing Education at the College of Veterinary Medicine at Cornell University. Duplication of these materials is prohibited.

Meals

Meal tickets are in the back of your nametag for:

- Lunch on Friday and Saturday. These lunch meal tickets are to be turned into the cafeteria cashier after you selected your lunch.
- Annual picnic on Friday. This picnic ticket will be collected, by a staff member, at the Lower South Pavilion at Robert H. Treman State Park.

Disclaimer

The lectures offered during this symposium will include some discussion of off-label use and commercial products and/or services. The opinion and recommendations expressed by the faculty are their own.

Agenda

15th Annual Fred Scott Feline Symposium
July 25-27, 2003

Friday, July 25, 2003

7:30 - 8:00 am	Registration Continental Breakfast	Atrium Hagan Room
8:00 - 8:15	Welcome - James Richards, DVM	Lecture Hall I
	Neurology	
	Alexander de Lahunta, DVM, PhD	
8:15 - 9:15	Feline Neurology - Part I	Lecture Hall I
9:15 - 9:30	Break	Atrium
9:30 -10:30	Feline Neurology - Part II	Lecture Hall I
10:30 - 10:45	Break	Atrium
10:45 - 11:45	Feline Neurology - Part III	Lecture Hall I
11:45 -1:15 pm	Lunch	Cafeteria
	Virology:	
	Diane D. Addie, BVMS, PhD, MRCVS	
1:15 - 2:15	Feline Coronavirus: Overview	Lecture Hall I
2:15 - 2:30	Break	Atrium
2:30 - 3:30	Feline Infectious Peritonitis: Diagnostic Workshop	Lecture Hall I
3:30 - 3:45	Break	Atrium
3:45 - 4:45	FCoV / FIP Prevention	Lecture Hall I
6:00 - 9:00	Annual Picnic - Lower South Pavilion	Treman State Park

Saturday, July 26, 2003

	Feral Cats	
	Margaret R. Slater, DVM, PhD	
8:00 - 9:00 am	An Overview of Feral Cat Issues	Lecture Hall I
9:00 - 9:15	Break	Atrium
9:15 - 10:15	Trap, Neuter and Return of Feral Cats: Efficacy and Rationale	Lecture Hall I
10:15 - 10:30	Break	Atrium
10:30 - 11:30	Veterinarians' Involvement in Feral Cat Control	Lecture Hall I
11:30 - 12:30 pm	Lunch	Cafeteria

Shelter and infectious disease issues

12:30 - 1:30	Feral Cats and Wildlife: What Do We Know and What Don't We Know? Jim Tantillo, PhD, MS	Lecture Hall I
1:30 - 1:45	Break	Atrium
1:45 - 2:45	Virulent Systemic Feline Calicivirus: An Emerging Concern Kate Hurley, DVM, MPVM	Lecture Hall I
2:45 - 3:00	Break	Atrium
3:00 - 4:00	Feline Population Medicine: A Herd Health Approach Kate Hurley, DVM, MPVM	Lecture Hall I
4:00 - 4:15	Break	Atrium
4:15 - 5:00	Epidemiology of Upper Respiratory Tract Infections in Cats in Animal Shelters Janet Scarlett, DVM, MPH, PhD	Lecture Hall I

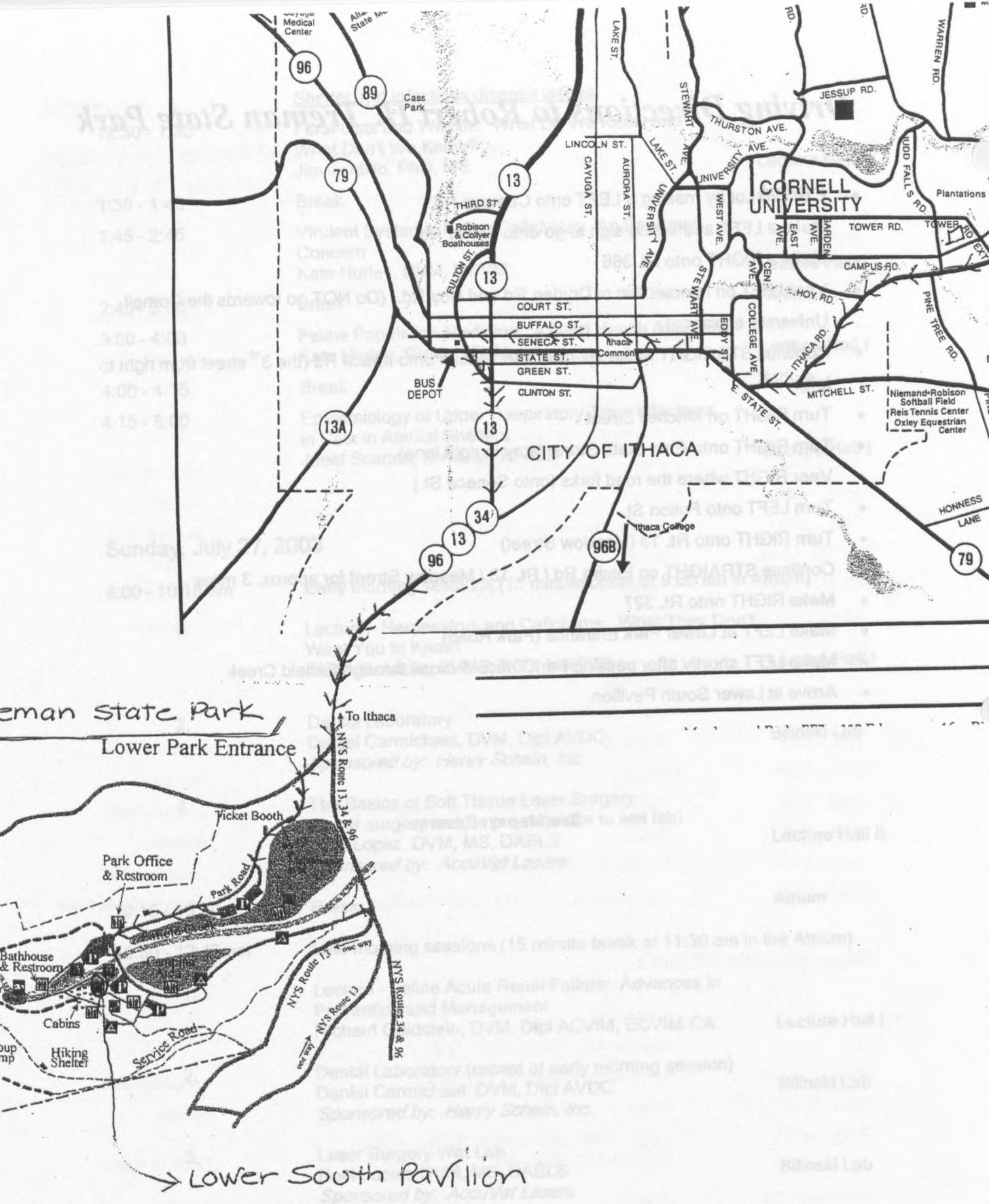
Sunday, July 27, 2003

8:00 - 10:15 am	Early morning sessions (15 minute break at 9:00 am in Atrium)	
1.	Lecture - Herpesvirus and Calicivirus: What They Don't Want You to Know! Diane D. Addie, BVMS, PhD, MRCVS	Lecture Hall I
2.	Dental Laboratory Daniel Carmichael, DVM, Dipl AVDC *Sponsored by: Henry Schein, Inc.*	Bilinski Lab
3.	The Basics of Soft Tissue Laser Surgery (Laser surgery lecture pre-requisite to wet lab) Noel Lopez, DVM, MS, DABLS *Sponsored by: AccuVet Lasers*	Lecture Hall II
10:15 - 10:30	Break	Atrium
10:30 - 12:45 pm	Late morning sessions (15 minute break at 11:30 am in the Atrium)	
1.	Lecture - Feline Acute Renal Failure: Advances in Prevention and Management Richard Goldstein, DVM, Dipl ACVIM, ECVIM-CA	Lecture Hall I
2.	Dental Laboratory (repeat of early morning session) Daniel Carmichael, DVM, Dipl AVDC *Sponsored by: Henry Schein, Inc.*	Bilinski Lab
3.	Laser Surgery Wet Lab Noel Lopez, DVM, MS, DABLS *Sponsored by: AccuVet Lasers*	Bilinski Lab

Driving Directions to Robert H. Treman State Park

- Exit O/B Lot by making a LEFT onto Campus Rd.
- Make a LEFT at the stop sign to go onto Tower Rd.
- Make a RIGHT onto Rt. 366
- Veer LEFT on intersection of Dryden Rd and Hoy Rd. (Do NOT go towards the Cornell University entrance)
- Continue STRAIGHT at the six-road intersection onto Ithaca Rd (the 3rd street from right to left).
- Turn RIGHT on Mitchell Street
- Turn RIGHT onto East State Street (stay in right lane)
- Veer RIGHT where the road forks (onto Seneca St.)
- Turn LEFT onto Fulton St.
- Turn RIGHT onto Rt. 13 (Meadow Street)
- Continue STRAIGHT on Elmira Rd / Rt. 13 / Meadow Street for approx. 3 miles
- Make RIGHT onto Rt. 327
- Make LEFT at Lower Park Entrance (Park Road)
- Make LEFT shortly after passing Park Office & cross through Enfield Creek
- Arrive at Lower South Pavilion

See Map on Reverse


Medical Center
96
89
Cass Park
79
13
LAKE ST.
CAYUGA ST.
AURORA ST.
LINCOLN ST.
STEWART AVE.
THURSTON AVE.
JESSUP RD.
WARREN RD.
UNIVERSITY AVE.
CORNELL UNIVERSITY
Plantations
THIRD ST.
Robison & Collyer Boathouses
FULTON ST.
WEST AVE.
EAST AVE.
GARDEN
TOWER RD.
CAMPUS RD.
HOY RD.
PINE TREE RD.
ITHACA RD.
COURT ST.
BUFFALO ST.
SENECA ST.
STATE ST.
GREEN ST.
CLINTON ST.
Ithaca Commons
BUS DEPOT
COLLEGE AVE
EDDY ST
MITCHELL ST.
E. STATE ST.
Niemand-Robison Softball Field
Reis Tennis Center
Oxley Equestrian Center
13A
CITY OF ITHACA
34
Ithaca College
96B
HONNESS LANE
96
To Ithaca
NYS Route 13, 34 & 96
Lower Park Entrance
Ticket Booth
Park Office & Restroom
Park Road
Bathhouse & Restroom
Cabins
Service Road
Hiking Shelter
NYS Route 13
NYS Routes 34 & 96
one way
Lower South Pavilion

Corporate Sponsors and Exhibitors

15th Annual Fred Scott Feline Symposium
July 25-27, 2003

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Annual Picnic
The wine provided at the Feline Symposium Annual Picnic was generously donated by two local vineyards: Six-Mile Creek Vineyard and Knapp Vineyards. Both vineyards are part of the Fingerlakes Wine Trail.

Exhibitors

AccuVet Lasers

Cornell Feline Health Center

Henry Schein, Inc.

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15th Annual Fred Scott Feline Symposium
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Faculty Disclosure Statement

The faculty presenting at this symposium have disclosed no financial interest or relationship with manufacturers or any of the product(s) or provider(s) of any of the services that may affect the content of their lectures.

Diane D. Addie, BVMS, PhD, MRCVS

Dr. Diane Addie graduated from Glasgow University Veterinary School. After 8 years in small animal practice, she returned to the University to work on feline coronavirus and feline infectious peritonitis (FIP) for her PhD. She is now a lecturer in veterinary virology and adviser to the University of Glasgow Companion Animal Diagnostics laboratory. She hosts a website dedicated to FIP: www.catvirus.com. In 2003 she received the Amoroso Award for outstanding contributions to small animal studies by a non-clinical member of university staff. Her major research interests are FIP, feline chronic gingivostomatitis, kitten mortality and the feline leukocyte antigen.

Contact information for Dr. Addie

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University of Glasgow
Department of Veterinary Pathology
Bearsden, Glasgow, G61 1QH
Glasgow, UK

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Email D.D.Addie@vet.gla.ac.uk

Daniel Carmichael, DVM, FAVD, Dipl AVDC

Dr. Dan Carmichael earned a Doctor of Veterinary Medicine degree from Cornell University in 1990. He then completed a residency program in small animal dentistry, and was granted diplomate status in the American Veterinary Dental College in 1997 (board-certified dental specialist). Dr. Carmichael is a member of the American Veterinary Dental Society, and a Fellow of the Academy of Veterinary Dentistry. He has published scientific papers and lectured extensively on the subject of Veterinary Dentistry. Currently, Dr. Carmichael practices at The Center for Specialized Veterinary Care in Westbury, NY and treats over one-thousand dental cases a year.

Contact information for Dr. Carmichael

The Center for Specialized Veterinary Care
609-5 Cantiague Rock Road
Westbury, NY 11590

Phone 516-420-0000
Email DCarmichael@vetspecialist.com

Alexander deLahunta, DVM, PhD, Dipl ACVIM

Dr. Alexander de Lahunta has been a faculty member of the Department of Anatomy at New York State College of Veterinary Medicine, Cornell University since 1960. He was chairman of the Department of Clinical Sciences from 1977 to 1986, and has been a consultant in clinical neurology to the Teaching hospital since 1963. His research includes the correlation of clinical neurological signs with specific anatomic locations of lesions in the nervous system, establishment of reliable data to differentiate between the various diseases that affect the nervous system in the different species of domestic animals, to recognize and publish new diseases of the nervous system, and to recognize diseases of the nervous system of domestic animals that are models for similar diseases in man.

Contact information for Dr. deLahunta

Department of Clinical and Biomedical Sciences
Cornell College of Veterinary Medicine, Box 18
Cornell University, Ithaca, NY 14853-6401

Phone 607-253-3547
Email ad43@cornell.edu

Richard Goldstein, DVM, Dipl ACVIM, ECVIM-CA

Dr. Richard Goldstein graduated from the Koret School of Veterinary Medicine, The Hebrew University of Jerusalem, Israel in 1993. He completed an internship at the same institution and completed a residency in Small Animal Internal Medicine at the University of California, Davis, in 1998. He spent two years in a private specialty practice in Southern California and one year as a faculty member at the Koret School in Israel, before joining the faculty at Cornell in September of 2001. Currently he is a lecturer of Small Animal Internal Medicine at Cornell. Dr. Goldstein is board certified in Small Animal Internal Medicine by the American College of veterinary internal medicine as well as the European College of Veterinary Internal Medicine - Companion Animals. His clinical and research interests are in small animal nephrology and the effects of renal disease on other body systems.

Contact information for Dr. Goldstein

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Phone 607-253-3060
Fax 607-253-3055
Email rg225@cornell.edu

Kate Hurley, DVM, MPVM

Prior to becoming a veterinarian, Dr. Kate Hurley worked as an adoption counselor, kennel attendant and state humane officer for the Santa Cruz SPCA. After graduation from the UC Davis School of Veterinary Medicine in 1999, Dr. Hurley worked as a shelter veterinarian prior to undertaking a residency in Shelter Medicine through the UC Davis Maddie's Shelter Medicine Program. She is currently in her third year of that program. Her interests include population health and infectious disease epidemiology.

Contact information for Dr. Hurley

Resident, Maddie's Shelter Medicine Program
Center for Companion Animal Health
UC Davis School of Veterinary Medicine

Phone 530-754-2117
Email kfhurley@ucdavis.edu

Noel A. Lopez, DVM, MS, DABLS

Dr. Noel Lopez is a 1988 graduate of Cornell University. He is first author of several scientific journal articles, and has made significant contributions to the veterinary profession in the realms of laboratory diagnostics and laser surgery. He is certified by the American Board of Laser Surgery and promotes the benefits of laser surgery when indicated for pets. He frequently lectures on this subject at National Conferences and wet labs. Utilizing his knowledge, experience and abilities he has compassionately eased the pain and suffering many pets normally experience following surgery. Dr. Lopez is a past president (2002) of the Veterinary Surgical Laser Society and now currently serves on its advisory board. He resides in Sutton, MA with his wife and three children where he owns and operates Boston Road Animal Clinic, a small animal practice that accepts referrals for laser surgery cases. Currently he is authoring a textbook of small animal laser surgery concepts and techniques, and Iowa State Press expects to release the material by the end of this year.

Contact information for Dr. Lopez

Boston Road Animal Clinic
239 Boston Road
Sutton, MA 01590

Phone 508-865-5500
Email drnoel@charter.net

James R. Richards, DVM

Dr. Jim Richards is the current Director of the Cornell Feline Health Center at the Cornell University College of Veterinary Medicine (CUCVM) and has held this position since 1997. In addition, he is the President-Elect of the American Association of Feline Practitioners (AAFP), Chair of the Education/Communication Subgroup, AVMA/AAHA/VCS Vaccine-Associated Feline Sarcoma Task Force, Editor-in-Chief of *CatWatch*, a publication of CUCVM, a faculty advisor for the student chapter of the AAFP, and a faculty advisor for the CUCVM Pet Loss Support Hotline.

Dr. Richards received his DVM from Ohio State University College of Veterinary Medicine in 1979. Prior to receiving his DVM he was a graduate student and teaching associate in the Department of Mathematics at Ohio State University and earned his Bachelor of Arts in Mathematics from Berea College in 1970.

Dr. Richards' current teaching responsibilities, at the Cornell College of Veterinary Medicine, include teaching a section on vaccine efficacy and adverse reactions, a bi-monthly feline health seminar presented to community practice service students, seminars on euthanasia, pet loss support, and feline health topics.

Contact information for Dr. Richards

Cornell Feline Health Center
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Cornell University
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Janet Scarlett, DVM, MPH, PhD

Dr. Jan Scarlett is a companion animal epidemiologist with a research interest in the study of the health of dogs and cats in animal shelters. She co-teaches a course regarding shelter issues and preventive medicine in animal shelters and is the faculty advisor for the student club, Veterinary Students for the Prevention of Cruelty to Animals. Her current research focus is on feline respiratory tract disease in shelter cats.

Contact information for Dr. Scarlett

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Margaret Slater, DVM, PhD

Dr. Margaret Slater began her career on a chicken farm in New Jersey. After obtaining her DVM from Cornell University in 1986, Dr. Slater spent a year in small animal practice before returning to Cornell to complete a doctorate in epidemiology in 1990. She then moved to the College of Veterinary Medicine at Texas A&M University to continue working on health and disease in companion animals, including research on nutrition, cancer and pet overpopulation. Her recent work has focused on the sources, problems and potential solutions for free-roaming cats and dogs in the US and Italy. She has written a chapter entitled: "Understanding and controlling of feral cats in practice", in the 4th edition of John August's Consultations in Feline Internal Medicine. In addition, her book, Community Approaches to Feral Cats: Problems, Alternatives, and Recommendations, was published by the Humane Society Press in 2002. She has been invited to speak at local, regional and national animal welfare meetings and presented a day long session at AVMA on free-roaming cat issues in 2002.

Contact information for Dr. Slater

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College of Veterinary Medicine
Texas A&M University
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James Tantillo, PhD

Dr. Jim Tantillo is a Lecturer in the Department of Natural Resources where he teaches courses in the humanities, particularly environmental history and environmental philosophy. He is a member of the American Philosophical Association and wrote his Ph.D. dissertation at Cornell in environmental ethics on the topic of the morality of sport hunting. Jim is active on the board of the Tompkins County SPCA, and in his spare time he enjoys reading and following his English setter in the autumn woods. Jim is allergic to cats, so he tries to keep his wife's four cats out of the house as much as possible.

Contact information for Dr. Tantillo

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Philadelphia, PA

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Plymouth, MA

Robert Bebko, VMD
Cat Clinic
Pittsburgh, PA

Keith Blackmore, DVM
Paris Hill Cat Hospital
Paris Hill, NY

Louis Borgia
Clarks Summit, PA

William Cadwallader, DVM
Homer and Tully Animal Clinic
Homer, NY

André Charlebois, DVM
Hilltop Mobile Vet Clinic
Watertown, NY

Elizabeth Colleran, DVM
Chico Hospital for Cats
Chico, CA

Ashley Cooper
Haskell Valley Vet Clinic
Portville, NY

Kathryn Dobyns, DVM
Middle River Veterinary Hospital
Verona, VA

Betsy Arnold, DVM
Caring for Cats
Rochester, NY

Lisa Baltes, DVM
Mapleview Animal Hospital
Norwalk, OH

Gerry Beekman, DVM
The Cat Clinic
York, ME

Douglas Boeckh, DVM
Merivale Cat Hospital
Ottawa, Ontario Canada

Catherine Buck, VMD
D. Charles E. London DVM PC
Red Lion, PA

Michael Casale, DVM
Palmyra, NY

Don Coats, DVM
Centreville Veterinary Hospital
Wilmington, DE

Melissa Connolly, DVM
The Visiting Veterinarian
Sherborn, MA

Lisa DeRosa, DVM
East Brook AH
Mansfield Center, CT

Lorri Dring, DVM
Just Cats Health Care
Port Jefferson Station, NY

Ray Dunnigan
Cortland SPCA
Cortland, NY

Janette Ellison, DVM
The Cat Hospital of Fairfax Inc
Fairfax, VA

Lewis Faulkner, DVM
Aliquippa, PA

Charles Fleming, DVM
Cats Exclusively
Pittsford, NY

Vicki Fowler, DVM
Wynantskill, NY

David Hagan, DVM
Waynesboro, PA

Myra Jones, DVM
The Cat Doctor
Indianapolis, IN

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Pittston, PA

Andrew King, DVM
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Sarasota, FL

Lana Kuenzi, DVM
Kuenzi Family Pet Hospital
Waukesha, WI

Gregory Leck, DVM
Bangor, PA

Jeanne Ludlow, VMD
Not Just Cats
Lansdale, PA

James Mac Donald
Red Hook, NY

Blair Ebert, DVM
Housecalls for Cats
Garden City, NY

Claire Ernst, DVM
Paris Hill Cat Hospital
Paris, NY

Carolyn Fisher, DVM
Suburban Animal Hospital
Rochester, NY

Sheila Forbes
Cornell University
Ithaca, NY

Patricia Gawley, DVM
Kirkland, WA

A.L. Hickman, DVM
Cat Hospital of Memphis
Memphis, TN

Carolyn Kadlec, DVM
Seneca Animal Hospital
East Aurora, NY

Lauren Keating, DVM
Natural Bridge Hospital for Animals
Natural Bridge Station, VA

Robin Knox, DVM
Sudbury, MA

Janice Lashmet, DVM
VCA Northside Animal Hospital
Bethlehem, PA

JoAnn Levy, VMD
Clark Animal Care Center
Penfield, NY

Amy Lynn
Cornell University
Ithaca, NY

Amy MacKay, DVM
Nine Lives Cat Clinic
Richmond Heights, OH

Kathleen Makolinski, DVM
SPCA in Cattaraugus County
Orchard Park, NY

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Roslyn Greenvale Vet Group
Greenvale, NY

K. Nasr, DVM
Corona, NY

Bonnie Norton, DVM
Milan Vet Clinic
Athens, PA

John O'Laughlin, DVM
Grace Veterinary Clinic
Mt. Pleasant, PA

Marjorie O'Neill, DVM
Islen Veterinary Hospital
Iselin, NJ

Alice Payton, DVM
Cranbury, NJ

Lisa Pohlman, DVM
Ottawa, Ontario Canada

Robert Reisman, DVM
New York, NY

Jan Rottenberg, DVM
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Rockledge Vet Clinic
Telford, PA

Jeanne Ramsey, DVM
Meadow Ridge Veterinary Clinic
Groton, NY

Anne Richards, DVM
The Cat Doctor
Bedford, MA

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Abbott Road Animal Hospital
Orchard Park, NY

Jennifer Saver, DVM
Catnip & Carrots Vet Hospital
New Hyde Park, NY

Linda Schoenberg, DVM
Just Cats Hospital
South Euclid, OH

Linda Senyshen, DVM
Ottowa, Ontario Canada

Kevin Shimel, DVM
Plainfield Animal Hospital
South Plainfield, NJ

Kelly St Denis, DVM
Bytown Cat Hospital
Ottawa, Ontario Canada

Heather Stewart, DVM
Guildcrest Cat Clinic
Scarborough, Ontario Canada

Marie-Helene Tetreault, DVM
Hospital Du Nord
St. Lambert, Quebec Canada

Carol Tice, DVM
Morrisville, NC

Katherine Trow, VMD
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Ardmore, PA

Daniel Viado, DVM
VCA Staten Island
Staten Island, NY

Rebecca Walley, DVM
Peace Plantation Shelter Medicine
Walton, NY

Teri White, DVM
Community Animal Hospital
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Kinsman, OH

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Haskell Valley Vet Clinic
Portville, NY

Pam Zaidspiner, DVM
Lawrenceville, GA

Arne Zislin, VMD
Fort Dodge Animal Health
Leawood, KS

Susan Shumaker, DVM
Branchport, NY

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Susan Thomas, DVM
The Cat Hospital
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Jean B. Townsend, VMD
Lutherville, MD

Ed Tudor, DVM
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Saipan, MP Northern Mariana Islands

Kathleen Wakefield, DVM
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Penfield, NY

Karen Ward, DVM
Toronto Humane Society
Missago, Ontario Canada

Gry Wildenstein, DVM
Meadowridge Veterinary Clinic
Groton, NY

Jeff Williams, DVM
Jamestown Veterinary Clinic
Kinsman, OH

Debbie Wismer, DVM
Millersville, PA

Karl Zaunbrecher, DVM
Zaunbrecher Vet Clinic
Saranac Lake, NY

Neurologic Gait Evaluation

Alexander deLahunta, DVM, PhD
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Unwilling or Unable - That is the first question to be answered when examining an animal with a gait abnormality. This is especially true when the patient is short-strided or does not support its weight on a limb. A loss of support from a femoral or radial nerve disorder will mimic a severe painful disorder causing reluctance to bear weight.

Patterns - With experience clinicians recognize specific "patterns" in abnormal gaits that suggest the anatomic diagnosis. There are 5 components to these patterns - 2 qualities of paresis and 3 qualities of ataxia.

Paresis and Ataxia - weakness and incoordination - these are synonyms respectively.

Paresis - In neurological terms paresis means deficiency in the generation of the gait or the ability to support weight. This definition covers the 2 qualities of paresis: upper motor neuron and lower motor neuron respectively.

Lower motor neuron (LMN) paresis reflects degrees of difficulty in supporting weight and varies from a slightly shortened stride (easily mistaken for a musculoskeletal lameness) to complete inability to support weight causing collapse of the limb whenever weight is placed on it. Occasionally animals with LMN - neuromuscular -disorders affecting the pelvic limbs will use them simultaneously - bunny hop.

Upper motor neuron (UMN) paresis causes a delay in the onset of protraction - the swing phase of the gait. Usually the stride will be longer than normal. Stiffness - spasticity - may be apparent in the stride. The UMN is comprised of numerous neuronal systems that initiate the gait via LMN recruitment and modulate muscle tone for normal posture and smooth locomotor function. In domestic animals most of these neuronal cell bodies are located in the pons and medulla and their processes descend the spinal cord in the lateral and ventral funiculi. Most lesions affecting these components of the UMN also affect the general proprioceptive sensory system which causes an ataxia (see below) because the involved tracts are adjacent to each other.

Ataxia has 3 qualities reflecting the functional system that is involved - general proprioception, the vestibular system and the cerebellum.

General proprioceptive (GP) ataxia - The GP sensory system has its dendritic zones in specialized receptors in muscles, tendons and joints and is responsible for "informing" the central nervous system (CNS) of the degree of muscle contraction (tone) at any time. It tells the CNS where the animal's parts are "in space" at any instant.
Loss of this system affects the gait by contributing to the delay in the onset of protraction , causing excessive adduction (swing in) or abduction (swing out) of the limb, occasionally overflexion in the swing phase and scuffing- dragging of the hoof and standing on the dorsal aspect of the hoof.

As stated above the GP system and the UMN are affected by the same lesions because of their close proximity and it is difficult but not necessary to differentiate between the UMN and GP signs.
Animals with UMN-GP deficits from a lesion anywhere between C1 and C6 have a tendency to overreach at the end of protraction causing a floating motion to the stride. I refer to this as a UMN-GP deficit because I do not know which system is responsible for preventing this action and that differentiation is not necessary in order to make the segmental anatomic diagnosis.

I can not and do not try to differentiate between conscious (cerebral) and unconscious (cerebellar) GP pathways. If an animal stands on the dorsal aspect of its digits, is this a LMN, UMN, conscious GP, or unconscious GP deficit? I can not differentiate the latter 3 and use other features of the exam to determine if this is LMN.

Vestibular (special proprioceptive) ataxia - This quality of ataxia reflects the loss of orientation of the head with the eyes, trunk and limbs - a loss of balance. Lesions in this system cause the animal to lean, drift, or fall to one side (usually the side of the lesion) .It is usually accompanied by a head tilt to that side and sometimes an abnormal nystagmus.

These same signs result from a lesion in any part of the vestibular system - peripheral or central. The difference is in the other clinical signs exhibited by the animal. A patient with only a vestibular ataxia and a facial paralysis most likely has otitis. These same signs with UMN-GP deficits indicate a pontomedullary lesion.

Cerebellar Ataxia - Classically animals with cerebellar disorders have a dysmetria characterized by sudden bursts of motor activity with marked overflexion on protraction - hypermetria. There are vestibular components in the cerebellum so there is usually some loss of balance and there may be an abnormal nystagmus.
A mild intentional head and neck tremor may also occur.

Notes

15th Annual Fred Scott Feline Symposium
July 25 - 27, 2003

Notes

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The Neurological Examination – deLahunta Version

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Learn to do the neurological examination on a cooperative small breed dog and then you can adapt it to accommodate the size and attitude of other dogs and cats as well as large animals and exotics. When asked how I do a neurological examination on a maniacal aggressive cat or dog my pat answer is --I don't! However you can make many reliable observations by just observing the animal while it is caged as long as you understand what the normal examination is determining.

Why do the neurological examination?

- To determine IF the nervous system is affected in a disease process.
- To establish as accurate an ANATOMIC DIAGNOSIS as possible when the nervous system is affected.

Making the anatomic diagnosis should always precede consideration of the differential diagnosis. Despite the tremendous contribution of imaging to neurological diagnosis and one can only guess what innovations lie ahead in the future, the basic hands on neurological examination is the most valuable cost effective determinant of the clinical diagnosis.

In most cases the anatomic diagnosis is a regional diagnosis and there are essentially 8 regions of the nervous system for consideration.
The PROSENCEPHALON (forebrain) - includes the two cerebrums and the diencephalon which is the most rostral part of the brain stem and is comprised of the thalamus and hypothalamus. The PONS and MEDULLA are usually considered together as they are the source of the upper motor neuron responsible for the generation of the gait. The CEREBELLUM may be the sole anatomic diagnosis or often is considered together with the pons and medulla This region is sometimes referred to as the caudal fossa which is the space these 3 anatomical areas occupy. The spinal cord is divided into 4 anatomical regions: C1 - C5 , C6 - T2 , T3 - L3 , L4 - Cd.
The peripheral nervous system components are usually considered collectively as the NEUROMUSCULAR system, realizing that there are important sensory systems here as well. Any of these 8 areas can be further divided into smaller components but this serves as a starting point in your anatomic diagnosis.

There are 5 components to the neurological examination:

1. SENSORIUM:

The owner is the best observer of any change in the patient's behavior. Significant changes will be obvious but you will often notice subtle changes as you examine the animal. Expressions used to describe these subtle changes include - a vagueness - seems out of touch with reality - is in a world of its own. More acceptable medical terms for the progressive loss of a patient's sensorium are: dullness, lethargy, obtundation , semicoma (stupor) and coma. In my experience the most common site for a focal lesion to cause progressive obtundation or stupor is the diencephalon - presumably from the interruption of the ascending reticular activating system (ARAS) at this level.

2. GAIT:

The most important aspect of the gait examination is to be able to walk the dog on a non-slippery surface. These are often scarce in most hospitals. If there is no convenient built in carpet in the hospital then purchase a reasonable sized indoor-outdoor carpet that can be rolled out for the exam and rolled out of the way afterwards and can be hosed off in your runs after your patient excretes on it - which is a guarantee. This is just as important for evaluating orthopedic lameness cases. A major objective of the gait evaluation is to determine if a lameness is caused by a neuromuscular disorder or an orthopedic problem. Lower motor neuron disease can mimic an orthopedic lameness and is often overlooked as the latter problems are so much more common. As you gain experience you will recognize specific gait. patterns that suggest the anatomic diagnosis ie - the "two engine" dog with short strides in the thoracic limbs and long delayed strides in the pelvic limbs that has a C6-T2 disorder.

The following is an attempt to dissect what it is that you are looking for when you evaluate the gait of a patient with a neurological problem. From a neurological perspective, you are assessing the gait for both paresis and ataxia.

A. Paresis (weakness) can be defined as a deficiency in the generation of the gait or the ability to support weight. There are two qualities of paresis - upper motor neuron and lower motor neuron.
   1. Lower motor neuron (LMN) paresis is seen as an inability to support weight and the patient walks short-strided - "lame". Other signs include a tendency to collapse, trembling, bunny hopping, and neck flexion.
   Thoracic limb support requires neurons in the radial nerve to be intact whereas those in the femoral nerve are necessary for pelvic limb support. Lesions that affect specific peripheral nerves to the limbs excluding the radial and femoral nerves will cause abnormal limb postures but still the ability to support weight.
   2.Upper motor neuron (UMN)paresis is seen as a delay in the onset of protraction (the swing phase) and a longer stride with a variable degree of stiffness - spasticity to the stride. Because the UMN tracts and the general proprioceptive (GP) tracts are adjacent to each other at every level of the spinal cord and caudal brain stem lesions at any of these levels usually will cause dysfunction in both systems therefore the gait reflects both deficits .GP lesions cause an ataxia in which the patient loses awareness of where its limbs are in space. This also may contribute to the delay in the onset of protraction and be the cause of excessive flexion, adduction or abduction of the limb during protraction and the tendency to bear weight on the dorsal aspect of the paw often referred to as "scuffing or knuckling".
   The pattern of gait observed reflects the loss of both of these functional systems and it is not necessary to distinguish between the two systems for your anatomic diagnosis.

   Patients that have C1-5 lesions and are still ambulatory often have a prolonged stride with the limb kept in extension that appears as if the patient is overreaching its landing site. This is especially evident on turns. Although by strict definition this is a form of hypermetria, I avoid calling it that as there is a strong tendency to relate any hypermetria to a cerebellar disorder which this is not. I refer to this overextension as overreaching or floating. Cerebellar hypermetria has a sudden bursting quality to the onset of protraction and an overflexion of the joints as opposed to the overextension seen here.

B. Ataxia is incoordination and comes in three qualities - general proprioceptive, vestibular (special proprioceptive) and cerebellar .
   1. General proprioceptive ataxia represents a loss of awareness of where the limbs are in space and was discussed above with the UMN system which it accompanies.
   2. Vestibular ataxia is a loss of balance reflected in a head tilt, and a tendency to lean, drift, fall or roll to one side. The ataxia is often accompanied by an abnormal nystagmus.
   3. Cerebellar ataxia reflects the inability to modulate the gait generating systems in the brain stem resulting in abnormal "uncontrolled" limb movements that usually are excessively abrupt in onset with an overflexion of the limbs on protraction and abnormal sites of limb placement .These excessive movements are usually referred to as hypermetria. This abnormal gait is usually accompanied by vestibular signs with a loss of balance because there are significant components of the central portions of the vestibular system in the cerebellum.

3. POSTURAL REACTIONS - MUSCLE SIZE

I usually assess these at the same time by standing over the patient with both of us headed in the same direction. In assessing muscle size it is important to try to have the patient bearing the same amount of weight on the two limbs that are being compared. I palpate both thoracic limbs simultaneously from proximal to distal and then flex and extend each for range of motion and as an assessment of muscle tone. When I place the paw back on the floor I place it on its dorsal surface to test for its return to a normal supporting position - the paw placement response. I then move caudally palpating the axial muscles and then palpate and move the pelvic limbs in a manner similar to the thoracic limbs and complete it with the paw placement test

To test the HOPPING RESPONSES I then move back to the thoracic limbs and while still standing over - straddling - the patient , I pick up one thoracic limb and hop the patient laterally on the other limb then I shift limbs and hop it back on the first limb. Only hop the dog laterally on the limb. I keep doing this back and forth shifting limbs when the patient reaches the limit of my stance. I do not move during this procedure and with heavy animals I brace my supporting elbow on my thigh to avoid the strain on my back. The patient does not have to be lifted off the floor for this, only supported so that it is bearing as much weight as possible on the limb being hopped.

To test the hopping responses in the pelvic limbs I stand beside the patient and place my forelimb between the thoracic limbs of the patient so I can lift it up off the floor by its sternum. I then pick up the pelvic limb on the side that I am on and push the dog away from me making it hop on the opposite pelvic limb. I have to change sides to test the other pelvic limb. For heavy animals these hopping responses can be evaluated as you make the patient hemiwalk. I stand beside the patient and pick up both limbs on that side and push the dog away from me. It is important to compare one thoracic limb with the other and one pelvic limb with the other as they are usually faster in the thoracic limbs normally.

These hopping responses essentially test all components involved in voluntary limb movement from sensory receptors in the limb ,to ascending spinal cord tracts , to medullary relay proprioceptive nuclei ,to thalamic relay nuclei to the thalamocortical pathways , to the internal capsule ,to the sensory cortex and the return of the UMN pathways .The latter begin in projection neurons in the adjacent motor cortex , pass into the internal capsule and crus cerebri ,to descending UMN pontomedullary systems, into the spinal cord in pyramial and extrapyramidal UMN tracts ,to the ventral grey column LMN ,to the muscles in the limb. One might conclude that this is fairly non-specific!! Correct - so why is it so useful? -Because first -it tells you if there is an abnormality somewhere in the nervous system and therefore is a reliable screening test. Second -its importance in localizing lesions is dependent on what else is abnormal. A patient with a normal gait in the environment of your examination that has hopping deficits on one side most likely has a contralateral prosencephalic lesion. This is a very common relationship and may be the only indication of a prosencephalic lesion. This is one of only 3 tests that you can use in your neurologic examination to determine if a prosencephalic lesion is present and on which side. If you have a patient with a head tilt and a mild loss of balance to one side but otherwise has a normal gait and there is a hopping deficit then if the lesion is focal it is central at the medulla and not in the inner ear. Postural reactions are normal with inner ear - peripheral vestibular system - disorders.

Misleading neurologic description: It is important to be clear and precise with your neurologic descriptions. What does it mean when you read a description in a published case study that describes an 8-year-old Beagle dog as having a right hemiparesis? If the author means that this dog has a normal gait but a right side postural reaction deficit then I would make an anatomic diagnosis of most likely a left prosencephalic lesion. If the author means this dog has a right sided gait deficit with a delay in protraction of the right limbs with spasticity and a tendency to float with the right thoracic limb then my anatomic diagnosis is right C1-C6.

It is important to remember that animals with neuromuscular disorders that still have voluntary movements will hop fast as long as their weight is held up because their proprioception is normal. This observation may help you distinguish between a subtle LMN and UMN paresis.

There are many other postural reactions that can be tested but in my experience the hopping responses are the most reliable and they are all that I routinely perform in this examination.

Many clinicians rely solely on what they call the CP - conscious proprioception response. This is an incorrect term as this tests more than just conscious proprioception. The late Ralph Kitchell published a paper in which he made a point of this common mistake describing that in this test there are somatic afferents that are responding to light touch and pressure in addition to the general proprioceptive neurons. In reality the failure to return the paw to its normal position can be caused by a LMN denervation of the digital extensors, an UMN paresis , or a loss of any of the sensory innervation just described. In addition to this lack of specificity it is my experience that there are some normal patients that when their paw is placed on its dorsal surface they will continue to stand on it until you make them move. This paw placement test should not be relied on in the absence of testing the hopping responses.

RECUMBENT ANIMALS: It is very important in evaluating these patients to pick them up and hold them in a standing position. Get help if it is a heavy patient. By holding them in this position and lifting them up and down you can determine the quality of muscle tone ie. whether they have a flaccid or spastic paralysis as well as determine if any voluntary movements can be elicited. If there are voluntary movements, while still supporting them you can determine the presence and quality of the hopping response.

4. SPINAL REFLEXES - MUSCLE TONE

Ideally these spinal reflexes and muscle tone will be diminished to absent in LMN disorders and increased in UMN disease. The degree of hypertonia that results from UMN disease will be determined by the amount the lesion interferes with the upper motor neurons that are inhibitory to extensor motor neurons. It is important to evaluate the tone and spinal reflexes together with the gait abnormality. Dogs can exhibit profound neuromuscular paresis with myasthenia gravis and still have normal tone and reflexes. Similarly some dogs with T3-L3 lesions often have normal muscle tone and reflexes

For evaluating the spinal reflexes the patient should be placed in lateral recumbency and be as relaxed as possible. The limbs can be flexed and extended to assess the degree of muscle tone that is present.

The only reliable tendon reflex in my experience and the only one that I routinely test is the PATELLAR REFLEX. Holding the stifle in partial flexion the patellar ligament is struck lightly with a hard object. The human pediatric patellar hammer is the best size for our small animals. Both the sensory and motor components of this reflex are contained in the femoral nerves and their components in the L4, 5 and 6 spinal nerves, roots and segments.
If you do not get this reflex in either the recumbent limb or non-recumbent limb do not consider it absent until you can not get it in the other position. For some reason that I do not know, this reflex is occasionally absent on either the recumbent or the non recumbent side. You only need to get it once to know it is intact. If the patient will not relax you may not be able to elicit this reflex. It is my experience that the other tendon reflexes are not consistently present in normal dogs and I do not routinely test them.

The WITHDRAWAL (FLEXOR) REFLEX is done on both limbs by squeezing a digit with enough pressure to elicit the reflex and a conscious response in a normal patient. Sometimes your digital pressure may be enough .Otherwise use a pair of forceps on the base of the toenail adding enough pressure to get the response or not get it if there is a lesion Remember that you can have a reflex loss without loss of nociception so you must use care in the amount of pressure you apply to avoid excessive discomfort to the patient and your injury by the patient. !! This is a more complex reflex. The sensory neurons tested depend on the digit being tested or the autonomous zone that you select for this stimulus and the motor response involves primarily the sciatic nerve in the pelvic limb (stifle flexion) and its branches, the tibial nerve (digital flexion) and peroneal nerve (tarsal flexion) . Beware that the hip flexion that results involves the femoral nerve and most all the ventral branches of the lumbar spinal nerves to the psoas major muscle. An animal with a complete sciatic nerve lesion can flex the hip when the medial aspect of the paw is stimulated (saphenous nerve - sensory branch of the femoral nerve). The segments of spinal cord, roots and spinal nerve ventral branches involved with the sciatic nerve are L6 L7 and S1.

In the thoracic limb there are multiple nerves involved with the withdrawal reflex thus it is a crude test of the entire brachial plexus and cervical intumescence. The sensory nerve or nerves tested depend on the autonomous or cutaneous zones selected.

Squeezing the base of the 2nd or 3rd digital nail stimulates the sensory components of the radial nerve dorsally and the median and ulnar nerves on the palmar aspect. The motor neurons involved are in the axillary nerve (shoulder flexion) musculocutaneous nerve (elbow flexion) and median and ulnar nerves (digital flexion) . Both the sensory and motor neurons that are involved are associated with the C6 to T2 spinal cord segments - the cervical intumescence.

These flexor responses only require the peripheral nerves and the segments of spinal cord where synapses occur between the afferent and efferent components. A transverse lesion in the spinal cord cranial to these segments that isolates the segments from the rest of the CNS will not cause a loss of these reflexes. They can persist independent of the rest of the CNS.

NOCICEPTION

By increasing the amount of pressure on the digit the stimulus becomes a noxious one and in the normal animal will elicit a conscious response. This response is the patient's manifestation of pain.

As an anatomist I try to strictly adhere to the approved nomenclature to avoid ambiguity. This is published in the Nomina Anatomica Veterinaria. No such Bible exists for medical terminology and is sorely needed. Therefore I appreciate it when I am corrected for an improper use of terminology. In my textbook I refer to this noxious stimulus as the pain stimulus which is incorrect. Once again, my neuroanatomical critic Ralph Kitchell pointed out to me the error of my ways and I applaud him for that. Pain is not a sensory modality. Pain is the subjective response of the patient to a noxious stimulus and varies between individual patients and is dependent on many other factors surrounding the origin of the noxious stimulus. We should all adhere to this terminology!! Having clarified that - the conscious perception of the noxious stimulus known as nociception is primarily at the level of the sensory (somesthetic) neocortex in the area of the postcruciate gyrus . To reach this level the entire pathway from the intumescence involved with receiving the noxious stimulus to this sensory cortex must be intact. In general when the afferents that have been stimulated by the noxious event enter the spinal cord dorsal grey column, they synapse on projection neurons there. Most of these will cross to form an ascending pathway in the oppsite lateral funiculus but some will form a similar pathway on the same side as the source of the stimulus In reality there are nociceptive pathways in all funiculi. However there are enough that are contralateral that in a cooperative patient with a prosencephalic lesion involving this pathway or the sensory neocortex there will be a degree of hypalgesia in the limbs on the opposite side. Only a transverse spinal cord lesion cranial to the intumescence involved will produce analgesia. Such a lesion in the cervical spinal cord is usually lethal due to the interruption of UMN respiratory tracts. Recognizing a hypalgesia in the limbs and trunk on one side in a patient with a normal gait is one of the 3 tests used to localize a prosencephalic lesion. It is easier to appreciate in the nasal mucosa which will be described with the cranial nerve part of this examination.

Because there is so much variation between animals in their response to noxious stimuli I do not believe I can reliably recognize the difference between the response to a mild and more severe noxious stimulus - referred to incorrectly as superficial and deep pain. Even if I could, I am not convinced it contributes to my ability to make the anatomic diagnosis.

Obviously the presence or absence of nociception with severe transverse thoracolumbar spinal cord lesions is important for prognosis as well as to specifically locate the site of the transverse lesion.

There is one more reflex that I usually test and always test when I am concerned about a possible transverse T3-L3 lesion in the spinal cord .This is the cutaneous trunci reflex - which I have incorrectly called the panniculus reflex. The sensory neurons stimulated by lightly squeezing or poking the skin over the epaxial muscles of the thoracolumbar vertebrae are contained in the dorsal branches of the spinal nerves innervating the skin at about the level of your stimulus. Synapse occurs in the spinal cord dorsal grey column on long interneurons that then project cranially in the fasciculus proprius . These interneurons terminate on LMN cell bodies in the ventral grey column at C8 and T1

which in turn enter the lateral thoracic nerve that innervates the cutaneous trunci muscle causing the skin to twitch. Rarely this reflex can not be elicited in a normal dog. Starting at the L7 region and stimulating the skin over each successive vertebra the reflex in most animals does not start to about the midlumbar level but there are many individual variations here. In patients with complete transverse T3-L3 lesions ,this reflex will be absent caudal to the lesion - and more specifically about 2 spinal cord segments caudal to the lesion because of the normal short caudal course of the dorsal branches after they leave the spinal nerve.
This reflex will also be absent with lesions that affect the lateral thoracic nerve or its origin from the C8 and T1 spinal nerves ie. avulsion of the roots of the brachial plexus, nerve sheath neoplasms of these spinal nerves.

The tail should be moved to assess the tone of its muscles and the anal tone should be determined. The PERINEAL REFLEX can be performed by mild digital pressure on the anus or with the blunt end of closed forceps or by squeezing the anal or adjacent perineal skin with forceps and observing contraction of the anal sphincter and tail flexion. The degree of stimulus can be gauged to avoid upsetting the patient when this innervation is still intact. This reflex is dependent on the sacral segments and their spinal nerves and the branches of the pudendal nerves. The tail response is dependent on the caudal segments and nerves. LMN bladder dysfunction is often indirectly assessed by loss of the perineal reflex because of similar involvement of sacral segments and the proximal sacral spinal nerves.

5. CRANIAL NERVES

The cranial nerve exam should be done when the patient is the most relaxed. With very young animals this is often before you handle them at all .In most instances with these young animals - the less restraint the better. For larger patients I prefer to do this cranial nerve exam while standing over the patient as I have been for the postural reactions. For small dogs and all cats I prefer to sit on the floor with my back against the wall - all very comfortable - flex my knees and place the patient with its back lying on my thighs. It is very easy to control the patient this way and especially its head that you are going to examine. Aggressive cats can be rolled in a towel before placing them in this position.
The cranial nerve exam can be done "by the numbers" or by region. I much prefer the latter. Either part or all of cranial nerves II thru VIII are evaluated in the region of the eyes.

MENACE - VISION - PUPILS

I always start with the menace response and cover one eye as I menace the other. This is a learned response and may not occur until 10 to 12 weeks in puppies and kittens in which case I have to use their ability to follow objects moving in their environment to assess vision . Anatomically this is a II - central visual pathway - VII response. The majority of the central visual pathway is contralateral to the eye being menaced. Some normal animals need a mild stimulus to get a response. I usually tap their orbital region with my hand a couple of times before I do the menace. Then be sure you are not too close with your menacing hand so that you avoid long vibrissae or a sudden air movement that stimulate sensory components of cranial nerve V. If I do not get a response then I immediately touch the eyelids and look for the palpebral response to be sure the facial nerve is functioning .If it is not, then I have to look for eyeball retraction or a head movement as a response to the menace if the patient is visual. Occasionally it is necessary to set up a maze of objects in the animal's environment to see if they can avoid the objects when walking around them.

Immediately following the menace test, the pupil size and response to light should be examined. Some pupil size can be seen in room light - I love cats that have a yellow iris. Most patients have a dark iris which will require some additional light to see the borders of the iris. Hold your pen light on the midline over the nose to give each eye the same amount of light to look at pupillary size and determine if any anisocoria is present. Then place the light source as close to the eye as possible and if no response occurs move the light around the fundus to be sure all areas are stimulated. After observing this in one eye quickly swing the light into the other eye, observe that eye's response and then swing the light source back to the first eye and keep repeating this. In the normal patient the pupil will constrict rapidly (depending on the species - this is always slow in horses) and as you move the light source from one eye to the other the pupils in both eyes will stay constricted.

This is how I observe the indirect or consensual response rather than try to see the response in the opposite eye while I hold the light in the stimulated eye. When I am teaching, writing examinations or publications I never use the terms direct response (eye stimulated) or indirect -consensual (the other eye) as these terms can be confusing unless you are very careful in your description and in many publications this care is absent. Avoid this confusion by indicating that when the light is directed into OS what happens to the pupil in OS and what happens in OD and do the same for the light directed into OD.

This light reflex is mediated thru the rostral brain stem. The retinal ganglion layer neurons involved with this reflex in each optic nerve presumably are directed at the chiasm either into the opposite optic tract (about 75% dog, 65% cat) and the remainder enter the ipsilateral optic tract. These light reflex processes pass over the lateral geniculate nucleus and enter the dorsal thalamus to synapse on neurons in the pretectal nucleus on that side. The majority of these pretectal neurons project thru the caudal commissure to terminate in the oculomotor nucleus on the opposite side of the rostral mesencephalon.

Based on this anatomy, light directed into one eye will have a greater influence on the ipsilateral oculomotor nucleus and the

response in the stimulated eye may be more rapid and complete than the indirect response in the opposite eye. This is not always obvious in your examination. You would also expect that a lesion limited to one optic tract would cause a decreased response when the contralateral eye was stimulated but this too may be difficult to appreciate.

Examples:

If the menace response is absent in one eye with a normal palpebral response and pupils are normal and equal in size and have normal pupillary light reflexes then the lesion causing the unilateral menace deficit is most likely in the contralateral optic tract, lateral geniculate nucleus, thalamocortical fibers, optic radiation part of the internal capsule or visual neocortex primarily in the occipital lobe. This is the central visual pathway for perception. In the dog about 75% of the pathway is contralateral to the eye menaced after the optic chiasm and about 25% remains ipsilateral. Therefore lesions in this central visual pathway on one side cause a 75% loss of vision in the contralateral eye and 25% loss in the ipsilateral eye but the owners rarely recognize this deficit. The menace test can only determine the contralateral 75% deficit and it is fairly reliable. Even though the contralateral optic tract contains the majority of the pupillary light reflex fibers - assuming that their portion that cross in the optic chiasm is similar to the visual perception pathway - there usually will be no recognizable loss of pupillary light response in the eye tested. This menace test is one of the 3 examinations to determine structural disorders in the prosencephalon

A patient has no menace response OS with a normal palpebral reflex. There is no anisocoria. Light directed into OS causes no response OU. Light directed into OD causes a normal response OU. As you swing the light from OD, where the pupil constricted, back to OS, the OS pupil which was constricted from the OD stimulation is now dilating back to its original size. This asymmetry is repeated as you swing the light back and forth between the two eyes. When you cover OD with your hand the OS pupil dilates to its full extent. Where is the lesion? Answer: In OS or the left optic nerve. Most of the time with these lesions there is enough room light entering the normal eye to keep the pupil in the abnormal eye constricted. Occasionally it will be slightly larger than the normal pupil in room light.

A patient has normal menace responses. The pupil OD is widely dilated. Light in OD only causes the pupil to constrict in OS. Light in OS only causes the pupil to constrict OS.

Where is the lesion?

Answer: Right oculomotor nerve - parasympathetic visceral efferent fibers, or ciliary ganglion or its ciliary nerve branches. Beware of this as the first sign of an extramedullary mass lesion ventral to the diencephalon compressing the oculomotor nerve with loss of the preganglionic parasympathetic function before the somatic efferent neurons to extraocular muscles are affected.

A patient has no menace OD with a normal palpebral reflex.

The OD pupil is widely dilated. Light directed into OD causes no response OU. Light directed into OS causes only the OS pupil to constrict.

Where is the lesion?

Answer: Right optic and oculomotor parasympathetic visceral efferent fibers or the ciliary ganglion or its ciliary nerve branches. A retrobulbar tumor or abscess could do this.

A patient is blind OU - no menace OU - with normal palpebral reflexes. In room light the pupils are mildly dilated. Light directed into OS causes the pupils to constrict OU .Light directed into OD causes the pupils to constrict OU.
Where is the lesion? Answer BOTH eyeballs, optic nerves optic chiasm or optic tracts. The two most common disorders that cause these specific signs are a retinal degeneration (SARDS-sudden acquired retinal degeneration) and optic neuritis.
From my clinical experience it appears that animals with lesions in the sites just described can lose their visual perception and be clinically blind but still have light responsive pupils when a bright light is directed into the eyes . However in room light there is insufficient light to permit normal constriction. This may reflect that the disease processes involved tend to spare the pupillary light reflex neurons in cranial nerve II or more likely to lose the light reflex completely it is necessary to interfere with the function of all of these neurons whereas vision is lost after a certain threshold percentage of retinal ganglion layer neurons are dysfunctional. In other words the pupillary light reflex neurons are the last to go when lesions disrupt the retina or optic nerve.

Anisocoria can result from many intraocular disorders. Iris atrophy is fairly common in older animals and creates dilated unresponsive pupils with no interference with vision. Neurological causes of anisocoria include disturbances to cranial nerves II, III and the sympathetic ocular innervation.
Complete sympathetic paralysis of the head (Horner's syndrome) causes a miosis, smaller palpebral fissure and a protuded third eyelid. Facial hyperthermia and decreased nasal air flow on the affected side are very difficult to appreciate in small animals. This sympathetic paralysis most commonly involves some component of the pre or postganglionic sympathetic LMN .In very acute severe C1-C8 spinal cord lesions an UMN Horner's syndrome may occur. This is most commonly seen in hemiplegic dogs associated with ischemia or infarction caused by fibrocartilaginous emboli. A persistently miotic pupil in a small animal with the signs of an avulsion of the components of the brachial plexus localizes the injury to the level of the roots or spinal nerves at the vertebral column.
It is important to remember that in general the size of the pupils represents a balance between the amount of light entering the eye and stimulating the oculomotor neurons that innervate the iris constrictor muscle and the emotional state of the patient which influences the sympathetic innervation of the iris dilator muscle.

STRABISMUS

While examining the eyes you can appreciate whether they are normally positioned in the orbits. Abnormal eye positions reflect a lack of innervation of the extraocular muscles or a disorder with the vestibular system. The latter is most common and the vestibular strabismus only occurs in some positions of the head. Somatic efferent neurons in the oculomotor nerve prevent a lateral and slightly ventral strabismus. The abducent neurons prevent a medial strabismus. The trochlear neurons prevent an excessive extorsion of the eye which can only be seen in the cat with the lateral positioning of the dorsal aspect of its vertical pupil. In the dog you would have to do a fundic exam and look at the position of the normally vertical superior vein at the optic disc.

A quick assessment of the function of the oculomotor nerve innervation to the medial rectus muscle and the abducent nerve innervation to the lateral rectus is to test for normal physiologic nystagmus by moving the head side to side. As you move the head to the right both eyes will move abruptly - jerk in that direction which tests the abducent nerve in the right eye and the oculomotor nerve in the left eye as both eyes will jerk together. On moving the head back to the left the opposite nerves will be tested. The stimulus for this response is the movement of fluid in the semicircular ducts and stimulation of vestibular nerve (VIII) receptors in the inner ear. These impulses will be projected thru the vestibular nuclei into the medial longitudinal fasciculus in the brain stem which projects to the somatic efferent neurons of the oculomotor and abducent nerves. This normal response can be readily elicited in most dogs but in some cats the eye movements will only occur at the end of the head excursion.
This takes along time to write and just a few seconds to do.

NYSTAGMUS

As I stand over the dog's head, following the menace and pupil examination, I look for any strabismus or any abnormal resting - spontaneous nystagmus. I then move the head side to side to

see if the eye movements are normal and then hold the head still in one lateral position and see if any abnormal positional nystagmus develops - then move the head to the opposite side and hold it still and look for abnormal nystagmus in that position and then I extend the head and neck and hold it still and look for abnormal nystagmus,. In dogs and cats when the head and neck are extended, the eyes normally elevate to stay in the center of the palpebral fissure. With vestibular system disorders the eye on the affected side usually fails to elevate completely giving you a vestibular strabismus.. When the head is held still there should be no nystagmus. Nystagmus is normal whenever the head is moved. In a severe vestibular system disorder a nystagmus occurs continuously regardless of the position of the head .This is an abnormal nystagmus referred to as a resting or spontaneous nystagmus. In less severe vestibular system disturbances an abnormal nystagmus may only occur when the head is held in various positions as just described This is referred to as an abnormal positional nystagmus.. Occasionally in very mild cases it will only show up when the patient is placed in dorsal recumbency with the head and neck extended.

The direction of the nystagmus is defined as the direction of the fast phase of the eye movements. The one rule that can be relied on is that when the vestibular disturbance involves the inner ear receptors or the vestibular part of the vestibulocochlear nerve (VIII) , the direction of the jerk nystagmus is always opposite to the side of the lesion The latter is the direction of the head tilt and loss of balance.

FACIAL AND TRIGEMINAL NEURONS

Although portions of these cranial nerves have already been assessed, I routinely reconsider them now. I gently touch the eyelids with a pair of forceps coming at the eyes from caudally so the forceps will not be seen. There is considerable overlap of the eyelid innervation by the trigeminal nerve ophthalmic branches medially and the maxillary branches laterally therefore I make no attempt to distinguish between the two. The sensory nerves stimulated are branches of the trigeminal nerve (CN V) and the motor response is via branches of the facial nerve (CN VII). Connections between the two involve the pons and medulla.. For other areas of facial nerve innervation I look for normal flaring of the nostrils on inspiration, hold the head and neck in extension and look at the corners of the lips for evidence of mucosa showing on the paretic side and abnormal drooling on that side. I also assess the abiltiy to move the ears in those patients with erect ears but do not spend much time on flop-eared dogs to avoid frustration. Sometimes a normal ear will move when you blow air into it. There is no need for the examiner to get excessively stressed in this process.
Remember that dogs and cats with facial paralysis will not have a smaller palpebral fissure (ptosis) unlike the herbivores nor will their nose deviate to the normal side.
When you see the nose deviated to one side in a dog this usually is a reflection of excessive contraction of the facial muscles on that side referred to as hemifacial spasm but it is not spasmodic-episodic as it is described in humans. It is a continual deviation. This is most commonly associated with a presumed irritation of the facial nerve from an otitis media. These dogs will have a narrowed palpebral fissure and an ear pulled dorsally and medially on that side .On testing the palpebral reflex there may be slight movement of the eyelids. This has been described as a denervation contracture of the facial muscles but these muscles will often relax during local or general anesthesia which refutes that consideration at least for most cases. This rarely occurs in the cat.

To complete my evaluation of the trigeminal nerve I palpate the muscles of mastication. The only evidence of a unilateral motor trigeminal nerve deficit will be the denervation atrophy that can be palpated. They can still bite!! You need bilateral loss of this mandibular nerve innervation to get a loss of the ability to use the jaw. When this occurs the lower jaw will be dropped so the mouth is continually open and can not be closed. The most common cause of a sudden onset of a dropped lower jaw is an immune-mediated trigeminal neuritis. The most common cause of unilateral atrophy of these muscles is a nerve sheath neoplasm in the dog and more likely lymphoma in the cat. Bilateral atrophy of the muscles of mastication is often seen in older dogs
with no evidence of any dysfunction . One cause may be a chronic myositis . Occasionally this atrophy and accompanying fibrosis is severe enough to prevent the jaw from opening.

On a routine examination I always touch the nasal septum with the end of my closed forceps as a test of both the sensory innervation by the trigeminal nerve - specifically ophthalmic branches via the ethmoidal nerve but also as a very sensitive test for nociception and therefore this projection pathway which involves the contralateral prosencephalon. This is one of the three tests that I have described that will evaluate prosencephalic function. With prosencephalic lesions this nasal septum

will never be analgesic just hypalgesic because some of the nociceptive pathway stays ipsilateral and the incomplete crossing occurs in the pons and medulla.

When you determine that there is nasal hypalgesia the lesion responsible for this can either be in the ipsilateral trigeminal nerve or in the contralateral prosencephalon. You differentiate between the two locations based on the rest of the clinical signs that are present. Are they related to the caudal brain stem and therefore this is a trigeminal nerve problem or are they prosencephalic and that is the basis for the hypalgesia ?

IX - X - XII

These 3 cranial nerves are examined together with the so-called "gag reflex". This is done rapidly as the patient usually objects to the manipulation that is necessary and especially cats. You grasp the upper jaw with one hand and pull down on the lower jaw with the other hand opening the mouth. This effort will test the tone- resistance in the muscles of mastication (CN V). You quickly look at the size of the tongue for atrophy - hypoglossal nerve (CN XII) and push the tongue with your finger to see if it moves. Then insert your finger deep into the oropharynx to assess the tone and sensory perception that you will stimulate. These latter functions are dependent on the innervation by the pharyngeal branches of the glossopharyngeal (IX) and vagal (X) nerves. This assessment of the gag response is difficult to evaluate and is usually very subjective. A more reliable indication of dysphagia usually comes in the form of a complaint by the owner as they watch their pet try to eat and swallow.

The following is an outline of the order of the cranial nerve exam just described.

Menace Response: II -central visual pathway to occiptal lobe-
VII = closure of palpebral fissure

Pupil Size- Light Response:II-brain stem-III parasympathetic
-ciliary ganglion - nerves = pupil constriction
direct and indirect

Eye Position: Strabismus - III = ventrolateral
VI = medial

Eye Movements: Normal vestibulo-ocular nystagmus
VIII - brain stem-III = adduction
-VI = abduction

VIII-Vestibular: Strabismus in some eye positions
Abnormal nystagmus (head not moving)

Facial muscles: VII-position,tone, movement :
eyelids, ears, lips, nose
Menace response: II - VII
Palpebral reflex: V - VII

Facial sensation: V
Palpebral reflex: V - VII --cutaneous, autonomus zones
Nociception: Nasal mucosa -Ophthalmic br. V

Masticatory muscles: V-Mandibular br. V
Muscle size, tone-jaw closure

Gag Reflex: Jaw tone - V
Tongue size movement - XII
Reflex gagging, swallowing - IX, X

Notes

Overview of FCoV/FIP

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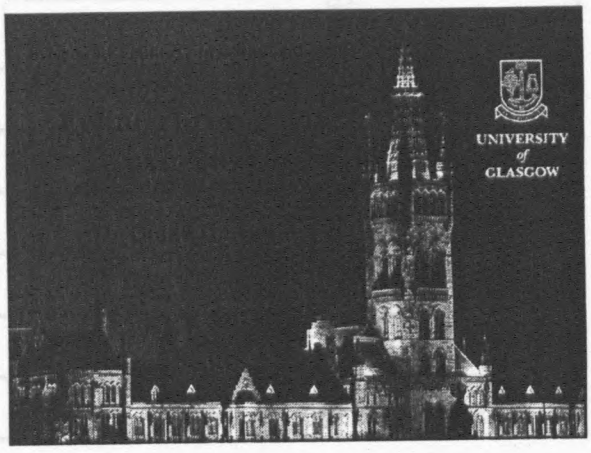

You can't get SARS from a cat!

Forget FECV/FIPV - ANY cat infected with FCoV can develop FIP

However, the majority of cats infected with FCoV remain perfectly healthy

Any age of cat can develop FIP

FCoV is shed in s- - t loads in faeces

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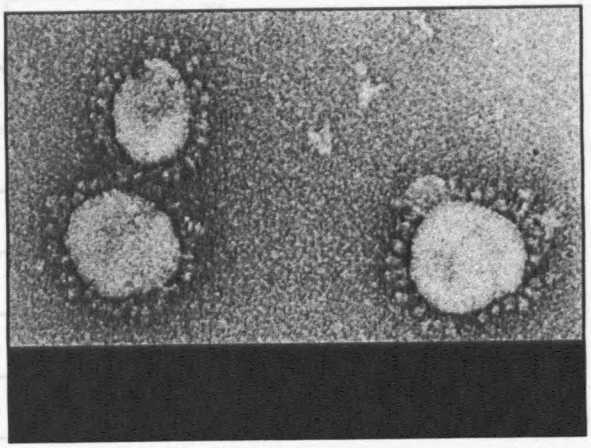

Overview of FCoV/FIP

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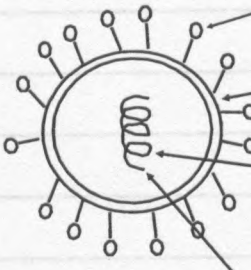

ɔronaviruses

	GEV	intestine
	RCV	respiratory tract
	EV	respiratory tract/CNS
	DV	intestine
MOUSE	MHV	liver/CNS
CHICKEN	IBV	respiratory/urogenital
DOG	CCV	intestine
CAT	FCoV	intestine/macrophages
HUMAN	HCV 229E HCV OC43 SARS	respiratory

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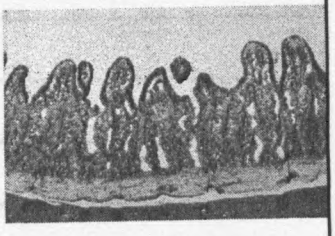
Aminopeptidase-N (CD 13)

an enzyme highly expressed at brush borders of:

respiratory epithelium
gut epithelium
nerve cells



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Overview of FCoV/FIP

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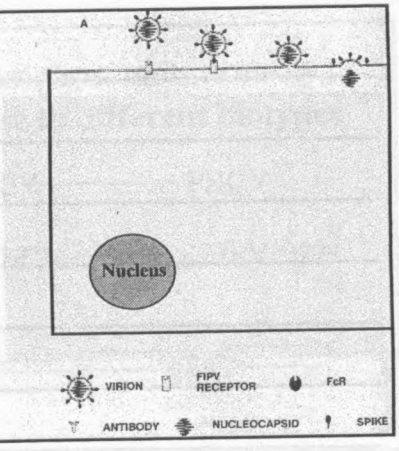
A: normal infection, attachment to cellular receptor



Coronavirus Groups

SARS - ? Group 4???

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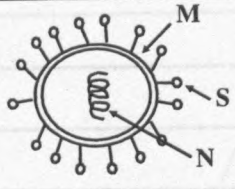

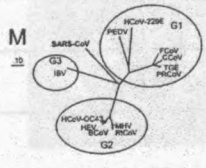

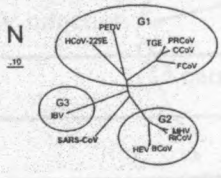

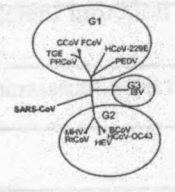

Rota *et al*. Epub 1 May 2003 Characterization of a novel coronavirus associated with severe acute respiratory syndrome. Science. 2003 May 30;300(5624):1394-9.

Overview of FCoV/FIP

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Type I FCoV

- Purely feline

- Most prevalent in field
- Possibly cell-associated
- Difficult to grow in cell culture

Type II FCoV

- Arise from recombination events between type I FCoV and CCV
- Less than 10% of field isolates, but most laboratory isolates
- Grow in cell culture

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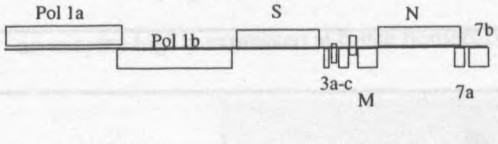
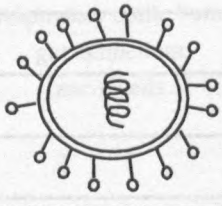
FCoV genome





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Overview of FCoV/FIP

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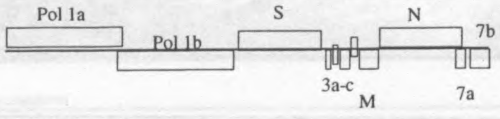
Coronaviruses mutate easily giving rise to different biotypes

TGEV ⟶ PRCV

FCoV/FECV ⟶ FIPV?????



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Coronaviruses mutate easily giving rise to different biotypes

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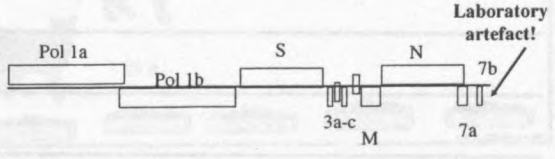

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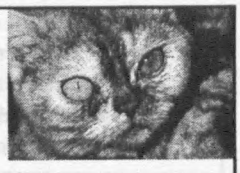

FIP

FCoV infection ⟶ transient infection

carrier/persistent infection

resistant

www.catvirus.com Addie D.D., Jarrett O. 2001 Veterinary Record. **148** 649-653

Overview of FCoV/FIP

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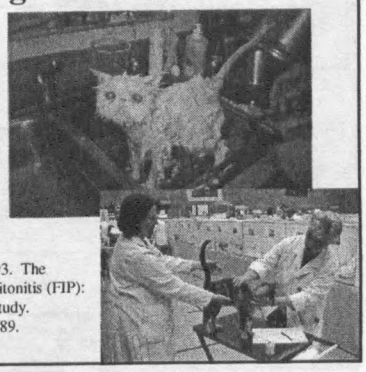
Pathogenesis of FIP

? viral change

? virus dose

history of stress



Rohrer C. Suter PF. Lutz H. 1993. The diagnosis of feline infectious peritonitis (FIP): a retrospective and prospective study. Kleinterpraxis 38 6 379-389.

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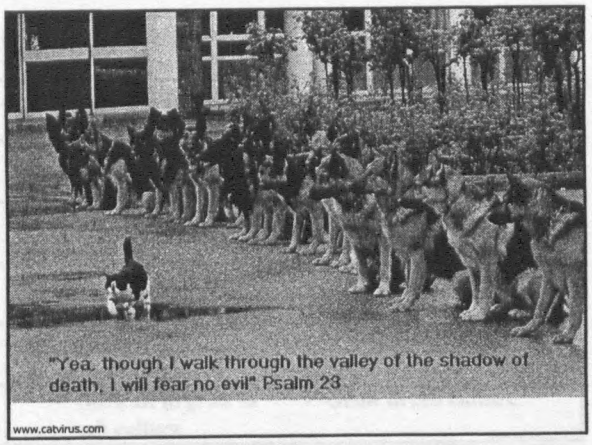

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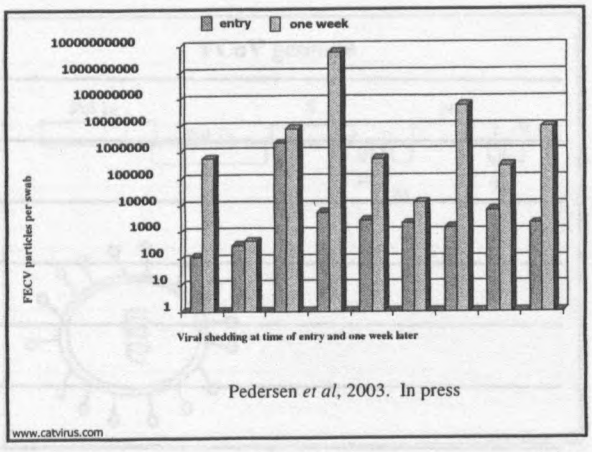

Pedersen *et al*, 2003. In press

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Overview of FCoV/FIP

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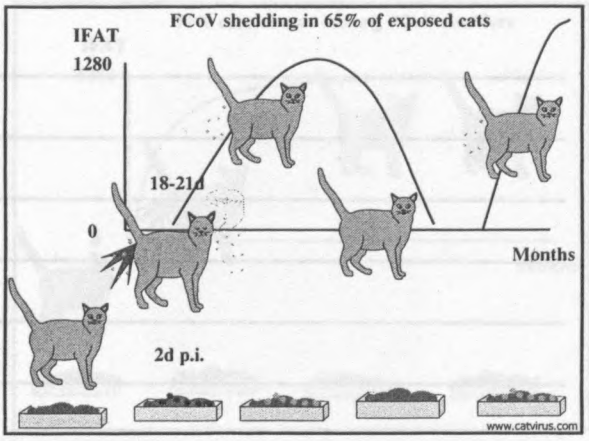

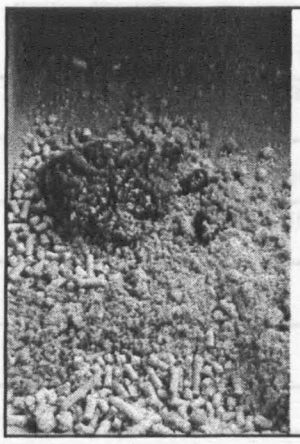

The main source of FCoV infection is contact with faeces or the used cat litter of infected cats.

FCoV can survive up to 7 weeks in dried up cat litter.

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Overview of FCoV/FIP

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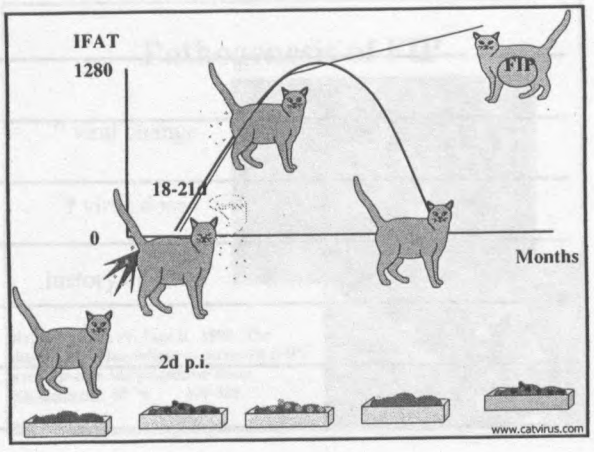

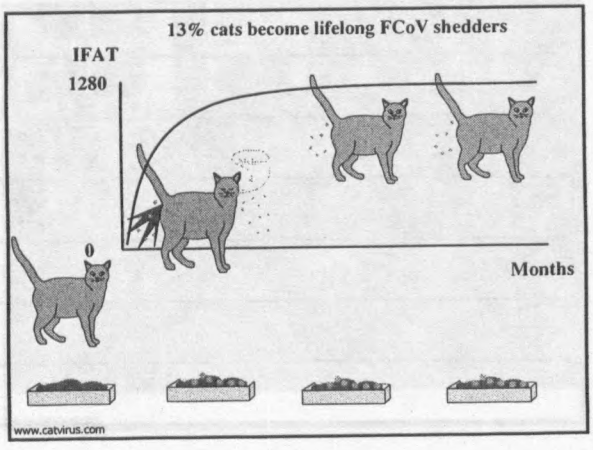

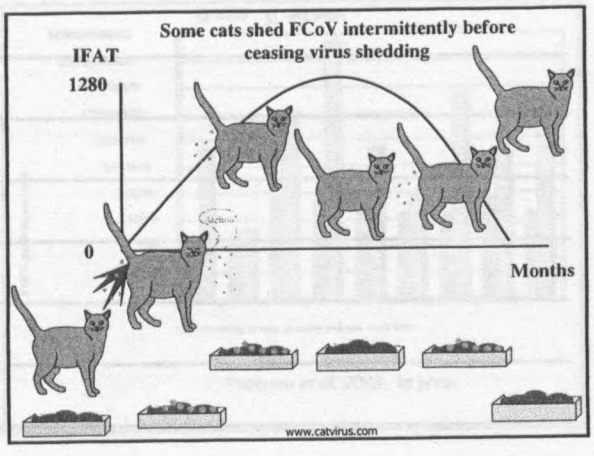

A single negative FCoV RT-PCR does not mean that a cat is not infectious:

- either the cat should also be seronegative
- or the cat should have had FIVE monthly negative RT-PCR tests

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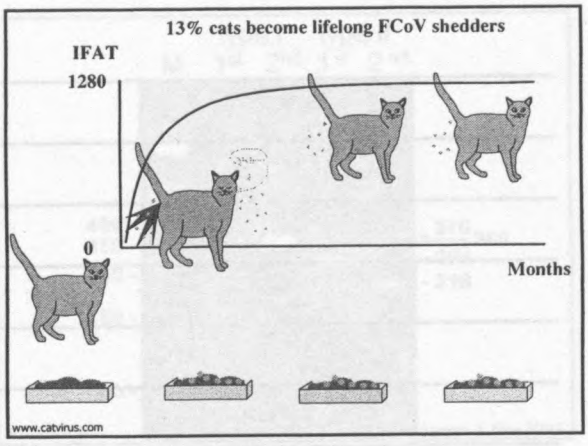

A single positive FCoV RT-PCR does not mean that a cat is a carrier:

- 9 consecutive monthly positive RT-PCR tests identifies most carriers

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Overview of FCoV/FIP

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Patterns of FCoV shedding

Transient shedders: cats which become infected for a short period of time (usually < 3m)

Intermittent shedders: indistinguishable from cats which become reinfected (unless antibody titre declines then increases)

Carrier cats: shed FCoV continually for the rest of their lives

Resistant cats: despite continual exposure, do not become infected

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Type I FCoV

- **Purely feline**

- **Most prevalent in field**
- **Possibly cell-associated**
- **Difficult to grow in cell culture**

Type II FCoV

- **Arise from recombination events between type I FCoV and CCV**
- **Less than 10% of field isolates, but most laboratory isolates**
- **Grow in cell culture**

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Question

Are UK cats infected with type I or type II FCoV??

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Overview of FCoV/FIP

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FCoV shedding survey

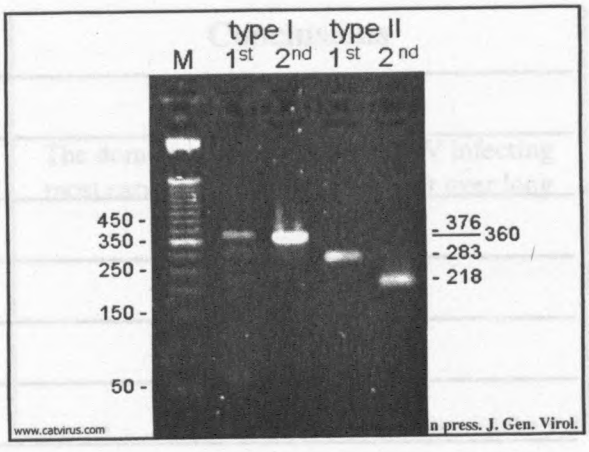
29 households

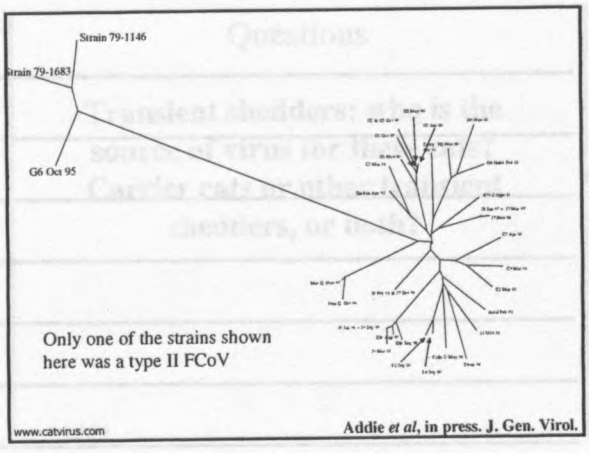
155 cats

7 dogs

Funded by Winn Foundation

Addie D.D., Jarrett O. 2001 Veterinary Record. **148** 649-653





Question

Are UK cats infected with type I or type II FCoV??

Answer:

28 of 29 (97%) isolates from 43 cats in 15 UK households were type I

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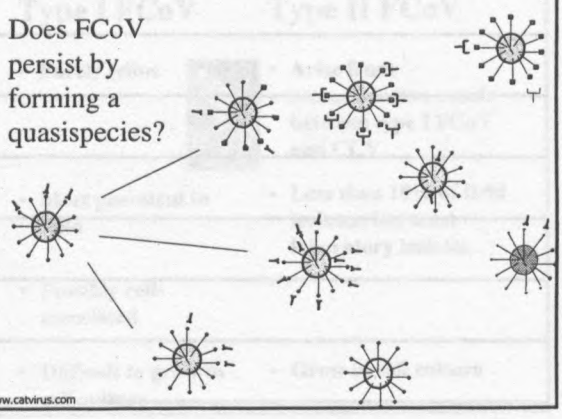
Addie *et al*, in press. J. Gen. Virol.

Does FCoV persist by forming a quasispecies?



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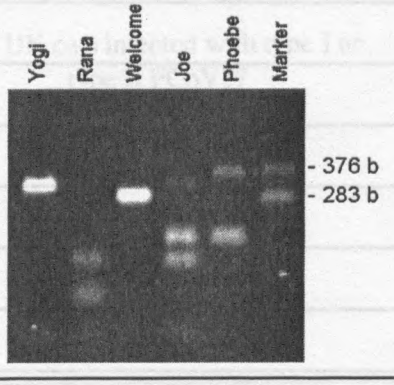
Alu I restriction enzyme digestion



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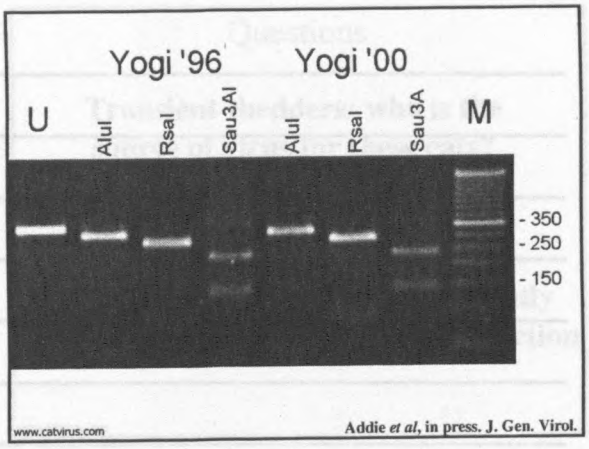

Conclusions

The dominant population of FCoV infecting most carrier cats remained similar over long periods of time

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Addie *et al*, in press. J. Gen. Virol.

Questions

Transient shedders: who is the source of virus for these cats? Carrier cats or other transient shedders, or both?

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Overview of FCoV/FIP

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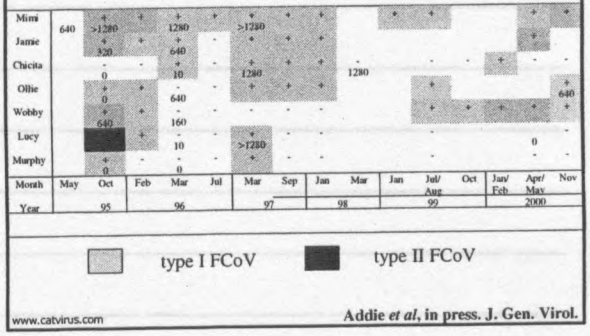
Reinfection event where the carrier is the source of the FCoV

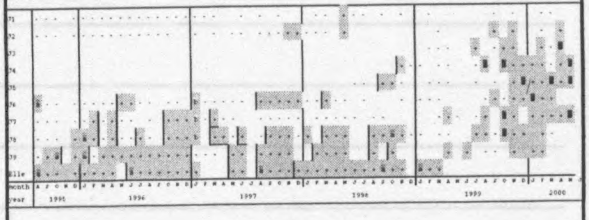


www.catvirus.com

Addie *et al*, in press. J. Gen. Virol.

Cats could be reinfected with the same or another strain of FCoV

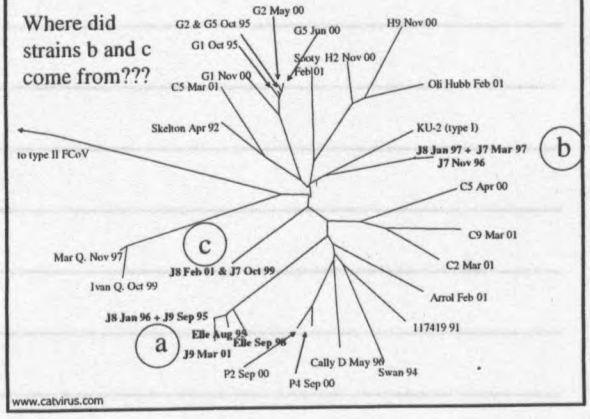
Where did b and c come from???



www.catvirus.com

Addie *et al*, in press. J. Gen. Virol.

Where did strains b and c come from???



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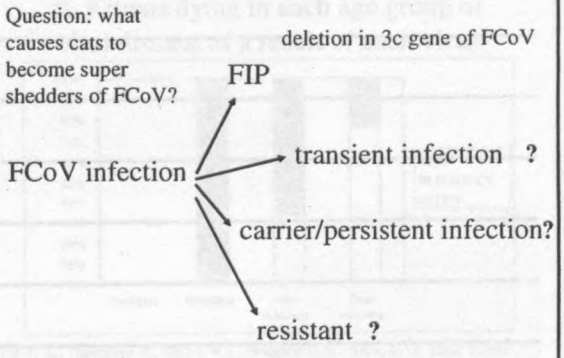
Questions

Transient shedders: who is the source of virus for these cats? Carrier cats or other transient shedders, or both?

Answer: carrier cats AND transiently infected cats are both sources of infection

www.catvirus.com

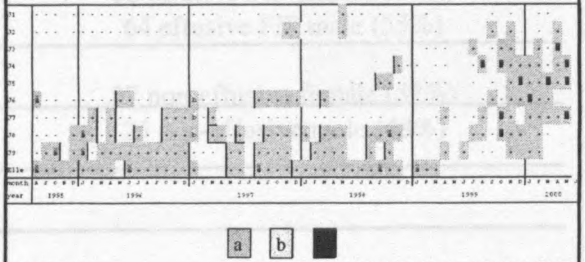
Question: what causes cats to become super shedders of FCoV?



www.catvirus.com Addie D.D., Jarrett O. 2001 Veterinary Record. **148** 649-653

Does the FCoV strain determine carrier status?

Subjected to the same strain, cats react differently.



www.catvirus.com Addie *et al*, in press. J. Gen. Virol.

Overview of FCoV/FIP

Diane D. Addie
www.catvirus.com

Feline coronavirus carrier cats

- shed FCoV in faeces continually for life
- 13 % of exposed cats become carriers
- identify by over 9 consecutive monthly positive RT-PCR tests
- most carrier cats do not develop FIP
- carrier cats at risk of developing chronic diarrhoea

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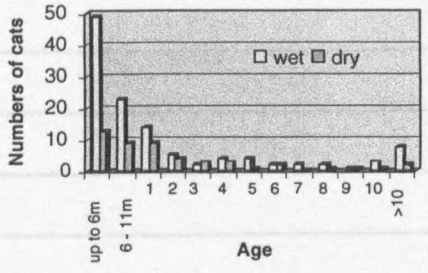
Age distribution of 119 cats with effusive FIP and 49 cats with non-effusive FIP



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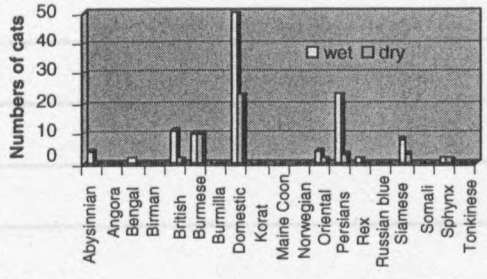
102 of 181 cats with FIP were purebred (56%)



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Overview of FCoV/FIP

Diane D. Addie
www.catvirus.com

Official GCCF news

Analysis of Breeds registered by the GCCº

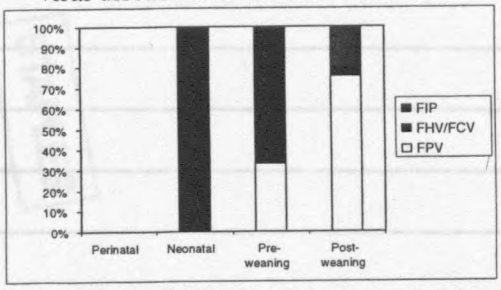
Breed Grouping 2001

British Short Hair	5,377	793	
Persian LH	5,062	Asian/Burmilla	622
Siamese	3,943	Devon Rex	587
Burmese	2,655	Tonkinese	441
Birman	2,223	Abyssinian	393
Bengal	1,942	Norwegian Forest	312
Ragdoll	1,398	Russian Blue	260
Maine Coon	1,377	Somali	244
Oriental Short Hair	1,310	Cornish Rex	193
Exotic Short Hair		Balinese	180
		Korat	148
		Egyptian Mau	109
		Turkish Van	107
		Tiffanie	106
		Ocicat	72
		Singapura	72
		Manx	36
		Angora	35
		Others	98

Total cats registered in 2001 - 30,093

www.catvirus.com

% Kittens dying in each age group of viral disease as a result of each virus



Cave T. A., Thompson H., Reid S.W.J., Hodgson D.R., Addie D.D. 2002 Kitten mortality in the United Kingdom: a retrospective analysis of 274 histopathological examinations (1986-2000). Veterinary Record. **151** 17 497-501

No sex predisposition for FIP

52 effusive FIP female (45%)
64 effusive FIP male (55%)

27 non-effusive female (52%)
25 non-effusive male (48%)

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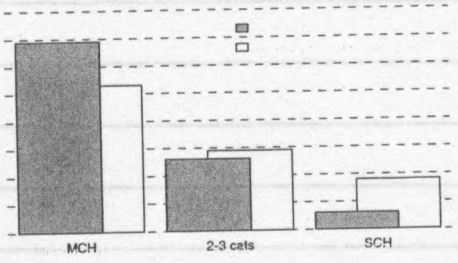
Home environments of cats with FIP and other conditions



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Cats from multicat environments are more likely to develop FIP for two reasons:

- increased viral dose
- increased stress

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The odds of FIP developing increases with the number of cats:

Number of cats	Increase in odds of FIP
8-20	two-fold
21-39 cats	three-fold
> 40	five-fold

Kass & Dent Feline Practice 23 27-32

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79 of 181 cats with FIP were domestic (44%)

102 of 181 cats with FIP were pedigree (56%)

… in a country where pedigree cats comprise 5-10% of the population

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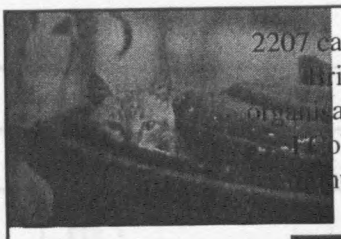
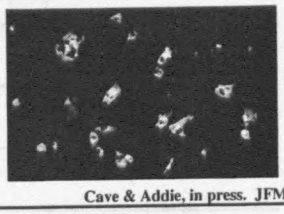


2207 cats relinquished to a British cat rescue organisation were tested for FCoV antibodies by immunofluorescence



www.catvirus.com

Cave & Addie, in press. JFMS

Pedigree cats were much more likely to be FCoV seropositive than domestic cats.

Domestic	18.3%	(182/994)
Pedigree	74.1%	(20/27)

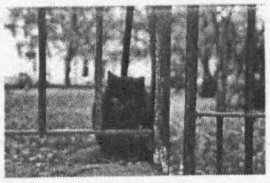
www.catvirus.com

Cave & Addie, in press. JFMS

Pet	20.2%	(129/638)
Stray	19.4%	(48/247)
Feral	11.5%	(12/104)

www.catvirus.com Cave & Addie, in press. JFMS

Feral cats were significantly less likely to be FCoV seropositive ...



... than pet or stray cats

www.catvirus.com Cave & Addie, in press. JFMS

Risk factors for developing FIP:

Young cats (under 2 yrs)
Pedigree (especially Persian)
History of multicat environment
History of stress

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Overview of FCoV/FIP

Diane D. Addie
www.catvirus.com

You can't get SARS from a cat!

Forget FECV/FIPV - ANY cat infected with FCoV can develop FIP

However, most cats infected with FCoV remain perfectly healthy

Any age of cat can develop FIP

FCoV is shed in s- - t loads in faeces

www.catvirus.com

Feline Coronavirus: Overview

Diane D. Addie, BVMS, PhD, MRCVS
University of Glasgow
Glasgow, UK

In August 2002, scientists and veterinary surgeons from around the world met in Scotland to share the latest information on feline coronavirus (FCoV). Although most cats infected with FCoV remain perfectly healthy, around one in ten cats infected with FCoV develops feline infectious peritonitis (FIP). FIP is now believed to be the leading infectious cause of cat death, yet it remains one of the most difficult of conditions to diagnose and treatment isn't curative, but only delays the need for euthanasia. Use of the only FIP vaccine, Primucell®, is controversial.

Although SARS is also caused by a coronavirus, it is phylogenetically very different from FCoV. It probably originated from the Civet cat, which isn't, in fact, a cat, but a mongoose. People cannot get SARS from their cats, but people with SARS can possibly infect cats.

FECV - FIPV - FCoV - Type I - Type II - what does it all mean?

A clinical syndrome involving peritonitis in cats was recognised by Jean Holzworth in 1963 and was named feline infectious peritonitis. When a coronavirus was discovered to be the cause it was naturally named feline infectious peritonitis virus (FIPV). Later, Pedersen showed that many healthy cats were seropositive to this coronavirus and postulated that these healthy cats were infected with an avirulent coronavirus which he named feline enteric coronavirus (FECV), stating that it lived only in the intestine and caused only mild diarrhoea in kittens. Later studies showed that there were no consistent differences between FIPV and FECV laboratory strains and that one in four healthy seropositive cats is also viraemic. Epidemiological studies showed that wherever coronavirus was present, FIP would develop in around 10% of cats and that the broader term FCoV was adopted because it more accurately describes the infection. It was then postulated that virulent FIPV arises by a mutation in FECV in the individual cat which develops FIP and it has been shown that the cats which develop FIP often have deletions in the 3c gene (Vennema *et al*, 1998). However, these deletions are not the same in all isolates and it is unlikely that a specific test based on this finding could be developed, which would be a specific test for FIP.

All FCoVs are either type I or II. Type I FCoV is the predominant field type and is wholly feline. Type II is rare in natural infections and its spike gene contains much canine coronavirus sequence (Herrewegh *et al*, 1998). Most laboratory strains of FCoV are type II.

There is no such thing as an avirulent FCoV in nature - all FCoV infections have the potential to cause FIP.

Transmission and FCoV shedding

The main source of virus is the faeces and infection is by accidental ingestion of particulate faeces (e.g. from grooming paws after using a litter tray, or airborne particles from litter tray contaminating food). FCoV is a fragile virus, surviving only days outside, but can survive up to 7 weeks in dried up faeces in cat litter particles. FCoV is shed only transiently in the saliva in a few cats. FCoV does not generally cross the placenta. Around 20% of cats have antibodies to FCoV, rising to over 50% of pedigree cats and 83% of cats at shows. FCoV is very infectious and once in a household, will infect 90% of cats.

After infection with FCoV there are 4 possible outcomes:

1. Develop FIP (about 10% of cats)
2. Become transiently infected, shed FCoV for many months, become seropositive, stop shedding virus, become seronegative. Be susceptible to re-infection. (Most cats.)
3. Become a healthy lifelong carrier cat (13%)
4. In addition, there is a tiny minority of cats who are naturally resistant to FCoV infection.

Transiently infected cats can be infected by a different strain of FCoV, or re-infected with the same strain. Therefore, control of FCoV (and ultimately FIP) is about interrupting the cycle of infection and re-infection. Carrier cats shed the same strain of virus for life.

Notes

15th Annual Fred Scott Feline Symposium
July 25 - 27, 2003


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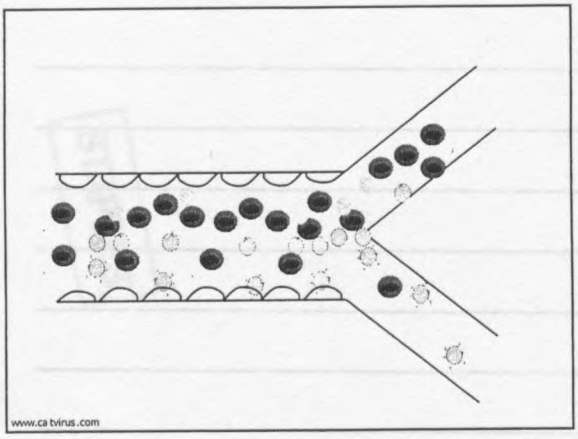
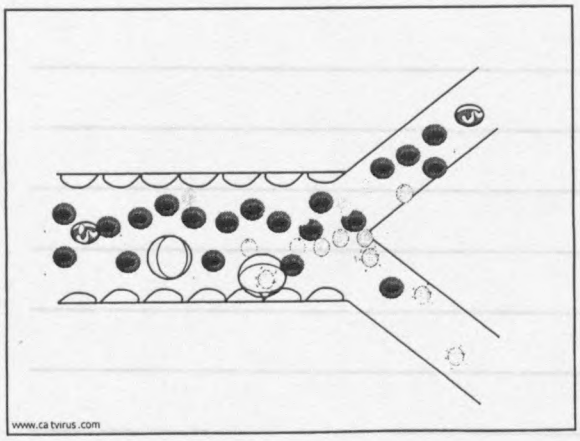

www.ca tvirus.com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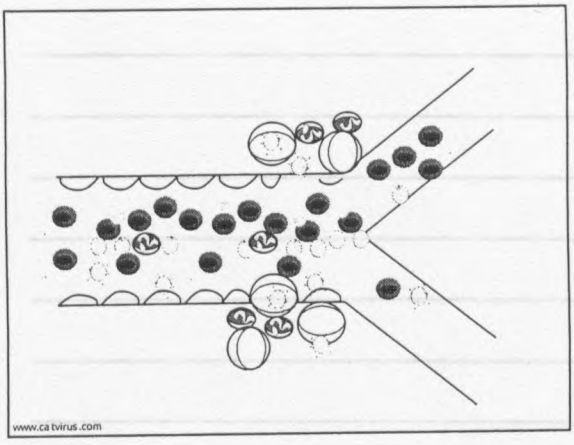
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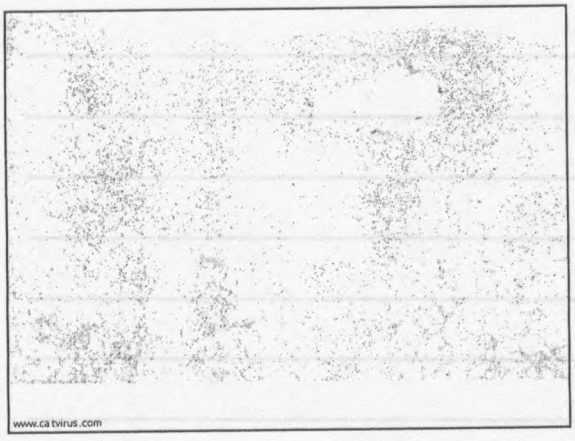
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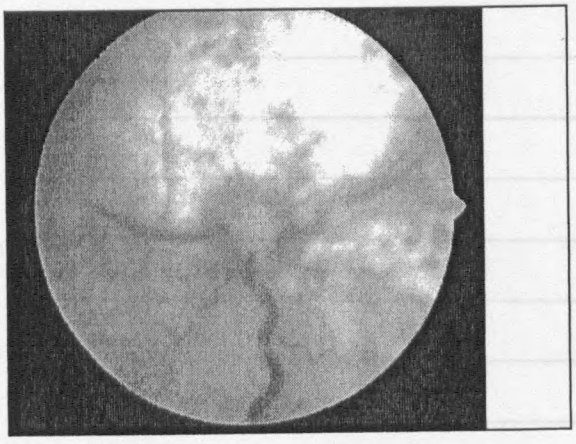

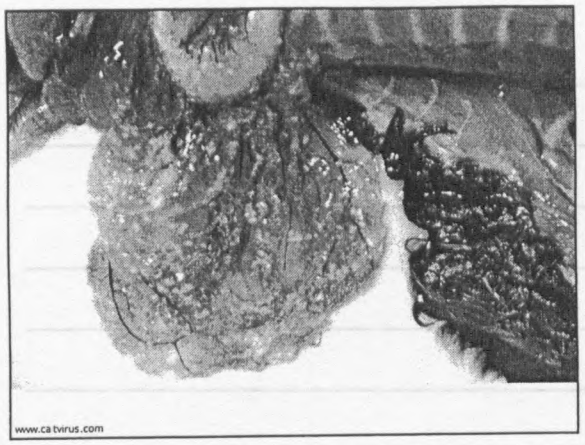
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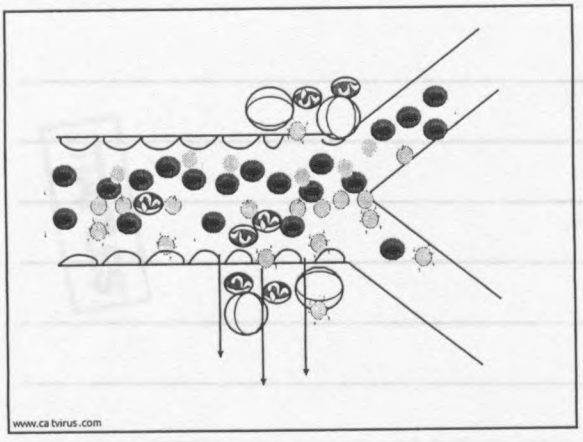
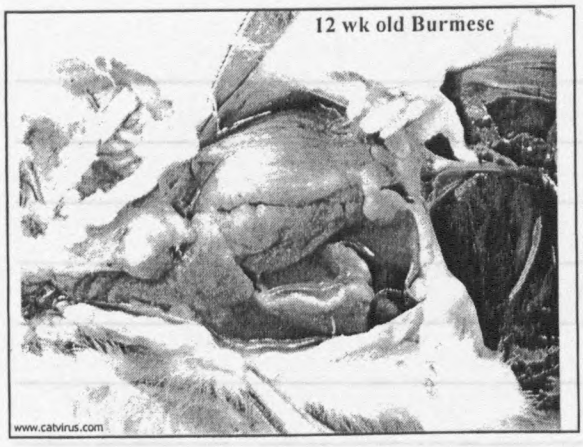

www.catvirus.com


12 wk old Burmese
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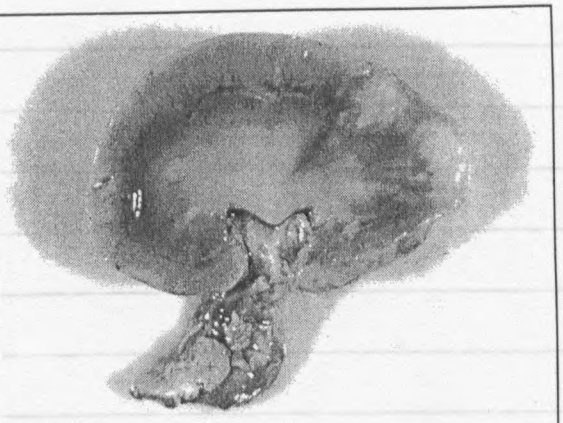

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Diagnosing FIP

First steps:

Presenting signs typical of FIP:
- **ascites**
- **pleural effusion**
- **intra-ocular signs**
- **neurological signs**
- **PUO and weight loss**

History compatible with FIP:
- **MCH environment**
- **purebred**
- **recent stress**

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Risk factors for developing FIP:

Young cats (under 2 yrs)
Pedigree (especially Persian)
History of multicat environment
History of stress

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Diagnosing FIP

Second step:

Clinical examination

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Pyrexia

Anorexia

Weight loss

Dull or bright

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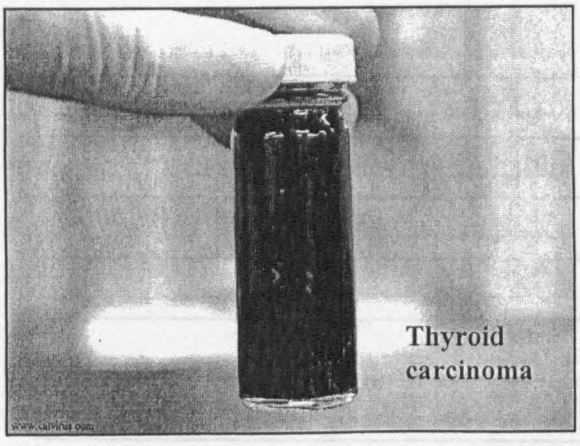

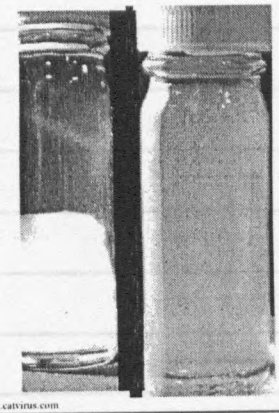
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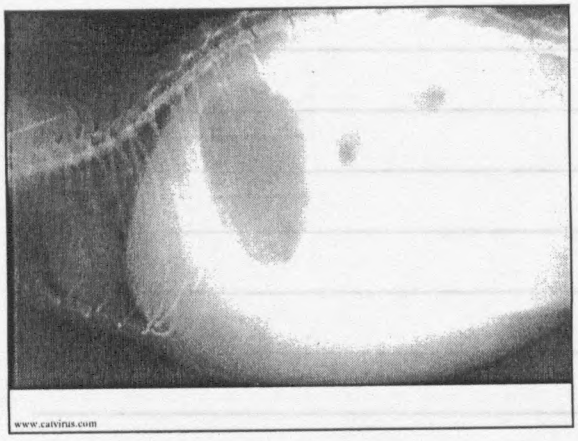
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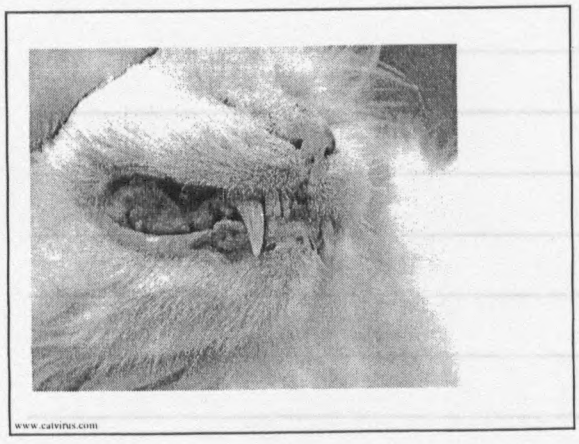
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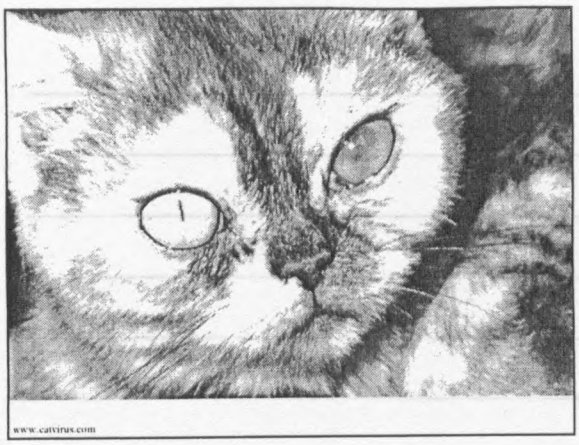

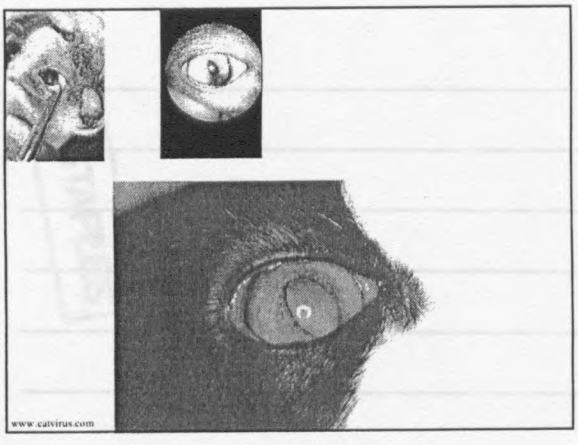

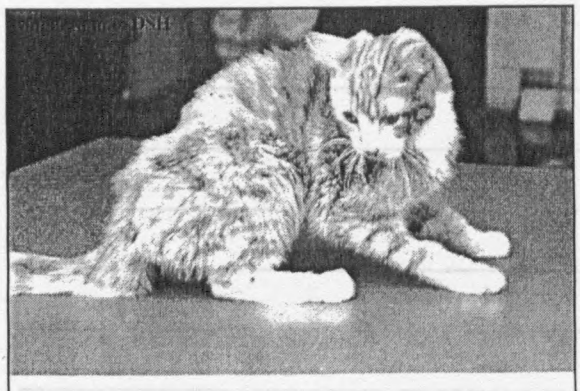

Neurological signs in 12% of non-effusive FIP cases

Diagnosing FIP

Third step:

Sampling for laboratory tests

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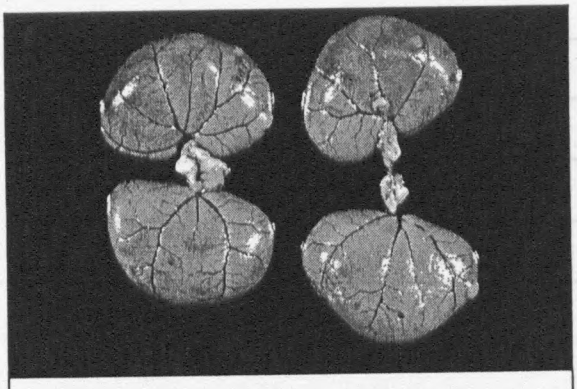

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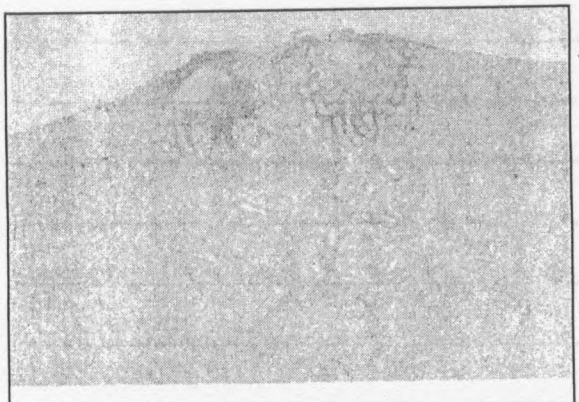

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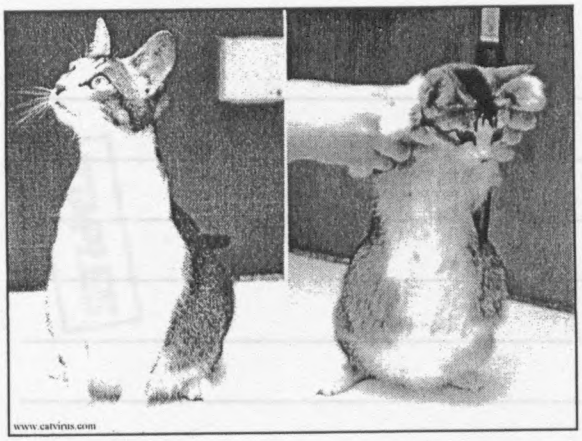
FIP diagnosis: FIP profile

FCoV antibody titre

Albumin:globulin

Haematology/cytology

α1- acid glycoprotein (AGP)

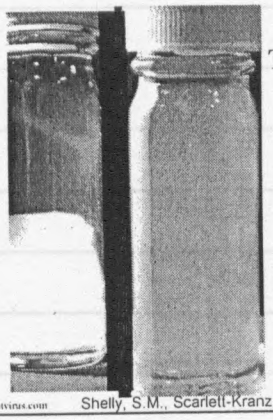


Differential diagnoses of effusive FIP

Adenocarcinoma	13
Lymphosarcoma	10
Other neoplasia	6
Bacterial peritonitis or pleurisy	9
Cardiomyopathy	7
Lymphocytic cholangitis	3
Cirrhosis	2

Pregnancy

Obesity

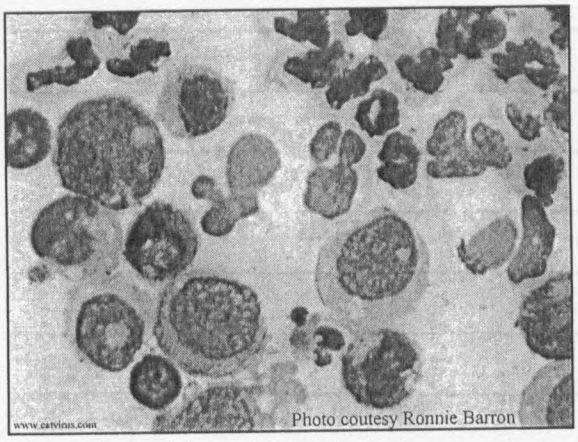


Total protein >35 g/l

Albumin:globulin ratio

< 0.4 FIP likely
> 0.8 FIP unlikely
0.4 - 0.8 see other parameters

www.catvirus.com Shelly, S.M., Scarlett-Kranz J., Blue J.T. 1988 JAAHA 24 495

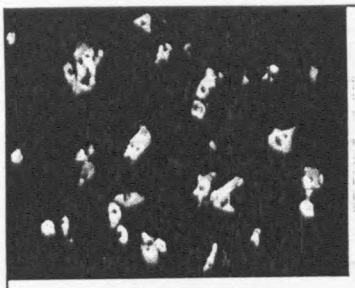


www.catvirus.com Photo coutesy Ronnie Barron

IF of macrophages - 100 % diagnostic if positive.

However, negative results does not rule out FIP.

www.catvirus.com Presented by K. Hartmann at SIFFS, 2002



FCoV IFAT

Internal negative control

Antibody titre: useful for comparison

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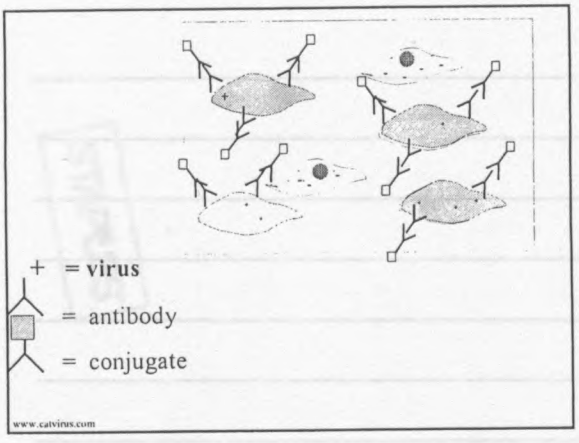

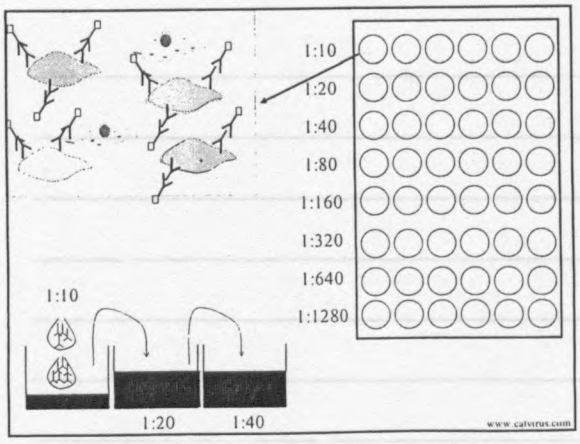

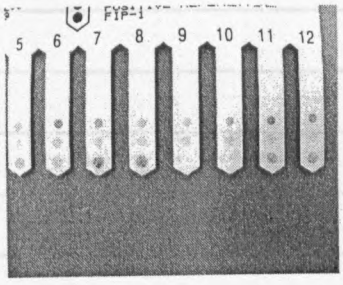

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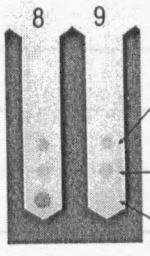

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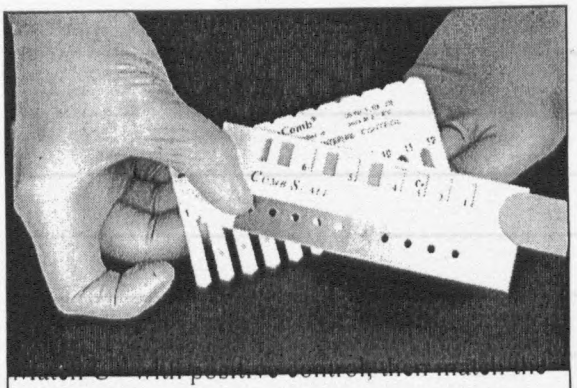
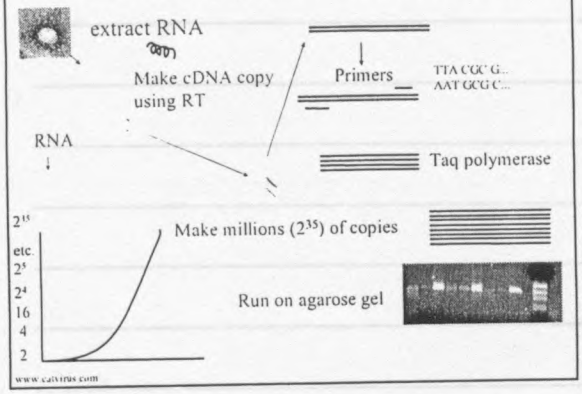


test spot.

www.catvirus.com

RT-PCR: polymerase chain reaction



FIP diagnosis: FIP profile

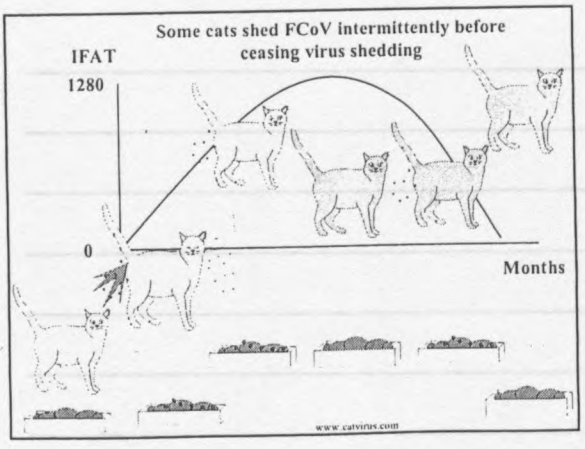
FCoV antibody titre
non-effusive FIP: >640
effusive FIP: almost any

Albumin:globulin
< 0.4 FIP likely*
> 0.8 FIP unlikely
0.4 - 0.8 see other parameters

Haematology/cytology
non-regenerative anaemia
neutrophilia with shift to left
lymphopenia
effusion: low wbc < 2.0 x 10^9/l
predominantly neutrophils and macrophages

α1- acid glycoprotein (AGP) > 1500μg/ml

* Shelly, S.M., Scarlett-Kranz J., Blue J.T. 1988 JAAHA 24 495-500



Uses of RT-PCR to detect virus

- cats can shed FCoV intermittently, so a single positive or negative does not mean a cat is a carrier or is free of FCoV
- carrier cats must shed FCoV continually for at least 8 months
- a positive result on an effusion can be diagnostic of FIP
- invaluable research tool
- mRNA detection may be diagnostic

www.catvirus.com

FIP profile

Ragbag, 13 m.o. FN DSH, from CP

FCoV antibody titre >1280 AGP >3600

Heparin plasma
Total protein 85 (60-85)
Albumin 31 (26-36)
Globulins 54 (27-45)
A:G 0.57

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FIP profile

Gizmo 2yo male DSH: wt loss. T103oF

FCoV antibody titre >1280 AGP 1600

Haematology
wbc 7.730 (5.5 -15.5)
rbc 9.020 (5.0 -10.0)
Hct 36.8 (30 - 45)

Total protein 118 (60-85)
Alb 25 (26-36)
Glob 93 (27-45)
Alb: glob 0.27

band neutrophils 4.3
neutrophils 2.01 (2.4-12.5)
lymphocytes 0.31 (1.5-7.0)

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FIP profiles

Zena 9mo F Maine Coon History: keratic ppts		Buffy 1yo Maine Coon in contact w. Zena
FCoV antibody titre >1280		>1280
AGP	1400	280
Hct	31.7	48.3
alb	23	38
glob	91	32
A:G	0.25	1.19

FIP profile

Jess, 12 y.o. FN DSH, from MCH

History: sudden onset laboured breathing.
Drained 195ml pleural fluid.

FCoV antibody titre 0

AGP 100

Pleural effusion
Total protein 48
Alb 26
Glob 22
A:G 1.2

Pleur effusion
wbc 0.6
rbc 0.2
neutrophils 0.05
lymphocytes 0.43
macrophages 0.12

FIP profile

Burre Bjom, 6 m.o. M Burmese

FCoV antibody titre >1280

AGP 2100

Ascites
Total protein 59
Alb 18
Glob 41
A:G 0.44

Ascites cytology
wbc 0.41
neutrophils 0.36
lymphocytes 0.02
macrophages 0.04

Neutrophils show degeneration and occasional toxic change.
No bacteria seen.

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FIP treatment

Prednisolone: 2mg/kg s.i.d.
reduce to 0.5mg/kg after remission

Virbagen Omega: 1M IU s/c e.o.d.
reducing to once weekly if remission occurs

4 cats of 12 recovered, 2 survived 4 and 5 months

From Ishida *et al*, presented at SIFFS, August 2002

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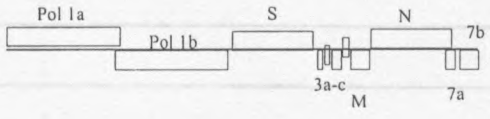
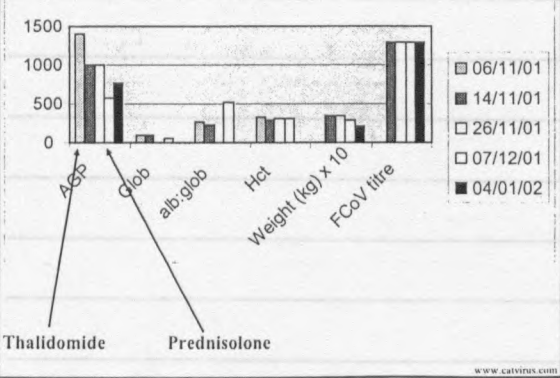


Pfizer: AG7088

???

Monitoring FIP treatment



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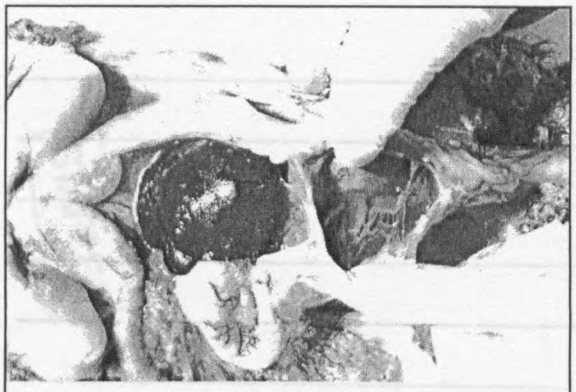
Diagnosing FIP

Fourth step:

Post mortem!!!

www.catvirus.com



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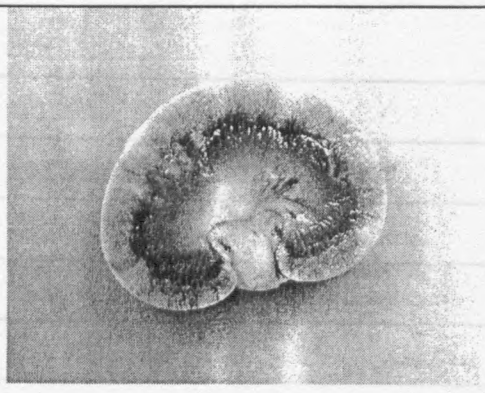
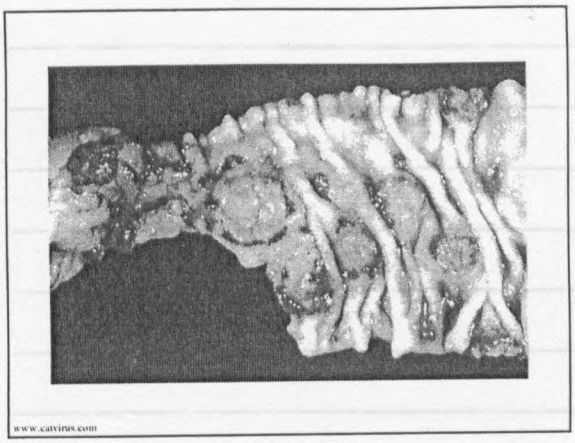
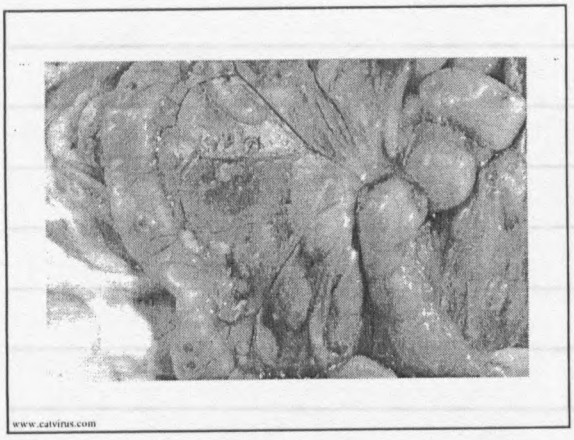


www.catvirus.com


www.catvirus.com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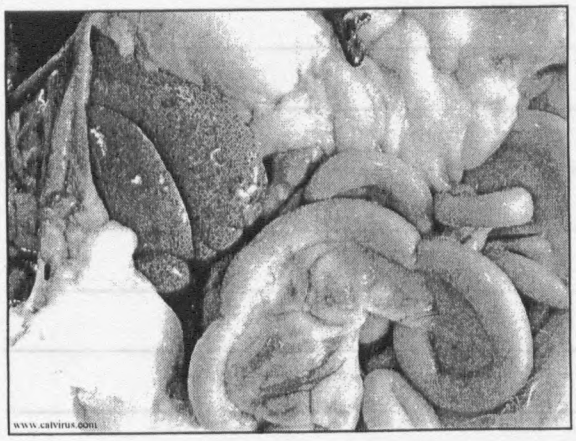
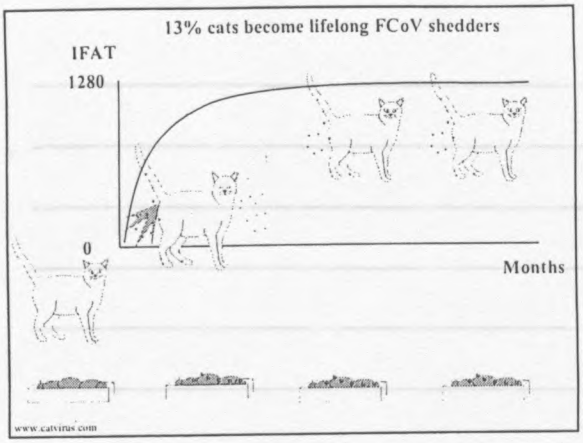

www.catvirus.com



Feline coronavirus carrier cats

- shed FCoV in faeces continually for life
- 15 % of exposed cats become carriers
- identify by over 9 consecutive monthly positive RT-PCR tests
- most carrier cats do not develop FIP
- carrier cats at risk of developing chronic diarrhoea

www.catvirus.com

Coronaviral diarrhoea

Diagnosis by default - all dietary, infectious and other reasons ruled out

Treatments

- **prednisolone**
- **? lactoferrin**
- **Probiotics (e.g. Protexin)**
- **? interferon omega**

- **(all plus bland diet)**

www.catvirus.com

www.Dr-Addie.com

www.gla.ac.uk/companion

www.felinecoronavirus.com

www.catvirus.com

Feline Infectious Peritonitis: Diagnostic Workshop

Diane D. Addie, BVMS, PhD, MRCVS
University of Glasgow
Glasgow, UK

Four steps to diagnosing FIP:

1. typical presenting signs (ascites, pleural effusion, PUO, weight loss, intraocular signs, neurological signs, icterus) and history (pedigree, recent history of multicat environment, recent history of stress)
2. clinical examination
3. samples for laboratory tests
4. post mortem confirmation of diagnosis

Although the majority of cats which develop FIP are young, any age of cat can develop FIP. Cats most at risk of developing FIP are pedigree and from indoor, multicat environments.

FIP clinical signs

If you understand that FIP is an immune-mediated vasculitis (inflammation of the blood vessels) it becomes easier to understand how it is able to manifest with so many varied clinical signs. Any blood vessel to any organ can be affected. FIP is generally defined as either "wet" (effusive) or "dry" (non-effusive) but neither is clear cut and an effusive case can become non-effusive or vice versa. Effusive FIP is the more acute condition - occurring within 4-6 weeks of a stressful event in the cat's life, whereas non-effusive FIP can incubate for months to years. In effusive FIP, many blood vessels are affected, allowing fluid to leak out into the abdomen or thorax or both. Thus the cat presents with ascites or pleural effusion. The ascitic cat may appear to have put on weight, although ribs are usually more palpable. The Orion Foundation call FIP "the purring disease" because the cat may still be bright and eating, though some are dull and anorexic. The temperature of cats with FIP rarely goes above 103°F. Cats with FIP frequently have palpably enlarged mesenteric lymph nodes. A cat with a pleural effusion will present with dyspnoea.

In the more chronic, non-effusive, FIP, fewer blood vessels are affected. The cat loses weight gradually, becomes dull and anorexic. Most cats with dry FIP have intraocular lesions. Clinical signs will depend on what organs are involved:

- liver → jaundice
- meninges/hydrocephalus → neurological signs (ataxia, nystagmus, fits, loss of reflexes)
- kidney → kidney failure (polydipsia, polyuria, vomiting)
- eyes → uveitis, aqueous flare, vitreous flare, retinal vessel cuffing, corneal precipitates, haemorrhage into anterior or posterior chambers

FIP diagnosis

Correct diagnosis is essential, we have found that only 18% of cats clinically diagnosed as having FIP were likely to really have FIP when tested by our FIP profile. There is no such thing as a single "FIP" test and it is unlikely that there ever will be - be very wary of any single test which claims to be diagnostic of FIP. The definitive diagnostic test remains histopathology of a biopsy and even then it can be difficult to interpret, when immunohistochemical staining might also be necessary. In our laboratory at the University of Glasgow, we use a careful selection of tests as indicated in table 1.

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Laboratory result	Effusive	Non-effusive
High FCoV antibody titre	Usually	over 640
High α1-acid glycoprotein (AGP)	Yes (>1500 μg/ml)	Over 1000 μg/ml
Non-regenerative anaemia (Hct 30 or less)	Possibly	Yes
Neutrophilia with shift to left	Probably	Probably
Cytology of effusion	Total wbc count < 2.0 x 10^9/l: neutrophils and macrophages. Sterile - no bacteria!	Not applicable
Raised globulins	In effusion: total protein > 35g/l, if less, it's not FIP	In plasma, globulins > 40g/l
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How to submit samples for an FIP profile

	Effusive FIP	Non-effusive FIP
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Send samples by air mail to:
Companion Animal Diagnostics,
University of Glasgow Veterinary School,
Bearsden
Glasgow, UK
G61 1QH

Tel: + 44 141 330 5777
Fax: + 44 141 330 5748
Email: Companion@vet.gla.ac.uk
www.gla.ac.uk/companion

The FIP profile costs £25.00 ($41.50).

FCoV tests

The indirect immunofluorescent antibody test (IFAT) is the gold standard. It gives an antibody titre which is useful for comparison in sequential testing and contains an internal negative control at each dilution, so that false positive results do not occur.

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Treatment

Since FIP is immune mediated, treatment aims at diminishing the immune reaction, usually using quite high doses of prednisolone, as a sliding dose (i.e. 4mg/kg/day for 10-14 days, reducing to 2 mg/kg/day for 10-14 days, and so on). However, at best one can only hope for a remission - FIP is, at time of writing, incurable. Most so-called "recovered" cats were probably simply misdiagnosed. A possible exception to what I have just written MAY be thromboxane synthetase inhibitors (used in humans with asthma), Watari et al (1998) describe remission of 2 cases of FIP using these substances: dose - ozagrel hydrochloride 5 mg/kg bid.

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FCoV/FIP Vaccination

Diane D. Addie
www.catvirus.com

Prevent FCoV and you prevent FIP

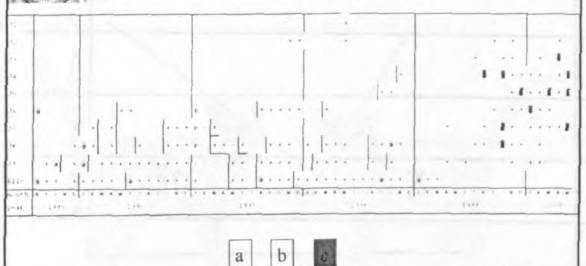
FCoV is shed in huge quantities in the faeces

The main method of control is good hygiene

FIP can be prevented in kittens by early weaning and isolation

www.catvirus.com

Panleucopenia vaccines work well because most cats in nature recover from the infection. Where hosts do not have good immunity, often do not have good vaccines, e.g. feline calicivirus. Cats do not appear to be resistant to re-infection with FCoV.



www.catvirus.com

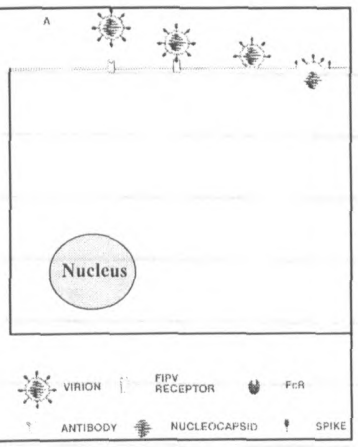
Addie D.D. *et al*, in press. J. Gen Virology.

FIP vaccines: a brief history

Inactivated FCoV	**ADE**
Avirulent strains of FCoV	**ADE**
Temperature sensitive mutant FCoV	**protects**
Heterologous virus:	
TGEV	**no protection**
CCoV	**no protection + ADE**
HCV 229E	**no protection**
Recombinant vaccinia expressing FCoV antigens:	
S	**ADE**
M	**?**
N	***no protection***
CCoV-S	**ADE**
Deletion variants without 3abc or 7ab	**protects**

www.catvirus.com

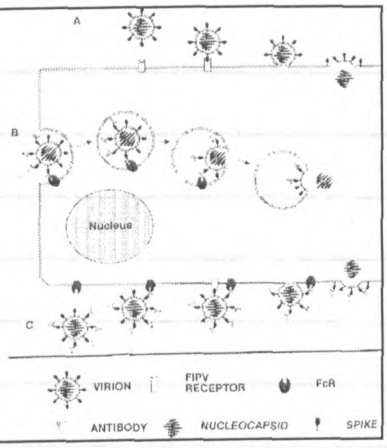
A: normal infection, attachment to cellular receptor



www.catvirus.com

A: normal infection, attachment to cellular receptor

B & C: ADE, absorption requires only Fc receptor



From: De Groot R.J., Horzinek M.C. 1995. *FIP. The Coronaviridae*, Ed S.G. Siddell., 293-315

Antibody dependent enhancement/early death syndrome

- more seropositive than seronegative cats develop FIP
- seropositive cats die of FIP more rapidly than seronegative cats
- sensitised cats sometimes exhibit bizarre pathology

..... in experimental infections, but not in natural infections

www.catvirus.com

Feline coronavirus survey

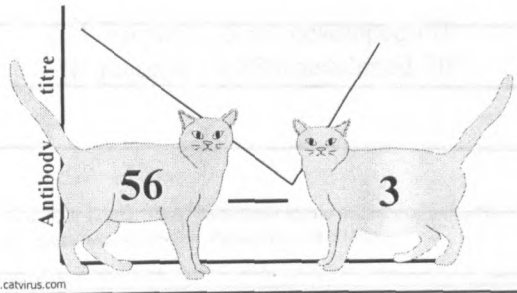
United Kingdom

1988 - 1994

820 cats
73 households

Addie D.D., Toth S., Murray G.D., Jarrett O. 1995 *The risk of feline infectious peritonitis in cats naturally infected with feline coronavirus.* Am. J. Vet. Res. **56** 4 429-434

Of 56 seropositive cats which were reinfected: only 3 developed FIP



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At the time of the index case

43 cats of 304 (14.0%) developed FIP

At the time of the subsequent case

14 cats of 158 (8.8%) developed FIP

Conclusion: ADE is a laboratory artefact

Addie D.D., Toth S., Murray G.D., Jarrett O. 1995 The risk of feline infectious peritonitis *in cats naturally infected with feline coronavirus.* Am. J. Vet. Res. **56** 4 429-434

www.catvirus.com

HE SURVIVED

THE NEIGHBOR KID'S BB GUN,

A DOBERMAN,

A GARBAGE TRUCK,

FALLING ASLEEP ON A CAR ENGINE

AND A FALL

OFF THE ROOF

IT'S A SHAME HE WASN'T

VACCINATED WITH PRIMUCELL® FIP.

www.catvirus.com

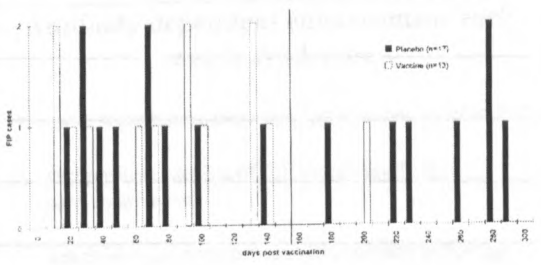
Primucell: intranasal temperature sensitive mutant

Protection correlated with IgA and lymphocyte proliferation response

Some questions about Primucell:

- does it work?
- does Primucell cause ADE?
- could Primucell stop FCoV shedding?
- does Primucell affect FCoV antibody titres?
- does Primucell recombine with endemic FCoV strains?

www.catvirus.com



Fehr D, Holznagel E, Bolla S, Lutz H., Hauser B., Herrewegh AAPM, Horzinek MC. 1995. Evaluation of the safety and efficacy of a modified live FIPV vaccine under field conditions. Feline Pract 23:83--88

www.catvirus.com

There is no point in Primucell vaccinating cats who have already been exposed to FCoV:

either they are incubating FIP....

..... or they are already immune!

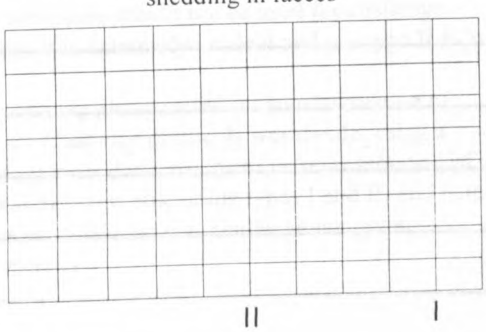
Rescue shelter: 500 cats

254 vaccine 0.8% developed FIP
246 placebo 3.25% developed FIP

Postorino Reeves N. 1995. Vaccination against naturally occurring FIP in a single large cat shelter. Feline Pract 23: 81--82

www.catvirus.com

Primucell vaccination does not prevent FCoV virus shedding in faeces



www.catvirus.com

Effect of Primucell on FCoV shedding:

FCoV shedder kept shedding	21
Negative cat stayed negative	18
Negative cat started shedding	12
Positive cat stopped shedding	13

www.catvirus.com

Effect of Primucell on FCoV antibody titre:

IFAT stayed the same	46
IFAT rose	3
IFAT fell	7
Seroconverted	3

www.catvirus.com

Some questions about Primucell:

- does it work? Yes
- does Primucell cause ADE? No
- could Primucell stop FCoV shedding? No
- does Primucell affect FCoV antibody titres? No
- does Primucell recombine with endemic FCoV strains? ??? Probably not

www.catvirus.com

Second International FCoV/FIP Symposium

4-7 August 2002

www.felinecoronavirus.com

Recommendations from SIFFS

The ideal vaccine:

- should protect against FIP
- give good mucosal immunity to
- prevent infection and reduce virus shedding

Development of a therapeutic vaccine should also be considered: both to treat cats with FIP and to attempt to stop carrier cats from shedding

In press: JFMS and to be published on www.felinecoronavirus.com

Recommendations from SIFFS

79-1146 strain should not be used for challenge studies: it is extremely virulent and is a type II FCoV

The working group called for standardisation of vaccine challenge protocols worldwide, using a constant virus dose, strains more representative of natural infection (including types I and II) and natural exposure challenge (i.e. challenge not given parenterally).

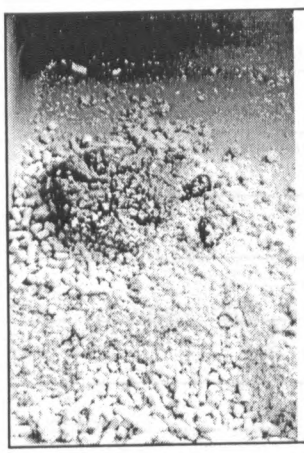
In press: JFMS and to be published on www.felinecoronavirus.com

Which cats need a FCoV/FIP vaccine?

- cats going into rescue or boarding catteries
- all pet cats
- cats going into rescue shelters
- pedigree/purebred cats

NOT seropositive cats

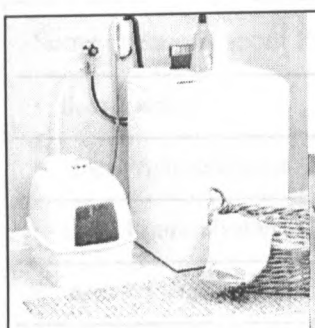
www.catvirus.com



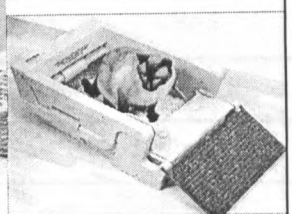
The main way to prevent FCoV infection is to prevent uninfected cats coming into contact with used cat litter of infected cats.

FCoV can survive up to 7 weeks in dried up cat litter.

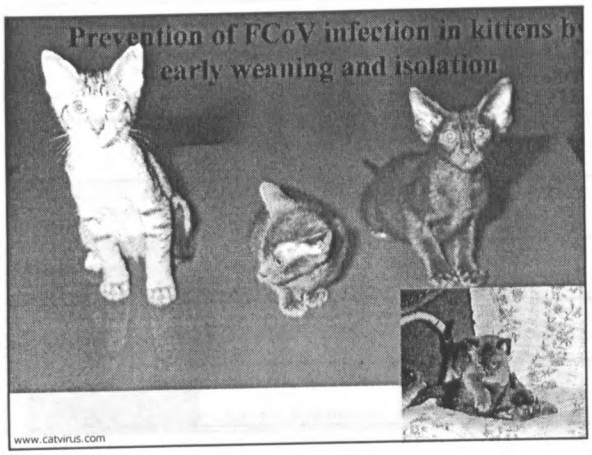
www.catvirus.com



LitterFree Self-Washing Cat Box



Mega Deluxe LitterMaid Automatic Self Cleaning Litterbox



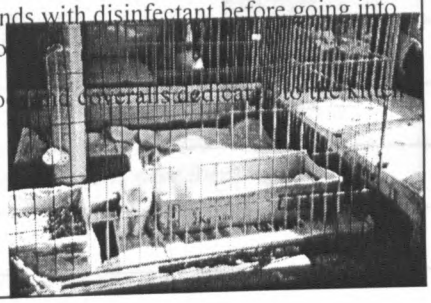
Prepare kitten room

1. **Remove all cats and kittens 1 week before putting queen in.**
2. **Disinfect room as far as possible using 1:32 dilution of sodium hypochlorite (Domestos or Milton).**
3. **Dedicate litter trays, food and water bowls to this room and disinfect with sodium hypochlorite.**
4. **Introduce queen 1-2 weeks before she is due to give birth.**

www.catvirus.com

Barrier nurse kittens

1. Deal with the kitten room before tending other cats.
2. Clean hands with disinfectant before going into kitten roo
3. Have sho overalls dedicat to the kitten room.



www.catvirus.com

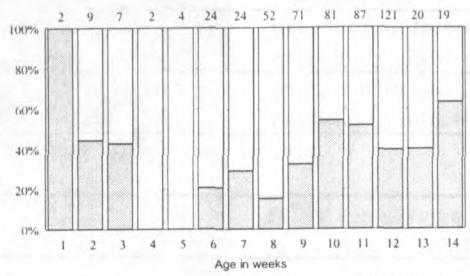
Early wean and isolate kittens

1. Test queen for FCoV antibodies either before or after kittening.
2. If queen's antibody titre is > 0, the kittens should be removed to another clean room when they are 5-6 weeks old.
3. If the queen has an antibody titre of zero, she can remain with the kittens until they are older.
4. Take care to socialise isolated kittens to accustomise them to humans during the 2-7 week old period.

Test kittens for FCoV antibodies at over 10 weeks of age to ensure that early weaning and isolation has worked.

www.catvirus.com

FCoV antibodies in kittens



www.catvirus.com

	Oct 87	Dec 87	Jan 88	Oct 88	Apr 89
5624	320	1280	40	0	20
5625	320	320	20	0	40
5626	160	1280	80	0	320
5627	160	320	80	10	80
5628	80	80	20	0	40
5629	40	20	20	0	160
5632	0	0	0	0	320
5334	1280	pts Nov 97, non-effusive FIP			Jan 89 new cat, 80

Rising antibody titres do not lead to the development of FIP

FCoV TESTED
STUD AND QUEEN REGISTER

www.felinebreeder.com

First USA registrant: Kit Humphrey, breeder of Siberians

Buber Mad[illegible]r

[illegible]evra of the Renaissance

	Mar 96	May 96	Jul 96	Aug 97	Jan 98	
Sooty	640	640	640	>1280	>1280	
	+	+	+	+	+	carrier
Todd	1280	1280	320	20	10	
	-	+	+	-	-	
Teafa	1280	>1280	640	640	160	
	-	+	+	-	-	
Garfield	640	640	640	160	20	
	+	+	+	-	-	
Skully	80	80	40	20	0	
	+	-	-	-	N/D	
Elsa	10	20	0	N/D	N/D	
	-	-	-			
Brewster	0	0	0	N/D	N/D	
	-	-	-			

Cats with antibody titres as low as 80 can shed FCoV

www.catvirus.com

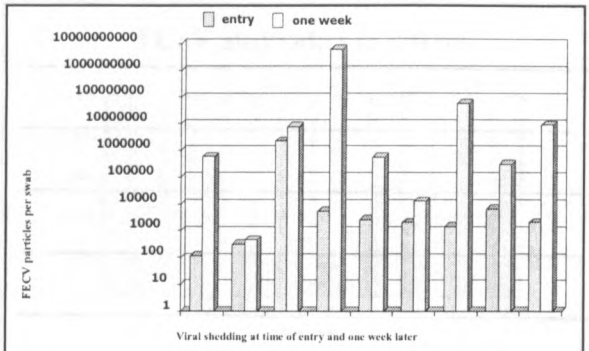
Good barrier nursing

- always tend the healthy animals before the sick ones
- tend young before old
- tend surgery cases, non-infectious medical before infectious (separate nurses if possible)

www.catvirus.com

Avoid stressing cats!

www.catvirus.com



Pedersen *et al*, 2003. In press

www.catvirus.com

Stress in the cat

being rehomed, moving house

new additions to house: baby, dog, cat

too many cats in one house (over 6)

going into cattery

surgery or trauma (e.g. RTA)

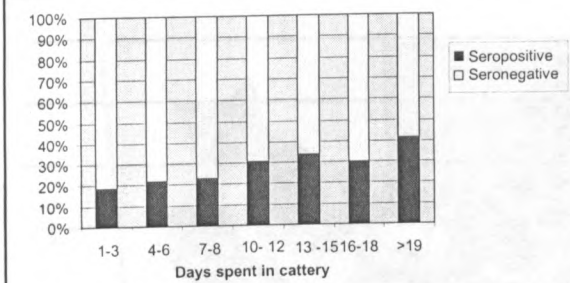
intercurrent illness

pregnancy, parturition, lactation

www.catvirus.com

"It would be better for people to go back to rehoming kittens themselves, instead of relinquishing them to cat shelters!"

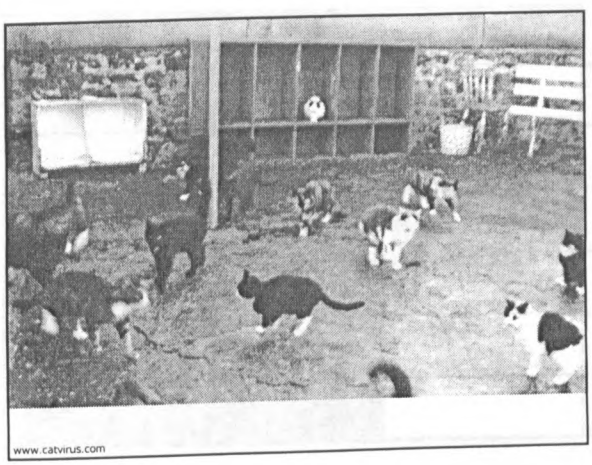
Prof. Niels Pedersen
(University of California)



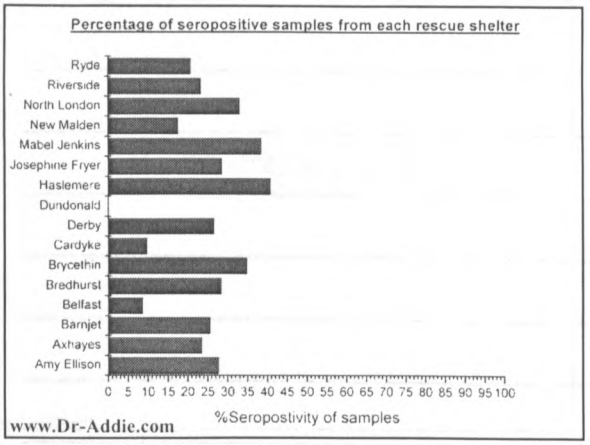
16.8% of cats were FCoV seropositive on day 1. Cats who had been in the cattery longer were more likely to be FCoV seropositive

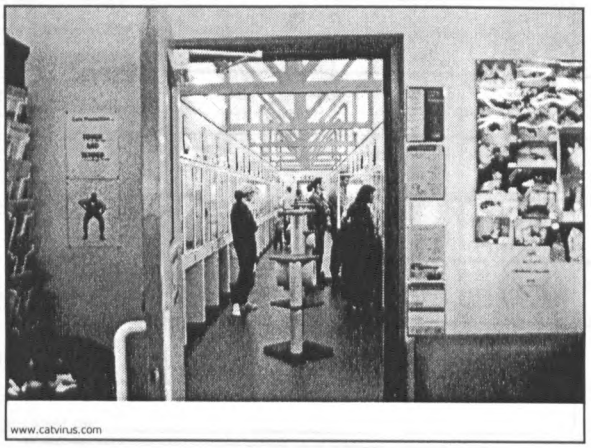
www.catvirus.com

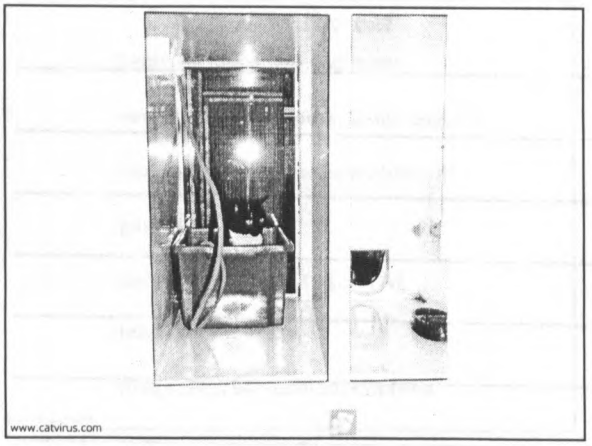
Cave & Addie, in press. JFMS

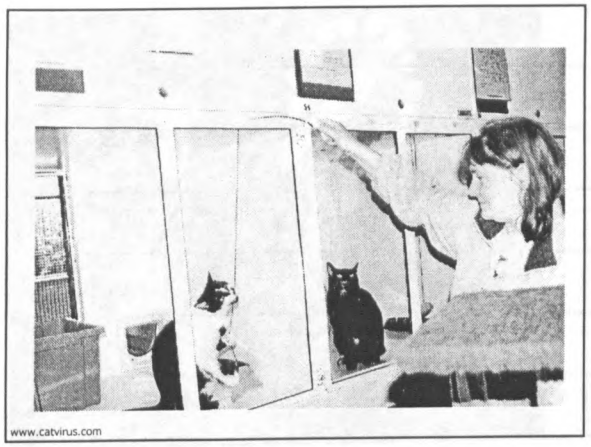


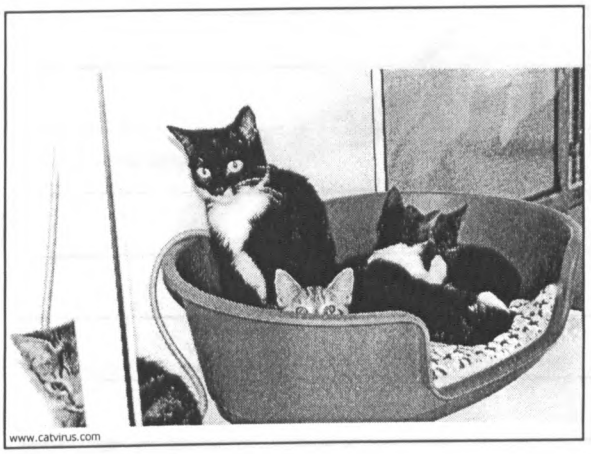
www.catvirus.com

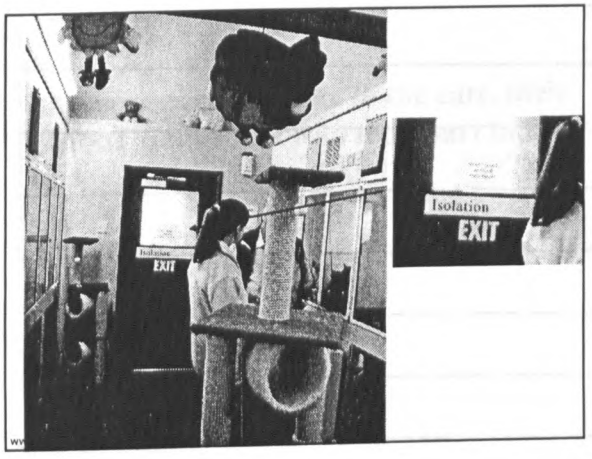

Percentage of seropositive samples from each rescue shelter
Ryde
Riverside
North London
New Malden
Mabel Jenkins
Josephine Fryer
Haslemere
Dundonald
Derby
Cardyke
Brycethin
Bredhurst
Belfast
Barnjet
Axhayes
Amy Ellison
0 5 10 15 20 25 30 35 40 45 50 55 60 65 70 75 80 85 90 95 100
%Seropostivity of samples
www.Dr-Addie.com

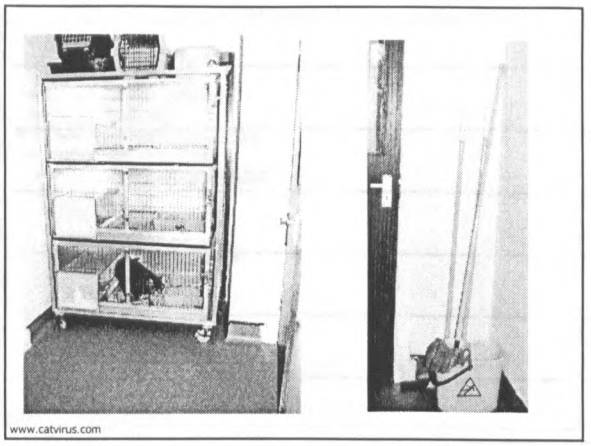

www.catvirus.com

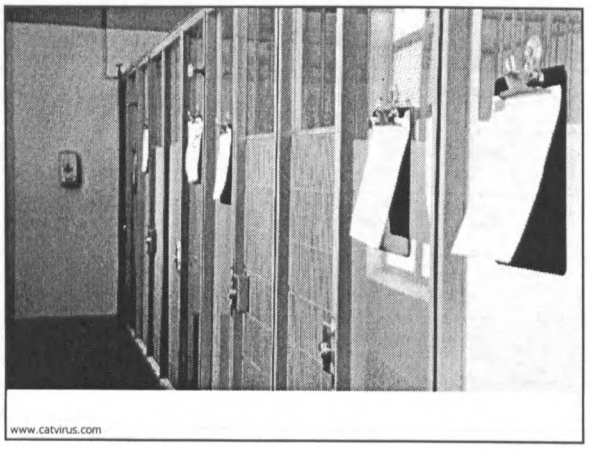

www.catvirus.com

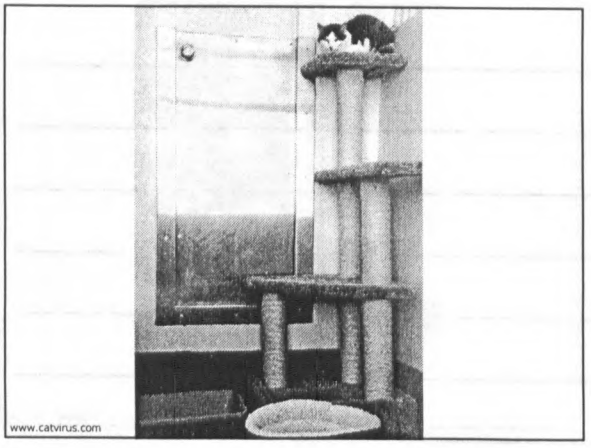

www.catvirus.com


www.catvirus.com


Isolation
EXIT
Isolation
EXIT


www.catvirus.com


www.catvirus.com


www.catvirus.com

A very special thanks to the cats, their humans and vets who took part in my studies!

www.catvirus.com

Thanks to the following for funding:

Winn Foundation
Orion Foundation
Companion Diagnostics
Wellcome Foundation
Royal College of Veterinary Surgeons

and many, many individuals

www.catvirus.com

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Os Jarrett
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Pablo Nart

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Melody Amundson

Administration

Maria Williams
Janet McGrane

www.catvirus.com

FCoV/ FIP Prevention

Diane D. Addie, BVMS, PhD, MRCVS
University of Glasgow
Glasgow, UK

The key to prevention of FIP is to prevent FCoV infection. Largely, this is done by keeping infected and uninfected cats apart, and by excellent hygiene. See table 2. FCoV infection is perpetuated by a cycle of infection, virus shedding, development of immune response, loss of immune response and re-infection. In breeding catteries and ordinary pet households, control has been effected by separating IFAT seropositive and seronegative cats. Cats are tested every 3-6 months and as their antibody titre falls to 20 or less, they are put into a seronegative group which is kept isolated from the seropositive group. The fewer cats the owner has, the better the chance of them eliminating FCoV.

Cat breeders should antibody test their cats for FCoV annually and only mate positive cats with other positive cats, and negative cats with negative cats. A new online register for cat breeders - www.catbreeder.com - allows stud owners to advertise their FCoV tested cats free. Kittens of positive to positive cat matings can be prevented from becoming infected by early weaning and isolation (see table 3).

Table 2. Protocol for minimising FCoV introduction or spread*

A. Reduce the numbers of cats in any area
♦ ordinary house owners should keep no more than 6-10 cats ♦ cats should be kept in stable groups of up to 3 or 4 ♦ in rescue facilities cats should be kept singly
B. In a FCoV eradication programme
♦ cats should be kept in small groups according to their antibody or virus excretion status antibody or virus negative cats together antibody or virus positive cats together
C. Avoid introducing virus to uninfected cats: antibody or virus testing
♦ incumbent cats should be tested before introducing new cats or breeding ♦ only antibody negative or virus negative cats should be introduced into FCoV-free catteries ♦ it is safer to introduce antibody positive cats than antibody negative cats into FCoV-infected households, but there is still a risk of FIP in both the newcomer and the incumbent cats
D. Prevention of kitten infection: early weaning and isolation
♦ both cat breeders and rescuers of pregnant cats should follow the protocol outlined in table 3.
E. Reducing fecal contamination of the environment
♦ have adequate numbers of litter trays - 1 tray per 1-2 cats ♦ litter trays should be declumped at least daily ♦ remove all litter and disinfect litter tray at least once a week ♦ site litter trays away from the food area ♦ vacuum around litter trays regularly ♦ clip fur off hindquarters of long-haired cats
F. Primucell vaccination
♦ at 16 and 19 weeks ♦ for all cats going into high risk environments, such as rescue shelters

*Based on recommendations from working groups of the international feline enteric coronavirus and feline infectious peritonitis workshop [Pedersen *et al*, 1995].

FCoV prevention in kittens

Kittens are protected from FCoV infection by MDA which probably wanes at around 5-6 weeks. This discovery enabled the breeding of uninfected kittens even in households where FCoV is endemic and their queen infected. FCoV free kittens can be advertised on the internet free at www.felinebreeder.com

Table 3. Protocol for prevention of FCoV infection in kittens

Prepare kitten room	1. remove all cats and kittens 1 week before putting queen in 2. disinfect room as far as possible using 1:32 dilution of sodium hypochlorite (Domestos or Milton) 3. dedicate litter trays, food and water bowls to this room and disinfect with sodium hypochlorite 4. introduce queen 1-2 weeks before she is due to give birth
Practise barrier nursing	1. deal with the kitten room before tending other cats 2. clean hands with disinfectant before going into kitten room 3. have shoes and coveralls dedicated to the kitten room
Early weaning and isolation of kittens	1. test queen for FCoV antibodies either before or after kittening 2. if queen's antibody titre is greater than zero, the kittens should be removed to another clean room when they are 5-6 weeks old 3. if the queen has an antibody titre of zero, she can remain with the kittens until they are older
Test kittens	1. test kittens for FCoV antibodies at over 10 weeks of age

Minimise stress in the seropositive cat

Around 1 cat in 10 who is infected with FCoV develops FIP. Cats which have FCoV antibodies should not be stressed if at all possible - for example, don't rehome them; delay having them neutered or any other operation which is not life-saving; if you have to leave them get somebody to look after them in their home rather than putting them into kennels.

References and further information

The proceedings of the Second International FCoV/FIP Symposium (SIFFS) will be published in the Journal of Feline Medicine and Surgery. The abstracts from SIFFS are on the website:

www.felinecoronavirus.com

http://www.Dr-Addie.com - a website devoted to FCoV/FIP

Addie, D.D. & Jarrett O. Feline Coronavirus. 1998 Infectious Diseases of the Dog and Cat. 2nd edition. Editor: Greene, Craig. Published by W.B. Saunders Company, The Curtis Center, Independence Square West, Philadelphia, Pennsylvania 19106. 58-68

Addie, D.D., and Jarrett, J.O. 2001. Use of a reverse-transcriptase polymerase chain reaction for monitoring feline coronavirus shedding by healthy cats. Veterinary Record. **148**:649-653.

Addie D.D., Schaap I.A.T.; Nicolson L., Jarrett O. 2003 Persistence and transmission of natural type I feline coronavirus infection. J. Gen Virol. In press.

Herrewegh A.A.P.M., Vennema H., Horzinek M.C., Rottier P.J.M, de Groot R.J. 1995 The molecular genetics of feline coronaviruses: comparative sequence analysis of the ORF7a/7b transcription unit of different biotypes. Virology. 212:622--631

Herrewegh A.A.P.M, Mahler M., Hedrich H.J, Haagmans B.L, Egberink H.F., Horzinek M.C., Rottier PJM, de Groot RJ. 1997 Persistence and evolution of feline coronavirus in a closed cat-breeding colony. Virology **234** 349-363

Herrewegh A.A.P.M, Smeenk I., Horzinek M.C., Rottier P.J.M, de Groot R.J. 1998 Feline coronavirus type II strains 79-1683 and 79-1146 originate from a double recombination between feline coronavirus type I and canine coronavirus. J. Virol. 72 5 4508-4514

Horzinek M.C. 1997 Update of feline infectious peritonitis. Feline Focus. 5 1-4

Pedersen N.C., Addie D., Wolf A. 1995. Recommendations from working groups of the international feline enteric coronavirus and feline infectious peritonitis workshop. Feline Practice 23:108-111.

Vennema H, Poland A, Foley J, Pedersen NC. (1998) Feline infectious peritonitis viruses arise by mutation from endemic feline enteric coronaviruses. Virology. 243 (1) 150-157

Watari T, Kaneshima T, Tsujimoto H, Ono K, Hasegawa A. Effect of thromboxane synthetase inhibitor on feline infectious peritonitis in cats. J Vet Med Sci. 1998 May; 60(5):657-9.

Weiss RC. Feline Infectious Peritonitis Virus: Advances in Therapy and Control. Consultations in Feline Internal Medicine 2 Edited by John R. August. Published by W.B. Saunders Company.

Information for clients

http://www.Dr-Addie.com

FCoV/FIP leaflet available from Cats Protection, 17 Kings Road, Horsham, West Sussex, RH13 5PP, United Kingdom.

Orion Foundation http://www.devonheaven.com/users/orionsociety/

Notes

15th Annual Fred Scott Feline Symposium
July 25 - 27, 2003

An Overview of Feral Cat Issues

Margaret Slater, DVM, PhD
Dept. of Veterinary Anatomy & Public Health
College of Veterinary Medicine
Texas A&M University

An Overview of Feral Cat Issues

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College of Veterinary Medicine
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Definitions

- Free-roaming cats
  - Any cat not confined to an owner's property
  - Indoor/outdoor cats
  - Stray (lost) or abandoned cats
  - Feral cats (unsocialized)
- Different groups need different solutions

Additional Definitions

- Sociability Spectrum
  - from social pets to no contact with humans (feral)
- Ownership Level
  - cherished pet/committed owner to no human involvement
- Location Description
  - barn cat, house cat, doorstep colony
- Colony
  - >2 reproductively mature cats

Cats in the US

- Most popular pet
  - At least 59 million (1996)
- 32 million households (1998)
- 78% have taken cat to vet in past year (2000)
- 63% believe overpopulation is most important pet issue

Importance

- Free-roaming cats seen in all companion animal practices
- Recent survey TX, TN
  - 2/3 of practitioners saw feral cats
- Related to strength of human-cat bond
- Key to cat overpopulation problem

Extent of the Feral Cat Problem

- 1993 CA: 10% fed unowned cats
- 1995 CA: 9% fed unowned cats
- Early 1990's MA: 15% fed unowned cats
- 1999 FL: 12% fed unowned cats
- Feral cats are both a cause and effect of pet overpopulation

Ferals and Overpopulation

1000 pet cats	1000 stray/feral
50% female	50%? female
80% spayed	0% spayed
16% litters before spay	
2 litter/y/cat=264	2 litter/y/cat = 1000
3 kittens to 1 yr	2 kittens to 1 yr
792 kittens/y/1000 cats	2000 kitten/y/1000 cats

Sources of Feral Cats

- Strays: about 20% may leave household as strays
- About 22% of cats acquired as strays
  - 1995-1996 data
- 32% of relinquished cats from a friend
- Feral cats are a people problem

Potential Sources

- Owned cats: 15-20% have litters before being spayed
  - cost and lack of knowledge about cat reproduction were major factors
- Abandonment
- Lack of animal shelter

Issues

- Public Health
- Impact on Wildlife and Birds
- Welfare of Cats
- People's Perceptions
- Management of Colonies

Public Health

- Rabies
  - In areas with high frequency
    - Cats are most common domestic species
  - Vaccination is very effective
  - May provide a barrier between wildlife and humans
  - No human cases from cats in decades

Public Health

- Toxoplasmosis
  - Not related to presence of cats in neighborhood
  - Shed mostly by young hunting cats
- Ringworm
- Bartonellosis
- Bites
- Larval migrans
  - Round and hookworms poorly controlled in all cats

Wildlife and Birds

- Cats are opportunistic hunters, they eat:
  - Common prey species
  - Carrion
  - Garbage and cat food
- International studies show they are rodent specialists
- Often large #s in urban or suburban areas with little native wildlife already

Philosophical Opposition

- Cats are a domestic species
  - Should not be allowed to hunt native wild species
    - Not regulated in the ecosystem (usually) in same way as wild predators
  - Should be controlled/confined
  - Are places where rodent control is important

Islands and Feral Cats

- Islands may have limited number of species and adaptations
- Cats may cause serious problems
  - Often deliberately introduced for rodents
- On large islands, other predators commonly introduced also
  - Foxes, mongooses, rats
- May be livestock, changes in habitat
- Predator-prey systems are complex

Common Wildlife Concerns

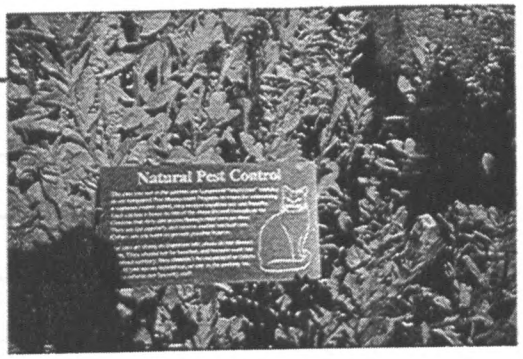
- Cats kill x millions of birds
  - Number 1 cause of bird and wildlife decreases are habitat destruction
  - Also, window strikes, other birds and predators, pollution kill many birds
  - No documentation of cat impact on species in mainland ecosystems
  - Arguments over numbers killed irrelevant
    - We all want there to be fewer feral cats
    - Usually numbers are extrapolated

Other Wildlife Problems

- Cat are exotic/introduces species, therefore they must be removed
  - But "Americans" are exotic to North America
  - So are livestock species, and native species are killed to protect them
  - Assumption is that if remove cats, ecosystem will return to "normal"

Wildlife Biologists and Feral Cat Activists

- Have much in common:
  - Keep cats indoors or confined
  - Convert outdoor cats to indoor
  - Spay/neuter before 4 months
  - Educate about abandonment and finding a new home
  - Support ordinances for id, vaccination



Welfare of the Cats Themselves

- Safety of any free-roaming cat
  - confinement to yard, enclosure, leash
- Quality of life
  - health
  - human contact
- Disease transmission
  - Between cats and cats and wildlife

Peoples' Perceptions

- From vermin to working animal to family member!
- Nuisance vs. pleasure
  - Footprints, feeding cats, etc.
- Anecdotal info and emotion

Management of Colonies

- Quality of life issues
- Level of care provided
- Effectiveness in population control
- As part of community level approach

Community Level Solutions

- Sterilization
- Education
- Adoption
- Fostering
- Identification
- Some laws/ordinances?
- Feral Cats/Caretakers

Conceptual Outline of Feral Cat Constituencies

- Grass-roots organizations
- Service providing and enforcement groups
- Resource and Teaching providers

Grass-roots Organizations

- Individuals, loose-knit groups and established non-profits
- Cat care, education, start new groups
- Two approaches for established groups
  - high volume subsidized spay/neuter
  - broad range of programs relating to cats

High Volume Feral Spay/Neuter

- Objective is to sterilize as many feral cats as possible
- Contact/visit programs: Feral Cat Coalition, Operation Catnip
- This will require
  - veterinary volunteers
  - a physical location
  - organizational skills

How to Start a TNR Organization

- Find a person with good leadership skills, patience, endurance (no cats required)
- round up individuals interested in cats
- explain TNR
- work with veterinarian/clinic
- divide the jobs up
- consider obtaining 501C3 status

Service Providing/ Enforcement Groups

- Animal Shelters
- Veterinary Hospitals
- Animal Care and Control Agencies
- Animal Industry Businesses
- Public Health agencies
- Others

Animal Shelter Involvement

- Any physical location for animals
- Involved with feral cats:
  - do not accept cats
  - accept and euthanize (+/-holding)
  - compete for adoptions with feral kittens
  - working with caretakers

Approaches for Animal Shelters

- Education (responsible pet ownership)
- Spay/neuter support
- Adoption of tame cats
- Services for feral cat caretakers
- Formal partnerships with established groups

Shelters and Caretakers

- loaning traps/assistance with trapping
- subsidized spay/neuter
- recognition of ear-tipped cats
- accepting tame cats and kittens for adoption
- foster home assistance
- education about caring for feral cats
- central referral source for info/contacts

Veterinary Involvement

- Education (spay/neuter, identification, pet overpopulation, etc)
- Spay/neuter surgeries
- General health care
- Euthanasia
- Spokesperson

Animal Care and Control Agency Issues

- May not deal with cats (no ordinances)
- May not be able to shelter cats
- Do get the complaints
- May not be well-funded
- Often reactive and not proactive

Animal Care and Control Agency Issues

- Legislation is not always helpful with feral cat issues
- Create written policy statements about feral cats
- Partner with shelters, caretakers to maximize community gains
- Flexibility is key; try to fix, not punish

Laws/Ordinances and Feral Cats

- Spay/neuter for pets from shelters
  - Most can agree to importance
  - May not have funds or veterinary support
- Definition of ownership
  - Varied by location, some no legal standing
- Identification vs licensing
  - Licensing viewed as revenue or control
  - Identification can be positive for all

Laws/Ordinances, Cont'd

- Registration of feral cat caretakers
  - Provides oversight by government
  - But viewed as "big brother" or out to get cats
  - Only will work in places with good relationship
- Nuisance vs. running at large, limit laws
  - Leash and limit laws can make TNR illegal
- Enforcement on complaint
  - Means that enforced irregularly

Resource and Teaching Providers

- Individuals or Groups
- Trappers
- Fund raisers
- Veterinarians
- Caretakers
- Scientists

Complicated Situation

- Critical for veterinarians to be informed of all aspects and views
- Must consider role of veterinary medicine in decreasing feral cat #s
- Do not accept simplistic or black and white explanations—need information on actual local situation

Acknowledgements

- Veterinarians around the country
- Animal protection professionals
- Caretakers
- Humane Society of the United States
- All the AFCAT coordinators and volunteers
- VMTH, CVM

Trap, Neuter and Return of Feral Cats: Efficacy & Rationale

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Choices for Control

- Trap and remove
  - For euthanasia
  - Into sanctuaries
  - Into new locations
- TNR
  - Including adoption!
  - Better community support
  - May be done with volunteers, donations

TNR vs Remove/Eradicate

- REMEMBER
- On Marian Island
  - 20 years to eradicate
  - Viral infections
  - Night hunting, trapping
  - Poisoning
- On Macquaire Island
  - $4.5 million dollars
  - 30 years

Trap, Neuter and Return

- A method to control and eventually reduce the numbers of feral (free-roaming) cats
- Often good public support
- Some demonstrated efficacy
- Trap and remove programs without habitat modification are unsuccessful
- Euthanasia of homeless cats is #1 cause of death

Trap, Neuter and Return

- Non-lethal control method for feral cats
- Long term goal is fewer cats
- Ongoing caretaker will be most effective
- Ideally includes adoption of tame cats in colonies and young kittens
- Must include vaccination for rabies
- Ear-tipping or notching for permanent identification

TTVAR-M Ideal

- Trap (adopt tame adults and kittens)
- +/- test for FeLV and FIV
- Vaccinate (at least for rabies)
- Spay or castrate (as early as 8 weeks/2 lbs)
- Ear tip or notch
- Provide with shelter, food, water, care
- Monitor daily by caretaker

TNR Benefits

- Sterilization decreases fighting, roaming, noise
- No breeding so no litters, less stress
- No kittens, no increase in number
- Some stability from incoming/new cats
- Overall improvement in health
  - Gain in weight, body condition after neutering
- Caretakers often report become more sociable, improved coat quality

TNR Efficacy

- Increasing data on control of cat #s
- Originally in Denmark and UK
- Regent's Park, London
  - 2 groups
    - 4 adults/6 kittens; 5 adults/2 kittens
  - No kittens born
  - Park authorities satisfied

University of Central Florida

- 1991-2002, 11 years, 155 cats total
- 75% were feral
- 56% were kittens (< 6 mo of age)
- At end of complete census (1996) down to 68 cats
- By 2002 down to 23 cats (one tame)

U of Central Florida, Cont'd

- 23 remaining cats (15%)
  - 7 year median duration of residency
- No kittens after 1995 (5 years)
- 47% of cats were adopted
- 15% disappeared
- 11% euthanized, 6% died
- 6% moved to nearby woods

Operation Catnip

- High volume spay/neuter for ferals in FL and NC
  - 57% female cats
    - 19% were pregnant, peak of 47% in April
  - Cryptorchid males 19.%
  - 1.9% already neutered
  - Unexpected deaths 0.3% (of 5323 cats)
  - Euthanasia 0.4% for serious diseases

Merrimack River Feline Rescue Society

- Tourist town with feral cat complaints
- Chamber of commerce, vets, public health
  - formal TTVARM program instituted
- Many tame cats/kittens with no local shelter
- Limited admission, cat only shelter
  - 8000 adoptions in 10 years
- Expanded programs, building, website

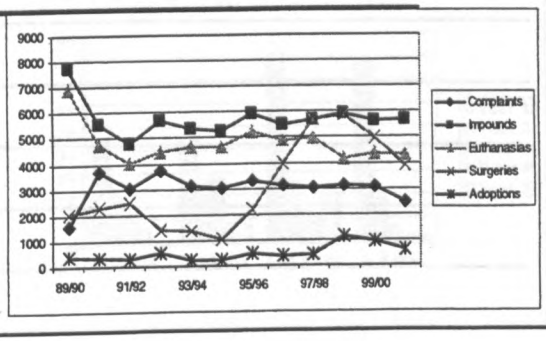
MRFRS

- About 4000 cats in TNR program in the region in past 10 years
- About 200 feral cats in town neutered originally
- About 20 feral cats in town now
  - All senior cats, most > 12 years
  - In some areas, there are no cats at all
- No kittens born on water front past 5 years

Orange County Florida

- Animal Control partnered with non-profit feral cat organization
- Cat group handles complaints, trapping
- Shelter does surgery, rabies, ear-tipping
- Cooperative agreement
- Improvement in community relationship
- Increase in human population >30%

Orange County Cat Problems



San Francisco SPCA

- Shelter with feral cat program
- Free spay/neuter with $5 to person
- Educational programs
  - 24 hour voice mail
  - Cat Workshops
- Advocacy and mediation
- Traps and food
- Expert advice and cat community network

SF/SPCA Cat Data (93-99)

- Impounds down 28%
- Total cat euthanasia down 71%
  - feral cat euthanasia down 73%
- 47,289 cat surgeries (increase of 23%)
  - increase in feral sterilization of 275% (~1900 feral cats in 1999)
- neonatal euthanasia dropped from >900 to 200 kittens

Aggie Feral Cat Alliance of Texas

- Managed feral cat colonies on Texas A&M University campus
- Campus volunteer feeders/caretakers
- Fourth year veterinary students do surgeries
  - PE, vaccines, testing for FeLV, FIV, ear tipping
- Graduate students do trapping/release
- Linked to other cat-related studies

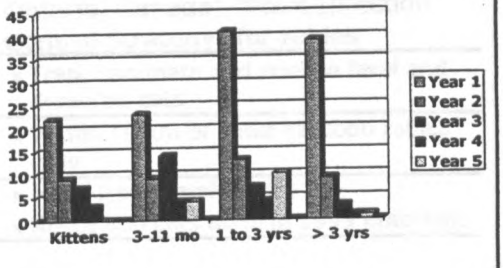
AFCAT (www.cvm.tamu.edu/afcat)

- Supported by:
  - Summerlee Foundation
  - ASPCA Partners in Caring
  - Ralston Purina/Friskies
  - Heska/Schering-Plough/Ft Dodge
  - AVID & Home Again microchip
  - Idexx laboratories
  - Veterinary Medical Teaching Hospital

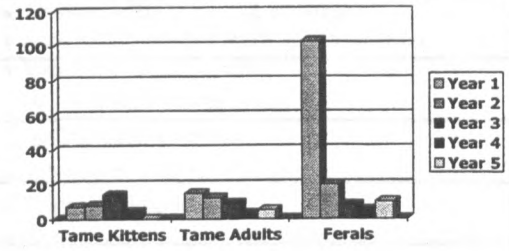
AFCAT (TAMU) Results

- Nearly 5 years duration
- 212 cats, 105 returned to campus
  - 13 "ferals" became socialized/adopted
  - 7 died of trauma or disease
- complaints on campus dropped
- no kittens born by second spring
- ~1/4 of cats due for revaccination re-caught
- much positive publicity for CVM

Age of Campus Cats at Trapping



Socialization Status



BFCA April 2000-2003

- 378 ferals neutered at veterinary college
  - About 20 done at local practice
- Over 100 adoptions
  - All health care through 3 local practices
    - Provide discounted service
    - BFCA brings vaccines, test kits, microchips
    - Do own worming, de-flea-ing
- More than 60 caretakers in area

Other TNR Issues

- Rabies and zoonotic diseases
- Cat health
- FeLV / FIV testing
- Human-cat bond

Feral Cats and Rabies Control

- Historically, rabies control:
  - Restrict movement of animals
  - Remove free-roaming animals
  - Vaccination of susceptible animals
- Current recommendations
  - Understand animal-human relationships
  - Develop culturally appropriate approach

Rabies Control in Dogs

- In countries with dogs as primary rabies reservoir
  - Elimination of dogs by any method had no long-term effect on population size
  - Animosity toward program personnel
  - Decreased cooperation with vaccination
  - **WHO no longer recommends removal**

Free-roaming/Feral Cats and Rabies

- Ontario, Canada: "Point Infection Control Strategy" for rabies
  - Trap, vaccinate and release feral and domestic cats
  - Within 10 km of initial raccoon rabies case
  - Many free-roaming cats
  - About 800 cats done in 1999 outbreak

Feral Cats and Rabies Vaccination

- Connecticut study of rabies vaccination compliance in pets
  - Vaccination is more common in homes where rabies is considered a problem
  - Cats vaccinated less often than dogs
- Early rabies vaccine, single dose
  - Provided complete protection > 3 years

Cat Health Issues

- In FL, fleas, ear mites common
  - Skin problems, ticks rare
  - Gain weight and body fat
  - Euthanasia other diseases 0.4% (FL)
- In TX, fleas common
- Overall, FeLV rates about 5-6%
- Overall, FIV rates about 2-8%
  - Comparable to owned cats

FeLV and FIV Testing in Feral Cats

- Cannot hold and retest like pets
- Cost may impede spay/neuter efforts
- Low frequency of positive cats: 4-6%
- Spaying prevents maternal infection of kittens
  - Major mechanism for FeLV spread
- Castration decreases fighting/breeding
  - Major mechanism for spread of FIV
- In these low prevalence populations, will likely have false positive test results

FeLV Calculations for 1500 Cats (Levy 2002)

Nothing done	Catnip, with FeLV testing	Catnip, no testing
1500 intact cats, 65% female, 60+ cats	667 intact cats, 434 female, 27 +	0 intact cats
	833 neutered, 33 + euth	1500 neutered, 60 + alive
6 kit/yr/fem =5850 kittens, =176+ kits	2604 kittens, 75% get FeLV from mom=78+	0 kittens
Total: 5850 extra cats, 236 FeLV+	2604 kittens and 105+ cats	60 + adults, no extra cats

Feral Cats and Caretakers

- Primarily female, middle aged, living with partner
- Employed
- 8-12% did not own pets
- 30-50% caring for > 2 years
- Sympathy/pity; love of animals
- Love of cats; opportunity for nurturing

Resources

- Alley Cat Allies www.alleycat.org
- Merrimack River Feline Rescue Society www.mrfrs.or
- Operation Catnip FL www.operationcatnip.com
- Operation Catnip NC www.operationcatnip.org
- San Francisco SPCA www.sfspca.org
- National Council for Pet Population Study and Policy www.petpopulation.org

References

- Island eradication
  - Bester et al. S Afr J Wildlife Res 32(1):65-73, 2002.
- Caretakers
  - Zasloff & Hart. Anthrozoos. 11(4): 242-48. 1998
  - Centonze & Levy. JAVMA. 220(11):1627-34. 2002.

TNR References

- Hughes & Slater. J Appl Anim Welf Sci. 5:15-27. 2002.
  - Also www.cvm.tamu.edu/afcat
- Hughes et al. J Appl Anim Welf Sci. 5:285-98. 2003.
- Levy et al. JAVMA. 222:42-6. 2003.
- Neville & Remfry. Vet Rec. 114:447-50. 1984.

Rabies References

- Beran & Frith. Rev Infect Dis. 10(supplement 4): S672-77 1988.
- Nelson et al. JAVMA. 212:1552-55. 1998.
- Rosatte et al. J Wildlife Dis. 37(2): 265-79. 2001.
- Soulebot et al. Cornell Vet. 71:311-25. 1981.
- WHO. WHO/Rab. Res./88.25. Geneva February 1988.

Cat Health References

- Akucewich et al. Vet Parasit. 109:129-39. 2002.
- Cuffe. Vet Rec. 112:129. 1983.
- Gibson et al. Can Vet J. 43:695-698.
- Hughes & Slater. J Appl Anim Welf Sci. 5:15-27. 2002.
- Lee et al. JAVMA. 220:620-2. 2002.
- Scott et al. J Appl Anim Welf Sci. 5:203-13. 2002.
- Scott et al. JAVMA. 221:1136-8. 2002.

Cat Health References

- Scott et al. J Appl Anim Welf Sci. 5:203-13. 2002.
- Scott et al. JAVMA. 221:1136-8. 2002.
- Levy. Proc AVMA 2002, Feral cats: Controversies and Controls

Veterinarians' Involvement in Feral Cat Control

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Definitions

- Free-roaming cats
  - Any cat not confined to an owner's property
  - Indoor/outdoor cats
  - Stray (lost) or abandoned cats
  - Feral cats (unsocialized)
- Different groups need different solutions

Additional Definitions

- Sociability Spectrum
  - from social pets to no contact with humans (feral)
- Ownership Level
  - cherished pet/committed owner to no human involvement
- Location Description
  - barn cat, house cat, doorstep colony

Veterinarians and Feral/Free-Roaming Cats

- Many needs are within the scope of daily practice
- Will be asked about the issues
- Need understanding to make choices about other kinds of involvement
- Cooperation with other groups will benefit all

Importance

- Numbers of cats
  - US residents own 65-70 million cats
  - Number 1 pet in US
- Millions of free-roaming cats
  - Some end up in shelters
    - > 70% of cats euthanized nationally
    - 2% returned to owners nationally

Veterinary Involvement

- As regular patients
  - Owned indoor/outdoor cats
  - Newly acquired stray cats (20-25%)
  - Adopted from a shelter (16-18%)
- In spay/neuter clinics
- In shelters
- With cat rescue organizations

Roles of the Veterinarian

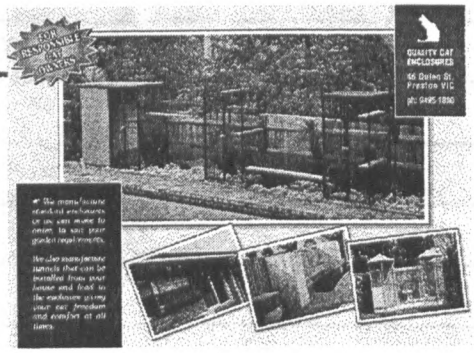
- Keeping cats in homes
- Assisting with adoption of cats
- Controlling the numbers of
  - Feral cats
  - Socialized homeless cats
- Education at all levels

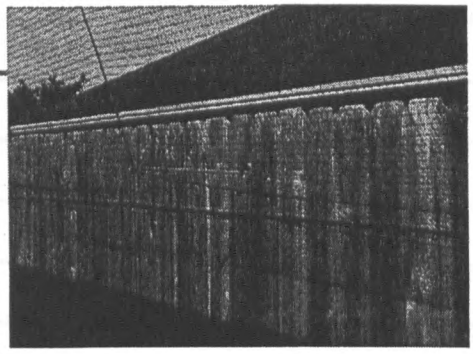
Controlling Free-roaming Cat Numbers

- Spaying and castrating client owned animals
  - Won't have litters outside
  - Won't have unwanted kittens/cats
- Working with homeless cats and groups
  - Individual clients
  - Shelters
  - Cat rescue
  - Feral cat trap, neuter and return programs

Veterinary Solutions For Free-Roaming Cats

- Owned cats
  - Spay/castrate
  - Identification
  - Confinement
    - Yard, porch, cat enclosure
    - Decrease time outside, unsupervised
  - Get owners to do a little more each visit





Veterinary Solutions

- Stray/abandoned cats (socialized)
  - Promotion of identification to prevent loss
    - Microchipping (shown to work in some locations with strong support)
  - Options if can't keep cat
    - How to find a home
    - Shelter, rescue group referrals
  - Promote bond, decrease problems
    - Behavior, socialization programs

Veterinary Solutions

- Feral cats
  - Eliminating the sources
    - Owned unspayed cats
    - Stray unspayed cats
  - Dealing with the existing cats
    - Euthanasia
    - Sanctuaries/rescue
    - Trap, neuter and return

Veterinarians and Feral Cats

- Euthanasia
  - For sick or injured cats
  - Sometimes with testing for FeLV, FIV
  - If in unsafe/inappropriate location
  - Can be a bad public relations option
  - Is almost never an effective long-term control of the population

Veterinarians and TNR

- Existing clients
- New clients
- Cat rescue or TNR group
- Outside of practice:
- Shelters
- Low cost spay/neuter clinics
- High volume TNR program

Trap, Neuter and Return

- Non-lethal control method for feral cats
- Long term goal is fewer cats
- Ongoing caretaker will be most effective
- Ideally includes adoption of tame cats in colonies and young kittens
- Must include vaccination for rabies
- Ear-tipping or notching for permanent identification

Working With Cat Groups

- They may or may not be organized
- They feel very strongly about their work and cats
- Finances are usually limited
- Knowledge may be limited
- Develop personal and practice philosophy and guidelines

Guidelines for Cat Groups

- What services to offer?
- FeLV, FIV testing policy?
- What sort of follow-up is needed?
- What is the financial structure (standard discounts, special pricing)?
- How many cats can be seen?
- What are the practice logistics for scheduling and handling cats?

Staff Considerations

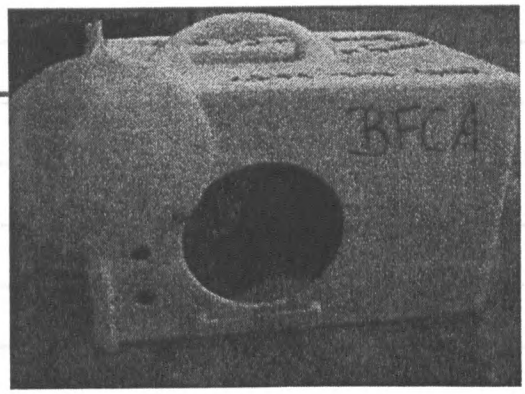
- Dealing with groups or individuals
  - Is there a specific person who schedules or guarantees payment?
- Appropriate behavior of people
- Special handling considerations for feral/frightened cats
- Rabies prophylaxis?

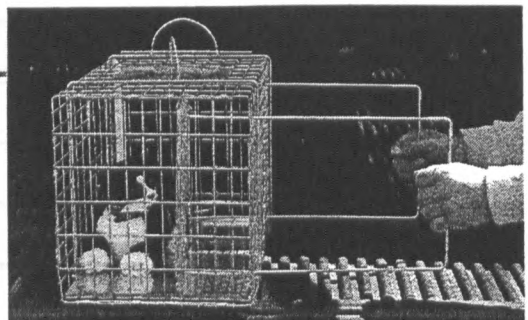
Special Feral Cat Considerations

- Arrival
  - Cages or traps
    - Traps are much easier to manipulate
    - 1 cat per container
    - May be held through hospitalization in trap
    - Cover immediately on arrival
  - Provide a cool down period
    - Dark, quiet area for 15-30 minutes

Special feral cat considerations

- Hospitalization
  - Consider a separate holding area
    - Quieter and for disease control
  - Care when cleaning, handling
    - Feral cat den
    - Squeeze cage
    - Confinement in end of trap
  - Transfer in closed location


BFCA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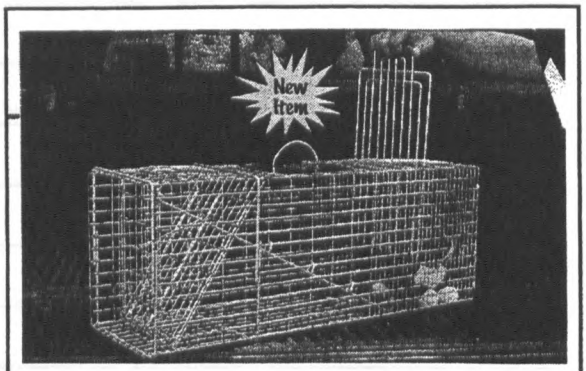
New Item

Special feral cat considerations

- Anesthesia
  - Must use for examination
  - Ketamine and xylazine
  - Ketamine and tiletamine-zolazepam
  - Plus or minus butorphanol
  - Other injectable combinations
- Euthanasia criteria
  - May be appropriate instead of surgery
  - Due to serious illness or injury

FeLV and FIV Testing in Feral Cats

- Guidelines for pet cats not applicable
  - Cannot hold and retest
- Cost of testing may impede spay/neuter efforts
- Low frequency of positive cats: 4-6%
- Spaying prevents maternal infection of kittens
  - Major mechanism for FeLV spread
- Castration decreases fighting/breeding
  - Major mechanism for spread of FIV
- In these low prevalence populations, will likely have false positive test results

Feral Cat Surgery

- Flank vs. ventral midline
  - Each has pros and cons
- Use absorbable skin sutures
- Ear-tipping or notching
  - Crucial for permanent identification
  - Avoid "re-spaying" cats and re-trapping neutered cats
- Release to caretaker after awake or hold
- Return to colony 12-48 hours later

Practitioner's Role

- As part of usual practice
  - Responsible pet ownership
- In being proactive to keep cats and people together
- By understanding the complex issues and sub-populations of free-roaming cats

Acknowledgements

- Veterinarians around the country
- Animal protection professionals
- Caretakers
- Humane Society of the United States
- Humane Society Press
- All the AFCAT coordinators and volunteers
- VMTH, CVM
- Fort Dodge, Idexx Laboratories, Purina

Resources

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Useful References

- Prepubertal spay/neuter
  - Howe. JAVMA 1997; 211:57-62.
  - Howe et al. JAVMA 2000;217:1661-1665
- Specific programs
  - Levy et al. Proceedings 2002 International symposium on non-surgical methods for pet population control. Page 101. Soon in JAVMA.
  - Scott et al. JAVMA 2002; 221:1136-1138.
  - Hughes, Slater. J Applied Anim Welf Sci 2002; 5:15-27
- Relationship to caretakers
  - Zasloff, Hart. Anthrozoos 1998; 11:242-248.

Useful References

- General information
  - Slater. Community Approaches to Feral Cats. Humane Society Press. 2002.
  - Slater. Chapter in August's Consultations in Feline Internal Medicine 2001; 561-570.
  - Patronek. JAVMA 1998; 212:218-226.
- Health
  - Scott et al. J Appl Anim Welf Sci 2002; 5:209-219.
  - Lee et al. JAVMA 2002; 220(5):620-622.

Feral Cats and Wildlife: What Do We Know and What Don't We Know?

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Feral Cats and Wildlife:

What Do We Know and What Don't We Know?

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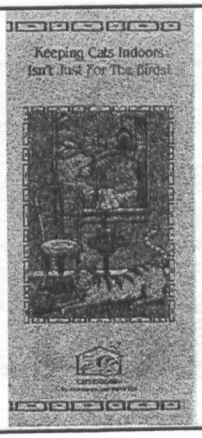
Department of Natural Resources
Cornell University
July 26, 2003

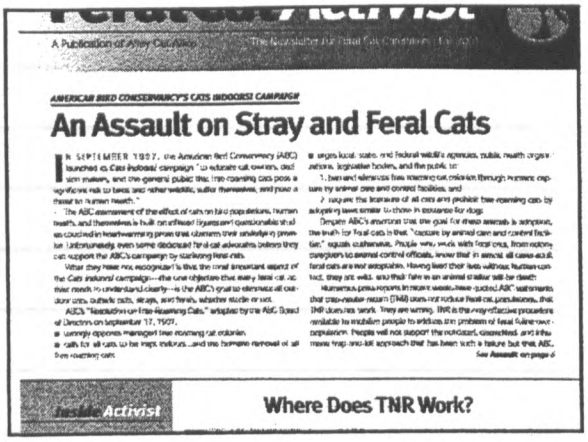
A feral cat problem?



Feral Cat Activist

An Assault on Stray and Feral Cats

Where Does TNR Work?



A Publication of Alley Cat Allies

AMERICAN BIRD CONSERVANCY'S CATS INDOORS! CAMPAIGN

An Assault on Stray and Feral Cats

IN SEPTEMBER 1907, the American Bird Conservancy (ABC) launched its Cats Indoors! campaign "to educate cat owners, decision makers, and the general public that free-roaming cats pose a significant risk to birds and other wildlife, suffer themselves, and pose a threat to human health."

The ABC assessment of the effect of cats on bird populations, human health, and themselves is built on offhand if unsupported assumptions that are couched in heartwarming prose that obscures their underlying unproven falsehoods. Even some dedicated feral cat advocates believe they can support the ABC's campaign by slandering feral cats.

What they have not recognized is that the most important aspect of the Cats Indoors! campaign—the one objective that every feral cat activist must understand clearly—is the ABC's goal to eliminate all outdoor cats: outside cats, strays, and ferals, whether sterile or not.

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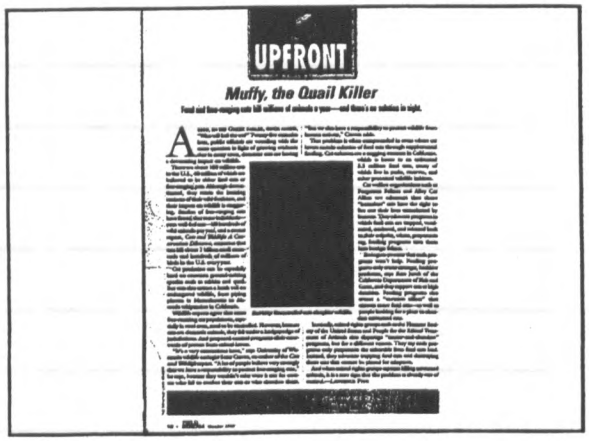
See Assault on page 6

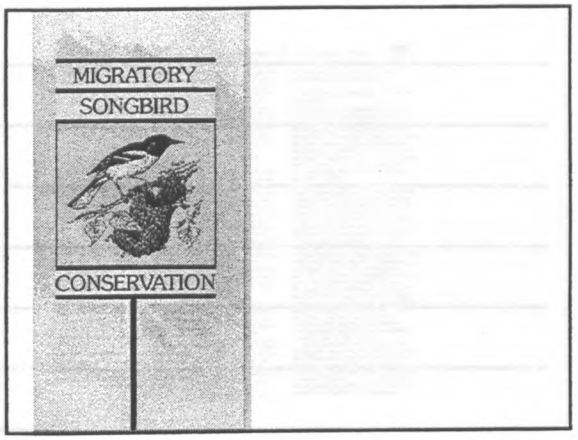
Where Does TNR Work?



UPFRONT

Muffy, the Quail Killer

Feral and free-ranging cats kill millions of animals a year—and there's no solution in sight.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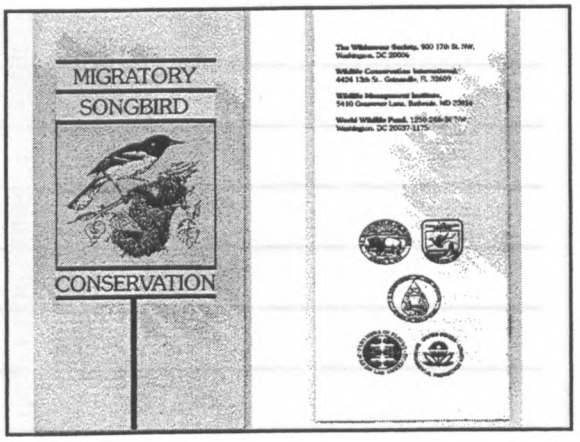
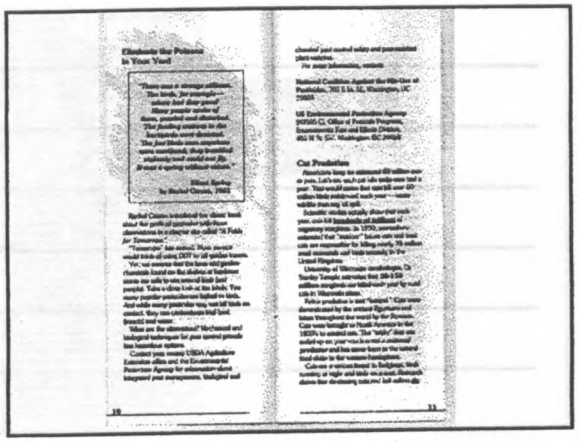

MIGRATORY
SONGBIRD
CONSERVATION
The Wilderness Society, 900 17th St. NW, Washington, DC 20006
Wildlife Conservation International 4424 13th St., Gainesville, FL 32609
Wildlife Management Institute, 5410 Grosvenor Lane, Bethesda, MD 20816
World Wildlife Fund, 1250 24th St. NW, Washington, DC 20037-1175


Cat Predation
10
11

Cat Predation

Americans keep an estimated 60 million cats as pets. Let's say each cat kills only one bird a year. That would mean that cats kill over 60 million birds (minimum) each year — more wildlife than any oil spill.

Scientific studies actually show that each year, cats kill **hundreds of millions** of migratory songbirds. In 1990, researchers estimated that "outdoor" house cats and feral cats are responsible for killing nearly 78 million small mammals and birds annually in the United Kingdom.

University of Wisconsin ornithologist, Dr. Stanley Temple estimates that **20-150 million** songbirds are killed each year by rural cats in Wisconsin alone.

Feline predation is **not** "natural." Cats were domesticated by the ancient Egyptians and taken throughout the world by the Romans. Cats were brought to North America in the 1800's to control rats. The "tabby" that sits curled up on your couch is not a ***natural predator*** and has never been in the natural food chain in the western hemisphere.

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11

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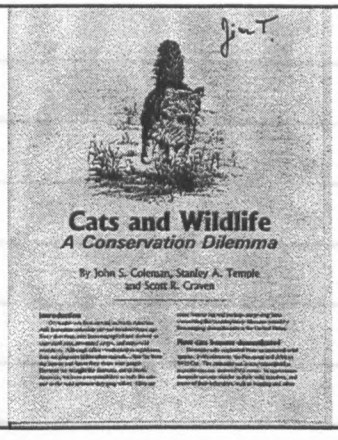

Cats and Wildlife

A Conservation Dilemma

By John S. Coleman, Stanley A. Temple and Scott R. Craven

activity patterns, remain essentially unchanged from their ancestral form. Cats were first domesticated in Egypt around 2000 BC [1]. Domestic cats spread slowly to other parts of the globe, probably because Egyptians prevented export of the animal they worshiped as a goddess. However, by 900 BC the Greeks had acquired domestic cats, and they spread rapidly throughout their sphere of influence. The Romans introduced the domestic cat to Britain by 300 AD. Domestic cats have now been introduced around the world, mostly by colonists from Europe.

How many cats are there in the United States?

The estimated numbers of pet cats in urban and rural regions of the United States have grown from 30 million in 1970 [2] to 60 million in 1990 [3]. These estimates are based on U.S. Census data and include only those cats that people claim to "own" as pets, not cats that are semi-wild or free-ranging. Nationwide, approximately 30% of households have cats. In rural areas where free-ranging cats are usually not regarded as pets, approximately 60% of households have cats. In the state of Wisconsin alone, with approximately 500,000 rural households, the number of rural free-ranging cats that humans own may be as high as 3 million [4]. The combined total of pets and free-ranging cats in the U.S. is probably more than 500 million. Because of bad press associations with humans, most of these cats are unmanaged by areas where people live either free to ensure undeveloped areas.

The legal status of domestic cats

The laws that relate to domestic cats vary by local government. In most areas, the person who provides care for a cat is legally responsible for its welfare and control. As with other domestic animals, if ownership can be established by collars or other means of identification, a cat is considered personal property [5]. It is usually the responsibility of the owner to control the cat's movements. In most areas, cats are to be trapped and either returned to the owner or turned over to authorities if they wander onto other peoples' property. Many municipalities have leash laws and require vaccination and neutering of pet cats. Because laws vary, one should check local ordinances for the appropriate way to deal with stray cats.

What effects do domestic cats have on wildlife?

Although rural free-ranging cats have greater access to wild animals and undoubtedly take the greatest toll, even urban house pets take their prey when allowed outside. Extensive studies of the feeding habits of free-ranging domestic cats over 20 rural and four suburban [6] indicate that small mammals make up approximately 70% of their cats' prey while birds make up about 20%. The remaining 10% is a variety of other animals. The diets of free-ranging cat populations, however, reflect the food locally available.

Observation of free-ranging domestic cats shows that some individuals can kill over 1000 wild animals per year [7], although smaller numbers are more typical. Some of the data on kills suggests that free-ranging cats living in small towns kill an average of 14 wild animals each per year. Rural cats kill many more wild animals than do urban or suburban cats [8]. Several studies found that up to 80% of free-ranging rural cats' diet was wild animals, and less than 10% of rural cats killed no wild animals [9]. Recent research [10] suggests that rural free-ranging domestic cats in Wisconsin may be killing between 8 and 217 million birds each year. The most reasonable estimates indicate that 39 million birds are killed in the state each year. Nationwide, rural cats probably kill over a billion small mammals and hundreds of millions of birds each year. Urban and suburban cats add to this toll. Some of these kills are house mice, rats and other species considered pests, but many are native songbirds and mammals whose populations are already stressed by other factors, such as habitat destruction and pesticide pollution.

Despite the difficulties in showing the effect rural predators have on their prey, cats are known to have serious impacts on small mammals and birds. Worldwide, cats may have been involved in the extinctions of more bird species than any other animal, except habitat destruction. Cats are contributing to the endangerment of populations of birds such as Least Terns, Piping Plovers and Loggerhead

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How many birds do cats kill?

Here are our best guesses at low, intermediate and high estimates of the number of birds killed by rural cats in Wisconsin based on the formula (number of rural cats) x (number of kills/cat/year) x (percentage of kills that are birds).

Low value:
(lowest population estimate) x (lowest kill rate by urban cats) x (our percentage of kills that are birds)
1.4 million cats x 28 x 20% = 7.8 million birds killed by rural cats annually.

Intermediate value:
(mean population estimate) x (intermediate kill rate) x (higher percentage of kills that are birds) 1.7 million cats x 91 x 25% = 39.7 million birds killed by rural cats annually.

High value:
(highest population estimate) x (highest kill rate) x (highest percentage of kills that are birds)
2 million cats x 365 x 30% = 219 million birds killed by rural cats annually.

Note, these estimates do not include predation by urban cats.

Also note, in northern states such as Wisconsin, most kills occur in spring and summer, though predation at winter feeders is substantial. Many of the spring kills would include nestlings and fledglings.

The densities of free-roaming cats in rural Wisconsin are several times higher than the typical outdoor densities of other mid-sized predators like foxes, skunks, opossums and raccoons. Clearly, free-ranging cats are a major predator of birds in rural Wisconsin.

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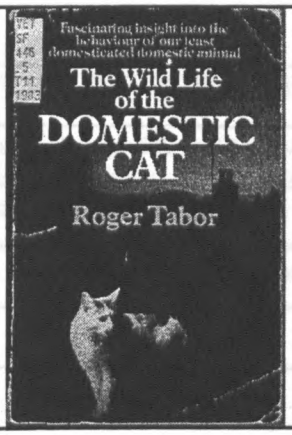
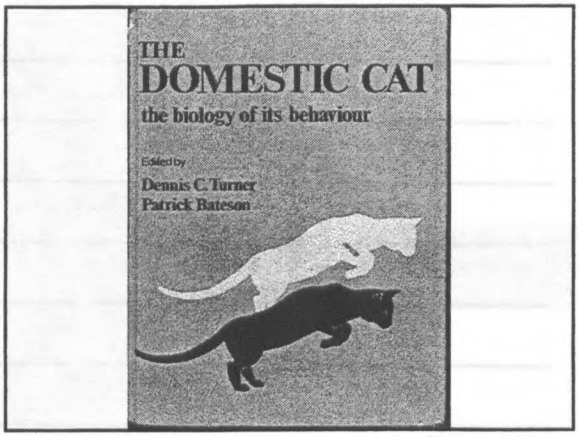


Tabor asks:

- "For as long as people have kept cats it would seem that they have appreciated that cats can catch birds. It is often suggested that cats take a heavy toll of bird-life but is this based on firm ground?" (p. 124)

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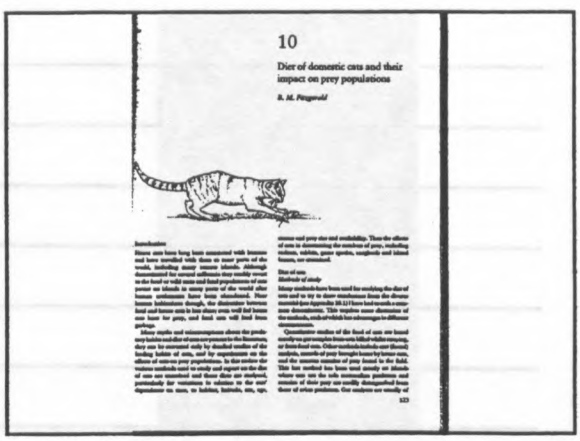
Hunting behaviour of the domestic cat

Dennis C. Turner and Odmar Mentre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Diet of domestic cats and their impact on prey populations

S. M. Fitzgerald

A sampling of the studies:

- British "What the cat brought in" survey (1987)
- New Zealand Orongorongo Valley study (1979)
- Southern Sweden study (1984)

- 1936 Southern Wisconsin survey
- 1941 Oklahoma report
- 1949 Michigan report
- 1950 Sacramento Valley study
- 1954 Pennsylvania food habits study

Cats and non-songbird impacts:

- William G. George (1974), "Domestic cats as predators and factors in winter shortages of raptor prey," *The Wilson Bulletin*
- Suppression of mammalian predators, e.g. rats, as suggested in the New Zealand study

Conclusions:

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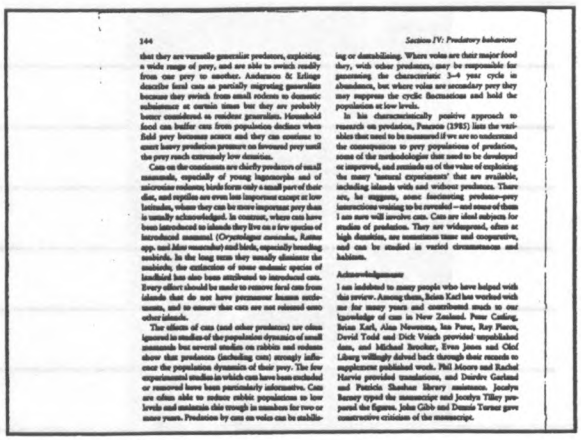
feral cats are present on virtually all islands inhabited by humans (Gibbons, 1984).

Concluding remarks

Predators can be categorised into several types, on the basis of their degree of specialisation (specialist or generalist) and mobility (resident or nomad) (Andersson & Erlinge, 1977). A review of the quantitative studies of the diet of house and feral cats shows



144 Section IV: Predatory behaviour

that they are versatile generalist predators, exploiting a wide range of prey, and are able to switch readily from one prey to another. Andersson & Erlinge describe feral cats as partially migrating generalists because they switch from small rodents to domestic substances at certain times but they are probably better considered as resident generalists. Household food can buffer cats from population declines when field prey become scarce and they can continue to exert heavy predation pressure on favoured prey until the prey reach extremely low densities.

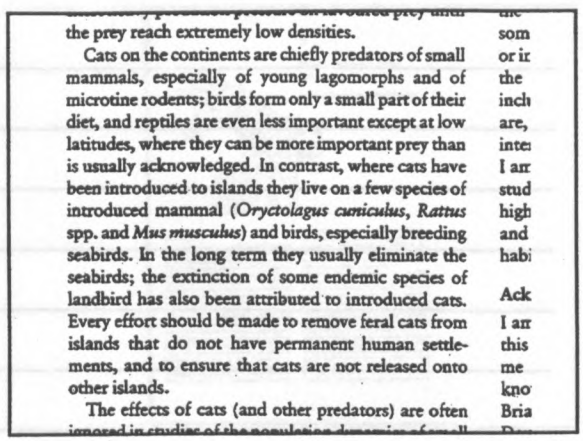
Cats on the continents are chiefly predators of small mammals, especially of young lagomorphs and of microtine rodents; birds form only a small part of their diet, and reptiles are even less important except at low latitudes, where they can be more important prey than is usually acknowledged. In contrast, where cats have been introduced to islands they live on a few species of introduced mammal (*Oryctolagus cuniculus*, *Rattus* spp. and *Mus musculus*) and birds, especially breeding seabirds. In the long term they usually eliminate the seabirds; the extinction of some endemic species of landbird has also been attributed to introduced cats. Every effort should be made to remove feral cats from islands that do not have permanent human settlements, and to ensure that cats are not released onto other islands.

The effects of cats (and other predators) are often ignored in studies of the population dynamics of small mammals but several studies on rabbits and rodents show that predators (including cats) strongly influence the population dynamics of their prey. The few experimental studies in which cats have been excluded or removed have been particularly informative. Cats are often able to reduce rabbit populations to low levels and maintain this trough in numbers for two or more years. Predation by cats on voles can be stabilizing or destabilizing. Where voles are their major food they, with other predators, may be responsible for generating the characteristic 3–4 year cycle in abundance, but where voles are *secondary* prey they may suppress the cyclic fluctuations and hold the population at low levels.

In his characteristically positive approach to research on predation, Pearson (1985) lists the variables that need to be measured if we are to understand the consequences to prey populations of predation, some of the methodologies that need to be developed or improved, and reminds us of the value of exploiting the many 'natural experiments' that are available, including islands with and without predators. There are, he suggests, many fascinating predator–prey interactions waiting to be revealed – and some of them I am sure will involve cats. Cats are ideal subjects for studies of predation. They are widespread, often at high densities, are sometimes tame and cooperative, and can be studied in varied circumstances and habitats.

Acknowledgements

I am indebted to many people who have helped with this review. Among them, Brian Karl has worked with me for many years and contributed much to our knowledge of cats in New Zealand. Peter Catling, Brian Karl, Alan Newsome, Ian Parer, Roy Pierce, David Todd and Dick Veitch provided unpublished data, and Michael Bomford, Erwin Jones and Olof Liberg willingly delved back through their records to supplement published work. Phil Moors and Rachel Norwiz provided translations, and Deirdre Garland and Patricia Shanhan library assistance. Jocelyn Barney typed the manuscript and Jocelyn Tilley prepared the figures. John Gibb and Dennis Turner gave constructive criticism of the manuscript.



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- From the known *fact* of predation they leaped to the unknown *effect* of predation, from the knowledge that some animals eat others to the assumption that this predation controls the population.

And recall Errington's influence on Aldo Leopold:

- Aldo Leopold was one of the first to call attention to Errington's research.
- In *Game Management* (1933), Leopold relied heavily on Errington's work to argue against predator control as a standard practice.

And recall Errington's influence on Aldo Leopold:

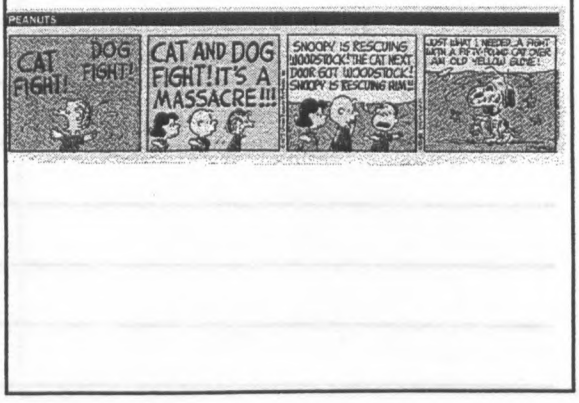
- There he said that predator control might be necessary, but it had to be shown to be correct in each case. It could not simply be assumed that killing predators would do any good.

FERAL AND FREE-RANGING DOMESTIC CATS

Position Statement

How did our hero make out?

- Snoopy versus the feral cat . . .



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- National Science Foundation, "Ethics of Wildlife Fertility Control"

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- Paul Curtis
- Bruce Lauber

FeralCatActivist

A Publication of Alley Cat Allies — The Newsletter for Feral Cat Caretakers / Fall 2001

AMERICAN BIRD CONSERVANCY'S CATS INDOORS! CAMPAIGN

An Assault on Stray and Feral Cats

IN SEPTEMBER 1997, the American Bird Conservancy (ABC) launched its *Cats Indoors!* campaign "to educate cat owners, decision makers, and the general public that free-roaming cats pose a significant risk to birds and other wildlife, suffer themselves, and pose a threat to human health."

The ABC assessment of the effect of cats on bird populations, human health, and themselves is built on inflated figures and questionable studies couched in heartwarming prose that obscures their underlying premise. Unfortunately, even some dedicated feral cat advocates believe they can support the ABC's campaign by sterilizing feral cats.

What they have not recognized is that the most important aspect of the *Cats Indoors!* campaign—the one objective that every feral cat activist needs to understand clearly—is the ABC's goal to eliminate all outdoor cats: outside pets, strays, and ferals, whether sterile or not.

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Numerous press reports in recent weeks have quoted ABC statements that trap-neuter-return (TNR) does not reduce feral cat populations, that TNR does not work. They are wrong. TNR is the only effective procedure available to mobilize people to address the problem of feral feline overpopulation. People will not support the outdated, discredited, and inhumane trap-and-kill approach that has been such a failure but that ABC

See **Assault** *on page 5*

Inside Activist

Where Does TNR Work?

THERE ARE THOUSANDS of successful TNR programs operating throughout the U.S. and Canada, ranging in size from the San Francisco SPCA to neighbors feeding small colonies in the back alley. Following are profiles of five programs that differ in scope but work toward the same end. A more comprehensive, although far from complete, list can be found at: www.alleycat.org by clicking on Natl. Feral Cat Day/ Where does TNR work?

Orange County (Florida) Animal Services

2769 Americana Boulevard, Orlando, FL 32839-2162; www.onetgov.net/Dept/gmer/animal/services.htm

Purpose (in part): Stabilize feral cat populations, control rabies, protect human health, reduce citizen complaints, and reduce costs by employing nonlethal population control methods that include TNR, early-age spay/neuter, managed feral cat colonies, and adoption.

Founded: 1995

Serves: Orange County, Florida (population: 896,344)

Funding source: Publicly funded, staffed by employees of Orange County Animal Services, with assistance of volunteers

Orange County provides traps to local residents; feral cats are tested for disease, vaccinated, and spayed/neutered. Healthy cats return to their colony. Kittens older than 8 weeks of age are not released to adoptive homes until sterilized.

Before implementing this program, Orange County Animal Services received 175 nuisance complaints a week. Nuisance complaints have dropped dramatically; cat adoptions have increased from 400 to more than 1,000 per year.

See **TNR** *on page 4*

UPFRONT

Muffy, the Quail Killer

Feral and free-ranging cats kill millions of animals a year—and there's no solution in sight.

AESOP, IN HIS GREEK FABLES, ONCE ASKED, "Who will bell the cat?" Twenty-five centuries later, public officials are wrestling with the same question in light of growing evidence that in many areas, domestic cats are having a devastating impact on wildlife.

There are about 100 million cats in the U.S., 40 million of which are believed to be either feral cats or free-ranging pets. Although domesticated, they retain the hunting instincts of their wild forebears, and their impact on wildlife is staggering. Studies of free-ranging cats have found that some individuals—even well-fed cats—kill hundreds of wild animals per year, and a recent report, *Cats and Wildlife: A Conservation Dilemma*, estimates that cats kill about 1 billion small mammals and hundreds of millions of birds in the U.S. every year.

Cat predation can be especially hard on common ground-nesting species such as rabbits and quail. But cats also extract a harsh toll on endangered wildlife, from piping plovers in Massachusetts to Alameda whipsnakes in California.

Bad kitty: Uncontrolled cats slaughter wildlife.

Wildlife experts agree that some free-roaming cat populations, especially in rural areas, need to be controlled. However, because cats are domestic animals, they fall under a hodgepodge of jurisdictions. And proposed control programs elicit caterwauls of protest from animal lovers.

"It's a very contentious issue," says University of Wisconsin wildlife ecologist Scott Craven, co-author of the *Cats and Wildlife* report. "A lot of people believe very strongly that we have a responsibility to protect free-ranging cats," he says, because they wouldn't exist were it not for owners who fail to confine their cats or who abandon them. "But we also have a responsibility to protect wildlife from human actions," Craven adds.

That problem is often compounded in areas where cat lovers sustain colonies of feral cats through supplemental feeding. Cat colonies are a nagging concern in California, which is home to an estimated 3.5 million feral cats, many of which live in parks, reserves, and other protected wildlife habitats.

Cat welfare organizations such as Forgotten Felines and Alley Cat Allies are adamant that these "homeless" cats have the right to live out their lives unmolested by humans. They advocate programs in which feral cats are trapped, vaccinated, neutered, and released back to their colonies, where, proponents say, feeding programs turn them into benign felines.

Biologists counter that such programs won't help. Feeding programs only create stronger, healthier predators, says Ron Jurek of the California Department of Fish and Game, and they support cats at high densities. Feeding programs also create a "vacuum effect" that attracts more feral cats—as well as people looking for a place to abandon unwanted cats.

Ironically, animal rights groups such as the Humane Society of the United States and People for the Ethical Treatment of Animals also disparage "neuter-and-abandon" programs, but for a different reason. They say such programs only perpetuate the miserable lives feral cats lead. Instead, they advocate trapping feral cats and destroying those cats that cannot be placed for adoption.

And when animal rights groups espouse killing unwanted animals, it is a sure sign that the problem is already out of control.—LAWRENCE PYNE

APH BY LANCE KRUEGER

You may send your comments on this article to 10/99 Backlash, FIELD & STREAM, 2 Park Ave.

Eliminate the Poisons in Your Yard

> ***"There was a strange stillness. The birds, for example — where had they gone? Many people spoke of them, puzzled and disturbed. The feeding stations in the backyards were deserted. The few birds seen anywhere were moribund; they trembled violently and could not fly. It was a spring without voices."***
>
> ***Silent Spring***
> **by Rachel Carson, 1962**

Rachel Carson introduced her classic book about the perils of pesticides with those observations in a chapter she called *"A Fable for Tomorrow."*

"Tomorrow" has arrived. Now, no one would think of using DDT to kill garden insects.

Yet, we assume that the lawn and garden chemicals found on the shelves at hardware stores are safe to use around birds (and people). Take a close look at the labels. Too many popular pesticides are **lethal** to birds. And while many pesticides may not kill birds on contact, they can contaminate bird food (insects) and water.

What are the alternatives? Mechanical and biological techniques for pest control provide less hazardous options.

Contact your county USDA Agriculture Extension office and the Environmental Protection Agency for information about integrated pest management, biological and chemical pest control safety and pest-resistant plant varieties.

For more information, contact:

National Coalition Against the Mis-Use of Pesticides, 701 E St. SE, Washington, DC 20003.

US Environmental Protection Agency (H7505 C), Office of Pesticide Programs, Environmental Fate and Effects Division, 401 M St. SW, Washington, DC 20460.

Cat Predation

Americans keep an estimated 60 million cats as pets. Let's say each cat kills **only one** bird a year. That would mean that cats kill over 60 million birds (minimum) each year — more wildlife than any oil spill.

Scientific studies actually show that each year, cats kill **hundreds of millions** of migratory songbirds. In 1990, researchers estimated that "outdoor" house cats and feral cats are responsible for killing nearly 78 million small mammals and birds annually in the United Kingdom.

University of Wisconsin ornithologist, Dr. Stanley Temple estimates that **20-150 million** songbirds are killed each year by rural cats in Wisconsin alone.

Feline predation is **not** "natural." Cats were domesticated by the ancient Egyptians and taken throughout the world by the Romans. Cats were brought to North America in the 1800's to control rats. The "tabby" that sits curled up on your couch is not a ***natural predator*** and has never been in the natural food chain in the western hemisphere.

Cats are a serious threat to fledglings, birds roosting at night and birds on a nest. Research shows that de-clawing cats and bell collars **do**

activity patterns, remain essentially unchanged from their ancestral form. Cats were first domesticated in Egypt around 2000 BC [*1*]. Domestic cats spread slowly to other parts of the globe, possibly because Egyptians prevented export of the animal they worshiped as a goddess. However, by 500 BC the Greeks had acquired domestic cats, and they spread cats throughout their sphere of influence. The Romans introduced the domestic cat to Britain by 300 AD. Domestic cats have now been introduced around the world, mostly by colonists from Europe.

How many cats are there in the United States?

The estimated numbers of pet cats in urban and rural regions of the United States have grown from 30 million in 1970 [*2*] to 60 million in 1990 [*3*]. These estimates are based on U.S. Census data and include only those cats that people claim to "own" as pets, not cats that are semi-wild or free-ranging. Nationwide, approximately 30% of households have cats. In rural areas where free-ranging cats are usually not regarded as pets, approximately 60% of households have cats. In the state of Wisconsin alone, with approximately 550,000 rural households, the number of rural free-ranging cats (not house pets) may be as high as 2 million [*4*]. The combined total of pets and free-ranging cats in the U.S. is probably more than 100 million. Because of their close association with humans, most of these cats are concentrated in areas where people live rather than in remote undeveloped areas.

The legal status of domestic cats

The laws that relate to domestic cats vary by local government. In most areas, the person who provides care for a cat is legally responsible for its welfare and control. As with other domestic animals, if ownership can be established by collars or other means of identification, a cat is considered personal property [*5*]. It is usually the responsibility of the owner to control the cat's movements. In most areas, cats can be live trapped and either returned to the owner or turned over to authorities if they wander onto other peoples' property. Many municipalities have leash laws and require vaccination and neutering of pet cats. Because laws vary, one should check local ordinances for the appropriate way to deal with stray cats.

What effects do domestic cats have on wildlife?

Although rural free-ranging cats have greater access to wild animals and undoubtedly take the greatest toll, even urban house pets take live prey when allowed outside. Extensive studies of the feeding habits of free-ranging domestic cats over 50 years and four continents [*6*] indicate that small mammals make up approximately 70% of these cats' prey while birds make up about 20%. The remaining 10% is a variety of other animals. The diets of free-ranging cat populations, however, reflect the food locally available.

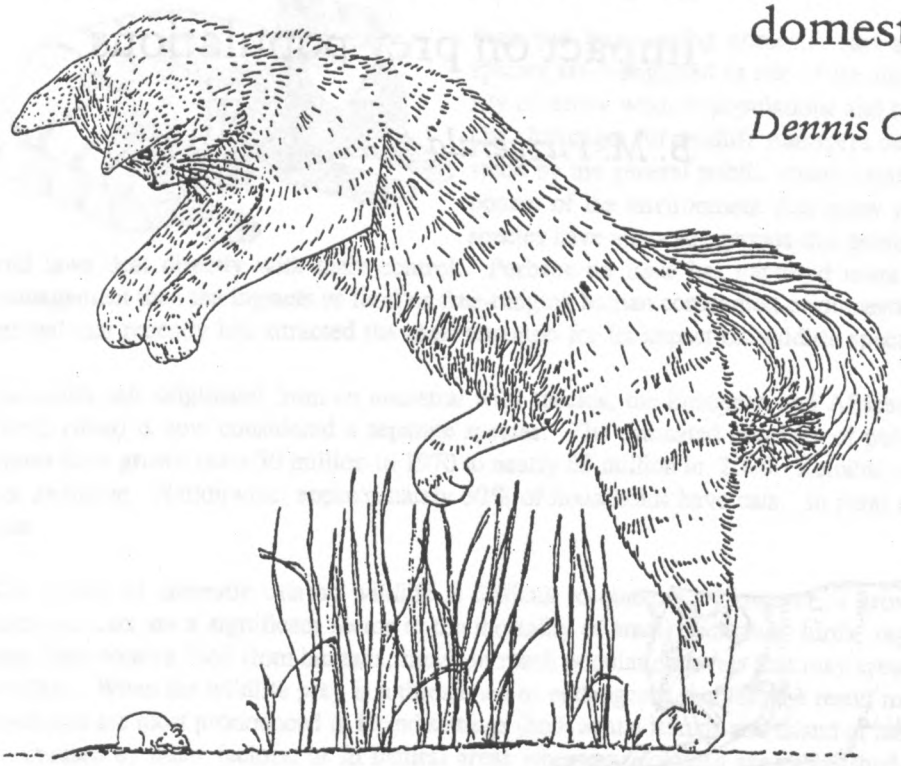
Observation of free-ranging domestic cats shows that some individuals can kill over 1000 wild animals per year [*7*], although smaller numbers are more typical. Some of the data on kills suggest that free-ranging cats living in small towns kill an average of 14 wild animals each per year. Rural cats kill many more wild animals than do urban, or suburban cats [*8*]. Several studies found that up to 90% of free-ranging rural cats' diet was wild animals, and less than 10% of rural cats killed no wild animals [*9*]. Recent research [*10*] suggests that rural free-ranging domestic cats in Wisconsin may be killing between 8 and 217 million birds each year. The most reasonable estimates indicate that 39 million birds are killed in the state each year. Nationwide, rural cats probably kill over a billion small mammals and hundreds of millions of birds each year. Urban and suburban cats add to this toll. Some of these kills are house mice, rats and other species considered pests, but many are native songbirds and mammals whose populations are already stressed by other factors, such as habitat destruction and pesticide pollution.

Despite the difficulties in showing the effect most predators have on their prey, cats are known to have serious impacts on small mammals and birds. Worldwide, cats may have been involved in the extinction of more bird species than any other cause, except habitat destruction. Cats are contributing to the endangerment of populations of birds such as Least Terns, Piping Plovers and Loggerhead

9

Hunting behaviour of the domestic cat

Dennis C. Turner and Othmar Meister



Introduction

Domesitic cats fulfil two very different roles in today's civilisation: they are kept as pets and/or for their propensity to hunt and kill agricultural pest species. This review concerns the latter aspect of their behaviour and we will describe the 'how, when and where' of hunting by cats on the following pages. Since many pet cats with outdoor access hunt, we hope to summarise available information of interest to the cat owner. But we are also interested in comparing the relative success of different hunting strategies and their application e.g. by male and female cats, for different prey types and in different habitats. Such questions are asked by the behavioural ecologist, and their answers also have practical consequences for those who keep cats for their pest-killing abilities, i.e. many farmers.

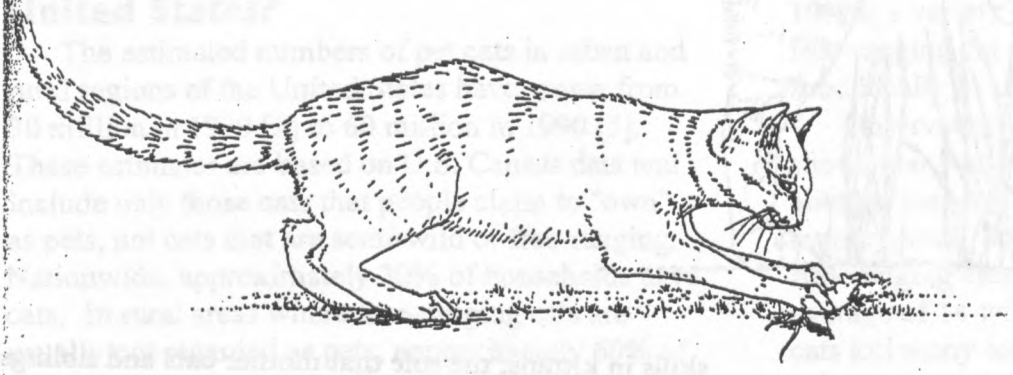
Two very important aspects of the cat's predatory behaviour are covered in other chapters of this volume: as part of their general treatment of behavioural development in cats, Martin & Bateson (Chapter 2) include the development of predatory skills in kittens, the role that mother cats and siblings play in that, and the role mothers play in the development of prey preferences of their offspring. And Fitzgerald (Chapter 10) discusses prey selection from an ecological viewpoint, including the impact domestic cats have on their prey populations.

Given the domestic cat's notoriety as an excellent hunter, it is surprising that very few studies have concentrated on its actual *hunting* behaviour. Most investigations on prey-related behaviour have dealt with the cat's activities after a prey item has been detected (or presented in an experimental situation), and many have concentrated on the acts of grasping, killing, handling and/or consuming the prey (see e.g. Caro, 1980a, b; Leyhausen, 1956c, 1965a, 1979). Although these are certainly important aspects of predation by cats, 'hunting' begins with the search for potential prey, and includes all behaviour leading to the successful capture of that prey and/or a renewed search for other prey items. Essentially, we agree with and will use Panaman's (1981) definition of hunting as (a) making a roving search of the environment, i.e.

10

Diet of domestic cats and their impact on prey populations

B. M. Fitzgerald



Introduction

House cats have long been associated with humans and have travelled with them to most parts of the world, including many remote islands. Although domesticated for several millennia they readily revert to the feral or wild state and feral populations of cats persist on islands in many parts of the world after human settlements have been abandoned. Near human habitations though, the distinction between feral and house cats is less clear; even well fed house cats hunt for prey, and feral cats will feed from garbage.

Many myths and misconceptions about the predatory habits and diet of cats are present in the literature; they can be corrected only by detailed studies of the feeding habits of cats, and by experiments on the effects of cats on prey populations. In this review the various methods used to study and report on the diet of cats are examined and those diets are analysed, particularly for variations in relation to the cats' dependence on man, to habitat, latitude, sex, age, season and prey size and availability. Then the effects of cats in determining the numbers of prey, including rodents, rabbits, game species, songbirds and island faunas, are examined.

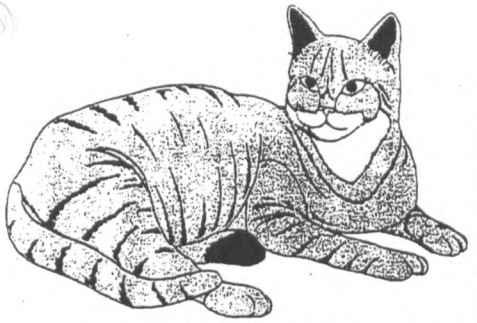
Diet of cats

Methods of study

Many methods have been used for studying the diet of cats and to try to draw conclusions from the diverse material (see Appendix 10.1) I have had to seek a common denominator. This requires some discussion of the methods, each of which has advantages in different circumstances.

Quantitative studies of the food of cats are based mostly on gut samples from cats killed whilst straying, or from feral cats. Other methods include scat (faecal) analysis, records of prey brought home by house cats, and the uneaten remains of prey found in the field. This last method has been used mostly on islands where cats are the sole mammalian predators and remains of their prey are readily distinguished from those of avian predators. Gut analyses are usually of

FERAL AND FREE-RANGING DOMESTIC CATS



Position Statement

Free and free-ranging domestic cats are exotic species to North America. Exotic species are recognized as one of the most widespread and serious threats to the integrity of native wildlife populations and natural ecosystems. Exotic species present special challenges for wildlife managers because their negative impacts are poorly understood by the general public, many exotic species have become such an accepted component of the environment that many people regard them as "natural," some exotic species have advocacy groups that promote their continued presence, and few policies and laws deal directly with their control. Perhaps no issue has captured more of the challenges for contemporary wildlife management than the impacts of feral or free-ranging human companion or domestic animals. The domestic cat is the companion animal that recently has attracted the most attention for its impact on wildlife species.

Domestic cats originated from an ancestral wild species, the European and African wild cat (*Felis silvestris*). The domestic cat (*Felis catus)* is now considered a separate species. The estimated numbers of pet cats in urban and rural regions of the United States have grown from 30 million in 1970 to nearly 65 million in 2000. Reliable estimates of the present total cat population are not available. Nationwide, approximately 30% of households have cats. In rural areas, approximately 60% of households have cats.

The impact of domestic cats on wildlife is difficult to quantify. However, a growing body of literature strongly suggests that domestic cats are a significant factor in the mortality of small mammals, birds, reptiles, and amphibians. Because free-ranging cats often receive food from humans, they can reach population levels that may create areas of abnormally high predation rates on wildlife. When the wildlife prey is a threatened or endangered species, the result may be extirpation or extinction. Effects of cat predation are most pronounced in island settings (both actual islands and island of habitat), where prey populations are already low or stressed by other factors, or in natural areas where cat colonies are established. Competition with native predators, disease implications for wildlife populations, and pet owners' attitudes toward wildlife and wildlife management also are important issues.

Extensive popular debate over absolute numbers or types of prey taken is not productive. The number of cats is undeniably large. Even if conservative estimates of prey taken are considered, the number of prey animals killed is immense. Feeding cats does not deter them from killing wildlife as they do not always eat what they kill. Humans introduced cats to North America and they must be responsible for the control and removal of cats that prey on wildlife.

The policy of The Wildlife Society in regard to feral and free-ranging domestic cats is to:

1. Strongly support and encourage the humane elimination of feral cat colonies.
2. Support the passage and enforcement of local and state ordinances prohibiting the public feeding of feral cats, especially on public lands, and releasing of unwanted pet or feral cats into the wild.
3. Strongly support educational programs and materials that call for all pet cats to be kept indoors, in outdoor enclosures, or on a leash.
4. Support programs to educate and encourage pet owners to neuter or spay their cats, and encourage all pet adoption programs to require potential owners to spay or neuter their pet.
5. Support the development and dissemination of sound, helpful information on what individual cat owners can do to minimize predation by free-ranging cats.
6. Pledge to work with the conservation and animal welfare communities to educate the public about the negative impact of free-ranging and feral cats on native wildlife, including birds, small mammals, reptiles, amphibians, and endangered species.
7. Support educational efforts to encourage the agricultural community to keep farm cat numbers at low, manageable levels and use alternative, environmentally safe rodent control methods.
8. Encourage researchers to develop better information on the impacts of feral and free-ranging cats on native wildlife populations.
9. Recognize that cats as pets have a long association with humans, and that responsible cat owners are to be encouraged to continue caring for the animals under their control.
10. Oppose the passage of any local or state ordinances that legalize the maintenance of "managed" (trap/neuter/release) free-ranging cat colonies.

Virulent Systemic Feline Calicivirus: An Emerging Concern

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Feline calicivirus review

Like other members of the family caliciviridae, feline calicivirus (FCV) is an unenveloped, positive strand RNA virus. As such, these viruses tend to be environmentally resistant and prone to mutation, two characteristics which make them good candidates to cause significant outbreaks of disease. Other members of the family include rabbit hemorrhagic disease and vesicular stomatitis virus of swine, both of which have historically been associated with widespread epidemics.

Infection with FCV is very common, and virus can be isolated even from clinically normal cats. Many cats continue shedding virus for weeks or months after recovery from clinical disease, and some go on to be lifelong carriers. Although vaccination is widely practiced, it is only partially effective. FCV vaccine does not prevent infection, and although vaccination mitigates severity of disease in some cases, vaccine resistant strains are known to occur.

Signs of FCV infection are variable, and include fever, rhinitis, conjunctivitis, oral ulcerations and/or chronic stomatitis, and lameness. Although morbidity is high, mortality due to uncomplicated FCV infection is usually low, and when death occurs it is typically due to pneumonia in very young or debilitated kittens.

Virulent systemic feline calicivirus

In 1998, an outbreak of unusually virulent feline calicivirus was recognized in northern California. In addition to signs more commonly seen with FCV, infection with this viral strain (FCV-Ari) was associated with signs of edema, skin crusting and ulceration, multiple organ involvement and death even in otherwise healthy, vaccinated adult cats. Since the northern California outbreak, at least 6 similar focal outbreaks of virulent, systemic FCV (VS-FCV) disease have been reported throughout the United States. In addition to similarity in clinical signs, common features of outbreaks include:

- In every outbreak where a suspect index case was identified (5/6 outbreaks), a cat from an animal shelter or rescue group appeared to be the source of infection.
- Otherwise healthy, adult, vaccinated cats were prominently affected.
- Spread occurred very readily, including via fomites to cats belonging to hospital employees and clients.
- Spread of disease was limited to the affected clinic(s) or shelter, with no spread within the community reported.
- The outbreak resolved within approximately two months.

In addition to known outbreaks, there have been reports of isolated case clusters of VS-FCV. Cats experienced similar clinical signs, but the disease in these clusters did not spread beyond a single affected household, in spite of infected cats being hospitalized for several days without strict isolation precautions. It appears that a combination of viral, host and environmental factors are required for an outbreak to occur.

Outbreak investigation: FCV-Kaos

The outbreak described here occurred in southern California in the summer of 2002, and was the largest outbreak of virulent systemic FCV yet reported, with 54 cases recognized at 3 private practices and a rescue group. The virus was named FCV-Kaos after an early case. The size of the outbreak and the fact that it was recognized relatively early afforded an opportunity to document the range of clinical signs, identify risk factors for disease, and characterize viral isolates from a relatively large number of cases.

Transmission

FCV-Kaos spread very easily. In addition to direct cat to cat transmission, disease spread between clinics and to homes via movement of infected cats and via fomites carried by owners and technicians. Subclinically as well as clinically infected cats were documented to transmit fatal disease, and continued spread of disease occurred in one clinic via a hospitalized cat with unrecognized infection. The outbreak was believed to start with a rescue group kitten who initially showed only mild upper respiratory signs. The median incubation period was 5 days, with a range of 1-12 days. Peracute disease and death occurred in some cases within 24 hours of infection. Incubation periods of greater than 5 days where seen only in cats exposed by another cat in the household; for hospital acquired infections, incubation was between 1-5 days.

As with field strains of feline caliciviruses, continued shedding may occur following recovery. One cat was documented to shed FCV-Kaos at least 16 weeks after recovery from disease. In some cases, shedding was intermittent. Therefore, multiple viral cultures (at least 2-3 obtained a week apart) are required to confirm negative viral status. However, no *fully recovered* cat has been shown to transmit disease, despite exposure in several cases to other naïve cats in the household or during subsequent hospitalization. The risk posed by recovered and chronically shedding cats remains uncertain.

Risk factors

Virtually all exposed cats became infected with FCV-Kaos. Unlike the pattern seen with most strains of calicivirus, adult cats were at significantly higher risk than kittens for severe disease and death. Overall mortality was 40% and mortality in cats over 6 months old was 60%. Although vaccination could not be specifically assessed as a risk factor (very few cats were known to be unvaccinated) at least 23 cases occurred in cats known to be currently vaccinated for FVRCP. There was no difference in severity of disease in kittens vaccinated intranasally versus parenterally.

Clinical signs

In order of frequency, clinical signs in the 54 cases of FCV-Kaos included:

Fever (median 104.9 F)	87%
Edema	50%
Oral ulcers	43%
Nasal or ocular discharge	26%
Icterus	19%
Respiratory distress	17%
Sores/alopecia	15%

Mild or subclinical disease occurred in 21% of cases. It is worth noting that, although serious, the prognosis is not hopeless; even in those cases with severe signs of disease, 13 of 35 cats survived.

Although the original reported outbreak of virulent systemic FCV (FCV-Ari) was described as a "hemorrhagic fever like" syndrome and the term has been used to describe such outbreaks subsequently, hemorrhage was reported in only two cases in this outbreak (from the nose in one case, and from the nose and rectum in another).

Clinical pathology

Clinical pathology findings were inconsistent and non-specific, and included:

Chemistry abnormalities (n=10):

- Hyperbilirubinemia (6/10, range 0.6-3.9 mg/dl))
- Hypoalbuminemia (5/10, range 1.1-2.1 gm/dl)
- Elevated creatine phosphokinase (5/10, range 639-10930)
- Elevated alanine aminotranferase (2/10, range 102 - 116)
-

Complete blood count abnormalities (n=8)

- Lymphopenia (5/8, range 180-1188)
- Neutrophilia (3/8, range 8549-11616)
- Mild anemia (2/8, HCT 25%)

Pathology

The most common findings on necropsy included:

- Edema and skin ulceration
  - More extensive than appreciated clinically
- Lesions at the junction of the footpad and haired skin
- Peracute hepatic necrosis with hepatocellular individualization
  - Not seen in all cases, but distinctive finding also reported in several other outbreaks of VS-FCV
- Variable involvement of other organs
  - Lung, spleen, pancreas and colon

No consistent cause of death was identified. Because lesions are inconsistent, full necropsies are preferred to specific tissue examination for diagnostic purposes. The junction of footpad and haired skin should be examined, even if lesions are not appreciated grossly.

Isolated case clusters

Follow-up surveillance has identified several cases or case clusters that did not appear to spread beyond the affected household, despite extensive exposure to other cats at veterinary hospitals prior to recognition of the infectious nature of the condition. Signs and epidemiology were otherwise similar; in each case cluster, disease was introduced after exposure to an infectious source (recent adoption of a shelter cat, recent hospitalization at a large veterinary hospital); cats developed signs of edema, high fever, and skin crusting and sores; and adult cats (and vaccinated cats in one case cluster) were severely affected. Cats within a household were variably affected, with 1 or 2 cats developing severe or fatal disease, while other cats from the same household showed mild or no signs. It is unknown what combination of viral, environmental and host characteristics are responsible for the development of major outbreaks versus isolated case clusters.

Diagnosis of VS-FCV

Diagnosis of virulent systemic FCV disease is based on positive viral isolation (culture or PCR) *and cDNA sequencing* of an identical viral strain from more than one affected cat. Acutely obtained sample are preferred, because sensitivity of viral isolation decreases later in disease. There is no test that can differentiate "every day" field strain calicivirus from more virulent forms based on a sample from a single affected cat. Many clinically normal cats are culture positive for field or vaccine strain calicivirus on oropharyngeal swab. A positive result on tissue or serum (as opposed to oropharyngeal swab) increases the index of suspicion for caliciviral infection causing clinical disease. Virus has been demonstrated in cases of VS-FCV disease in foot pads and other lesions via

immunohisochemistry. Visualization of virus in tissue outside the oral cavity may assist in diagnosis of VS-FCV.

Diagnostic summary

Suspicion of virulent systemic feline caliciviral disease increases when:

- Cat has a history of recent infectious disease exposure (shelter, cat show, boarding, vet hospital)
- More than one cat is affected
- General signs of feline calicivirus infection (upper respiratory infection, fever, oral ulceration, limping) *precede* characteristic signs of VS-FCV
- Characteristic signs of VS-FCV (as listed above)
- Other causes of systemic vascular damage (e.g. sepsis) ruled out
- Peracute hepatic necrosis with hepatocellular individualization in conjunction with consistent signs and history
- Positive viral isolation from serum or tissue
- Isolation and sequencing of identical viral strains from more than one cat

Treatment

As for any virus, treatment is primarily supportive care. Treatment with steroids and interferon have been reported. The efficacy of these treatments is unknown.

Prevention and control

We are still learning about the biology and epidemiology of virulent systemic feline calicivirus disease. These recommendations are suggested based on what has been observed so far, and may not be all-inclusive:

At all times:

- ✓ Handle cats with upper respiratory infection, high risk history or suspicious signs with careful infectious disease precautions, including use of a disinfectant proven against unenveloped viruses

In an outbreak:

- ✓ Thoroughly clean and disinfect entire premise with a disinfectant proven effective against unenveloped viruses (e.g. sodium hypochlorite, 5% diluted at 1:32)
- ✓ In areas that can't be effectively disinfected, clean as well as possible and close area to cat access for 2-4 weeks
- ✓ Strictly isolate ALL exposed cats
  - Gowns, gloves, shoe covers, separate instruments and supplies, minimal staff entry
  - Isolate asymptomatic/exposed separately from clinically ill cats
- ✓ Repeat viral culture at 1-2 week intervals on recovering cats until 2-3 negative cultures in order to confirm a cat is no longer shedding
- ✓ Contact owners of recently exposed cats, advise to monitor closely
  - Signs most likely to develop within 1-5 days
- ✓ If spread within hospital has occurred, close to cat admissions until above measures have been taken and outbreak is controlled
- ✓ Promptly communicate with area hospitals, shelters and public health officials when an outbreak is suspected

Summary

Outbreaks of virulent systemic feline calicivirus are recognized with increasing frequency. Although no specific treatment exists, spread of disease can be minimized by prompt recognition, clear communication, and implementation of effective control measures.

Feline Population Medicine: A Herd Health Approach

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Center for Companion Animal Health
UC Davis School of Veterinary Medicine

INTRODUCTION: HERD HEALTH VERSUS INDIVIDUAL ANIMAL MEDICINE

The purpose of this presentation is to suggest a format for a herd health approach to a feline population, following the components that are usually included in a typical patient encounter: history, physical exam, diagnostics, treatment, and recheck.

Caring for a population of animals requires the same familiar steps that are taken in caring for an individual patient, but applied in a different way. We would not think of initiating treatment without first taking a history and performing a physical exam and appropriate diagnostic tests. Similarly, interventions to promote herd health should be built on a foundation of understanding the goals and problems specific to that population. Once a problem is identified, individual animal medicine tends to focus more on treatment, while herd health focuses on prevention. Written policies and protocols are developed, similar to the prescription that details treatment for an individual patient. Finally, the herd health veterinarian monitors data that reflect disease and performance levels, just as the veterinarian treating an individual performs rechecks to ensure efficacy of treatment.

Of course, assessing a whole population is a complex and time-consuming process. Although the following outline is presented following the model of a single office visit, in reality these steps would more likely be accomplished over a series of herd health visits, tackling just one or two areas at a time.

HISTORY: ESTABLISHING PRODUCTION GOALS AND BASELINE PERFORMANCE MEASURES

Taking a careful history is an important part of an exam. One component of the history is establishing the purpose for which the animal is kept, whether as a companion, show animal, breeding animal, etc. This may in turn determine the recommendations made for prevention and treatment of various conditions. However, the majority of cats are kept purely for the pleasure of their company, and the our job is to make recommendations to optimize the cat's health and well-being. The owner decides how much they are willing and able to spend, and which recommendations they will implement.

In herd health, the "purpose" of the population receives more consideration. Few herds are kept exclusively for the simple pleasures of having the animals around. The herd medical program is designed not only to keep the animals healthy, but to help the herd perform optimally. Resources are limited: veterinarian and herd manager work together to ensure that resources are being divided among competing programs for optimal return on expenditures. A dairy herd that had perfectly healthy cows but lost money due to excessive medical costs would not be pleased with their medical program. Similarly, an animal shelter that had perfectly healthy cats but could not afford to hire staff to perform adoptions might not meet the goal of saving many lives.

Performance goal examples

Dairy herd:

- Milk production that maximizes economic return for the farmer
- Provision of care commensurate with reasonable animal welfare/ in compliance with applicable laws

- Minimization of pollution by animal wastes
- Prevention of zoonoses
- Avoidance of contamination and residues in animal products

Animal shelter:

- Ending euthanasia of adoptable animals
- Ensuring health and comfort of animals in the shelter
- Protecting public health and safety
- Providing adopters with appropriate, healthy pets and a positive adoption experience.
- Educating the public and modeling good animal care practices

Performance targets

When treating an individual patient, we look for abatement of symptoms or changes in lab values towards normal as an indication that treatment is working. In herd health, we identify *performance targets*, measurable factors we can track to see if we are making progress towards our goals. These will be revisited later in the section on data collection.

Dairy cow performance target example:
- Reproductive performance
- Incidence and prevalence of mastitis
- Calf health and survival
- Milk production score

Shelter performance target example:
- Percentage and number of animals leaving shelter alive
- Incidence and prevalence of important diseases
- Number and reasons for animal surrender
- Frequency and reason for returns after adoption
- Frequency of disease immediately after adoption

HERD PHYSICAL EXAM : MAJOR COMPONENTS OF A PREVENTIVE MEDICINE PROGRAM.

When performing a physical exam, we are careful to observe all major body systems which, functioning together, make up a healthy organism. Similarly, major components of a preventive medicine program must be functioning adequately in order to maintain a healthy population. Components of the herd health "physical" include:

- Population management/crowd control
- Cleaning/disinfection
- Vaccination
- Nutrition
- Parasite control
- Screening and diagnostic testing
- Population segregation
- Behavioral health/environmental enrichment
- Facility health
- Specific disease considerations

A more detailed description of each of these elements of a herd health exam follows. This is primarily based on an assessment of an animal shelter; slightly different questions may pertain to other cat populations, but the general categories remain the same.

Crowd control

Overcrowding is the single biggest contributor to poor health in many feline populations. This is particularly common in shelters, where it often seems that more lives could be saved if only a few more cats could be squeezed in. In fact, overcrowding has the potential to cost many lives through

increased disease and stress-induced behavioral disorders, compromise of staff's ability to care for animals and provide good customer service, and the subsequent loss of community support and trust. Crowding is not simply a matter of cage space; overcrowding occurs whenever the animal population outstrips staff's ability to provide appropriate care.

Population and crowding questions include:

- What is the annual population served?
- What is the average daily population?
- What is the ideal capacity of the facility? What is the actual population today?
- If there is group and individual housing, what is the capacity of each? What is the size of group housing areas, and the maximum number of cats housed there?
    - Maximum group size of 10 cats, no more than .6 cats per m^2 (3.2 ft^2) recommended [1-3]
- What is the average length of stay ("turnover time")? (This question is specifically important in shelters which perform cat adoptions.)
    - Estimated by: daily capacity * 365 / annual population
      (gives maximum turnover time - most accurate in a facility that's usually close to full) For example, shelter with a daily capacity of 50 cats, took in 300 cats last year: 50 cat/day * 365 days / 300 cats = average stay of 60 days per cat
    - Decreasing turnover time increases the number of animals that can be served with the same space, staff and cost
    - Turnover time is decreased by speeding adoption, reclaim, rescue, and by eliminating un-necessary delays in making cats available for adoption.
    - Turnover time should be considered when building in programs that delay availability for adoption, such as lengthy quarantine or extensive pre-adoption workups. **Not** to say that such programs are wrong, just that the effects on turnover time should be considered.

Cleaning and disinfection:

Products used, cleaning schedule, techniques and "cleanability" of the environment should be assessed. Questions include:

<u>Areas to be cleaned/frequency of cleaning:</u>

- How often are cat holding areas cleaned?
    - For facilities with multiple sub-populations of cats, in what order are cat holding areas cleaned?
    - Ideally, cleaning proceeds from healthiest/most vulnerable to least healthy, e.g. proceed from kittens to owned/adoptable adults to strays/recent admissions to quarantine to isolation.
- How often are common areas (lobbies, hallways, visiting rooms, etc.) cleaned?
- How often are air vents, furnace filters, drains and other easily overlooked areas cleaned?
- Is deep cleaning of all areas performed on a regular basis?
- Are there areas of particular concern for cleaning? Carpeted areas, furniture, unfinished wood, grassy outdoor areas, etc.? What is the plan for preventing contamination and/or for cleaning these areas?

<u>Cleaning products and methods:</u>

- What products are being used for cleaning?
    - **Detergent?** A detergent (or a disinfectant with detergent properties) is required for effective cleaning.
    - **Disinfectant?** A product proven effective against unenveloped viruses[4-6] (e.g. feline panleukopenia and feline calicivirus) should be used on a daily basis in facilities with moderate to high population turnover, and at regular intervals (e.g. weekly) in other cat facilities.
    - **Degreaser/deep cleaner?** A degreaser should be used at regular intervals (depending on degree of soiling)
    - Special cleaning products for hard-to-kill agents (e.g. ringworm) or hard-to-clean areas?

- At what concentration is the disinfectant used? Is it measured or just eye-balled?
- What is the method of application (spray, rags, paper towels, etc.)?
- How long is the disinfectant left in contact with the surface? Consistent with manufacturers recommendations? (Most disinfectants require ten minute minimum)
- Are there any factors, such as hard water, excessive organic matter or exposure to light that may render the chosen disinfectant ineffective?
- Are special provisions made for cleaning up after an exposure to an unusually durable agent such as panleukopenia or ringworm?
- How are dishes, litter boxes, animal bedding, carriers, toys etc. cleaned?
- How often are litter boxes scooped, and how often are they emptied and thoroughly scrubbed?
- If cats do not remain in the same cage during cleaning, where are they held (carrier, temporary cage, etc.)? Is the holding area sufficiently cleaned between cats?

If infectious disease spread continues in the face of a seemingly adequate cleaning program, it is worth actually watching cleaning from start to finish. Sometimes the cleaning protocol actually in use is quite different from the one the manager believes is being followed. Also consider fomites such as exam surfaces, carriers, vehicles, door knobs and other areas that may escape daily attention.

Vaccination

If vaccines are given in-house by shelter or cattery staff (as is often the case), the herd health vet should ensure that vaccines are being chosen, stored, and administered for maximum efficacy and safety. Questions include:

- What vaccines are used routinely?
- For each vaccine:
  - Route? (intranasal versus parenteral)
  - Location? (for parenteral vaccines - are AAFP guidelines followed?)
  - Killed versus live?
  - Manufacturer?
- How often/at what age are vaccines administered? At intake? Are boosters given?
- How/where are vaccines stored?
- Are vaccines recorded in a permanent record?
- Are written instructions provided for recognition and response to adverse vaccine reactions? If in-house treatment is provided for emergency response to vaccine reactions, are all necessary drugs available and current?

Nutrition

A well thought out feeding program is an important part of a comprehensive herd health plan. The diet should be consistent and of good quality, with a system in place to ensure adequate food and water intake. Both overfeeding and anorexia (due to stress, unfamiliar diet, competition for food in group housing, or concurrent upper respiratory or GI disease) are common in many cat populations. Questions to ask include:

- What diet is routinely fed? Is it always the same brand?
- Are different diets fed for different life stages?
- Are special diets used for cats with GI disease, skin disease, etc.?
  - Some shelters have a wide variety of donated special diets hanging around; it is important to ensure these are used appropriately.
- Is food measured out or are animals free-fed? If measured, how much per cat?
- In group housing, how many feeding stations are there?
- Is appetite and water consumption/urine output monitored on a formal basis?
- Are cats weighed (and/or body condition score assessed) on intake and on a regular basis?
  - This is especially important in shelters and boarding catteries which house cats for longer than a week or two.

Parasite control

In addition to improving the health and comfort of the feline herd, internal and external parasite control is important to reduce spread of vector borne disease (e.g. tapeworm, bartonellosis), prevent environmental contamination with durable agents (e.g. roundworm), and control zoonotic diseases carried by ticks and fleas or caused by internal parasites. In addition, animals free of parasites make more appealing pets for adopters. Parasite control questions include:

- What products are routinely used for external parasite control?
  - Which animals are treated? All, or on an as-needed basis?
- What products are routinely used for internal parasite control?
  - Which animals are treated? All, kittens only, or on an as-needed basis?
  - Are repeat treatments given, or just a single dose at intake? (i.e. follow up 2 weeks later?)
- Are fecal floats or other diagnostics routinely performed?

Even if extensive diagnostics can not be performed routinely, it is worth periodically working up multiple fecal samples (at least five) to get a feel for prevailing pathogens in that population.

Screening and diagnostic testing

Some shelters and catteries perform in-house diagnostic testing such as FeLV/FIV ELISA, woods lamp screening and/or ringworm culture, fecal floats and others. In shelters, life and death decisions may be made on the basis of test results. Determining what tests are being used is important in order to provide appropriate counseling about test administration, interpretation and sources of false results. Diagnostic testing questions include:

- What tests are used routinely? (record type and brand)
- For each test, are all animals tested, animals of a certain age, or only certain animals at higher risk?
- At what point are animals tested?
  - Intake, prior to moving into a group housing area, prior to adoption?
- Is confirmatory testing performed?
  - e.g. IFA to confirm positive ELISA for FeLV?
- How are test results recorded?
  - In addition to recording test results in the cat's record, it is helpful to keep a log for each test documenting the number of positive and negative results.
- Where are tests stored?
  - Are tests stored and used according to manufacturer instructions? The importance of this should be emphasized.

Monitoring and documentation

In order to facilitate communication between multiple caretakers and ensure that each cat in a population is being checked regularly, a formal system of monitoring is helpful. Basic indicators of the cats' physical and behavioral well being should be noted on a regular basis (daily in a population with high turnover, perhaps less often in a more stable population). This also provides the veterinarian a record of the cats past health and behavioral status in the event of a problem, in lieu of a doting owner available to speak on the cat's behalf. Monitoring questions include:

- Is a written record maintained of:
  - Appetite
  - Water intake
  - Urination
  - Defecation
  - Physical symptoms (e.g. upper respiratory signs)
  - Behavior (e.g. hiding, hissing at other cats, withdrawn, friendly, etc.)

- Are all cats specifically observed on a daily basis ("daily rounds")?
  - Who is responsible for this?

Population segregation

Segregation of population sub-groups is an important tool in maintaining herd health. Populations can be isolated by age, health status, temperament, date of intake (e.g. cohort admission) or simply divided into smaller groups to reduce overall disease transmission. However, quarantine and isolation can increase the time and expense of caring for a cat in a shelter or introducing a new cat into a cattery. In order to make it worthwhile, population segregation must be practiced *effectively*: quarantine of adequate length for the purpose, control of fomites in additional to physical separation of cats, etc. Segregation questions include:

Sub-populations and group housing:

- What sub-populations are housed separately?
  - Cats versus kittens?
  - Stray versus adoptable/owned?
  - Sick versus healthy?
  - Where are non-infectious cats requiring medical treatment housed?
- What screening is done prior to introducing cats into group housing?
  - Are cats held for a certain length of time, either in quarantine or individual housing, before being moved into a group area?
  - What testing, vaccination, and other prophylactic measures are required prior to introduction to group housing?
  - Are group housed cats all-in all-out or is there a constant flux in and out?

Quarantine:

- What is the purpose of quarantine?
  - Quarantine may serve multiple purposes.
    - Identify cats that may be incubating disease
    - Complete vaccination and parasite control prior to general exposure
    - Perform screening tests (e.g. ringworm culture) prior to introduction into general population or hard to clean areas.
- What is the length of quarantine?
  - Greater than the incubation period for the disease being screened for?

Isolation:

- Under what circumstances are cats placed in isolation?
  - e.g. every cat that sneezes versus only severely ill cats
- Are there separate isolation areas for other infectious conditions besides URI (such as diarrhea or ringworm) or are all infectious cats housed together?
- For both quarantine and isolation, which of the following are in place:
  - Protective garments worn only in that area (gloves, lab coats/jumpsuits/old surgery gowns).
  - Foot baths and/or disposable/dedicated boots
  - Dedicated cleaning and animal care supplies used only in that area.
  - Minimal traffic in and out
  - Ventilation and drainage separate from rest of building (ideal, but uncommon!)

Behavioral health/environmental enrichment

Apart from control of overcrowding, reduction of stress is probably the single biggest factor in promoting feline population health. Enrichment programs should recognize variations in individual temperament; what is relaxing and pleasant for one cat may be stressful for another. It is also important that cat socialization programs strike a balance between preventing disease transmission and allowing cats to experience socialization, petting, toys and time out of their cage. This can be accomplished through effective quarantine of cats with unknown health status, careful observation

and hygiene on the part of cat handlers, and quick identification and isolation of sick cats. Enrichment questions include:

Individually housed cats:

- Do cats have a hiding space?
- Do cats have a bed or place to curl up other than the litter box?
- Are there toys in the cages?
- Are the feeding and litter areas separated as widely as possible?
- Is the litter box of adequate size to accommodate the cat?
- Do cats remain in the same cage throughout their stay at the facility?
  - Moves from cage to cage can be stressful as well as promoting disease spread.

Group housed cats:

- Are cats selected for group housing based on compatible temperament?
  - For cats not previously socialized to other cats, housing in a group environment can be significantly more stressful than single cat housing [7]
- Are cats gradually introduced and monitored after introduction?
- Are stable groups maintained, or is there constant flux in and out?
- Are there multiple perches and hiding spaces?
- Are there multiple litter boxes in several different parts of the group cat area?
  - Guarding may occur if the litter boxes are all clumped in one location.
- Also see density considerations discussed in crowd control section above.

All cats:

- What is the noise level like in the cat housing areas? Is there lots of noise from barking dogs?
- Are lights turned off at night?
- Is the temperature comfortable?

Facility health

The condition of the facility can have a significant effect on the health of the population. Take a walk through the facility and look for hazards or problems such as blocked vents, peeling paint, cracked floors that could harbor germs, etc. Facility questions include:

- Ventilation
  - What is the source of fresh air flow?
  - How many air exchanges per hour are there in the cat housing areas?
  - Is the ventilation system routinely serviced and checked?
  - Are air filters used?
    - What type? (The EPA has a good web site on air cleaners at http://www.epa.gov/iaq/pubs/residair.html)
    - Are the filters changed on a regular basis?
- What is the general humidity level in the cat housing areas? Are cat areas constantly moist or do they dry out on at least a daily basis? (Low humidity is much preferable for disease reduction.)
- Are there any problems with rodent or insect infestation? Is there a pest control program in place? (Rats, flies and cockroaches can transmit some infectious agents from cage to cage.)

Specific disease considerations

Notice I put this last. Of course, as you work with a population, individual cases of illness will be treated as they arise. However, it is important not to get so caught up in putting out fires that you lose track of the big picture - the preventive measures that keep animals from getting sick in the first place. However, at some point in a herd health visit, the vet should review with the manager any unusual or serious disease occurrences since the last visit. If any data regarding disease occurrence has been collected (see below), it should be analyzed and compared to previous data collection periods. If endemic disease such as URI or diarrhea seems more severe than usual or has an atypical presentation, samples should be taken for analysis. More information on collecting herd health diagnostics can be found at http://www.vetmed.ucdavis.edu/CCAH/Prog-ShelterMed/Diagnostics.htm

HERD TREATMENT PLAN: POLICY AND PROTOCOL DEVELOPMENT

Answers to the questions raised above will most likely suggest areas for improvement or change. Because multiple people with widely varying levels of medical knowledge and training will be responsible for implementing changes, development of written protocols to cover common situations is vital. In addition to general protocols for cleaning, vaccination, test administration and the like, specific protocols should be developed for the most common and serious diseases encountered in that population. The veterinarian may not be the one to actually write all this, but should at least encourage and consult on development of such documents. A protocol for an infectious disease in a shelter should include the following information (not all categories will apply to other feline populations):

- Basic disease description (ie it's a virus, it causes diarrhea, it can be fatal, etc.)
- General policy regarding admission, treatment, adoption or euthanasia
- How recognized/diagnosed (case definition)
- Who to notify
- Where to house the animal (general population, isolation, test and remove, etc.)
- How to clean contaminated cages, exam surfaces, etc.
- Which animals will be treated? (if applicable)
- Treatment (if standard treatment is initiated by other than vet)
    - Who can initiate treatment
    - Circumstances under which standard treatment initiated
    - Side effects/contra-indications to standard treatment
- Monitoring
- Recovery/treatment failure
    - How defined
    - Who can determine
        - If other than vet, standardized protocol for determining recovery/treatment failure
- Adoption
    - Will animal be adoptable prior to recovery?
    - Adoption release required notifying adopter of medical condition?
    - Medications to go home with animal?
- Documentation

For a truly exhaustive discussion of policy and protocol development, see our website at http://www.vetmed.ucdavis.edu/CCAH/Prog-ShelterMed/pdfs/ID_protocols_KFH.pdf

HERD FOLLOW UP: MONITORING THE RESPONSE TO INTERVENTION

Resources are limited, and if changes cost money or time and don't result in a measurable improvement, the approach may need rethinking. Measurable values, such as CBC, chemistry, pulse and heart rate, assist in following the course of illness in a patient. Success of treatment is assessed by following the trend of these values towards normal. Similarly, data collection and analysis helps determine the success of intervention in a herd.

Examples of performance measures revisited

Some measures of herd health or performance are fairly simple to collect. For a cattery, important numbers to track may include number of live births per litter and per queen, proportion of kittens surviving to weaning, and occurrence of heritable defects and infectious disease. For an animal shelter, numbers to track may include number of intakes, number of adoptions, number and cause of disease and death/euthanasia. Results of all routinely administered tests should be recorded, both positive and negative, to allow an estimate of the true prevalence of the disease in the tested population and watch for trends. In general, data is best collected and displayed with both a numerator and denominator: number of adoptions out of total intake is more informative than simply the number of adoptions in a shelter, for instance.

Animal risk factors

Taking data collection one step further, it is often valuable to distinguish *risk factors* for various outcomes, such as disease, heritable defects, small or large litter size, adoption or euthanasia, etc. Animal risk factors to consider may include age, breed (or genetic line, in a cattery), color, location in facility, vaccination status, reproductive status, source (in an animal shelter, e.g. stray, feral, owner surrendered), and presence of concurrent disease. Availability of such information is particularly helpful in tracking an outbreak. A sample outbreak tracking form is attached in appendix 1.

Disease occurrence

There are two common methods used specifically to describe disease levels in a population: incidence and prevalence. Each measure has certain strengths and weaknesses.

Prevalence is defined as *the number of cases of disease present in a population at risk at a given point in time*, and represents a "snapshot" of disease.

Example: 20 cases of URI out of a total of 100 cats in the shelter that day = 20/100 = .2 = 20% prevalence.

Prevalence is fairly simple to calculate: count total sick and total population daily, weekly, etc, and plot results over time. Prevalence goes up with an increase in either the number of *new cases* of disease *or* increased *duration* of disease. For instance, if a shelter becomes able to treat cats with URI rather than euthanizing them immediately, prevalence will increase because the cats will be present in the population longer, even if the number of new cases of URI does not change. On the other hand, if a more effective treatment for URI is found such that cats recover more rapidly, prevalence will decrease even if the number of new cases remains the same. *Because prevalence is influenced by duration, it should not be used to measure a disease for which animals are frequently euthanized, sent to foster care or otherwise removed from the population before recovery.* In such cases, "duration" is artificially controlled by facility policy, and resulting prevalence levels will be misleading.

Incidence is defined as *the number of new cases of a disease occurring in a population at risk over a period of time*.

Example: Shelter that cared for 100 cats for 10 days apiece in a given month (100*10 = 1000 cat days at risk). 20 cats developed URI out of 1000 cat days at risk that month = 20/1000 = .02 cases of URI per cat day at risk.

Incidence is independent of disease duration, and is therefore a useful measure in cases where prevalence is unreliable as discussed above. A minor disadvantage is that it requires calculation of population time at risk. This is defined as the total time contributed by all disease-free individuals in the facility during the period under consideration. Population time at risk is obtained by taking a daily head count of "healthy cats" (cats without the disease in question) and summing that for the time period under consideration. If that is impractical, population time at risk can be estimated by taking a periodic count (at the beginning and end of the time period or at regular intervals during the time period) and using the average. This is fairly accurate if there is not much fluctuation in the daily population. In a facility where cages for healthy cats are almost always full, the population time at risk can also be estimated by simply multiplying the number of cage spaces for healthy cats by the number of days in the time period under consideration.

Duration of disease

Neither incidence nor prevalence indicates duration (or severity) of disease. Duration should be considered when assessing the benefits of some interventions. For instance, some vaccines claim to decrease duration and severity of disease, although a greater number of mild cases of disease/vaccine reaction may be seen. If one considered only incidence, such a vaccine would seem harmful, but considering duration will give a more realistic assessment of possible benefit. Note that

duration cannot be calculated for animals that are euthanized, adopted or otherwise lost to follow-up prior to recovery.

Statistical analysis

Tracking data as described above is useful for determining trends and suggesting areas for further investigation or action. However, it does not demonstrate the cause of the observed changes. A certain amount of variation between animals or within a population over time is expected due to chance alone. Additional variation is likely to be the result of more than one factor. A change in season, an increase in the average age of the population at risk, and a change in vaccination protocol could all contribute at once to a decrease in URI levels, for example. Establishing the significance of observed changes requires statistical analysis, which is well beyond the scope of this presentation. A full chapter is devoted to use of statistical testing in herd health in the book "Herd Health: Food Animal Production Medicine"[8]; much of this information is equally applicable to the feline herd.

The big picture: Feline "meta-population" health

Most feline populations are not closed systems. The health of the population is influenced by the health of the surrounding community with which it interacts, and in turn influences that community. As more and more people get their cats from concentrated sources such as shelters and catteries, the health of these populations exerts an ever greater influence on the health of cats in general. If an animal shelter draws from a community in which overpopulation is rampant, few cats are vaccinated or identified, and disease is common, even a well run shelter will most likely be a crowded, unhealthy place. Concentrating a vulnerable population in the shelter furthers the opportunity for spread of disease, and cats are likely to leave the shelter and re-enter the community (and veterinary practices!) carrying contagious, chronic and/or zoonotic conditions. On the other hand, reducing feline overpopulation, increasing owner retention and improving the condition of the greater feline population relieves pressure on the shelter, which in turn leads to healthier cats re-entering the community. The private practitioner's role in improving shelter population health is a topic worthy of whole lecture in its own right (and an article[9]); suffice it to say here that what goes on *outside* the shelter is as important as what goes on inside the shelter in determining the health of the feline community.

References

1. Dowling, J., *All Together Now: Group Housing for Cats*, in *Animal Sheltering*. 2003. p. 13-26.
2. Kessler, M.R. and D.C. Turner, *Stress and adaptation of cats (Felis silvestris catus) housed singly, in pairs and in groups in boarding catteries.* Animal Welfare, 1997. **6**(3): p. 243-254.
3. Kessler, M.R. and D.C. Turner, *Effects of density and cage size on stress in domestic cats (Felis silvestris catus) housed in animal shelters and boarding catteries.* Animal Welfare, 1999. **8**(3): p. 259-267.
4. Eleraky, N.Z., L.N. Potgieter, and M.A. Kennedy, *Virucidal efficacy of four new disinfectants.* J Am Anim Hosp Assoc, 2002. **38**(3): p. 231-4.
5. Kennedy, M.A., et al., *Virucidal efficacy of the newer quaternary ammonium compounds.* Journal of the American Animal Hospital Association, 1995. **31**(3): p. 254-8.
6. Scott, F.W., *Virucidal disinfectants and feline viruses.* Am J Vet Res, 1980. **41**(3): p. 410-4.
7. Kessler, M.R. and D.C. Turner, *Socialization and stress in cats (Felis silvestris catus) housed singly and in groups in animal shelters.* Animal Welfare, 1999. **8**(1): p. 15-26.
8. Slenning, B., *Quantitative Tools for Production Oriented Veterinarians*, in *Herd Health: Food Animal Production Medicine*, O.M. Radostits, Editor. 2001, W.B. Saunders. p. 47-107.
9. Scarlett, J.M., et al., *The role of veterinary practitioners in reducing dog and cat relinquishments and euthanasias.* Journal of the American Veterinary Medical Association, 2002. **220**(3): p. 306-311.

Appendix 1: Outbreak tracking form

Animal ID	Date entered shelter	Date of vaccine	Date diagnosed	Cage/run # at time of diagnosis	Animal description (sex, spay/neuter, age, breed)	Symptoms*/test results (SNAP test, necropsy, other)

Symptom codes: D=diarrhea, V=vomit, B=blood in diarrhea/vomit, L=lethargy, N=no abnormal signs

Epidemiology of Upper Respiratory Tract Infections in Cats in Animal Shelters

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Introduction

Veterinarians working in or with shelters are well aware of the high incidence and often devastating consequences of upper respiratory tract infections (URTD) in feline shelter populations. In contrast to client-owned cats, URTD are often a major cause of feline morbidity and mortality in shelters. Variable vaccination histories, stress, debilitation from malnutrition, other diseases or injuries, high URI agent concentrations, cat densities, declining maternal antibodies in kittens, and other factors contribute to the high rates of URTD.

For many cats in full admission shelters, the development of even mild signs of URTD becomes a death sentence, as sick cats are often selected first when euthanasia decisions must be made in shelters where cage space is at a premium. Also, infected cats serve as a source of infection for healthy animals in the shelter (Sinclair, 1997).

The literature regarding agents causing feline URTD is extensive. The feline calicivirus (FCV) and the feline herpervirus-1 (FHV-1) are believed to account for approximately 80% of clinical cases of URTD (Knowles & Gaskell, 1991). Other agents such as *Bordetella bronchiseptica*, *Chlamydia psittaci*, Mycoplasma spp, and possibly others also cause URTD signs (Gaskell & Dawson, 1998), and there is mounting evidence that some of these may act as both primary and secondary pathogens (Chandler and Lappin, 2002; Foster et al, 1998).

The prevention and control of respiratory agents in animal shelters is complicated by the carrier states associated with FCV, FHV-1 and *Bordetella bronchiseptica* (August, 1984; Coutts et al, 1996). Subclinical infections and chronic carriage of FCV in clinically unaffected cats makes it impossible to identify and isolate all cats shedding virus into their environment. In some experimental studies, most FCV-infected animals shed virus 30 days and 50% continued to shed 75 days after recovery from clinical signs (Gaskell & Dawson, 1998); a small proportion became lifetime shedders. FCV strain differences also influence the frequency and severity of signs associated with infection (Pedersen et al, 2000; Pedersen & Hawkins, 1995). Among cats infected with FHV-1, at least 80% of cats infected will become latent carriers, displaying no clinical signs or evidence of viral shedding. Following stressful events (such as entering an animal shelter), however, these cats often begin shedding again with or without manifesting clinical signs (August, 1984; Stiles, 2000). Experimental studies suggest that Bordetella can be recovered from infected cats for at least 19 weeks following infection (Coutts et al, 1996).

Vaccines for FCV and FHV-1 generally provide protection against severe clinical signs, but do not prevent infection (Gaskell & Dawson, 1998). This is probably also true of the vaccine for *Bordetella bronchiseptica*. While reducing the frequency and severity of clinical signs and probably reducing the amount of virus shed, they fail to block viral shedding altogether into the shelter environment. To complicate prevention and control efforts further, recent work by Foley et al in California suggest that at least some of the modified-live FCV vaccines may actually contribute to clinical signs and shedding in vaccinated shelter cats (personal communication, 2000). Intranasal vaccines, designed to provide immunity at the respiratory mucosal surface within 2-4 days of vaccination, can produce mild clinical signs, complicating the distinction of naturally-infected from vaccinated cats. In environments where clinical signs can mean the difference between life and death, intranasal vaccination may contribute to deaths due to URTD.

The nature of the common respiratory agents of cats, the stress inherent to animal shelters, imperfect vaccines, staff turnover, and other factors make the elimination of upper respiratory tract disease impossible. The goal instead is to reduce the incidence of URTD to the lowest possible level. In light of the many factors influencing disease occurrence, including the physical facility and resource availability, the target "lowest" level of URTD among shelters may vary.

Despite its acknowledged high frequency in shelters there are few scientifically collected data from shelters relating to its occurrence and management in cats. Data from our on-going studies of URTD in shelter cats will be summarized.

References

August, J.R. (1984) Feline viral respiratory disease: the carrier state, vaccination and control. Vet Clin No Amer: Sm Anim Pract 14:1159-1171.

Chandlers, J.C. and M.R. Lappin. (2002) Mycoplasmal respiratory infections in small animals: 17 cases (1988-1999). J Amer Anim Hosp Assoc 38:111-119.

Coutts, A.J., S. Dawson, S.H. Binns, C.A. Hart, C.J. Gaskell, and R.M. Gaskell. (1996) Studies on natural transmission of bordetella bronchiseptica in cats. Vet Microbiol 48:19-27.

Foster, S.F., V.R. Barrs, P. Martin, R. Malik. (1998) Pneumonia associated with Mycoplasma spp. in three cats. Aust Vet J 76:460-464.

Gaskell, R. and S. Dawson. (1998) Feline respiratory disease In: Greene, C.E. (Ed.) Infectious Diseases of the Dog and Cat. W.B. Saunders Co.: Philadelphia, pp 97-106.

Gaskell, R.M. (1992) Upper respiratory disease in the cat (including chlamydia); control and prevention. Feline Pract 20:7-12.

Kleinbaum, D.G., L.L. Kupper, and H. Morgenstern. (1982) Epidemiologic research: principles and quantitative methods. London: Lifetime Learning Publications, pp 448-456.

Knowles, J.O. and R.M. Gaskell. (1991) Control of upper respiratory diseases in multiple cat households and catteries. In: August, J.R. (Ed.) Consultations in feline internalmedicine, Edition 1. W.B. Saunders:Philadelphia, pp 563-569.

Pedersen, N.C. and K.F. Hawkins. (1995) Mechanisms for persistence of acute and chronic feline calicivirus infections in the face of vaccination. Vet Microbiol 47:141-156.

Pedersen, N.C., J.B. Elliott, A. Glasgow, A. Poland, K. Keel. (2000) An isolated epizootic of hemorrhagic-like fever in cats caused by a novel and highly virulent strain of feline calicivirus. Vet Microbiol 73:281-300.

Sinclair, L. (1997) Controlling upper respiratory infections in your shelter. Animal Sheltering (Jan/Feb), 5-12.

Stiles, J. (2000) Feline herpesviruses. Vet Clin No Amer: Sm Anim Pract 30:1001-1004.

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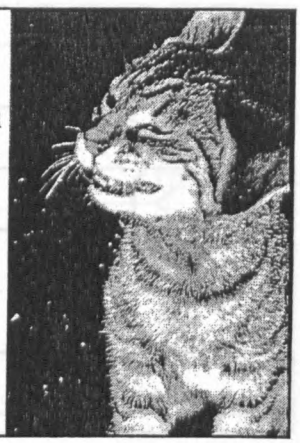
URT in the cat is controlled by cat flu vaccines .....

..... or IS it??????



www.catvirus.com

Major suspects in feline URT:

Feline calicivirus (FCV)

Feline herpesvirus (FHV)

Chlamydophila felis

Bordetella bronchiseptica

www.catvirus.com

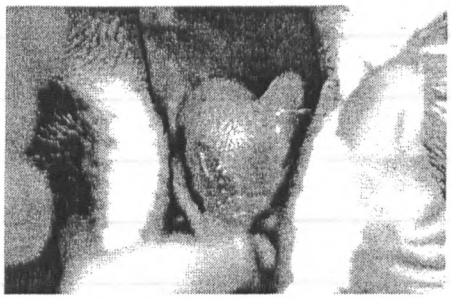
Minor suspects in feline URT:

Allergy

Foreign body

Haemophilus felis

www.catvirus.com



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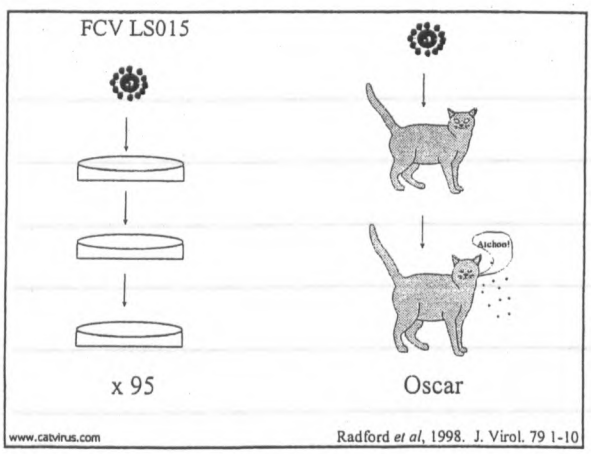


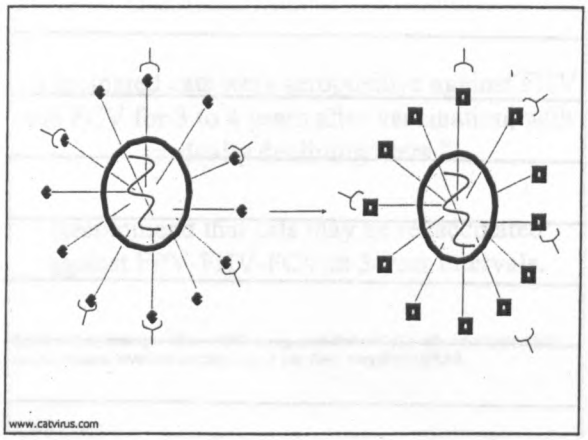
www.catvirus.com

Radford *et al*, 1998. J. Virol. 79 1-10

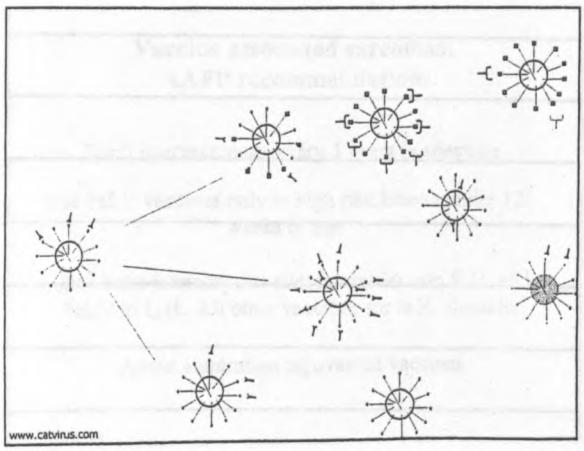
Neutralizing antibody response of Oscar to FCV

www.catvirus.com

Radford *et al*, 1998. J. Virol. 79 1-10



www.catvirus.com



www.catvirus.com

Neutralization of field strains of FCV by antibody to anti-F9 (vaccine) FCV strain

10 units anti-F9 antibody

1958 - 1979

1980 – 1989

1990 – present (USA)

1990 – present (UK)

Lauritzen *et al*, 1997. Veterinary Microbiology 56 55-63

www.catvirus.com

Sequence analysis of feline caliciviruses isolated from the oral cavity of clinically normal domestic cats (Felis catus) in Florida.

"This work provides support to the idea that currently circulating FCV strains may differ substantially from presently used vaccine strains."

Weeks ML, Gallagher A, Romero CH.Sequence analysis of feline caliciviruses isolated from the oral cavity of clinically normal domestic cats (Felis catus) in Florida. Res Vet Sci. 2001 Dec;71(3):223-5.

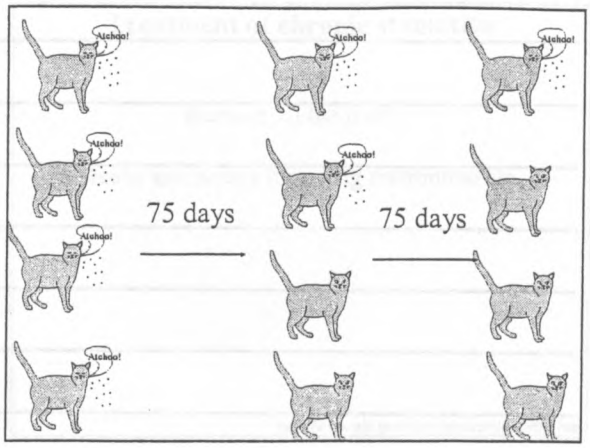
www.catvirus.com

Neutralized field strains

F9	43%
225	67%

Mochizuki M, Kawakami K, Hashimoto M, Ishida T.Recent epidemiological status of feline upper respiratory infections in Japan. J Vet Med Sci. 2000 Jul;62(7):801-3.

www.catvirus.com



"Vaccinated cats were seropositive against FHV and FCV for 3 to 4 years after vaccination, with gradually declining titers."

Recommend that cats may be revaccinated against FPV-FHV-FCV at 3-year intervals.

Scott FW, Geissinger CM. 1999 Long-term immunity in cats vaccinated with an inactivated trivalent vaccine. Am J Vet Res May;60(5):652-8 .

www.catvirus.com

Vaccine associated sarcomas: AAFP recommendations

Don't overvaccinate: every 3 years is adequate

Use FeLV vaccines only in high risk kittens under 12 weeks of age

Don't use interscapular site: put rabies into R.H. and FeLV in L.H. All other vaccines s/c in R. shoulder.

Avoid aluminium adjuvanted vaccines

www.catvirus.com

Oral cavity disease in cats in the USA

Healthy 34%

Healthy 19%

Healthy 12%

Ear mites 4%

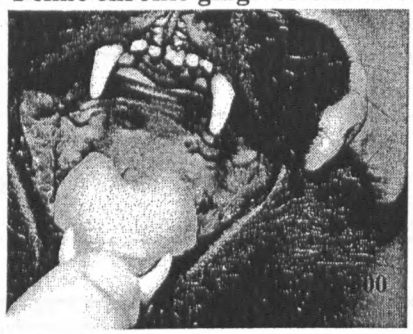
Cat bite abscess 2.5%

Chronic renal failure 2%

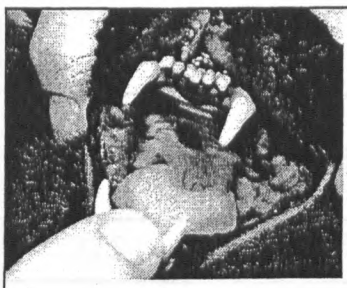
Johnson, 1998 In Practice 20 4 171-179

www.catvirus.com

Feline chronic gingivostomatitis



Addie D.D., Radford A., Yam P., Taylor D.J. 2003 Cessation of feline calicivirus shedding coincided with resolution of clinical signs in a case of chronic lymphocytic plasmacytic gingivostomatitis. Journal of Small Animal Practice. 44 (4) 172-176



Feline calicivirus isolated from almost 100% of these cases.

Feline herpesvirus isolated from 92%.*

*Lommer MJ, Verstraete FJ.Concurrent oral shedding of feline calicivirus and feline herpesvirus 1 in cats with chronic gingivostomatitis. Oral Microbiol Immunol. 2003 Apr;18(2):131-4.

Treatment of chronic stomatitis

Remove all the teeth

6 weeks antibiotics including metronidazole

http://www.gla.ac.uk/companion/stomatitis.htm

Orbital Penetration Associated with Tooth Extraction

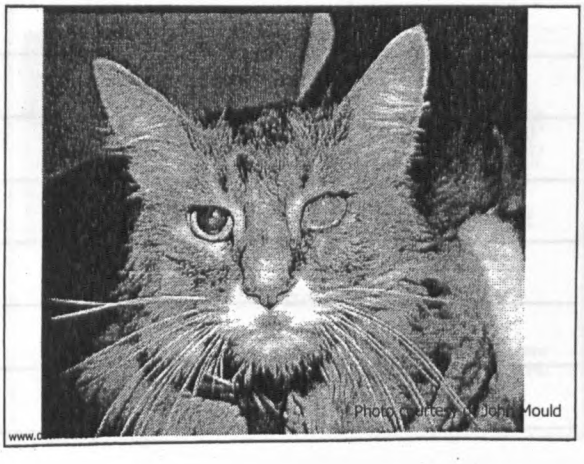
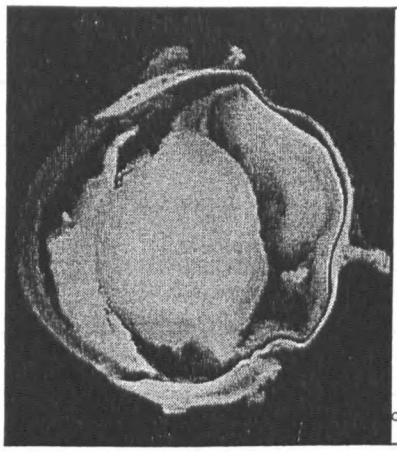
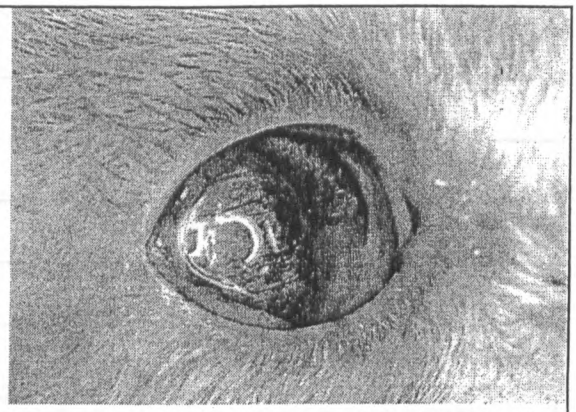


Photo courtesy of John Mould

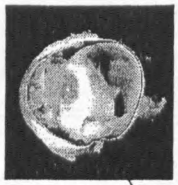


o courtesy of John Mould



www.catvirus.com

Photo courtesy of John Mould



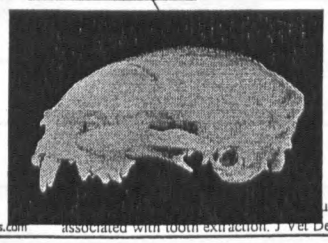
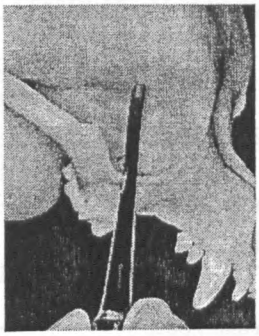


Photo courtesy of John Mould

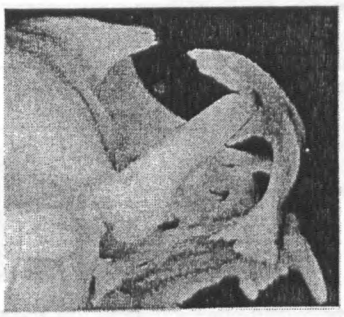
www.catvirus.com

uld J. Orbital penetration associated with tooth extraction. J Vet Dent. 2003 Mar;20(1):8-17.



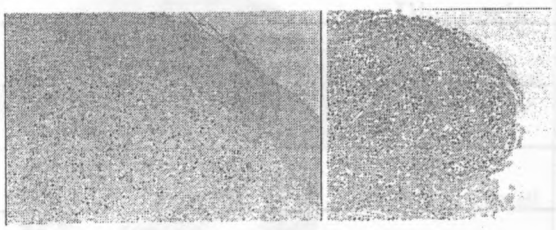
Smith MM, Smith EM, La Croix N, Mould J. Orbital penetration associated with tooth extraction. J Vet Dent. 2003 Mar;20(1):8-17.

Smith MM, Smith EM, La Croix N, Mould J. Orbital penetration associated with tooth extraction. J Vet Dent. 2003 Mar;20(1):8-17.



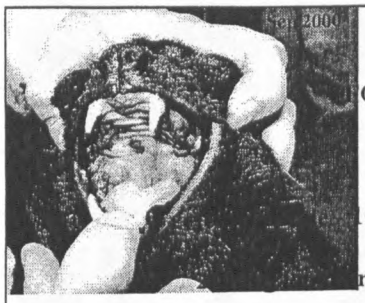
Smith MM, Smith EM, La Croix N, Mould J. Orbital penetration associated with tooth extraction. J Vet Dent. 2003 Mar;20(1):8-17.

Smith MM, Smith EM, La Croix N, Mould J. Orbital penetration associated with tooth extraction. J Vet Dent. 2003 Mar;20(1):8-17.



Shift from Th 1 to mixed Th 1 and Th 2

Harley R, Helps CR, Harbour DA, Gruffydd-Jones TJ, Day MJ. 1999 Cytokine mRNA expression in lesions in cats with chronic gingivostomatitis. Clinical and Diagnostic Laboratory Immunology. 6 4 471-478



CV shedding ceased

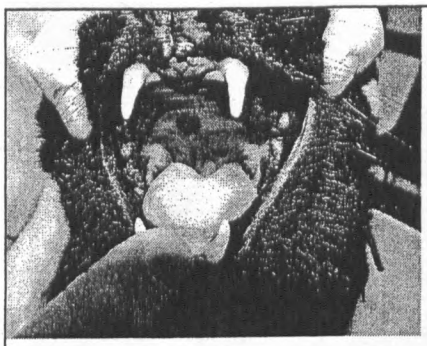
100mg at night

rrin applied directly

Butchers Classic cat food

Addie D.D., Radford A., Yam P., Taylor D.J. 2003 Journal of Small Animal Practice. 44 (4) 172-176

www.catvirus.com



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Treatment of chronic stomatitis

~~Remove all the teeth~~

6 weeks antibiotics including metronidazole

Anti-inflammatories: aspirin 10mg/kg every 2-3 days
Metacam
Thalidomide

50% of cases euthanased within 18 months

Steroids or Ovarid 5 mg 1/2 twice a week only if the alternative is death!!!

www.catvirus.com

http://www.gla.ac.uk/companion/stomatitis.htm

Treatment of chronic stomatitis

Interferon:

Virbagenomega

Intron A } 30 i.u. daily orally

Roferon }

http://www.virbagenomega.com

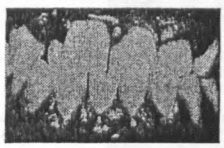
Antioxidants: vitamins C and E

Butcher's Classic cat food ???

www.catvirus.com

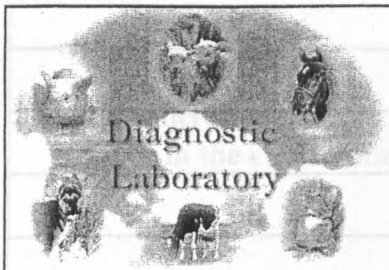
Butchers Classic cat food

Hills a/d



http://www.gla.ac.uk/companion/stomatitis.htm

www.catvirus.com



Diagnosis

Oropharyngeal swab for FCV isolation

$50.00 at Cornell

http://diaglab.vet.cornell.edu/dl_home.html

http://www.gla.ac.uk/companion/stomatitis.htm

www.catvirus.com

Diagnosis

Biochemistry:

high globulins

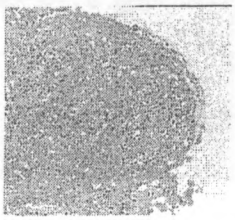
high α1-acid glycoprotein (AGP)

http://www.gla.ac.uk/companion/stomatitis.htm

www.catvirus.com

Diagnosis

Biopsy



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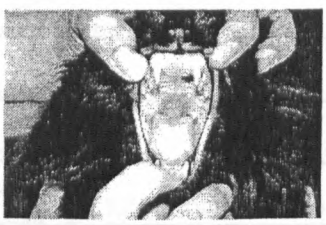


Eosinophilic granuloma

www.catvirus.com

Monitoring treatment

Weigh cat each visit

Photograph mouth

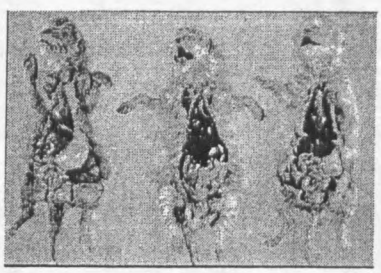
Monitor globulins

Monitor FCV shedding

Monitor AGP

www.catvirus.com http://www.gla.ac.uk/companion/stomatitis.htm

3 BSH faded kittens



2d	1wk	2d
NI	FCV	NI

www.catvirus.com

Feline calicivirus persists 24-48 hours in the environment

www.catvirus.com

Feline calicivirus isolated

from 24% of cats

at UK cat shows

Coutts *et al*, Veterinary Record 1994 135 555-556

www.catvirus.com

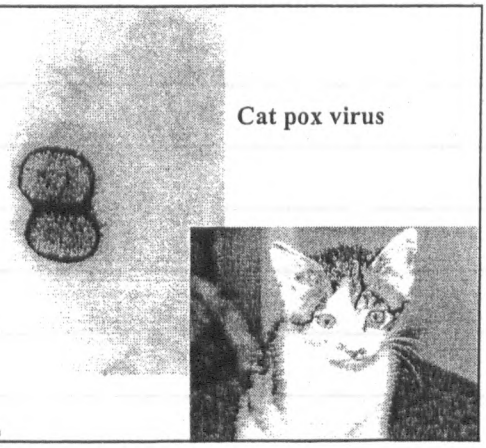
Differential diagnoses of oral ulceration:

FCV

poxvirus

something caustic

www.catvirus.com



Cat pox virus

www.catvirus.com

Feline poxvirus

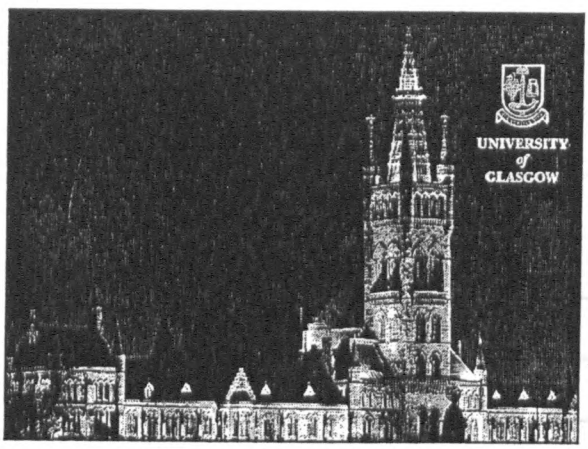
Occurs late summer, early autumn

Only free-ranging and usually hunting cats affected

Treatment: supportive

NEVER use corticosteroids

www.catvirus.com



Feline herpesvirus

aka Feline Viral Rhinotracheitis (FVR)

alphaherpesvirus (DNA)

over 90% of cats have been exposed to FHV-1

80% of exposed cats become lifelong carriers

www.catvirus.com

Feline herpesvirus

latency within trigeminal ganglia

reactivation after stress: lag period of 1 wk, then viral shedding for 1-2 weeks

www.catvirus.com

Diagnosis

Oropharyngeal swab

Conjunctival swab (always take BEFORE using fluorescein)

www.catvirus.com

Stress in the cat

being rehomed, moving house

new additions to house: baby, dog, cat

too many cats in one house (over 6)

going into cattery

surgery or trauma (e.g. RTA)

intercurrent illness

pregnancy, parturition, lactation

www.catvirus.com

FHV, FCV and FCoV shedding in a California shelter

Weeks after admission	FCV	FHV	FCoV
0	10/162	4/162	33/162
1	10/60	30/60	36/60
2	1/5	2/5	2/5

Pedersen *et al*, 2003. In press: Journal of Feline Medicine and Surgery

www.catvirus.com

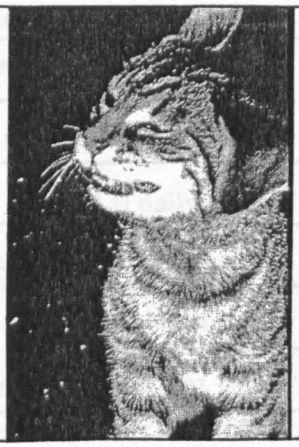
Feline herpesvirus

survives 12-18 hours in the environment

sensitive to all disinfectants

www.catvirus.com

Sneezed droplets can travel 1.3m



www.catvirus.com

Feline herpesvirus

Clinical signs:

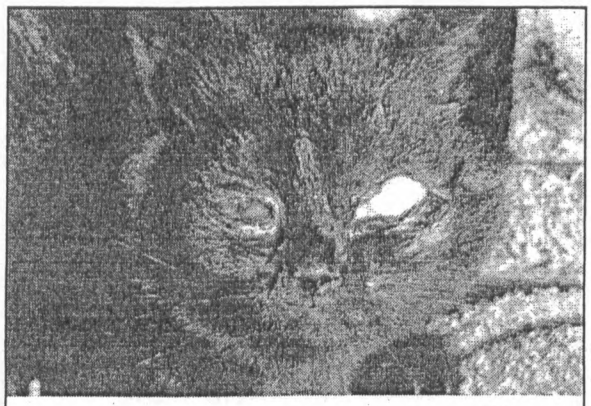
Cat flu
Fading kittens
Ocular lesions
Chronic rhinitis/sinusitis

www.catvirus.com

Ocular manifestations of FHV

corneal ulceration
chronic conjunctivitis
symblepharon
keratoconjunctivitis sicca
eosinophilic keratitis
stromal keratitis
corneal sequestrum

References:
D.J. Maggs 2001 Update on the diagnosis and management of feline herpesvirus-1 infection. Consultations in Feline Internal Medicine 4. Editor John August
S.E.Andrew Ocular manifestations of feline herpesvirus. JFMS 2001 3 9-16



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Treatment of ocular FHV

Trifluorthymidine ocular ointment

? Interferon

Human disposable contact lenses to protect debrided ulcer

??? drop of cat's own serum or plasma into eye

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Differential diagnoses of nasal discharge

Feline herpesvirus chronic rhinitis

Foreign body (e.g. grass blade)

Cryptococcus neoformans

Polyp

Tumour - usually squamous cell carcinoma

Treatment of FHV

Lysine – l-lysine 250mg orally bid

Human IFN α – 30 i.u. per day, orally

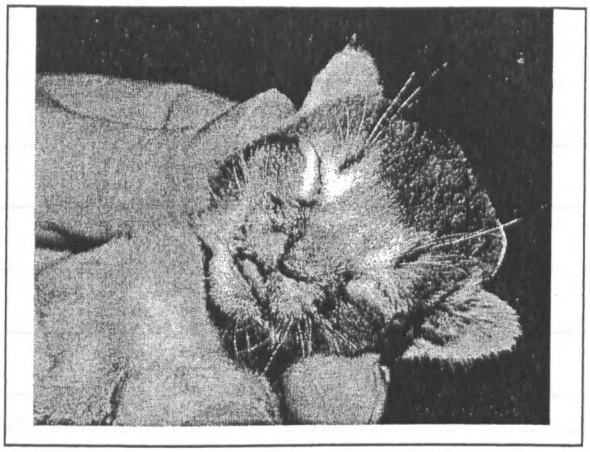
Feline IFN omega - 10,000 U daily, orally

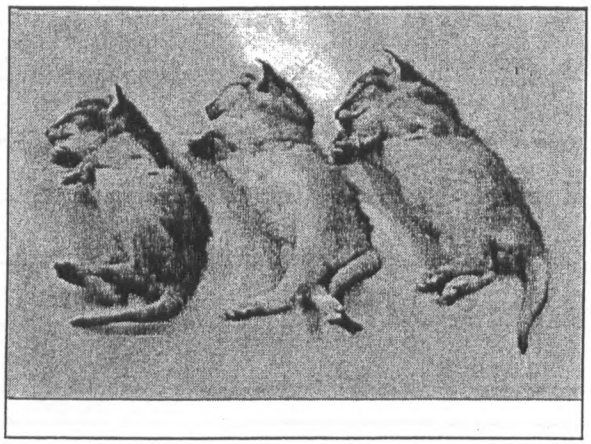
Steamy bathroom

Eucalyptus oil (or Vick's vaporub) on soft collar

Aromatic foods: sardines, chicken liver, tuna

www.catvirus.com





FHV control in breeding queen

Ideally, use virus free cats

Change housing of queen 3 weeks before parturition to allow for reactivation of latent virus and FHV levels should fall before birth

? Use L-lysine throughout pregnancy?

Feline herpesvirus vaccination

Essentially only one serotype

Vaccinate kittens at 8 and 12 weeks, boost at 1 year

Ameliorates clinical signs

Doesn't prevent latency

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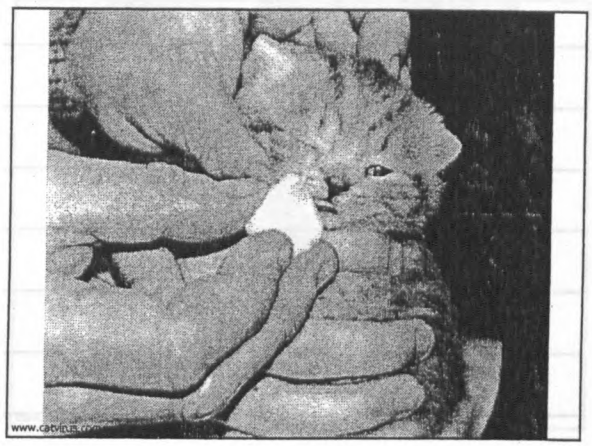

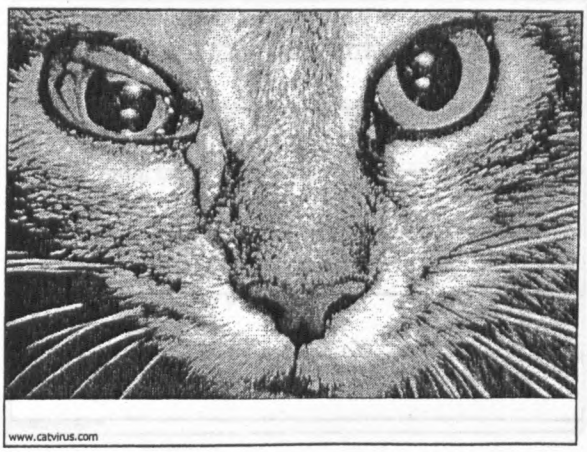

conjunctivitis

chemosis (swelling of the conjunctiva)

serous to mucopurulent ocular discharge

unilateral becoming bilateral

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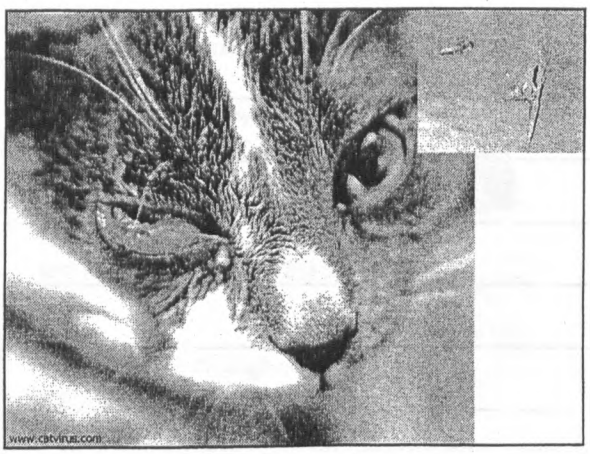
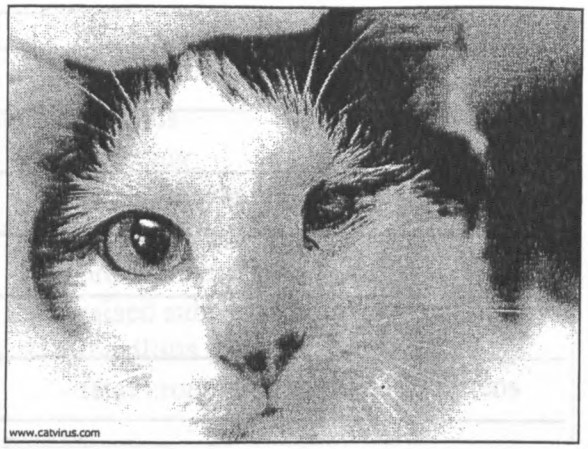
Chlamydophila felis

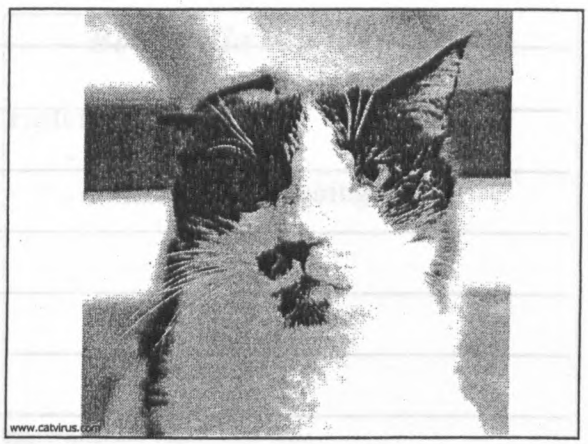
The obligate intracellular parasite

formerly known as *Chlamydia psittaci*

www.catvirus.com







Differential diagnosis of conjunctivitis:

- cat flu: feline herpesvirus (feline calicivirus)
- bacterial conjunctivitis
- foreign body (e.g. grass seed)
- corneal ulcer
- blocked nasolacrimal duct
- squashed up face - Persians
- (cryptococcus neoformans)
- (tumour)
- (pemphigous)

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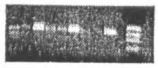

- sensitive
- not dependent on organism viability
- rapid
- specific
- possibility of false positive results
- organism can die rapidly in transit

NOT RECOMMENDED

- useful to rule out exposure
- does not correlate with *Chlamydophila* shedding

M. McDonald, B.J. Willett, O. Jarrett, D.D. Addie. 1998 A comparison of DNA amplification, isolation and serology for the detection of *Chlamydia psittaci* infection in cats. Veterinary Record. 143 97-101

Treatment of *Chlamydophila felis* infection

- doxycycline (Ronaxan) at 10mg/kg
(i.e. 2 x 20mg tablets s.i.d., or one bid for a 4kg cat)

- 1% chlortetracycline ointment (Aureomycin opthalmic ointment) q.i.d.

Treat for at least 4 weeks or until 2 weeks after the end of clinical signs

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Prevention

Test new cats before allowing them to mix with existing cats

Good hygiene between handling cats

Vaccination - does not prevent infection

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Bordetella bronchiseptica

Clinical signs
- sneezing
- ocular discharge
- harsh cough
- productive: white froth
- raised submandibular lymph nodes
- tonsillitis
- fatal bronchopneumonia of kittens

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Bordetella bronchiseptica

History
- in a cattery
- contact with coughing dog

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Bordetella bronchiseptica prevalence

Rescue catteries	19.5%
Breeding catteries	9.0%
Research colonies	13.5%
Household pets	0%

www.catvirus.com

Binns *et al*, Vet Rec 1999 May 22;144(21):575-80

Bordetella bronchiseptica - a zoonosis

Bordetella bronchiseptica has been isolated from immunosuppressed humans (HIV positive) with respiratory signs ranging from mild URT signs to pneumonia.

www.catvirus.com

Treatment

Doxycycline (Ronaxan)

20mg bid for 5 days

Trimethoprim sulfadiazine
(Tribrissen 20 or Trimacare 20 sid 5-7d)

? enrofloxacin (Baytril) - works in vitro

www.catvirus.com

Bordetella vaccination

Protex Bb

Uses of Bordetella vaccine

- all cats going into boarding catteries
- all cats going into rescue catteries
- cats going to cat shows
- cats going to live in multicat environment

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Differential diagnoses of Bordetellosis

- fur ball
- *Aelostrongylus abstrusus*
- intratracheal or bronchial foreign body

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Jerry, 1 y.o.

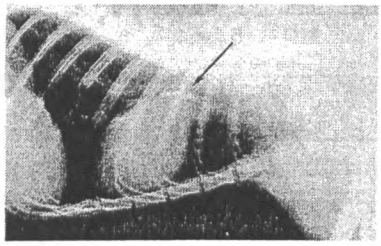
Harsh lung sounds

Dull

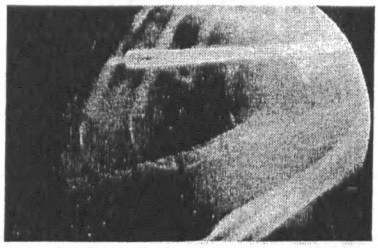
Breathing rapidly, open mouth

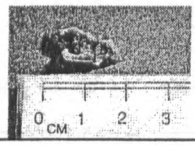
Cannon M. 2000 FAB Journal Vol 38 (3)

www.catvirus.com



www.catvirus.com





www.catvirus.com

Lungworm

Aleurostrongylus abstrusus

Coughing

Transmission: from infected intermediate host (slug, snail) or transport host (mouse)

Prevention: keep cat in, regular worming (Panacur)

www.catvirus.com

Vaccine associated sarcomas: AAFP recommendations

Don't overvaccinate: every 3 years is adequate

Use FeLV vaccines only in high risk kittens under 12 weeks of age

Don't use interscapular site: put rabies into R.H. and FeLV in L.H. All other vaccines s/c in R. shoulder.

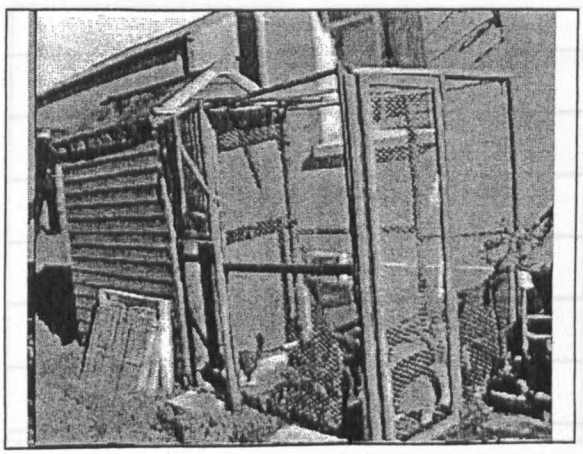
Avoid aluminium adjuvanted vaccines

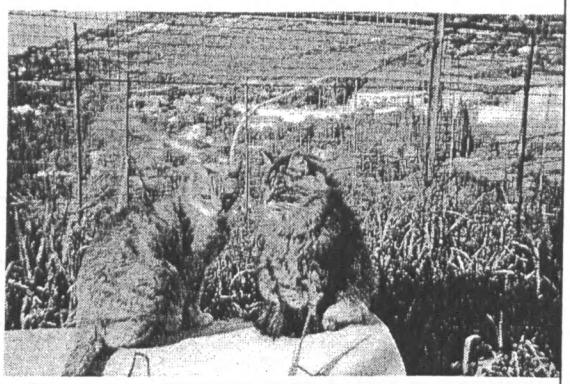
www.catvirus.com

"Because most client-owned cats had detectable serum antibodies suggestive of resistance to infection, use of arbitrary booster vaccination intervals is likely to lead to unnecessary vaccination of some cats."

Lappin MR, Andrews J, Simpson D, Jensen WA.Use of serologic tests to predict resistance to feline herpesvirus 1, feline calicivirus, and feline parvovirus infection in cats. J Am Vet Med Assoc. 2002 Jan 1;220(1):38-42.

www.catvirus.com





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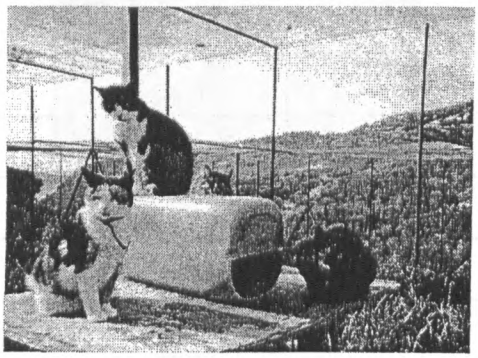

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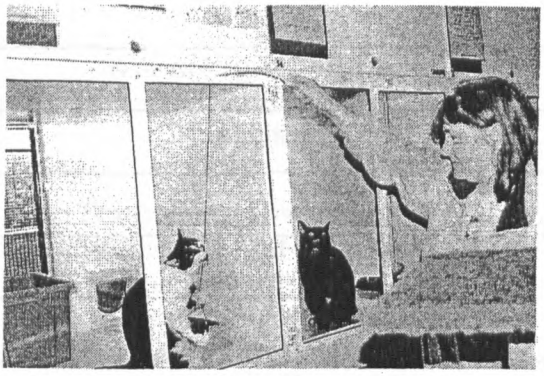
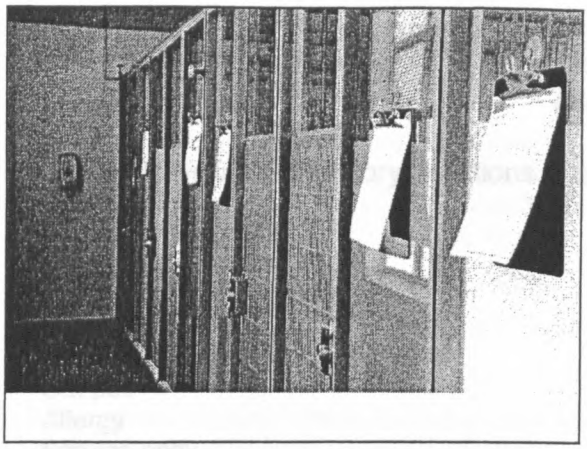


www.catvirus.com



For keeping up to date on feline chronic gingivostomatitis:

http://www.gla.ac.uk/companion/stomatitis.htm

For FCV and FHV isolation

http://diaglab.vet.cornell.edu/dl_home.html

Herpesvirus and Calivirus: What They Don't Want You To Know!

Diane D. Addie, BVMS, PhD, MRCVS
University of Glasgow
Glasgow, UK

The major feline respiratory infections of cats are:
Feline calicivirus
Feline herpesvirus
Chlamydophila felis
Bordetella bronchiseptica

Minor suspects in feline URT:
Cat pox
Allergy
Foreign body
Haemophilus felis
Aelurostrongylus abstrusus
Corynebacterium spp

Feline calicivirus (FCV)

FCV is less severe than FHV infection, but is still a cause of fading kittens, and cat flu in kittens over 2-3 weeks old and adult cats.

Signs: FCV commonly causes lingual ulceration, sneezing, anorexia, depression and oculonasal discharge. Fading kittens - post mortem shows thymic atrophy, congested lungs, the body will probably be underweight for the kitten's age.

Other signs in FCV: shifting lameness (especially some vaccines implicated). Acute haemorrhagic diarrhoea.

Virus shedding: in oropharyngeal secretions, shedding is continuous. Half life of 75 days.
Transmission: mainly direct, but FCV can survive up to 7 days in the environment so fomite transmission is possible.

Prevention

Test breeding queens by virus isolation or RT-PCR from an oropharyngeal swab. FCV is shed continuously, with a half-life of 75 days. Preferably breed using only negative queens. If not possible, MDA lasts up to 2-3 weeks, so kittens can be early weaned and kept in isolation from infected individuals. Use bleach diluted 1:32 in water with washing up liquid to disinfect. Serious cat breeders should try to prevent this virus from entering their household by testing all new cats and prospective mates of their cats before admitting them into their premises.

Vaccines do not protect against the majority of field strains, though Fort Dodge claim that their FCV strain (strain 255, which is also in the Merial vaccine) covers over 90% of field strains. Vaccines do not prevent asymptomatic carrier state.

Chronic stomatitis in the cat

While the exact aetiology of feline chronic lymphocytic plasmacytic stomatitis is not proven, it is probably caused by combination of FCV infection and the cat's inappropriate immune reaction within the stomatitis lesions (shift from normal Th type 1 response (cellular) to a mixed Th type 1 and 2 (humoral) response. (Harley *et al*, 1999)).

Treatment is aimed at restoring normal mouth flora (healthy cat's mouths have predominantly Pasteurella multocida, 50% of cats with stomatitis have spirochaetes), trying to eliminate FCV, and shifting the immune response back to type 1.

Diagnosis

Send an oropharyngeal swab in viral transport medium (vtm) to Cornell for FCV isolation ($50.00). You may also wish to send a biopsy of the lesion in 10% formol saline.

To request vtm, email Cornell: diaglab@cornell.edu or telephone on 607-253-3900

Treatment

Antibiotics
Metronidazole 4-6 weeks

Interferon
Intron A obtained as 3M I.U. from local pharmacist (write a prescription). Dilute in one litre of saline, aliquot into 1ml volumes, freeze for up to a year. Defrost as required, dilute 1:100 to get 30 I.U. per millilitre, keep refrigerated for up to a week.

Dose: 30 I.U. per day orally

New Virbagen Omega not yet assessed for clinical efficacy in these cases. However, we are about to begin a clinical trial using the following protocol:
Dose: 1.0 x 10^6 U/kg by injection every other day for 5 treatments
followed by 10,000 U orally daily until FCV shedding ceases.

Reconstituted solution can be kept in the fridge for up to 3 weeks.
We will also be using an anti-inflammatory and Butcher's food or Hills a/d.

Thalidomide
Fax prescription for 100 x 50 mg thalidomide (Sauramide) capsules to:

Dose: 1-2 x 50 mg capsules per day given in the evening
NOT to be used in pregnant queens

Food

It is uncertain what influence, if any, food plays in this condition, however, prior to the recovery of one case, the cat had been changed to Classic Cat Food (a tinned food made by Butcher's, available in Sainsbury's). After dentistry, cats fed on Hills a/d diet gained more weight and had shorter lesions than those fed on a control diet (Theyse *et al*, 2003).

Don't use a live FCV vaccine in these cats after recovery.

Further reading

Addie D.D., Radford A., Yam P., Taylor D.J. 2003 Cessation of feline calicivirus shedding coincided with resolution of clinical signs in a case of chronic lymphocytic plasmacytic gingivostomatitis. Journal of Small Animal Practice. 44 (4) 172-176

Harley R, Helps CR, Harbour DA, Gruffydd-Jones TJ, Day MJ. 1999 Cytokine mRNA expression in lesions in cats with chronic gingivostomatitis. Clinical and Diagnostic Laboratory Immunology. 6 4 471-478

Lauritzen A., Jarrett O., Sabara M. 1997. Serological analysis of feline calicivirus isolates from the United States and United Kingdom. Veterinary Microbiology. **56** 55-63

Theyse LFH, Logan EI, Picavet P. 2003. Partial extraction in cats with gingivitis-stomatitis-pharyngitis complex - beneficial effects of a recovery food. Proceedings Hill's European Symposium on Oral Care. Amsterdam 2003 64-65

Veterinary Clinics of North America. Feline Dentistry

http://www.gla.ac.uk/companion/stomatitis.htm

Feline herpesvirus

A more severe infection than FCV, causing fading kittens and cat flu. Affected kittens can be left with lifelong chronic sinusitis. Over 90% of cats have been exposed to FHV-1 and 80% of exposed cats become lifelong carriers.

Virus shedding: in oropharyngeal secretions, shedding is intermittent and occurs about a week following stress, lasting 1-2 weeks.

Examples of stress in a cat:

- being rehomed,
- moving house
- new additions to house: baby, dog, cat
- too many cats in one house (over 6)
- going into cattery
- surgery or trauma (e.g. RTA)
- intercurrent illness
- pregnancy, parturition, lactation

Transmission: mainly direct, FHV can only survive up to 12-18 hours in the environment, fomite transmission is possible only within a household.

Clinical signs

Fading kittens - post mortem shows thymic atrophy, congested lungs, the body will probably be underweight for the kitten's age. Histopathology shows acidophilic intranuclear inclusion bodies. Kittens which survive to 14 days may have very swollen eyes, with corneal ulceration or even ruptured eyeballs under their still closed eyelids. Kittens over 2-3 weeks old show typical cat flu signs: sneezing, anorexia, depression, oculonasal discharge and pneumonia.

Ocular manifestations of FHV:

- corneal ulceration
- chronic conjunctivitis
- symblepharon
- keratoconjunctivitis sicca
- eosinophilic keratitis
- stromal keratitis
- corneal sequestrum

Diagnosis

Send an oropharyngeal swab in viral transport medium (vtm) to Cornell for FHV isolation ($50.00). Remember that FHV shedding is intermittent, so while a positive result is definitely positive, a negative result may simply mean that you have missed the shedding time. Try to time swabbing to be either at the beginning of the animal being symptomatic, or a few days to a week after stress occurring.

To request vtm, email Cornell: diaglab@cornell.edu or telephone on 607-253-3900

Treatment

Supportive: the cat should be tempted to eat with small but frequent portions of aromatic foods such as sardines, roast chicken or liver. In order to clear the nasal passages it is beneficial to the cat if he can be confined to a steamy bathroom for an hour each day. Vick Vapourub can be applied to the chin or a few drops of eucalyptus oil put on the cat's bedding. The cat should be cleaned gently with a cloth and warm water, especially if he can no longer groom himself, and kept warm until dry.

Cats with ocular discharge should have their eyes bathed three or four times a day with a warm solution of salt and water, using one teaspoonful of ordinary table salt (sodium chloride) in one pint (half a litre) of water.

Chronic sinusitis:

- antibiotics for long periods often helps,
- human interferon at 30 i.u. per day orally
- feline interferon at 10,000 i.u. per day orally

Lysine - l-lysine 250mg orally sid prevents successful FHV viral particle assembly.

Prevention

Test breeding queens by virus isolation from an oropharyngeal swab but bear in mind that FHV shedding is intermittent and occurs post-stress. Use the feature of virus reactivation by stress to advantage: stress carrier queen 2-3 weeks prior to kittening by moving her into her kittening room. This will cause viral recrudescence **before** the kittens are born, so shedding will be stopped when they are born. It will also boost her antibodies, giving the kittens increased MDA and a longer period of protection.

Cats entering a disease-free colony should be quarantined for 3 weeks and virus isolation attempted at least twice a week before being allowed to mix with the other cats. Vaccination does not prevent induction of a carrier state, so a carrier cat may never have shown clinical signs. Clean contaminated food bowls, etc. using bleach diluted 1:32 in water with washing up liquid to disinfect.

References and further reading:

D.J. Maggs 2001 Update on the diagnosis and management of feline herpesvirus-1 infection. Consultations in Feline Internal Medicine 4. Editor John August

S.E.Andrew Ocular manifestations of feline herpesvirus.
JFMS 2001 3 9-16

Website for cat owners

http://www.gla.ac.uk/companion/ocatflu.htm

Dental Laboratory

Daniel T. Carmichael DVM, Dipl. AVDC, FAVD
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(516) 420-0000

The focus in this year's dentistry laboratory will be on feline oral and dental surgery. Experience in performing oral and dental surgical procedures will be gained through performing the following procedures:

1. Surgical extraction of a maxillary canine tooth.
2. Crown amputation for FORL.
3. Partial rostral mandibulectomy.

Extraction of a Maxillary Canine Tooth

Introduction

Dental extraction should be considered an end-stage or salvage procedure. Once the tooth is gone–that's it. Unfortunately, we frequently get cases where teeth are already in the end-stage of disease, and extraction is not only advisable but also necessary. As veterinary dentists, our first goal is to relieve pain and prevent infection.
Veterinarians performing surgery in the mouth should adhere to the same general principles of surgery elsewhere in the body.

General Principles to Consider:

1. Go slowly--take your time. The time it takes to fix mistakes (like retrieving a fractured tooth root) is always more than the time to carefully avoid them.
2. Remove as little bone as needed to get the job done, but do not hesitate to remove as much bone as needed to get the job done.
3. Maintain hemostasis. In almost all cases, application of direct pressure for three to five minutes will stop oral bleeding.
4. Never suture under tension.
5. Always suture fresh-cut epithelium to fresh-cut epithelium.
6. Place your suture line over bone, not over holes.
7. Treat your gingival flaps with respect.
8. Do not traumatize the adjacent teeth that are not being extracted.
9. A pre-treatment radiograph is always indicated.

Extraction is indicated for teeth in the end-stage of periodontal disease, fractured teeth with exposed pulp (when endodontic treatment is not possible), teeth with FORL, retained deciduous teeth, crowded or malpositioned teeth, and in other select cases. Always get the client's consent prior to performing extraction to avoid misunderstandings.

Equipment

- #15 scalpel blade
- Dental elevators and luxators of various sizes
- Extraction forceps
- Suturing equipment (4/0 chromic gut is this author's preference)
- Gauze sponges
- Dental drill with water cooling; round (#'s 2 and 4) and cross-cut fissure burs
- Periosteal elevator (Freer or Molt)

Extraction procedure

1. Mucoperiosteal flap

A mucoperiosteal flap is created by making two full-thickness incisions (through mucosa and periosteum) with a #15 blade starting at the gingival margin on either side of the tooth to be extracted and extending them apically. The incisions should diverge so that the base of the flap is wider than the gingival edge. The scalpel blade is then used to incise the epithelial attachment of the flap at the gingival margin. Next, the flap is raised with a ST-7 (Henry Schein), Molt, or Freer periosteal elevator. When using the elevator to create the flap, keep the pressure angled into the bone to incorporate periosteum in the flap. The flap should contain mucosa and periosteum. Once raised, the flap should expose the buccal cortical bone overlying the tooth root.

In cats, the mucoperiostium is quite thin and fragile. Use extra care not to rip or tear the flap.

2. Buccal cortical bone removal

Once exposed, the buccal cortical bone overlying the root is removed with a round bur on a high-speed dental drill. Water-cooling is essential to prevent heat necrosis of the surrounding bone. The juga is the bony prominence overlying the root. Palpation and visualization of the juga gives landmarks for bone removal. As the bone is removed, the root can be visualized as a color change. Use the bur with a gentle touch, and keep it moving.

3. Elevation

The canine tooth root must now be separated from its alveolar attachment. Teeth are held in the socket by the periodontal ligament. Therefore, the periodontal ligament must be severed to accomplish extraction. The first step is to stretch the periodontal ligament fibers. These is done by inserting the elevator in a "fulcrum" position and rotate or lever it to apply stretching pressure to the periodontal fibers. This also causes hemorrhaging in the periodontal space, further stretching the fibers (by hydraulic force). The tooth should be held in the distracted position for 10 seconds. Next, find another fulcrum point, and repeat the procedure again, holding the tooth in the distracted position for 10 seconds. This will eventually stretch the periodontal fibers to a point of fatigue, at which time the tooth will give. .

Elevation is the step where the majority of root fracture complications occur. Go very slowly--a few extra minutes being careful avoids many extra minutes digging out broken root tips.

4. Alveoloplasty

Alveoloplasty is the procedure to remove any rough or sharp bony projections in the extraction site. It is best accomplished with a large (#4) round bur operated on a high-speed handpiece with water-cooling. Alveoloplasty is complete when digital palpation of the extraction site reveals no sharp projections.

5. Suturing

The final step in the surgical extraction procedure is suturing the extraction site. Suturing reduces postoperative hemorrhage and allows primary healing. The general rules of surgery apply for oral surgery as well; especially, that the sutures should not be under tension. To relieve tension, the flap may be rendered more pliable by fenestrating the periosteum. This is performed by gently incising the periosteal fibers on the underside of the flap with a #15 blade. Be careful not to sever the flap.

The suture material of choice for oral surgery should be absorbable and not bulky. 4/0 Chromic Gut and 4/0 Monocryl (Ethicon; Somerville, NJ) are good choices.

Crown Amputation for treatment of Feline Odontoclastic Resorptive Lesions

Crown amputation is a procedure where tooth roots are intentionally left in the mouth. The only indication for this procedure is for treatment of teeth affected with feline odontoclastic resorptive lesions with no evidence (verified radiographically) of periodontal disease. Improper use of this

technique can lead to disastrous consequences to the patient. Therefore, case selection is extremely important.

Start by making a mucoperiosteal flap ("mini-flap") that exposes the junction of tooth and alveolar bone. Next, using a high-speed handpiece with water cooling, section off the crown with a #2 round bur. Perform this sectioning right at the level of the alveolar bone/tooth junction, trying not to traumatize the alveolar bone. Finally, suture the flap closed with #4/0 absorbable suture material.

Partial Rostral Mandibulectomy

A partial rostral mandibulectomy is indicated for definitive treatment of certain neoplastic processes involving the rostral mandible. In cats, the majority of oral neoplasms are squamous cell carcinoma and carry a poor prognosis, even with radical surgical intervention and adjunct therapies.

Partial rostral mandibulectomy would be indicated for feline rostral mandibular neoplasms where *en bloc* resection of the mass can be curative. Treatment planning prior to surgery is paramount, but beyond the scope of this laboratory.

Surgical Procedure:

1. Establish margins:

   Incise the oral epithelium to the level of the bone circumferentially around the mass. The margin of normal tissue appropriate for the tumor type being excised can vary from 5 mm to 20 mm. Do not encroach on or manipulate the tissue within this circle for the remainder of the procedure.

2. Create incisions for exposure of mandible:

   Starting on the above-created circle, extend incisions along the dental arch rostrally to the midline (symphysis), and caudally to the distal aspect of the molar tooth. Repeat this procedure on the lingual aspect as well.

3. Elevate mucoperiostium:

   With a periosteal elevator, reflect buccal and lingual mucoperiostium to the level of the ventral mandible. In the area between the canine tooth and third premolar on the buccal surface of the mandible, you will encounter the middle mental artery that can be ligated.

4. Separate the mandibular symphysis:

   With a #15 scalpel blade, incise the cartilaginous attachment of the mandibular symphysis. Then, insert a dental elevator into the symphysis and rotate it to complete the separation.

5. Incise the ventral attachments to the mandible:

   Grasp the mandible near the symphysis with a bone-holding forceps or a dental extraction forceps. Apply outward traction on the mandible. With Mayo scissors, keeping right on the periosteum of the ventral mandible, incise the muscular attachments in a rostral to caudal direction to the level of the proposed caudal sectioning.

6. Incomplete sectioning of the mandible:

   With a #2 or #4 round bur (surgical length is ideal), section the mandible at the caudal site of the proposed partial mandibulectomy starting dorsally (where the teeth are) and progressing ventrally about one-half way. Next, using the same bur, make fenestrating holes around the ventral one-half of the mandible, taking care not to perforate deep into the mandibular canal.

7. Completing the partial mandibulectomy:

   Insert a dental elevator or osteotome into the slot created in the dorsal half of the mandible and rotate it to fracture the mandible along the line of fenestrations. Be careful

to preserve the mandibular astery by not creating excessive distraction of the two segments.

8. Ligate the mandibular artery:

   Carefully distract the two segments of mandible to expose the mandibular artery. Gently place a mosquito forceps on the artery, and double ligate it. Sever the mandibular artery and monitor for hemorrhage. Remove the resected partial mandible.

   Using a #4 round bur, smooth the edges of the remaining caudal mandible.

9. Closure:

   Suture the opposing epithelial surfaces in one layer with fine, absorbable suture.

10. Crown amputate or extract the contralateral mandibular canine tooth:

    Following partial rostral mandibulectomy, the opposite mandible will drift toward the midline. The mandibular canine tooth on the opposite side will then traumatize the hard palate. To avoid this post-operative complication, crown amputation of the mandibular canine tooth and performing a direct pulp capping procedure should be done. Alternatively, the tooth can be extracted.

Post operative pain management and appropriate antibiotic therapy should be continued for 5 days.

The Basics of Soft Tissue Laser Surgery

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Introduction

Today, veterinary medicine is rapidly accepting laser surgery as a surgical therapeutic modality due to the decreased postoperative pain, bleeding, and swelling associated with its use.[1-5] Presently, the most commonly used form of laser for small animal soft tissue surgery applications uses CO_2 gas as a medium. When CO_2 is stimulated by electricity within a resonant cavity a coherent, collimated, monochromatic beam of light is produced at an invisible wavelength of 10.6 µm. This wavelength is ideal in small animal soft tissue surgery due to the fact that it is highly absorbed by water (which constitutes 90% of the volume of most soft tissues cells) and does not scatter within the tissue. The energy absorbed by the tissues is rapidly converted to heat and causes instantaneous boiling of intracellular water. The rapidly expanding volume of steam causes lysis of the relatively weak cell membranes. The resulting desiccated cellular solids are transformed into a plume of smoke. This process is called vaporization.

CO_2 laser energy offers several advantages over surgery with a scalpel. CO_2 laser light can incise and ablate tissue cleanly when the beam is focused on the tissue surface. Moving the focal area of the laser beam proximally or distally relative to the tissue surface will cause the beam to be defocused at that point and can produce cauterization of tissue when used in this mode. The CO_2 laser provides moderate hemostasis, whereby small blood vessels that are less than half the diameter of the laser beam (up to 0.5-0.6 mm diameter) are coagulated and sealed. Lymphatics are similarly sealed following laser vaporization, causing less post-surgical swelling.[5] Most notably, there is less pain following CO_2 laser surgery since vaporized small nerve endings are sealed and do not initiate action potential at the endings of pain sensors. Laboratory evidence of reduced uptake of horseradish peroxidase following transection by CO_2 laser energy supports this theory.[6] Finally, bacteria, fungi and viruses are also vaporized with CO_2 laser energy, so tissues may be disinfected during surgery, making it a useful tool in wound debridement.[1-4,7,8]

Semiconductor technology has also made its appearance in laser surgery applications and is available for surgical use in several distinct near-infrared wavelengths of intense light. Near infrared light (810-1064nm) is delivered to tissues *via* a quartz fiber. At the tissues, this energy is moderately absorbed by pigments and is poorly absorbed by histologic water. However, laser light at these wavelengths also scatters within the tissue quite readily. Melanin rich tissues and vascular lesions are ideally suited as targets for diode laser surgery procedures. These tissue pigments absorb the laser energy of near-infrared wavelengths quite readily, where it is transformed into heat and rapidly conducted to surrounding tissues. The resulting heat distribution causes prolonged coagulative necrosis and thermal injury up to 10mm away from the originally targeted area, making it a bit more difficult to predict the depth of thermal injury of a given amount of laser application time. In contrast,[9] CO_2 laser energy rarely causes thermal necrosis beyond 1mm from the incised or ablated tissue. Consequently, diode lasers and the Nd:YAG laser are ideally suited for cauterization and coagulation of highly vascular tissue, whereas CO_2 lasers are ideal for ablative applications where minimal collateral thermal damage is desired, or for incisions in the absence of large vessels.

Presently, diode surgical lasers do have some notable advantages. Diode laser energy may be transmitted by á quartz optical fiber. Quartz will transmit laser wavelengths between 300-2100nm. Outside of this range the light is unacceptably attenuated and not usable at the distal end.

These fibers are easily passed through flexible fiberoptic endoscopes and have been used in large animal upper respiratory tract surgeries.[10-12] Although diode laser energy can be passed through an endoscope, a CO_2 laser would be more appropriate for surgery in this area. Since laser light wavelength of 10.6 µm will not pass through optical quartz fibers, veterinary endoscopic surgeons have had to use near infrared wavelengths. If a suitable transmitter of far infrared light were available, then it would be more sensible to use it in conjunction with a flexible endoscope to avoid surgical complications associated with diode lasers.

To deliver the light energy generated by a CO_2 laser, a suitable reflective light pipe must be used to transmit the beam from its source to the distal end where it is used. Usually a series of articulated arms are utilized to allow such transmission by a combination of highly reflective mirrors perfectly aligned within the mechanical system. The mirrors may be fixed in position, or they may be mounted within jointed elbows having an exceptionally high flexibility. Generally, transmission systems using sequential mirrors within articulated arms will produce a collimated laser beam at the distal end. Collimated beams do not allow for changes in power density with distance from the target. *Power density* is defined as the power that is delivered over a given area. This means that at great distances from the working handpiece, the power density of the collimated beam is still as high as it was at its origin (unless mechanically focused).

The major disadvantage of an articulated arm for transmitting laser light is its fragility. They may also be much less flexible and more unwieldy to manipulate compared to flexible optical quartz fibers. It is extremely difficult and costly to maintain these devices in perfect alignment for proper use as well. The great advantage of using an articulated arm, however, is that it is the most efficient mode of radiant energy transmission, allowing preservation of beam geometry and the ability to employ extremely high power emitted from a laser.

Recently, commercially available hollow reflective light guides[a] have been produced for transmitting laser wavelengths not suited for fiberoptic transmission. They are generally highly reflective, slender, hollow, cylindrical tubes of 1.0m or 1.5m lengths that are very flexible. They are frequently misnamed "fibers", and may actually lose a great deal of energy due to repeated internal reflections within the lumen of the hollow waveguide. Additionally, these flexible waveguides do not produce a collimated beam at the distal end. The end of the flexible hollow waveguide must have a convergent handpiece and focusing tips to be used properly.

Focusing tips for a CO_2 laser are a necessity. They are placed within a handpiece that attaches to the end of the flexible hollow waveguide. They are named according to the diameter of the beam at its focal area, and come in diameters of 1.4mm, 0.8mm, 0.4mm and 0.3mm. The tips are also straight, curved, extremely curved and also are cut to several different lengths. When these tips focus laser light, it can interact with tissue and create a circular "spot" that is equivalent to the diameter of the focusing tip when the handpiece is held at the focal distance, usually 1mm-3mm away from the target tissue. Since the area of this circular spot is related to the square of the diameter, by increasing the spot size using the same power setting, the power density of the beam will diminish exponentially.

For example, the power density of 8W focused by the 0.8mm tip is the same as the power density of 2W focused by the 0.4mm tip. Because the focused beam becomes divergent distal to the focal area, it is possible to reduce the power density of the spot by moving the handpiece farther away from the tissue. Since coagulation is the predominant tissue effect when using lower power densities, and vaporization is predominant with higher power densities, it is possible to have great control over the desired tissue effect with a focused laser beam. Neither collimated beams nor fiberoptic laser delivery systems provide this instantaneous user-directed change in power density for surgical uses.

The availability of relatively inexpensive medical lasers has made it more practical for the veterinarian to own and operate this surgical tool. After first becoming educated in laser physics and safety, the veterinary practitioner can quickly master its use. Currently, one manufacturer of medical lasers[a] has directed energy to developing the veterinarian's accessibility to laser technology in private practice. This author's experience has been favorable. Using the following basic preparations, the surgical CO_2 laser can be used every day.

Equipment and Preparation

Standard surgical decorum is followed in the laser surgery suite. Only specifically trained veterinarians should perform laser surgery. A few simple accessories will enhance the efficiency of any laser-assisted procedure. It is absolutely imperative that all veterinary technicians are trained in laser hazards and safety as well. Veterinary technicians should also be well trained in maintaining the equipment and laser surgery procedure logs.[1] A standard "laser in use" warning sign should be highly conspicuous near the operating room as a warning to passers-by.[13-15] I keep all of the following items readily accessible as the laser is not used as a specialty instrument, but is used in most of the procedures I perform.

A smoke evacuation system must be used to remove the plume created by laser interaction with tissue. Carcinogens, noxious gases, irritating suspended particles, fungi, viruses and bacteria should not be inhaled by anyone in the surgery suite.[16-20] A laser surgery mask should be worn over the nose and mouth during any procedure. These masks have very fine pores (<0.5µm) that filter out any suspended airborne particles missed by the smoke evacuator and also protect the patient from the contaminated gases exhaled by the surgeon. Veterinary technicians should monitor the time that the smoke evacuating systems have been used, and ensure that this vital safety equipment is functioning properly.[1]

Direct or indirect laser light can be permanently harmful to the eye. All persons near the surgical suite should wear goggles or other eyewear that will attenuate harmful laser wavelengths and intensities. These protective goggles are specific for the wavelength of laser light they can filter,[15] and are also rated by the diminished ten-fold factor of attenuated light intensity they provide to the user *e.g.*, OD 6 goggles diminish the intensity by a factor of 10^6. Contact lenses alone are NOT adequate. The eyes of the patient should also be shielded from potential stray laser beams. Observation windows require a protective covering that provides an adequate margin of safety.[13-15]

The straight handpiece and the angled handpiece are the most common attachments to the hollow flexible fiber waveguide. Any of the required focusing tips will fit snugly into the end of the handpieces to focus the laser beam emerging from the fiber. Both handpieces and tips are autoclavable, gas sterilizable, or can be soaked in disinfecting solution. Having several handpieces previously sterilized will help smooth the preparation for sterile surgical procedures. Other techniques to keep the handpiece sterile include tying a sterile latex glove around the handpiece, placing a plastic sleeve over the handpiece, or using a handpiece shroud.[21]

The scanner is a pattern-generating device that is an optional accessory providing an interesting benefit. It delivers an effective zone of vaporization of 3mm diameter by using an instrument specific 0.8mm tip that is eccentrically positioned around a rotating axial head. The scanner has an autoclavable shield that can be used to keep the surgeon's hand and field sterile. It is most useful for large areas of tumor ablation, entropion repair, scar reduction, skin graft host site preparation, acral lick granuloma treatment and removal of multiple benign cutaneous masses. Purchasing a CO_2 laser delivery system with enhanced capabilities is required to use the scanner.

The laser assisted uvular palatoplasty (LAUP) kit is recommended if the surgeon wishes to perform correction of elongated soft palates in brachycephalic breeds.[22,23] This kit also has a longer handpiece without a backstop for use in areas that are deep. These handpieces are also autoclavable, gas sterilizable, and can soak in disinfecting solution. They each have a special port at the end for a smoke evacuator hose. This tool is most useful in the oropharynx where there is limited room for maneuvering instruments. The smoke evacuator port obviates the need for a technician to operate the larger wand usually employed for plume removal. A variety of disposable sterile tips are used with a surgical CO_2 laser. They are individually packaged in γ-irradiated sterile wrappers. They are opened using sterile technique, and may be either disposed after one use, or reprocessed and used again. They can be gas or steam sterilized, and they can also be soaked in disinfectant solution. Excess char buildup will eventually end the life of the tip, its performance waning steadily until that point. A solution of 1:10 bleach will disintegrate most char that builds up on the ceramic tips, but may permanently damage metallic tips. Detergent enzyme solutions[b] are also available, which take longer to digest the char but are gentle to the focusing tip. An ultrasonic scaler-bath is also a useful way to dislodge any adherent char from the aperture of a focusing tip. Spraying pressurized air[c] through the tip after its use can also eliminate some char. Tips can be cold sterilized

between uses in an appropriate disinfectant solution, but must be completely dry before use. The increasing supply of tips has lowered their cost, so it is wise to discard the tip after a few uses and incorporate that cost into the operating expense of the laser. It is contraindicated to use faulty tips that will cause excessive heating of target tissues or damage to the hollow flexible waveguide. Finally, for the purpose of convenience, it is practical to keep a tacklebox nearby to neatly organize the tips and other accessories in one compact area.

Sterile gauze and sterile saline should always be available at the beginning of every laser surgery procedure. It is essential to have this ready to wipe away any char that forms at the edges of the incision. Since char is a tissue irritant it is undesirable to leave it within incisions because doing so may lead to wound dehiscence. Moistened gauze also is used as a barrier to absorb any stray beams of laser energy during a procedure. This serves to protect nearby structures from inadvertent thermal injury.

It is also impressive to use an octylated cyanoacrylate[d] following CO_2 laser surgery. It is most useful in feline declaw procedure and routine spay and neuter procedures. It is a clear tissue adhesive which functions differently than the regular blue, butylated cyanoacrylate. Because a laser incision that is made correctly will be clean and dry, this clear, uncolored skin closure product will allow the incision edges to remain in opposition. If the wound edges are oozing or bloody it will not be effective. Also, it is not to be buried under the skin, therefore, when used in the feline declaw procedure it must be applied to the dry and clean incision edges only. If the incision is not made correctly, or if there is remaining char at the wound edges, there will be surgical complications *e.g.* wound dehiscence, and accordingly poor technique should be improved.

In general, any incision is enhanced with the superpulse (SP) temporal pattern, available with an appropriately equipped CO_2 laser unit. Because the superpulse temporal pattern creates sharp and rapid peaks of intensity followed by intermittent rests, there is an optimal balance of vaporization and thermal relaxation of target tissues. To create an incision with the laser the surgeon should produce tension at the leading edge of the beam that vaporizes tissues. This causes the characteristic clean and dry charred separation of tissue at the zone of vaporization. The zones of thermal necrosis and thermal conduction are thus greatly diminished and create a superior incision having negligible thermal effects to surrounding tissues.[24-26] There is some superficial char formation when superpulse mode is used, however, it is almost completely removed following gentle wiping of wound edges with sterile moistened gauze. Some surgeons may also prefer to repeat pulse the superpulse mode (30 Hz, 25msec pulse width = 75% power) to add an additional degree of control in making an incision in a delicate anatomical area (*e.g.* for stenotic nare correction or ferret adrenalectomy).

In contrast, when continuous wave (CW) or repeated pulse temporal patterns (PW) are used there is greater carbonization of tissues and more char remains. These settings will also cause slightly larger zones of thermal necrosis and thermal conduction for an equivalent zone of vaporization, thus a higher degree of incisional hemostasis should be expected compared to an incision made using superpulse mode. If superpulse is not available, the surgeon may still wish to minimize char formation created with incisions. Several repeat pulse settings are available on all CO_2 laser units. After experimenting with several settings, I have found that 40% power repeat pulse (20 Hz, 20 msec pulse width) is the closest setting to superpulse available at this time. A table of common settings is available in the user's manual supplied by the manufacturer.

Summary

There are many surgical procedures not described herein that are performed better with a surgical CO_2 laser. I am continually impressed with the efficiency of this surgical laser wavelength. Many areas of the body that are generally perceived as difficult by other means due to bleeding tendencies are now manageable: the eye, ear, nose and throat being the most commonly cited. Skin fold-plasty, incisions for lipoma removal, treatment of furunculosis are all common cosmetic procedures easily performed with a laser. Besides skin incision for surgical sterilization, my favorite laser surgical procedures are cystotomy, thyroidectomy, elongated soft palate correction, anal sacculectomy, perianal adenoma removal, acral lick granuloma and indolent ulcer treatment, because there is minimal or no hemorrhage during the procedure and visibility is unsurpassed. The potential for important future innovative procedures is enormous.

The author has observed that by using a CO_2 laser the patient's post-surgical comfort is dramatically improved over traditional techniques. More detailed studies are needed to organize and document these findings. A new discipline of veterinary surgery is unfolding before us. At no other time in history have pets been anthropomorphized as the present. It is every veterinarian's responsibility to minimize animal suffering in any way possible. Therefore, if CO_2 laser surgery is truly a superior technique in its appropriate application, it should be utilized as an every day tool for surgical procedures such as skin tumor removal, sexual sterilization, and feline declaw.

References

1) Lucroy, M. D. & Bartels, K. E., Using Biomedical Lasers in Veterinary Practice. Veterinary Medicine Special Supplement. Oct. 2000, pp. 4-9.

2) Lucroy, M. D. & Bartels, K. E., *Surgical Lasers, Textbook of Small Animal Surgery.* Slatter, 3rd Edition, 2001.

3) Bartels, KE, Current Techniques in Small Animal Surgery IV, Williams & Wilkins, Baltimore, MD, 1998

4) Carruth, J. A. S. & McKenzie, Medical Lasers: *Science and Clinical Practice.* Adam Hilger, Bristol, 1986.

5) Durante EJ, Kriek NP, Clinical and Histological Comparison of Tissue Damage and Healing Following Incisions with the CO_2 Laser and Stainless Steel Surgical Blade in Dogs, JSAVA, 1993 Sep;64(3):116-20

6) Fligny, I, et al., Comparative Study of CO_2 Laser and Scalpel Nerve Transections, Lasers Surg Med 1992;12(1):43-50.

7) Wright VC, Laser Surgery: Using the Carbon Dioxide Laser, Can Med Assoc J 1982 May 1;126(9):1035-9

8) Verschueren RJ, Oldhoff J, The Carbon Dioxide Laser: A New Surgical Tool, Arch Chir Neerl 1975;27(3):199-207

9) Fisher JC, Basic Laser Physics and Interaction of Laser Light with Soft Tissue. In: Shapshay SM ed. Endoscopic Laser Surgery Handbook, New York, Marce Dekker, 1987.

10) Tate LP et al Transendoscopic Nd:YAG Laser Surgery for Treatment of Epiglottal Entrapment and Dorsal Displacement of the Soft palate in the Horse. Vet Surg. 1990 Sep-Oct;19(5):356-63..

11) Tate LP. Application of Lasers in Equine Upper Respiratory Surgery. Vet Clin North Am Equine Pract. 1991 Apr;7(1):165-95.

12) Tulleners E. Instrumentation and Techniques in Transendoscopic Upper Respiratory Tract Laser Surgery. Vet Clin North Am Equine Pract. 1996 Aug;12(2):373-95.

13) Sliney, D. H. & Wolbarsh, M. L., *Safety with Lasers and Other Optical Sources.* Plenum Pub. Corp. New York, 1980.

14) Sliney, D. H. & Trokel, S. K., *Medical Lasers and Their Safe Use.*Springer-Verlag, New York, 1992.

15) American National Standards Institute, *Safe Use of Lasers*, ANSI Standard Z136.1-3

16) Baggish MS, Elbakry M. The Effects of Laser Smoke on the Lungs of Rats. Am J Obstet Gynecol. 1987 May;156(5):1260-5.

17) Nezhat C, et al., Smoke From Laser Surgery: Is There a Health Hazard? Lasers Surg Med. 1987;7(4):376-82.

18) Kunachak S, Sobhon P.The Potential Alveolar Hazard of Carbon Dioxide Laser-Induced Smoke. J Med Assoc Thai. 1998 Apr;81(4):278-82.

19) Kokosa JM, Eugene J, Chemical Composition of Laser-Tissue Interaction Smoke Plume, J Laser Appl, 1989;000:59-63

20) Smith JP, et al, Evaluation of a Smoke Evacuator Used for Laser Surgery, Lasers Surg Med, 1989;9:276-281

21) Lopez, NA. How Do I Use the Laser and Keep My Hands Sterile? AccuVet® LaserPoints. 2000 Aug (2)

22) Wykes PM. Brachycephalic Airway Obstructive Syndrome. Probl Vet Med. 1991 Jun;3(2):188-97.

23) Clark GN, Sinibaldi KR, Use of a carbon dioxide laser for treatment of elongated soft palate in dogs. JAVMA. 1994 Jun 1;204(11):1779-81

24) Walsh JT, et al, Pulsed CO_2 Laser Tissue Ablation: Effect of Tissue Type and Pulse duration on Thermal Damage, Lasers Surg Med 1988;8(2):108-18.

25) Venugopalan V, et al, The effect of CO_2 Laser Pulse Repetition Rate of Tissue Ablation Rate and Thermal Damage, IEEE Trans Biomed Eng 1991 Oct;38(10)1049-52

26) Kirn DS, et al, Skin Contraction with a Pulsed CO_2 and Erbium:YAG Laser, Plast Reconstr Surg 1999 Dec;104(7)2255-60

Footnotes

a) AccuVet® Novapulse® CO_2 Laser. ESC/Sharplan. Bothell, WA

b) Detergezyme®, Metrex Research Corp., Parker, CO

c) Dust-Off®, Falcon Safety Products, Intl., Branchburg, NJ

d) Nexaband® S/C, Veterinary Products Laboratories, Phoenix, AZ

Using CO_2 Lasers to Perform Elective Surgical Procedures

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Today's demanding pet owner desires the finest care available through advancements in medical technology and surgery. The recent availability of more affordable, compact CO_2 surgical laser technology for use in veterinary surgery has provided practitioners with a new and exciting way to satisfy their clients' desire for high quality medicine. Today's veterinary CO_2 laser equipment is relatively easy to master as a surgical tool, and the reduction in post-operative bleeding, pain, and swelling[1-3] resulting from proper use in the hands of a trained veterinarian engenders a lasting impression of compassionate care in the pet-owner's mind.

The purpose of this paper is to introduce the reader to some suggested techniques for elective surgical procedures that all veterinary practitioners can perform everyday using a CO_2 laser. In general, the techniques vary little from standard protocol; with the exception that a scalpel-like handpiece delivering focused laser energy replaces the traditional steel scalpel.[1-3]

This author uses a 20W CO_2 laser.[a] This surgical device produces an intense, monochromatic, coherent, collimated beam of infrared light at a wavelength of 10.6µm. The laser beam is delivered to the surgical site *via* a hollow flexible wave-guide that terminates at a scalpel-like handpiece. The laser energy exits the handpiece through exchangeable tube shaped tips (various diameters ranging from 0.3mm to 1.4mm). The various suggested focusing tip sizes will be pointed out, so that proper power density[2] can be delivered. The power settings that are given in this paper are suggested guidelines for use and **not** absolute values for the given procedure. The mode of energy delivery is described as either CW (Continuous Wave), meaning that the laser is delivering a continuous stream of laser power, or as CW SP (Continuous Wave, Superpulse), meaning a stream of laser energy characterized by a pattern of sharp and rapid peaks and rests of laser energy intensity. Superpulse provides an optimal balance of vaporization (peaks) and thermal relaxation (rests) of target tissues thereby minimizing tissue charring and collateral thermal necrosis.[4,5] If Superpulse is not available, then a continuous wave, pulsed wave (CW PW) can be delivered, which also provides for a period of rest and cooling between pulses, but is not as effective in reducing heat and char formation.

The CO_2 laser wavelength has a high absorption coefficient in water that makes it ideal for soft tissue incisions and ablations when used properly, because it results in the least amount of collateral tissue damage and post-operative coagulative necrosis due to heat. Because this particular wavelength does not pass through, but strongly reacts with cellular water, it vaporizes tissue layer-by-layer minimizing energy transmission to underlying cellular structures.

To the contrary, wavelengths generated by diode or Nd:YAG (between 810nm-1064nm) lasers are poorly absorbed by cellular water, and react more with the pigments present in tissues. Due to the fact that cells are comprised more of water than pigments, these wavelengths more readily transmit through and scatter deeper within the tissue. Coagulative necrosis and deeper, more prolonged thermal conduction characterize the effects to tissue following irradiation at these wavelengths. The ability to deliver these wavelengths *via* optical quartz fiber makes them well suited to flexible endoscopic applications, and their high absorption in pigmented tissue makes them more efficient coagulators of larger blood vessels.[1] However, the CO_2 laser's more predictable, controllable, soft tissue effect, coagulative properties, broader range of applications, and inherently

shorter learning curve make it the ideal laser for the general veterinary practitioner, and is the reason this author prefers it over other wavelengths.

Cutaneous Mass Removal

Small benign papillomas can be ablated using a 0.8mm tip, using 6-8W CW. In favorable anatomic areas of cooperative patients, a local anesthetic alone can be used to remove one or two small lesions. Larger masses require general anesthesia before removal is attempted.

Lasers provide the veterinary surgeon with unprecedented speed, accuracy and cleanliness when multiple cutaneous mass removals are desired. With cutaneous masses of less than 0.5 cm in diameter, the wound resulting from growth removal can be left open without the need for sutures. This is accomplished by "caramelizing", or mildly charring, the tissue using a defocused beam of low power intensity (*e.g.* 0.8mm tip, 4-6W CW). The resultant caramelized tissue acts as a bandage. If a scanner is available, many of these tumors can be rapidly ablated with 6-10W of power in CW SP mode. Scanner technology enhances laser ablation by generating a scanned laser pattern of up to 3mm in diameter versus the standard spot size of 0.3 - 1.4mm, making quicker work of ablating larger surface areas. An antibiotic ointment applied twice daily for one week will keep the healing tissues moist and encourage rapid re-epithelialization.

Any wound created with a CO_2 laser that is greater than 0.5 cm in diameter should be wiped free of char with moistened gauze, and then sutured closed. To excise these larger masses, a 0.4mm tip using 6-8W CW SP is used to make an elliptical incision around the mass. Appropriately sized tissue margins are excised as well. Once the edges are defined, a 0.8mm tip using 6-12W CW can be used to more rapidly excise and ablate tissue to be removed by cleanly dissecting away tissue. When submitting the specimen for histopathology, larger tissue margins should be submitted since some shrinkage due to desiccation will occur.

The main advantage of using a CO_2 laser in these procedures is diminished post-operative surgical pain. Additionally, the hemostatic action of the laser enhances intra-operative visualization and increases surgical speed by decreasing bleeding and the time usually spent during the procedure keeping the site free of blood. Furthermore, in patients where anesthesia is a concern due to age or other factors, less invasive laser surgery under local anesthetic provides a safer surgical alternative for the patient.

Canine Scrotal Open Castration

A routine neuter in any patient is improved using a CO_2 laser by diminishing the pain, bleeding and swelling usually experienced post-operatively. The fur surrounding the entire scrotum is clipped and prepped atraumatically. Excessive or close clipping will initiate a cycle of irritation and self-mutilation in the patient, and should be avoided. The scrotum and surrounding exposed area is surgically prepped with a chlorhexidine scrub solution. One testicle is advanced to the anterior scrotal median raphe and used to create tension on the thinned scrotum where the incision is planned. A midline anterior scrotal incision is created with a 0.4mm tip using 6-8W CW SP. As the testicle tenses the scrotal skin, the incision is continued until the testicle begins to exteriorize. The testicle is manually extracted through an incision in the scrotal subcutaneous tissue. The entire testicle and contents of the tunic are exteriorized and the connective tissue and any fat is wiped out and away with sterile gauze. The vaginal tunic is opened to clearly visualize the pampiniform plexus, vas deferens and cremaster muscle. The cremaster muscle is ligated proximally with absorbable suture material and transected, or in dogs with light musculature the cremaster can be cleanly divided using the laser beam. The pampiniform plexus and vas deferens are ligated and transected proximally as well.

The open castration of the second testicle is then performed through the same scrotal incision. A second incision is made in the scrotal subcutaneous tissue using the same surgical laser settings. It, likewise, is then surgically removed by open technique. Before final closure, the scrotum is examined for any bleeding or remaining fatty connective tissue. Any char is wiped from the incision edges. A small bead of tissue adhesive[b] is placed over the incision to complete the procedure.

When performing laser-assisted castration on a mature dog, use the same technique as in a young dog neuter. A preputial incision to advance the testicles through can be used, but this would require placement of a non-irritating subcutaneous absorbable suture to keep the incision edges opposed during healing. A caudal scrotal incision should be avoided because the incision may become traumatized when the patient assumes a normal sitting posture. Proper use of the laser will produce a clean and dry incision that heals rapidly. However, some dogs insist on grooming, which must be prevented to allow normal healing. An Elizabethan collar may be worn to prevent self-mutilation

Feline Castration

The patient is anesthetized and the scrotum is surgically prepped. A single pass is made on the scrotal median raphe using a 0.3mm tip and 3-4W CW SP, or a 0.4mm tip and 6-8W CW SP. Both testicles can be manually extracted, one at a time through the same scrotal incision. Two separate incisions may be required in the scrotal subcutaneous tissues to completely exteriorize each testicle and supporting structures. Tension is applied to the testicular vessels by gentle traction of the testicle. Sterile moistened gauze is placed under the tensed scrotal cord to protect the surgeon. A single pass is made through the tunic to divide the testicular artery and vein as well as the vas deferens and cremaster muscle. The proximal remnant of the transected structures will retract through the inguinal ring into the abdomen. . The vessels and nerves will be sealed immediately causing hemostasis and diminished or absent pain.

Occasionally a mature cat that has well-developed gonadal tissue and broad vasculature will bleed from the vaporizing pass made with a laser if the surgeon does not consider the diameter of the vessel. This occurs because the CO_2 laser will seal only vessels that are smaller than the diameter of the laser tip being used for the incision. To vaporize the spermatic cord the surgeon may wish to use a 0.8mm tip, or use a ligature to be safest.

Any char remaining on the skin incision is wiped away with sterile moistened gauze. The edges are left in opposition and neither sutures nor surgical adhesive is required. The predominant benefit of using a laser in this technique is patient comfort and diminished bleeding. The surgeon should choose technique carefully in a mature cat, and consider ligating the testicular vessels if they are large. Either way, if the scrotal incision is made using a laser, the patient will be more comfortable following the procedure.

Ovariohysterectomy

A routine spay in any patient is improved using a laser because it reduces the post-operative pain, bleeding and swelling experienced by the patient. Make a midline skin incision using 0.4mm tip, 6-8W CW SP. The surgeon may wish to use 7-9W SP pulsed 30Hz, 25msec pulse width (75% power) to further reduce char formation, thermal conduction through tissue and creation of laser surgical plume. After wiping away any char, the subcutaneous tissues are dissected away to clearly expose the linea alba. The abdominal muscles and peritoneum are tented up with forceps and a single cranial stab incision is created with a scalpel blade. A grooved director is introduced into the abdominal incision to serve as a backstop for the CO_2 laser-assisted continuation of the incision. All internal structures must be protected from stray laser beams. Inadvertent lasing of the spleen, bladder, or mesenteric blood vessels could prove disastrous and should be avoided. The abdomen is incised using 0.4mm tip 6-8W CW over the backstop, which is lifted upward to tense the targeted muscles that are vaporized. Any char is wiped away from the clean and dry muscle edges. The procedure is then continued as previously performed by the surgeon's routine technique. If desired, a surgeon may wish to vaporize tissues of the ovarian pedicle and/or cervical stump following ligation, although it is not recommended due to the larger diameter of the target vessels.

Following routine closure of the abdominal muscles and subcutis using absorbable suture material, the skin can be opposed with sutures, however, no exterior sutures may be necessary if tissue adhesive[b] is used as a protective bandage. This technique has two benefits: it gives the client an added convenience as there is no need for a second visit to remove sutures, and the appearance of the surgical site is more pleasing. Because there is no swelling of the incision edges, incisions made with a surgical laser oppose cosmetically.

Feline Onychectomy

When elective feline declaw must be done for the owner's benefit, the CO_2 laser declaw procedure is the most humane technique in that it significantly reduces post-operative bleeding, pain, and swelling, providing a quicker return to normal activities.[6,7] After careful client counseling the cat is induced with general anesthetic agents that include an opioid.[8,9] The success of this procedure is largely dependent on the experience and skill level of the laser surgeon. My preference is to trim the pointed toenails before pre-surgical cleansing. All toes are cleansed with a dilute chlorhexidine solution then rinsed with sterile saline. A tourniquet is not required and may be contraindicated as it causes anoxia and reperfusion injury to the tissues of the antebrachium and digits. Oschner (rat toothed) hemostats are used to clamp P3 just proximal to the ungual crest. To dissect P3 cleanly from P2, this technique is as follows: a single laser pass with rapid hand speed using a 0.4mm tip 6-8W CW is performed on the lateral surface of the ungual crest from dorsal to ventral. A similar single laser pass immediately follows on the medial surface of the ungual crest so that the two incisions meet at its dorsal most point. If necessary, a small burst of laser energy can be used to connect the two incisions dorsally. Be cautious not to overuse the laser during this procedure since the resulting excessive char formation will impair healing and cause irritation that defeats the benefits of laser declaw procedure. The thin skin covering P3 can now be advanced proximally (pushed back) to better expose the distal interphalangeal joint.

To continue the procedure, the Oschner forcep holding P3 is rotated to flex the joint. With the laser tip pointed away from P2 to avoid inadvertent charring of the remaining phalanx, the laser makes a single pass over the dorsal elastic ligament and extensor tendon and transects it using 0.4mm tip 6-8W CW. It is important to continually flex P3 while applying laser energy to cause separation of P3 from P2 as previously illustrated. Some surgeons have found it is easier to perform this technique with the angled handpiece used so frequently in the oral cavity. It is also advisable to make clean passes with the laser and avoid going back and forth over the same area. Excessive lasing of charred tissue will cause undesirable heating of tissues and result in post-operative pain and swelling. While continuing to laser-cut the lateral collateral, then medial collateral distal interphalangeal ligaments the surgeon should continually flex the joint to ensure that enough tension is applied to separate P3 from P2. At this point the distal interphalangeal joint should be completely exposed. In a final aggressive flexing maneuver, the laser is directed distally toward the backside of P3 to vaporize the deep digital flexor tendon attachment from the flexor process of P3. The laser beam can now be directed onto P3, which is being removed with tension, and should help in completely avoiding P2. The digital pad should also remain completely intact due to the aggressive flexing action during the amputation. A hole that is clean and dry will measure 2-3 mm in diameter if this procedure is followed correctly. Defocusing the beam and applying a short burst of laser energy onto any culprit vessel can control any small bleeding.

It is essential that all of P3 be removed and that P2 not be charred during the laser declaw procedure. Any char on the skin incision is wiped away with sterile moistened gauze, and then dried with sterile gauze. Lastly, a small amount of tissue adhesive[b] is applied to the skin edges only, and then sealed closed. Avoid using excessive amounts of this bonding agent, as this can lead to postoperative inflammation, granulation tissue, and discomfort. It is preferable to shape the toes to a natural and pleasing appearance. Following ten seconds of gentle pressure, the surgical site is examined for any problems before moving on to the next toe. An injectable broad-spectrum antibiotic may be administered, and the patient is sent home with shredded newspaper[c] to use as litterbox liner. Bandaging of the feet is not required.[10] The patient should recover normally and walk comfortably. It is wise to allow the cat several days to adjust to its "new shoes", and although not painful, the cat will need to learn to walk and support weight slightly differently.

Brachycephalic Breed Upper Airway Syndrome Correction

Old English Bulldogs commonly have many features of brachycephalic upper airway syndrome.[11] Other popular breeds of dogs such as the French Bulldog, Lhasa Apso, Pekingese, Pug, and Shi-Tzu are similarly grouped. Stenotic nares and tracheal hypoplasia may also be present in many of these dogs having this congenital complex. These dogs generally tire easily during minor exercise and have a pronounced inspiratory stridor. The negative pressures exerted upon the oropharynx during inspiration can cause these tissues to swell and further occlude the normal flow of air to the lungs. Chronically affected animals may develop secondary acquired airway restriction from everted laryngeal saccules, and terminally, laryngeal collapse. To prevent a future respiratory

crisis it is prudent to correct these anatomic problems in symptomatic animals when they have reached satisfactory size and age for a safe anesthetic episode.

Elongated Soft Palate Correction

This procedure requires a backstop on the laser delivery handpiece, or wet gauze placed behind the soft palate to prevent inadvertent lasing of tissues in the back of the oropharynx.[12] In sternal recumbancy the pre-medicated patient is pre-oxygenated prior to anesthetic induction with either propofol or pentothal. If ultra-short acting injectable anesthesia is not available, then inhalant anesthetics are satisfactory, however, the surgeon will need to repeatedly intubate and re-extubate to complete the procedure. A single stay suture is placed in the distal midline of the elongated soft palate. With the tongue in a relaxed position, the surgeon makes a visual estimation of the amount of tissue to remove, or can make a low power pass on the elongated palate to mark the area to be excised. The goal is for the soft palate to conform to the shape of the epiglottis and just barely make contact with it. Using a laser assisted uvular palatoplasty kit,[d] 10-15W laser power is applied to the redundant tissue using the stay suture for traction. Begin at the lateral margin of the palate and vaporize tissue in a path heading to the contralateral side. It is extremely important to keep the laser beam perpendicular to the target tissue. Tangential beams are less efficient at vaporization and will cause thermal necrosis, post-operative discomfort, and potentially disastrous bleeding into vital supporting tissues (*e.g.* the palatine artery). After the desired tissue is excised, visual inspection confirms the soft palate to rest just above the epiglottis. Sutures are neither required nor recommended.

The dog is then intubated to enhance oxygenation during recovery from the anesthetic. Perioperative broad-spectrum antibiotics and glucocorticoids are recommended. The patient should be able to eat and drink normally following complete anesthetic recovery. There should be a noticeable decrease in both respiratory effort and inspiratory stridor following the procedure.

Correction of Stenotic Nares

Relieving stenotic nares with a CO_2 laser is a clean, easy and highly effective procedure for allowing greater airflow through the nostrils and reducing oronasopharyngeal negative pressure. Usually bilateral and congenital, stenotic nares frequently occur in brachycephalic breeds. Affected dogs and cats will produce a loud noise on inspiration, although their owners do not usually recognize this. Many affected dogs (Lhasa Apso, Old English Bulldog, Pug) will also snore at night or have respiratory stridor due to an elongated soft palate. Many cats (Himalayan, Persian) will also have multiple changes around the nose and eyes such as epiphora, conjunctivitis, face-fold pyoderma, and entropion. The reduced airflow through the nostrils cause the affected patient to require a greater inspiratory effort, which in turn creates a greater negative pressure within the oropharynx, thus exacerbating brachycephalic upper airway syndrome.

No preparation of the surgical area is required with the patient under general anesthesia. The surgeon envisions the final nasal opening as a circular conduit for airflow. To do this, a conical-wedge shaped portion of the stenotic nare is removed using a 0.3mm tip 4-5W CW SP or 0.4mm tip 5-7W CW SP. Continue the incision of the nostril from the philtrum to the alar fold to make the circular shape complete. To excise the redundant tissue that causes the stenosis, apply tension as the laser dissects deep into the nostril space. The anatomical continuation of the nasal passage courses caudoventromedially. Any remaining tissue in this area may also be excised until the surgeon is satisfied sufficient tissue has been removed to ameliorate the stenosis. There should be no bleeding in this highly vascular area, however, if minor hemorrhage occurs the surgeon should apply a defocused low power laser beam for hemostasis. Sutures are neither required nor indicated unless the surgeon desires to perform the standard vertical wedge resection or the horizontal wedge resection instead. A pleasing cosmesis will result, and the patient will breathe easier upon anesthetic recovery. This in turn reduces cardiorespiratory stress and will prolong the life of the patient, especially if performed at a young age. Antibiotics are usually not necessary, but the owners should be instructed to keep the area clean and moist for several days to prevent buildup of mucus or scabs.

Tonsillectomy

The pre-medicated patient is pre-oxygenated prior to anesthetic. An endotracheal tube is lubricated and placed comfortably. The endotracheal tube is then packed off with moistened gauze to protect it against perforation by the laser beam, which can result in a fatal flame hazard. Forceps are used to gently grasp the tonsil and slightly elevate it from its crypt. Both tonsils are excised using 0.8mm tip 10-15W CW (LAUP extension without the backstop). There should be no bleeding, and the airway should be much larger following removal of both tonsils. In giant breeds, or when tonsils are grossly enlarged and inflamed, some minor bleeding may occur. Direct pressure for a few minutes, cautery, a ligature, or a defocused laser beam may then be used for hemostasis.

Everted Laryngeal Saccule Ablation

The saccules are usually very edematous clear to pale white bulges of tissue cranial to the vocal folds. To access this area, the patient must be anesthetized with propofol following pre-oxygenation. This is a most difficult area to reach. Smoke evacuation will be tricky, and the surgeon must coordinate every move with the technician operating the plume evacuator. Fortunately, this portion of the procedure is rapid and not much smoke will be created unless the membranes are enormous. Depress the epiglottis with Allis tissue forceps and then carefully introduce the tips into the larynx. Then dilate the larynx by opening the jaws to visualize the saccules laterally. It is more effective to permanently vaporize the saccules. It is not practical to try excising them since the working area is very deep and tight. Ablating the saccules with a curved 120mm long tip, 0.8mm spot size is much better than trying to excise them with this size tip. Ablate the saccules with 4-6W CW power during an expiration to avoid allowing the patient to inhale their own smoke. Carefully wipe away any char with a moist, cotton tipped applicator.

Conclusion

Veterinary CO_2 laser surgery equipment provides a general practitioner with the instrumentation for dramatically improved post-operative outcomes. This equipment is now more affordable, easier to use, and is in great demand by clients. The techniques described in this paper are the author's preference for elective procedures. The settings that were discussed are only examples of the typical power settings required to accomplish the surgical task. To have excellent post-surgical outcomes it is important to posses a mastery of anatomy, surgical experience and advanced training. The application of a surgical laser does not guarantee success, but its judicious use will certainly improve surgical results.

After considering the different types of laser delivery systems available to veterinary medical care providers, it was clear that the CO_2 laser would be the most sensible choice for small animal practice. The decision against using a diode laser or an Nd:YAG laser was made due to their undesirable tissue interactions and cost. Surgeons should promote more compassionate post-surgical care by diminishing the pain, swelling and bleeding associated with most procedures. Using a CO_2 laser is the ideal surgical tool to expand a veterinary practice's capabilities, and it is easy to acquire the proper skills to master its use. Incorporating a laser into a practice provides additional opportunities for surgical services that can be offered to clients for the benefit of their pets. The general public definitely perceives this as the highest level of quality care and they demand it.

Every day, more veterinarians are beginning to manage challenging surgical cases in their own practices without the need to outsource the labor to provide the service. Practitioners using this current technology are enjoying the excitement of the near effortless solutions that a CO_2 laser offers to problems that were not as easily managed before. Additionally, many common procedures when performed with a CO_2 laser result in improvements in surgical outcomes unequalled by any other single surgical technology available to the veterinary profession. The result is happier pets and clients, improved job satisfaction to all employees of a veterinary practice, and increased practice revenue. Laser surgery is certainly here to stay, and prudent practitioners will avail themselves of its benefits.

References

1) Lucroy MD & Bartels KE, *Surgical Lasers, Textbook of Small Animal Surgery*. Slatter, 3rd Edition, 2001.
2) Lopez NA. The Basics of Soft Tissue Laser Surgery. Vet Med. 2002 April 97(4):292 - 301
3) Durante EJ, Kriek NP, Clinical and Histological Comparison of Tissue Damage and Healing Following Incisions with the CO_2 Laser and Stainless Steel Surgical Blade in Dogs, JSAVA, 1993 Sep;64(3):116-20
4) Venugopalan V, et al, The Effect of CO_2 Laser Pulse Repetition Rate on Tissue Ablation Rate and Thermal Damage, IEEE Trans Biomed Eng 1991 Oct;38(10)1049-52
5) Walsh JT, et al, Pulsed CO_2 Laser Tissue Ablation: Effect of Tissue Type and Pulse duration on Thermal Damage, Lasers Surg Med 1988;8(2):108-18.
6) Cambridge AJ, et al, Subjective and Objective Measurements of Postoperative Pain in Cats, JAVMA 2000 Sep1;217(5):685-90
7) Mison MB, et al., Use of CO_2 Laser in Feline Onychectomy: A Prospective, Randomized, Blinded Clinical Trial. Abstract, Resident's Forum, ACVS Symposium Sept. 2000.
8) Franks JN, et al, Evaluation of Transdermal Fentanyl Patches for Analgesia in Cats Undergoing Onychectomy, JAVMA 2000 Oct 1;217(7):1013-20
9) Carroll GL, et al, Evaluation of Analgesia Provided by Postoperative Administration of Butorphanol to Cats undergoing Onychectomy, JAVMA 1998 Jul 15;213(2):246-50
10) Anderson DM, White RA, Ischemic Bandage Injuries: A Case Series and Review of the Literature, Vet Surg 2000 Nov-Dec;29(6):488-98
11) Wykes PM. Brachycephalic Airway Obstructive Syndrome. Probl Vet Med. 1991 Jun;3(2):188-97.
12) Clark GN, Sinibaldi KR, Use of a Carbon Dioxide Laser for Treatment of Elongated Soft Palate in Dogs. JAVMA. 1994 Jun 1;204(11):1779-81

Footnotes

a) Accuvet,® Novapulse®, ESC / Sharplan, Bothell, WA
b) Nexaband® S/C, Veterinary Products Laboratories, Phoenix, AZ
c) Yesterday's News®, Cat Litter, Canbrands International Ltd., Moncton, New Brunswick, Canada
d) LAUP kit, LXT120ST 0.8mm tip and backstop, ESC / Sharplan, Bothell, WA

Feline Acute Renal Failure

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Azotemia refers to a state where there is an increase in non-protein associated nitrogen in the blood. Both increases in urea and creatinine are commonly assessed when determining if a patient is azotemic. This is different then uremia which refers to a clinical syndrome caused by renal failure. Azotemia does not have to be a primary kidney disorder. It may be:

Pre-renal, caused by
- Dehydration
- ↓ perfusion
- Renal
- Post renal, caused by a urinary obstruction or à ruptured renal pelvis, ureter, bladder or urethra.

Acute renal failure (ARF) is characterized by a rapid onset of renal insufficiency, reduction in glomerular filtration rate (GFR) and renal plasma flow. It results in an acute uremic syndrome with potential severe hematological and systemic effects. In some patients this syndrome is accompanied by insufficient quantities of urination (oliguria or anuria), although this is not pathognomonic, and likely true for less than half of the patients seen. The excretory failure is identified by rapid (hours to days) increases in BUN, serum creatinine and phosphate, and variable hyperkalemia and metabolic acidosis. Urine concentration capability is impaired. Acute renal failure is a tenuously reversible state, which must be treated aggressively. Failure to initiate therapy may result in irreversible parenchymal damage and death.

Stages of acute renal failure:

Three stages are classically described in ischemic renal failure although not always clinically evident.

1. The initial phase lasts from the onset of the ischemic insult until the development of azotemia and oliguria if it develops. This is the stage where the kidney is damaged and is characterized by acute cell injury leading to decreasing urine concentration capability, sloughing of casts and cellular debris and finally azotemia. If aggressive therapy is implemented in this stage there may be a complete and rapid return to normal function.

2. The maintenance phase is characterized by increasing azotemia loss of concentrating ability and in some cases oliguria, along with increasing percentages of cell death. Intervention in this phase may result in complete resolution and return to normal function although the improvement will take longer than in the previous phase. Without treatment this phase will end in death if the damage is extensive enough.

3. The recovery phase includes recovery of the vascular blood supply but still with tubular dysfunction resulting in diuresis and polyuria. Our therapeutic goal is to convert our patients in the maintenance phase to the recovery stage and allow time for renal healing. This phase can last many days. The degree of return of function depends on the severity of the tubular injury sustained in the prior two phases.

The pathophysiology of acute renal failure:

In general acute renal injury results from either toxic exposure or ischemic insult. Both may result in primary vascular (hemodynamic) or tubular renal damage. Several mechanisms are thought to be involved in each case of renal insult. There are 6 major sites of impairment:

- Afferent arteriole vasoconstriction disrupts glomerular flow. This frequently happens as a result of decreased systemic blood pressure, hypovolemia and dehydration
- Mesangial cell contraction causes a reduction in glomerular filtration surface area. This is commonly a result of ischemia, humural agents or toxins.
- Vasodilation of the efferent arteriole causes decreased glomerular capillary pressure and GFR. This is most commonly a result of pharmacological blocking of the production angiotensin II with an ACE inhibitor.
- Reduced tubular reabsorption of NaCl causes excessive Na delivery to the distal tubule (macula densa) and causing tubular glomerular feedback and increased afferent vasoconstriction.
- Damage to the tubular epithelial cells disrupts the integrity of the tubular lining and may result in tubular backleak, reducing excretory capacity and the effective GFR.
- Tubular damage can also cause tubular flow obstruction by sloughing cellular casts and debris, reducing GFR by reducing glomerular filtration gradient.

The intracellular results of ischemia are ATP depletion, increased intracellular calcium concentrations, increased free radical production causing cellular dysfunction and if severe cell death.

Recognizing patients at risk for developing acute renal failure is a crucial role of the clinician. Many cases of acute renal failure are preventable and occur in the hospital setting or in a patient receiving veterinary care.

These are just some of the potential risk factors for acute renal failure:

<u>Preexisting diseases</u>

Renal insufficiency
Pancreatitis
Hepatic insufficiency
Diabetes mellitus
Heart disease
Trauma

<u>Clinical conditions</u>

Volume depletion
Electrolyte abnormalities (Na+, Ca2+,K+)
Hypoalbuminemia
Hyper or hypotension
Fever
Sepsis
Anesthesia
Surgery
Radio contrast media
NSAIDs
Nephrotoxic drugs

<u>Intrinsic Renal Disease (partial list)</u>

Infectious
FIP, pyelonephritis, sepsis...
Glomerular disease
SLE
Vascular (thrombotic)
Urinary outflow obstruction
urethral obstruction
ureteral obstruction
Toxic - huge list

Ethylene glycol, cisplatin, amphotericin B
Pigmenturia
Neoplasia
Lymphoma
Adenocarcinoma
Hypercalcemia

Clinical Presentation

The clinical presentation of patients with acute renal failure varies based on the cause, severity, previous therapy and associated diseases predisposing to the renal injury. Consistent and characteristic signs of ARF include the sudden onset and rapidly progressive development of listlessness, depression, anorexia, vomiting and diarrhea. Oliguria (<0.27ml/kg/hr) and anuria may or may not be present, more commonly occurring in ischemic than nephrotoxic disease. Obtaining a thorough history especially of any possible exposure to nephrotoxins or medications is crucial for accurate diagnosis and therapy.

Physical examination commonly demonstrates dehydration (prior excessive fluid therapy makes overhydration a common presentation at referral centers), hypothermia, oral ulceration, "uremic breath", scleral injection, tachycardia or bradycardia, tachypnea, abdominal pain, rarely seizures and enlarged and painful kidneys on abdominal palpation. Melena resulting from GI bleeding is a common finding on rectal palpation.

Because of the abrupt onset of uremia, patients are often of good body condition, good hair coats and normally pink or injected mucous membranes when compared to the general poor condition of chronic renal failure patients on presentation. When the acute disease comes "on top" of a chronic condition the presentation becomes quite confusing.

Laboratory evaluation

The initial data base should include a CBC, biochemical profile (including HCO3- or TCO2 or central venous blood gas), urinalysis (if urine is obtainable) and urine culture.

CBC

The hemogram is generally non-specific. A non-regenerative anemia would be more suggestive of underlying chronic renal disease, or other chronic disease, although can occur with acute GI bleeding as well. Hypovolemia and dehydration may induce hemoconcentration and increased serum proteins, potentially masking pre-existing anemias. Therefore, the PCV should be reassessed once rehydration has been achieved.

Chemistry panel

Azotemia is the biochemical hallmark of renal failure. It is common for the azotemia to be marked in cases of acute renal failure (BUN above 100mg/dl and creatinine above 6mg/dl), although it is not possible to definitively distinguish pre, renal, and post renal azotemia based on the degree of azotemia.

Serum phosphate is regulated primarily via urinary excretion and tubular reabsorption and is heavily dependent on glomerular filtration. In acute renal changes increases in serum phosphate concentrations are often times marked, more than with the same degree of azotemia in chronic disease. This is a valuable tool in our battle to differentiate acute from chronic renal disease.

Additional electrolyte abnormalities include hyperkalemia. This can be severe in oliguric or anuric renal failure and is exacerbated by concurrent acidosis. This is a life threatening condition and necessitates rapid therapeutic measures when present. Hypocalcemia is also a common finding in acute renal failure, it too can be life threatening, although is usually not. The hypocalcemia results mainly from the acute rise in serum phosphorus without time for secondary hyperparathyroidism to develop. Treatment may be warranted in severe cases or if clinical signs of hypocalcemia are evident. The acidosis typically seen with acute renal failure is primary metabolic in origin and results mainly from decreased renal bicarbonate production and reabsorption, vomiting, diarrhea and a respiratory component may induce a mixed acid-base disturbance that requires careful blood gas analysis.

Increases in additional values in the chemistry panel may be increased as well, if they are dependent on GFR. Pancreatic enzymes amylase, lipase and TLI (trypsin like immunoreactivity) are all typically elevated. Although ARF and pancreatitis can occur concurrently it is important to remember this fact and not to suspect that every case of renal insufficiency also has biochemical evidence of pancreatitis.

Urinalysis

Obtaining urine for analysis prior to initiation of fluid therapy is extremely valuable in differentiating acute renal failure from pre-renal azotemia. If there is no urine at that time it should be obtained as soon as possible after initiation of therapy. Concentrated urine (>1.025) is consistent with pre-renal disease, whereas azotemia in the presence of non concentrated urine is suggestive of at least some degree of renal insufficiency, drug therapy (diuretics) or concurrent disease (hyperadrenocortism, diabetes insipidus, etc....). Proteinuria and glucosuria (indicative of tubular damage) are commonly seen in ARF. The sediment is frequently "active", containing RBCs, WBCs, epithelial cells, and casts - reflecting active tubular damage. An abundance of oxalate crystals, bacteriuria may predict the cause of the renal disease.

Imaging

On routine radiographs kidney size can be assessed although masses and cysts may not be differentiated from renal parenchyma. Ultrasound evaluation is superior for the evaluation of renal parenchyma and is indicated in acute renal disease. Kidneys are generally normal to enlarged and may be hyperechoic in certain disease states (ethylene glycol toxicity for example). Uroliths and signs of urinary obstruction may be seen in radiographic or ultrasonographic evaluations. Ultrasound also is necessary for percutaneous renal aspirates or biopsies in most dogs but is not usually necessary in cats). Additional studies such as contrast radiography, computer tomography and nuclear scintigraphy are useful in some cases.

Renal aspirate & biopsy

Indicated in many cases of acute renal disease. The information obtained from these procedures may be crucial in understanding the etiology of the renal disease (neoplasia, inflammation, infection) as well as the duration of the disease and the prognosis. Specific therapy in the case of neoplasia for instance can only be given if a histological or cytological diagnosis is obtained, and is only effective if performed early in the disease process.

Additional diagnostics

Additional tests are available and valuable in achieving a diagnosis depending on the appropriate history, clinical signs, season and geographic location.
In cases where ethylene glycol toxicity is suspected then measuring the serum osmolality and comparing it to the calculated osmolality.
Calc osmolality = 2(Na +K) + glu/18 + BUN/2.8 If the measured serum osmolality is greater than the calculated then there is a non-calculated solute contributing, possible ethylene glycol and it's metabolites. Ethylene glycol and metabolites can also be measured in blood or urine to achieve a definitive diagnosis.

Consequences of ARF

Fluid balance

The clinical presentation of dogs and cats in acute renal failure can be complicated and require intense monitoring and a complex therapeutic regime. Problems likely to occur should be anticipated and prevented. This type of strategy will carry a much higher success rate than treating the problems as they are identified. Most cases in acute renal failure will present initially in a state of hypovolemia and dehydration, usually contributed to by anorexia, vomiting and diarrhea. Rapidly worsening azotemia will be a consistent finding in serial blood evaluations. Inappropriate and overzealous fluid therapy usually in the face of oliguria/anuria will produce overhydration and hypervolemia. It is not uncommon to receive the cases in this state in a secondary or tertiary referral

center. At this time they may have peripheral or pulmonary edema, ascites and or pleural effusion. This overhydrated state will potentiate systemic hypertension which will likely be present. In cases with underlying cardiac insufficiency fluid therapy may cause "full blown" congestive heart failure.

Oliguria (<0.3ml/kg/hr) & Anuria

Oliguria or anuria are present in up to 50% of ARF cases seen at veterinary centers. Before making this clinical assumption one must try and rule out pre-renal causes of oliguria (as long as a normal animal is dehydrated urine output should be minimal) as well as post renal causes such as obstruction or tear in the urinary system. (DO NOT act upon oliguria until rehydration has been established!)

When oliguria is present it is devastating and life threatening as it does not allow for conservative medical management via diuresis and electrolyte therapies. The solute retention increases the uremia, hyperkalemia results as well as worsening acidosis and death. Inappropriate attempts at diuresis cause life threatening overhydration. If oliguria or anuria are present in a hydrated animal despite fluid therapy in a patient with ARF every attempt must be made to re-establish adequate urine production. If this is not achieved then there is no chance of improvement with continued conservative medical therapy.

Additional consequences of ARF

Neurological complications have been associated with:

- Uremic encephalopathy
- Weakness, lethargy
- Seizures
- Hypertension
- Drugs (H2 blockers?)

- Bleeding disorders
- Platelet dysfunction
- Vasculitis (Lepto.)
- DIC

- GI disorders
- Uremic ulcers
- Vomiting
- Hypergastrinemia

- Electrolyte imbalances
- Hyperkalemia
- Hyperphosphatemia
- Increased Phosphorous X calcium product
- Hypocalcemia
- Severe metabolic acidosis

Prevention, prevention, prevention!!!

Many cases of ARF occur in the clinic or in animals under ongoing veterinary care!

Think about ARF as likely complication in CRF, surgery, trauma, heatstroke, sepsis, hypovolemia, pancreatitis, any severe systemic disease!
Prevent ARF today!!!

Avoid nephrotoxins
Managing hypovolemia
Maintaining blood pressure

Use reno-protective agents - mannitol or Lasix and dopamine in animals predisposed to ARF or doing any surgery on a patient predisposed.
Monitor hydration and urine production during surgery or in any sick animal

Acute Renal Failure - Management

Prior to therapy try to differentiate pre-renal/renal/post-renal azotemia
Obtain blood for PCV, BUN, creatinine (a full CBC and chemistry panel when possible)
Obtain urine for specific gravity and ideally a full urinalysis and culture
Physical examination and good palpation of the urinary bladder to try and rule out post renal disease.

Steps to take -

- Achieve rehydration, adequate colloid oncotic pressure.
- Correct electrolyte imbalances
- Hypocalcemia
- Hyperkalemia
- Hypo/hypernatremia
- Correct metabolic acidosis
- Give a lot of bicarb!

Assess blood pressure and begin to correct severe hypertension. Hypertension is common with acute and chronic renal disease. Systolic pressure should be lowered to no more than 160mmHg. Drugs in common use today include

- Amlodipine
- Hydralazine
- ACE inhibitors (Enalapril, Benazepril)

After Rehydration and mild volume expansion!!! Document urine production
And then - treat oliguria/anuria

Mannitol - Mannitol can be given as an initial bolus and then as a constant rate infusion. It causes an osmotic diuresis, vascular volume expansion and increased GFR. It also has antioxidant properties.
Lasix (furosemide) and dopamine.

When possible - treat underlying disease

- Antidote (ethylene glycol)
- Antibiotics (pyelonephritis, leptospirosis)

Treat GI manifestations

- Metoclopramide
- H2 blocker

If oliguria/anuria cannot be converted to polyuria

Do not overhydrate!!!!!
Consider peritoneal/pleural/hemodialysis

Renal Transplantation

Outcome

Kidney transplantation prolongs the life of cats with chronic renal failure, but by no means are cats expected to have a normal lifespan. Average age of cats at the time of kidney transplant in a recent study was 7 years. Average survival for those cats after the transplant was 21 months. As might be expected, older cats had significantly shorter survival than younger cats. Reasons for death in the first 6 months after surgery included infection, kidney rejection, and neurologic problems. Long-term complications included cancer and diabetes, which may be related to side effects of the immunosuppressive drugs given to transplant recipients.

WHAT ARE THE CONTRAINDICATIONS TO KIDNEY TRANSPLANTATION?

Certain pre-existing problems can negatively affect outcome of kidney transplantation. These problems include:

- Extreme age
- Severe weight loss
- Heart disease
  - Unregulated hyperthyroidism
- Feline leukemia or FIV
  - Toxoplasmosis
- Urinary tract infection
  - Urinary calculi
  - Systemic disease
  - Inflammatory bowel disease
  - Neoplasia
  - Fractiousness

Table 1. Diagnostic tests prior to referral	
Laboratory tests	Complete blood count
	Serum biochemistry panel
Cardiovascular	Thoracic radiographs
	Electrocardiography
	Echocardiography
	Systolic blood pressure
Infectious	FeLV,FIV
	Toxo IgG, IgM
Urinary	Urinalysis
	Urine culture
	Abdominal ultrasound
	Urine protein:creatinine
Immune	Feline blood typing
	Major, minor blood cross match
Endocrine	Thyroid evaluation