### Friday September 8

**PLENARY**

- Species Spanning Medicine: Veterinary Responses to Animal Cruelty, Abuse and Neglect  
  Phil Arkow
- What Canadian Veterinarians Need to Know and Explain about AMR: A Take-Home Tool Kit  
  John Prescott

### Saturday September 9

**LARGE ANIMAL**

- Demographics and Health Status of Geriatric Horses  
  Katharina Lohmann
- Selected Health Concerns of the Geriatric Horse  
  Katharina Lohmann
- Outpacing the Resistance Tsunami: AMR in Equine Medicine  
  John Prescott
- Troubleshooting Micronutrient Status in Cow-Calf Herds  
  Cheryl Waldner
- Investigating Poor Reproductive Performance in Beef Cows  
  Cheryl Waldner
- Small Flock Poultry Disease Basics  
  Victoria Bowes
- Keeping the Small Poultry Flock Healthy  
  Victoria Bowes
- Small Flock Poultry Zoonoses and Food Safety  
  Victoria Bowes

**COMPANION ANIMAL**

- Current Therapy for Canine Chronic Bronchitis  
  Stephan Carey
- Feline Chronic Nasal Disease: Pathophysiologic Basis of Diagnosis and Therapy  
  Stephan Carey
- Canine Infectious Respiratory Disease Complex: Host, Pathogen and Environmental Interactions  
  Stephan Carey
- Antimicrobial Stewardship in Companion Animals: Welcome to a Whole New Era  
  John Prescott
- Practical Analgesia and Anesthesia in Exotic Pets  
  James Morrisey
- Common Emergencies in Exotic Pets  
  James Morrisey

**WELLNESS CAFE**

- “Should you lead with your heart or your head?”  
  Jayne Takahashi

### Sunday September 10

**LARGE ANIMAL**

- Establishing Diagnosis in Cattle: Physical Exam  
  Allen Roussel
- Establishing Diagnosis in Cattle: Clinical Laboratory Data  
  Allen Roussel
- Effective Stockmanship as a Dimension of Beef Production management  
  Tom Noffsinger
- BRD Case Definition  
  Tom Noffsinger

**COMPANION ANIMAL**

- ‘Ears looking at you.’ Diagnosis and Treatment of Canine Otitis Externa  
  Charlie Pye
- ‘You are what you eat!’ Diagnosis and Management of Food Allergies in Companion Animals  
  Charlie Pye
- ‘I’ve tried X,Y and Z... why is nothing working?’ Common Reasons for Dermatologic Treatment Failures  
  Charlie Pye
- Local Anesthetics and Local Anesthetic Techniques  
  Cate Creighton
- Pain Physiology and Recognition  
  Cate Creighton
- CRIs during Anesthesia  
  Cate Creighton
Pets can have positive impacts on families, therapeutic interventions, and community well-being. However, there is also a “dark side” of the human-animal bond, when animal abuse serves as a bellwether for potential or co-occurring interpersonal violence as well as adversely affecting the animals’ welfare.

Paralleling the One Health movement linking veterinary and human medicine, the Link model recognizes that animal abuse is closely tied to domestic violence, child maltreatment and elder abuse and is another form of family violence. When animals are abused people are at risk, and when people are abused animals are likewise at risk.

Forms of The Link

The Link is typically manifested in six ways:

1. **Domestic violence:**
   Abusive partners threaten, harm or kill pets or livestock to punish, warn or retaliate against their human victims. This form of coercive control is one of the four greatest risk factors for someone becoming a batterer and one of the three greatest risk factors for lethality in domestic violence crises. Children trapped in these polyvictimizations are at further risk of carrying forward an intergenerational cycle of violence.

2. **Child sexual abuse:**
   Similarly, harm or threats to children’s pets intimidate them into silence or compliance.

3. **Child abuse of animals:**
   Youths’ committing or witnessing acts of animal abuse may lead to desensitization to violence and concurrent or future antisocial behaviors.

4. **Animal hoarding:**
   This multidimensional animal abuse issue frequently necessitates interventions from mental health, public health, social services and law enforcement agencies as well as veterinary and animal care/control officials.

5. **Animal fighting:**
   This serious crime often co-occurs with human trafficking, gambling, narcotics, weapons and other offenses, exposing children to numerous unhealthy and illegal activities.

6. **Animal sexual abuse:** Growing awareness of bestiality and its links to child pornography have prompted legislative efforts to ban the practice.
With animal abuse considered “the tip of the iceberg,” animal care/control investigators and veterinarians are often the first responders to situations where other family and community violence is present. Interagency referrals and reports are encouraged and, increasingly, becoming mandated.

Research illuminating these principles includes studies in Saskatchewan, Alberta and Prince Edward Island reporting that significant numbers of women remain in abusive situations to protect their pets and livestock, and that service gaps and a lack of collaborative partnerships separate domestic violence safe houses and animal shelters.

Meanwhile, Link awareness is changing public expectations of veterinarians and animal welfare agencies. The former are now more likely to be seen as resources in animal cruelty cases; the latter are recognizing that traditional humane campaigns are merely stopgap solutions and that addressing underlying family dysfunction and violence would be more effective.

Six Stages of Veterinary Involvement

In the midst of these new dynamics, veterinarians face confounding clinical, legal, ethical and moral dilemmas to respond to suspected animal abuse. Studies confirm that practitioners will see cruelty cases: though incidence may be infrequent, each case is problematic and raises many concerns. A six-stage process has marked veterinary medicine’s great advances in recent years to resolve these challenges:

1. Awareness and Responsibility
   Recent studies suggest widespread awareness that animal abuse is a legitimate concern and that Non-Accidental Injury must be included in the differential diagnosis. Studies have identified the most common reasons why practitioners report suspicions or choose not to. A large majority of veterinarians support laws mandating them to report suspected animal abuse, especially if immunity from civil and criminal liability is included. However, there is still considerable uncertainty among practitioners as to their jurisdictions’ specific reporting requirements and which investigatory agency receives these reports.

2. Professional Support
   Revised codes of professional conduct, policy statements and veterinary oaths have been promulgated by national veterinary associations in Canada, the U.S., the U.K., and New Zealand. These policies consider it the responsibility of the practitioner to report suspicions to appropriate agencies. Veterinary associations in Scotland and New Zealand have gone even further and emphasize responding to suspected domestic violence. The American Academy of Pediatrics promotes pediatricians’ screening patients for suspected animal abuse, based on empirical data demonstrating increased risks to children in homes where pets are abused. The veterinarian should be as proactive in the response to animal abuse as physicians have been in responding to child maltreatment.

3. Legislative support
   35 U.S. states, and most Canadian provinces, mandate or permit veterinarians to report suspected abuse with immunity from civil and criminal liability. Such laws help practitioners avoid contentious ethical dilemmas over whether to report, fears of violating client confidentiality, and worries about damaging the reputation of the client or the finances, safety and reputation of the practice.

4. Diagnostic tools
   Practitioners accustomed to well-defined clinical diagnoses may be uncomfortable attempting to identify the amorphous presentations of animal cruelty, particularly when personal, professional, community, and legal standards vary widely. Veterinarians need to be reminded that they are medical, not legal, experts, and that the determination of whether a situation may be prosecuted as abuse or neglect can be determined only by the courts. Veterinarians do not have to “know” if a condition is cruelty: their role is to serve as the animals’ first line of defense; to report findings to the appropriate agency for further investigation; to document their findings objectively; and to present them in a court of law if necessary.
Numerous “red flag” indicators, injuries and conditions should arouse an Index of Suspicion suggestive of Non-Accidental Injury. These include:

a. Client’s profile  
b. Client’s behavior  
c. Patient’s medical history  
d. Family dynamics  
e. Nature of patient’s injuries  
f. Signs of animal fighting  
g. Signs of animal sexual abuse

Other conditions are highly suggestive of animal neglect, unsuitable living environments and animal hoarding situations.

Resources now available to assist the veterinarian in making these diagnoses include: an assortment of textbooks; training conferences; the University of Florida’s distance-learning courses in veterinary forensics; and the International Veterinary Forensic Sciences Association.

5. Practice Management Guidances
Protocols have been developed in the U.S., U.K., New Zealand and elsewhere to help practitioners resolve practice management dilemmas regarding potential liability, loss of clientele, security concerns, and confrontational interactions with clients. Preparing the practice for an animal abuse reporting policy involves:

- Establishing, in advance, a reporting protocol for practice owners and staff  
- Personnel safety concerns  
- Treating the animal without compromising potential criminal evidence  
- Documentation and evidence preservation  
- Risk assessment for all parties involved  
- Determining whether the best response is client education, case monitoring, or reporting

A flow diagram of potential responses, and a client risk-assessment questionnaire administered at intake, can facilitate these decisions. Interpersonal communication and listening skills to approach problematic clients are essential.

6. Know Who to Call
Regulations in each province determine which agencies have authority to investigate suspected animal abuse and to lay charges. In almost all cases, the local or provincial SPCA or humane society is involved in the investigation, and any charges are usually laid either by the police or in conjunction with the police. If you suspect that an animal is being abused or neglected in Saskatchewan, call your local SPCA or humane society. In areas where there is none, call Animal Protection Services of Saskatchewan or your local RCMP.

Conclusion
Animal maltreatment is one of the most challenging diagnoses in clinical work, requiring time, experience, emotional energy, sensitivity, tact, and not a small measure of courage. Such cases are invariably problematic and difficult to resolve. A proactive response has the potential to save human lives and reduce animal suffering. Veterinarians are ideally placed as sentinels and can be an essential public health component to break the cycles of violence. In so doing, veterinarians can work within a One Health approach in common concern for the vulnerable, victimized and at-risk. By directly addressing abuse or neglect, veterinarians can help animals and their families, and be part of the solution to the problems of violence in our communities.
WHAT CANADIAN VETERINARIANS NEED TO KNOW AND EXPLAIN ABOUT AMR:
A TAKE-HOME TOOL-KIT

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Major changes are underway in how agricultural use of antibiotics is regulated in food animals in Canada. All such use will become veterinary prescription only, growth promotion use of medically important antibiotics and Own Use Importation will stop, and compounding will be more strictly regulated. Veterinarians will need to understand more about antimicrobial resistance (AMR) so that they can explain to clients what resistance is and why it’s important that these changes have been made, and the national and international context in which they have been made.

Although the international and national focus has been on agricultural use of antibiotics, the “post-antibiotic era” is more likely to emerge in companion animal medicine, and has actually already arrived in the form of the ESKAPE pathogens. There has been considerable blaming of agriculture for the AMR crisis in medicine, but a more balanced view is emerging with the general (but not totally accepted) embrace of a “One Health” approach. This is a “we’re-all-in-this-together (rather than blaming) approach” but this demands that everyone using antimicrobials accepts responsibility for their stewardship. In agriculture, a stewardship approach will be a partnership of owners with veterinarians, although ultimately the legally mandated responsibility for stewardship has shifted to veterinarians.

There is a now an overwhelming amount of material on the AMR crisis and on the response to the crisis. It’s a fast moving field that’s hard to stay up with. This talk will provide veterinarians with a “tool kit” of the tools and resources, including a PowerPoint, successfully to start engaging with the era of antimicrobial stewardship and the changes in attitudes, behaviors and practices that this will require. The intent is to provide access to some of the best freely downloadable resources about the issue, so that veterinarians may both understand the dimensions of the issue and defend successfully some of the regulatory changes that position them to help address the issue in the food animal sector.

What is antimicrobial resistance, how does it develop and spread?

There’s a large YouTube resource on AMR, including full feature documentaries, on the development and spread of antimicrobial resistance. Some good short ones are referenced.

Antibiotic resistant bacteria often have numerous other traits that make them “fit” as pathogens, and the “clonal” nature of successful high-risk AMR pathogens, and their global spread, is increasingly recognized. This spread highlights continuing issues of poor infection control in numerous health settings.

The Antibiotic Apocalypse Explained: https://www.youtube.com/watch?v=xZbcwi7SfZE
What causes antibiotic resistance? https://www.youtube.com/watch?v=znnp-lvj2ek&t=31s

What is the resistance crisis?

AMR is one of the greatest global challenges of our time. Modern medicine is built on, and inconceivable without, the ability to control bacterial infections. Without this ability, numerous medical and surgical procedures now regarded almost routine will become impossible; even infections following minor cuts may become life-threatening.

The AMR resistance crisis is the emergence of difficult-to-treat infections because of the development and spread of AMR. The most difficult, and in some cases impossible to treat, infections are associated with
hospitals (the “ESKAPE pathogens”: *Enterococcus faecium*, methicillin-resistant *Staphylococcus pseudintermedius* (and *S. aureus*), *Klebsiella pneumoniae*, *Acinetobacter baumannii*, *Pseudomonas aeruginosa*, and *Enterobacter* species) but resistance is increasingly a problem in “community acquired” infections. This crisis of resistance in human medicine is occurring across a broad range of pathogens and in a broad range of settings. It is a global problem because of the ready, non-prescription, availability of antibiotics in many countries, the rapid movement of peoples, and for other reasons. This tsunami of resistance has developed when the approval of new systemic antibiotics has declined to virtually nil in the last 30 years. Unless we act now, AMR bacterial infections are predicted to become the biggest single killer of humans by 2050.

Veterinary medicine has an increasing resistance problem across all fields of its activities, with impacts on human health. The emergence of livestock-associated MRSA and of MRSP in companion animals is just one example of the emergence and spread of resistance, with human health significance, but there are many more.


Who’s to blame for the resistance crisis?

The development and spread of resistance has many causes and influences, and is the cumulative effect of widespread use antibiotics for over 60 years for numerous purposes and in numerous settings. It’s been estimated that 50% of antibiotic use in humans is unnecessary and of the other 50% the antibiotic chosen is inappropriate; it seems likely that similar figures would be true for animals, but agriculture also has a special feature of the use of medically-important antibiotics for growth promotion and “subtherapeutic” purposes. We’ve become so used to antibiotics as part of the modern world that we’ve taken them for granted. Most of the “blame” however lies in the ability of many bacteria to survive antibiotics through the ability to adapt and also to spread resistance genes through mobile genetic elements, such as plasmids. Adaptation is what many successful pathogens do naturally. Resistance itself can be infectious. Rather than assign blame (agriculture, medicine) the consensus view is that we have to totally change the way we use antibiotics, and in other ways. A “One Health” approach to AMR is increasingly being adopted.

What is the global response to the resistance crisis?

There has been a massive global response to the AMR crisis. The G8 countries are addressing it politically as an issue on a par with climate change and international terrorism. The World Health Organization has shown considerable leadership in this area, and is a good source for downloadable materials. The mobilization of different national and international groups in medicine, public policy, veterinary medicine, and agriculture has been unprecedented, and is continuing, with considerable media and public attention to the issue. All agree on the scale, complexity and multidimensional nature of AMR, but there is increasing focus on the multiple international, national and local actions, including the coordination and resource allocation, required to address the issue.

What is agriculture’s role in the resistance crisis?

There is overwhelming evidence that antibiotic use in animals selects for AMR in animal bacterial pathogens and commensals; a strong causal relationship has been shown between antimicrobial use (AMU) and AMR in commensal enteric bacteria in food animals. There is also overwhelming evidence that antibiotic use in animals (companion, food) can lead to spread of resistant pathogens or their resistance genes to humans and that this can make these infections more difficult to treat. Examples include resistant *Campylobacter jejuni*, extraintestinal pathogenic *Escherichia coli* (ExPEC), *Salmonella*, methicillin-resistant *S. aureus* or *S. pseudintermedius*, vancomycin-resistant enterococci, and others.

The ways in which these infections, or resistance genes from animal-derived bacteria, can reach people is shown in the Figure. What is unclear, and what has been highly contentious, is the scale of this contribution. It is hard to get an overall sense of this, and new data are still emerging, in part because agriculture has been sometimes been the whipping boy for AMR issues in human medicine and in part because AMU issues have been a way to attack aspects of modern agriculture that some groups don’t like. It is however generally now accepted that most AMR in human pathogens is the result of AMU in humans, but it is also accepted that there is a contribution from animals (especially farm animals) to AMR in selected important human pathogens. Continuing to argue about this misses the point that the resistance genie is out of the bottle and won’t be got back in, and the train has left the station long ago.

Given the mobile nature of many resistance genes, resistance anywhere is potentially resistance everywhere.

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Figure. Epidemiology of antimicrobial resistance and movement of resistant bacteria (and resistance genes) between humans and animals. The figure illustrates the complexity of the interaction and the idea that “resistance anywhere is potentially resistance everywhere”. After Linton (1997) as modified by Rebecca Irwin, PHAC.


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What is Canada’s response to the resistance crisis?

Early response: The Canadian Integrated Program for Antimicrobial Resistance Surveillance, CIPARS

Following the 1997 Health Canada Consensus Conference on antimicrobial resistance in Montreal, an early response was the report to HC in 2002 on Uses of antimicrobials in food animals in Canada: Impact on resistance and human health. Still a very useful document, one of the very few recommendations acted on, and that was probably underway anyway, was the development of the Canadian Integrated Program of Antimicrobial Resistance Surveillance (CIPARS), modelled partly on the US NARMS program. CIPARS, which is highly respected internationally, integrates national resistance surveillance data in selected enteric pathogens in animals and humans, and monitors resistance in selected enteric “indicator” bacteria such as E. coli from animals at slaughter and animal products at the retail level. CIPARS is moving from resistance surveillance to use surveillance in animals. CIPARS became well known internationally through its work on ceftiofur resistance in Salmonella Heidelberg in chicken hatcheries and the impact on serious infection in humans with an organism resistant to the drug of choice (Dutil et al., 2010).

CIPARS published quarterly and annual surveys of resistance in selected pathogens, focused on the human-agricultural use, and on use in humans and farm animals: http://www.phac-aspc.gc.ca/cipars-picra/index-eng.php


Recent response: The pan-Canadian Framework for Action

Canada has responded relatively late to the AMR crisis but has been spurred to action by a 2015 report from the Auditor-General that criticized the Public Health Agency of Canada (PHAC) and Health Canada (HC) for lack of a national strategy to address AMR. The Federal government released a federal Framework for Action on AMR and AMU (antimicrobial use) in Canada in 2014, addressing the issue in three pillars: Surveillance, Stewardship and Innovation, and committing PHAC, HC, Agriculture and AgriFood Canada (AAFC), the Canadian Food Inspection Agency (CFIA), and the Canadian Institute of Health Research (CIHR) to addressing AMR. The 2015 Federal Action Plan committed the government to leadership on AMR. HC announced a process to remove the growth promotional use of medically important antibiotics, to bring antimicrobials used in food animals under veterinary prescription, to closing down the “own use importation” loophole, and
to tightening up regulations around use of active pharmaceutical ingredients. Since medicine, veterinary medicine and pharmacy are provincial responsibilities, subsequently, PHAC has worked with the provinces and territories, and numerous stakeholders, to develop a pan-Canadian Framework for Action (draft May 2014) based on Surveillance, Infection Prevention and Control, Stewardship and Innovation which requires to be endorsed by all the provincial and territorial Ministers of Health and Ministers of Agriculture. Once it is, then these groups will work on the pan-Canadian Action Plan. The pan-Canadian Framework is taking a “One Health” approach (minus the environmental piece) to AMR.


What is antimicrobial stewardship?

**Antimicrobial stewardship** is a coordinated program that takes a multifaceted approach to sustaining the efficacy of antibiotics and minimizing the emergence and spread of resistance. It promotes the appropriate use of antimicrobials (by promoting the selection of the optimal antimicrobial drug regimen, dose, duration of therapy, and route of administration), improves patient outcomes, reduces microbial resistance, and decreases the spread of infections caused by multidrug-resistant organisms. It’s an evolving concept that replaces the older terms of “prudent use” or “judicious use” since it. Aspects are illustrated in the Figure below.

The practices of antimicrobial stewardship are perhaps best developed in human hospitals, originally as a cost saving measure, but the area is exploding as stewardship becomes a requirement for hospital licensing. Nevertheless, there is increasing focus in both human and veterinary medicine on primary care physicians and veterinarians, since they are the major users of antimicrobials. The general mindset of **good stewardship practice (GSP)** is a “**5R**” approach: Responsibility, Reduction, Refinement, Replacement, and Review. A 5R stewardship approach is an active, dynamic, process of continuous improvement in AMU, a pragmatic ethic with many steps of different sizes. The Figure is an illustration of some of the different elements of GSP, many of which are discussed in the two other talks given at this conference. It seems likely that, within a short time, provincial veterinary regulations will require all practices to develop AMS policies and that regulators will monitor and evaluate AMU by veterinarians against agreed benchmarks.

**Practice guidelines:** The British Equine Veterinary Association (BEVA) has developed an award-winning approach to AMS, Protect ME, and is the best veterinary stewardship approach currently available: [www.beva.org.uk/protectme](http://www.beva.org.uk/protectme). The British Small Animal Veterinary Association (BSAVA) has developed a practical and accessible approach to AMS, called PROTECT: [https://www.bsava.com/Resources/Veterinary-resources/PROTECT](https://www.bsava.com/Resources/Veterinary-resources/PROTECT). PROTECT offers a comprehensive approach for a practice to develop its AMS policies and practices: the acronym stands for **P**ractice policy; **R**educe prophylaxis; **O**ther options (eg, lavage, topical use); **T**ypes of drug and bacteria (drug properties, likely bacterial agents); **E**mploy narrow spectrum; **C**ytology and culture; **T**reat effectively; For example, under Practice policy it recommends making a list of **first-line**, **second-line and third-line drugs**, where culture and sensitivity is used for second- and third-line drugs, and the latter are only used for life-threatening infections where first- and second-line drugs are not appropriate.

**Infection control:** Since AMR is essentially an infection control issue, infection control is an essential part of GSP. See comments on important AMR infections in the other two talks.
Resistance and Use Surveillance: Benchmarking: “Benchmarking”, the quantitative determination of norms for antibiotic use by veterinarians or at the farm level has been a powerful driver in reduction of antibiotic use in agriculture in countries which has significantly reduced AMU. There are also increasing reports surveying AMU in companion animals, some through capture of digital records through commonly used practice software. Benchmarking of AMU by veterinarians and at the farm level has been important in the reductions of AMU achieved in Denmark, where it forms the basis of the “Yellow card” system, and in Holland, where it has been an integral part of the 65% reduction in AMU on farms between 2009 and 2016. It seems likely that there will be farm and food animal veterinarian benchmarking in Canada as part of the pan-Canadian Antimicrobial Resistance Action Plan.

Danish Veterinary and Food Administration: The Yellow Card initiative on Antibiotics: https://www.foedevarestyrelsen.dk/english/Animal/AnimalHealth/Pages/The-Yellow-Card-Initiative-on-Antibiotics.aspx


Will making antimicrobials in food animals in Canada veterinary-prescription-only have any impact on resistance?

In Canada, the move to veterinary-prescription-only for antimicrobials in food animals is a major shift in responsibility for stewardship and GSP from agriculture to veterinarians. There would seem to be considerable scope to reduce the quantities of antimicrobials used in food animal production to where the benefits are clear and substantial, a process that is now underway. The increased emphasis on documenting use rather than simply resistance is appropriate, since use drives resistance and use is more easily managed than resistance. In Holland, which has intensive animal agriculture generally similar to that of Canada, there has been a 65% reduction in AMU on farms between 2009 and 2016. There will likely be a pan-Canadian
process that will need to monitor and report AMU provincially, nationally and internationally as part of the process of prescription filling and the requirements for benchmarking. If you can’t measure AMU, you can’t manage AMR. Major food retailers are now making changes in AMU requirements for farms from which they obtain animal products, and may become ahead of veterinary and other regulatory bodies. However, failure of veterinarians to rise to the challenge will result in loss of self-regulation in this regard.

What’s the role of the Canadian Veterinary Medical Association (CVMA) and the provincial veterinary regulators?

The CVMA continues to take a major leadership role on AMR on behalf of the veterinary profession in Canada. It is revising and expanding prudent use guidelines and developing numerous other resources including educational material around stewardship. One very important initiative around AMR has been the development by CVMA, together with the Council of Canadian Veterinary Registrars, of a pan-Canadian Framework of Professional Standards for Veterinarians on Veterinary Oversight of Antimicrobial Use. It is expected that these will become the common standard adopted by all the veterinary licensing bodies.

All provincial licensing bodies are engaging with AMR issues, and will do so more in the future, particularly in relation to development of the pan-Canadian Action Plan. Québec has the most experience, since veterinary prescription only of antimicrobials in food animals has been a provincial requirement for at least 20 years.


What’s the role of specialty veterinary groups?

All specialty veterinary groups nationally and internationally are engaging with antimicrobial stewardship, including development of practice guidelines. It is beyond the scope of this review to list examples because they are too numerous.

What’s the role of Canadian farmers and the farm organizations?

Major Canadian farm groups (aquaculture, beef feedlots, dairy, poultry, pork) are engaged with AMR and stewardship issues through the different national on-farm food safety and quality assurance (FSQA) programs. The smaller farm groups (eg smaller cow-calf operations) are probably less engaged and less accessible to educational initiatives. They will be forced to engage further with AMR as a result of the move to prescription only of antibiotics for food animals and what will almost certainly to documentation of AMU and benchmarking initiatives. All groups will benefit by educational actions by veterinarians. The move to stewardship standards in animal agriculture that reaches international standards will have to be an on-going partnership between veterinarians and farmers, and will require monitoring by regulatory groups.

The Chicken Farmers of Canada have developed the most rigorous antibiotic stewardship programs of all the major farm groups, a response to the problems identified ceftiofur resistance in Salmonella Heidelberg and E. coli through extra-label use of ceftiofur in hatcheries, as well as market demand for poultry raised without preventive use of medically-important antibiotics.
**National Farmed Animal Health and Welfare Council:** The NFAHWC is an advisory council of the Federal-Provincial-Territorial Regulatory Assistant Deputy Ministers of Agriculture Committee that works with industry, commodity groups and animal health organizations to develop advice on matters of national importance relating to animal agriculture. Addressing agricultures role in antimicrobial stewardship is a matter of active current interest to the Council and was the subject of a useful report in 2016 and of on-going activities.

Chicken Farmers of Canada: [http://www.chickenfarmers.ca/what-we-do/antibiotics/](http://www.chickenfarmers.ca/what-we-do/antibiotics/)


**Other groups involved in the changing nature of animal agriculture’s antibiotic use**

**Canadian Animal Health Industry:** The Canadian Animal Health Industry (CAHI has taken a critical and indispensable leadership role in supporting responsible changes in the use of antimicrobials in animal agriculture. This on-going work includes providing documentation of AMU in the provinces and by different user groups, as well as discussion of the logistics of the move to veterinary prescription only for food animals.


**The complexity of leadership on animal AMR issues in Canada:** Major changes will have been instituted in food animal use of antimicrobial once the federal action plan is implemented around or after December 2017. The pan-Canadian Framework for Action is setting the scene for development of a pan-Canadian Action plan to take a One Health approach to AMR. There are numerous issues to resolve, including notably the pan-Canadian leadership: Who do you phone to find out what’s happening or discuss issues? Who’s monitoring and reporting AMR and AMU, and to whom? How will antimicrobials be distributed to farms and farmers? Who’s monitoring appropriate use, and benchmarks against which possible AMU and AMR problems can be identified? The Figure below shows the many actors involved and the complexity of the integration that will be required for an effective pan-Canadian approach to addressing AMR that meets international standards. We are underway but there’s lots to be integrated.
What are the critically-important antibiotics for human medicine?

The World Health Organization (WHO), and Health Canada, categorize and prioritize antibiotics into different categories based on their importance in treating bacterial infections in human medicine. Those of the Very high importance (HC) are drugs regarded as critical for the treatment of very serious infections for which alternates are not available. The purpose of categorization is to assist in risk assessment for determination of the approval process for drugs used in veterinary medicine, for example restricting these in different ways. Health Canada’s intention is that categorization is a dynamic process depending on changing circumstance. It’s a somewhat contentious process with differences in the categories (and antimicrobials listed) between Health Canada and the WHO (and other groups). What is obvious is that humans and animals share the same antimicrobials and that there is a “hierarchy” of importance of antimicrobials, to some extent based on when they were first introduced. The general approach is an important part of the strategy to preserve antibiotics.

Examples for Health Canada’s categories of “Very high importance” are third and further generation cephalosporins, carbapenems, penicillin-beta-lactamase inhibitor combinations; fluoroquinolones; glycopeptides; polymixin. Examples of “High importance” are aminoglycosides, first- and second-generation cephalosporins, lincosamides, macrolides, penicillins, and trimethoprim-sulfamethoxazole. Examples of “Medium importance” are bacitracin, phenicols, sulfonamides and tetracyclines. Examples of “Low importance” are ionophores.

An alternative “unofficial” approach to categorization is of “First line”, “Second line, and “Third line”, and Restricted drugs. Primary or first line drugs are those used in initial treatment in advance of or in lieu of culture and susceptibility testing; such drugs are those less important for treating serious human infections. Examples include penicillins, first- and second-generation cephalosporins, trimethoprim-sulfonamides. Secondary or second line drugs are used when culture and susceptibility, plus patient and infection factors, indicate that no first line drugs are reasonable options. These drugs may be more important in treating serious human infections. Examples include fluoroquinolones and third- or fourth-generation cephalosporins. Tertiary or third-line drugs are those used for serious, life-threatening infections with support by culture and susceptibility, and where no first or second line drugs are indicated. An example is carbapenems. Restricted drugs are those that are either never used or only under the most dire circumstances. An example is vancomycin.

An example of the use of this categorization approach has been the voluntary ban on third-generation cephalosporins in swine production in Denmark (Agerso and Aarestrup, 2013).


World Health Organization, 2016: Critically important antimicrobials for human medicine: http://apps.who.int/iris/bitstream/10665/255027/1/9789241512220-eng.pdf?ua=1

What does the future look like?

The future is always hard to predict, but we are going to have a very different relationship with antimicrobial drugs in the future. The final O’Neill Report identifies the top ten approaches to addressing AMR globally, one of which is reduction of antibiotic use in agriculture and reducing antibiotic contamination of the environment. AMR will continue to adversely affect how we treat bacterial infections. Bacteria can readily change to resistance, but people, groups and institutions are highly resistant to change. We have however no choice.

http://apps.who.int/iris/bitstream/10665/44812/1/9789241503181_eng.pdf


References available for purchase


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Disclaimer: The perspectives expressed in this summary are mine alone, and almost certainly do not adequately acknowledge the effort or thought put into AMR by numerous different individuals and groups in Canada.
Many publications have emphasized the increasing role of aged horses in veterinary medicine, and this is supported by some data. Publications concerning geriatric horses need to be interpreted carefully, however, as the definition of a geriatric horse, the inclusion criteria, and the consideration for confounding factors such as co-morbidities may vary among studies.

Horses are frequently considered aged or geriatric when they are at least 15-20 years of age. (1) With an average life expectancy reported as 19 years, (2) these cut-offs may appear appropriate; in addition, some authors feel propose that an additional category of the “very old” horse deserves to be considered separately. Based on the most recent NAHMS study in the United States, the percentage of the horse population that is older than 20 years has increased since 1998 and now constitutes approximately 11% of the horse population, (3) while “very old” horses greater than 30 years of age represent 1.5% of the population. Even higher numbers have been reported from the UK, where approximately 25-28% of the equine population was older than 15 years. (1) Aside from improved health care, an increasing number of aged horses may be attributable to changing owner attitudes towards the “usefulness” of an equine and, interestingly, 56% of horse owners queried for the 2010 Canadian Horse Industry Profile study indicated that they intended to own their horses for the horses’ lifetime.

Visible signs of aging in horses include graying of the face and muzzle, development of a swayback appearance, drooping lips, marked supraorbital grooves and increased dental and hoof wear. Of importance to veterinarians, horse owners may misinterpret clinical signs of disease as those merely related to aging in geriatric horses, and may not perceive alterations in hair coat, weight changes, stiffness or reduced joint flexibility, or reduced exercise tolerance as cause for concern and veterinary consultation. (1) Veterinarians should therefore take any opportunity for thorough history taking and physical examination, e.g. during annual dental or vaccine appointments, in order to adequately counsel owners on potential causes for changes in their aging horse’s health. Chronic pain from lameness or dental disease may be an under-recognized serious welfare concern in the aging horse and may result in secondary problems such as weight loss, e.g. if the horse cannot move adequately towards a feed source or cannot comfortably eat off the ground, or sleep deprivation, which may present as episodes of collapse or near collapse. In horses that continue to perform athletically into advanced aged, as well as in those housed outside in cold climates, physiologic changes such as less efficient thermoregulation need to be taken into account in order to provide adequate care.

With the exception of routine dental care, there is limited information concerning the need to alter general management practices such as vaccination and deworming for the aging horse. Horses may undergo changes similar to immunosenescence in human beings, which results in a reduced response to vaccination, increased susceptibility to infection and a generalized pro-inflammatory state. (4) Evidence for direct clinical correlates is lacking at this time, however, and specific information for the healthy aging horse as opposed to the horse that may also be impacted by a poor nutritional status or concurrent diseases such as severe endoparasitism is not sufficiently available. General recommendations for the use of core and risk-based vaccines, such as those published by the American Association of Equine Practitioners (www.aaep.org) apply and likely do not need to be adapted in most situations. Importantly, horse owners may perceive the risk of infectious disease to be decreased in aging horses, which may lead them to omit vaccination entirely or change vaccination schedules arbitrarily for these animals.

An increased risk of endoparasitism in aged horses has been stated in several publications, (reviewed in 1) but the available evidence if not clear-cut and situation-specific investigation appears more appropriate than blanket changes to deworming schedules in horses of increasing age. Endoparasitism may become a problem
in horses with pituitary pars intermedia dysfunction (PPID, Equine Cushing’s Disease), however, and should always be considered in the management of these patients.

Several studies have addressed the prevalence of different diseases in the aging equine population; (reviewed in 1) however, differences in study design and inclusion criteria make it difficult to directly compare or summarize the results. Conditions that are commonly mentioned as problems for geriatric horses include musculoskeletal conditions (primarily arthritis), laminitis (especially in connection with PPID and insulin dysregulation), certain causes of colic (impactions and strangulating lipomas), equine asthma or heaves, weight loss, dental diseases and sinusitis, degenerative valvular disease, ocular disorders and neoplasia (including PPID, benign thyroid adenomas and lipomas but also melanomas, squamous cell carcinomas and lymphosarcoma). Uterine artery rupture is reported as more common in aging broodmares. There is no direct evidence for an increased risk of infectious diseases such as strangles or influenza (4) although an increased case-fatality rate of West Nile virus infections, (5,6) and an increased susceptibility to the neurologic form of EHV-1 infections (7) in aged horses have been reported.

It may be difficult for horse owners to make decisions regarding the health care of their aging horse, be it in emergency situations such as the need for immediate colic surgery, or when faced with chronic health concerns such as lameness or weight loss. Several studies have evaluated the outcome of colic in aged horses, and while reduced short-term survival and reduced survival of colic surgery in horses older than 20 years were reported by some authors (8,9) one study found no difference in the long-term outcome after colic surgery. (10) Perceptions on the quality of life of aged horses may also vary among horse owners and while this certainly needs to be respected by the attending veterinarian, veterinarians play an important role in advocating for the welfare of the horse. The AAEP euthanasia guidelines (https://aaep.org/horsehealth/aaep-guidelines-euthanasia-2011) are a useful tool in this regard as they contain statements such as a recommendation for euthanasia of horses that require pain medication for the remainder of their lives in order to stay comfortable.

References


Weight loss and obesity are both reported in geriatric horses and may occur with equal frequency. Each can be a serious health concern and veterinarians should be prepared to counsel horse owners on ways to maintain their animals in appropriate body condition throughout their lives. The 2013 Code of Practice for the Care and Handling of Equines states that geriatric horses should “receive a diet that is adequate for maintaining health and vigour” (2). According to the code, corrective action must be taken for horses with a body condition score of 3/9 or less, or 8/9 or higher, and veterinary advice must be sought for emaciated geriatric horses (body condition score of 2/9 or less). (2)

While some differences between studies exist, current research does not suggest that there is a significant change in the digestibility of energy, fiber, crude protein, fat or minerals and micronutrients in aging horses. Maintenance energy requirements may decrease with age, as they do in other species such as humans, which may be attributable to decreased physical activity, decreased lean muscle mass and less metabolic turnover. (4-6) In horses, these physiologic changes may be off-set by age-related changes in mastication, however, or by medical conditions such as endoparasitism or neoplasia that affect feed efficiency or increase energy requirements. (7,8) As body fat distribution can also change, especially in horses with pituitary pars intermedia dysfunction (PPID) or metabolic syndrome, assessment of physical condition should take into account actual or estimated (e.g. by weight tape) body weight in addition to the body condition score and the visible appearance of the animal. (6)

Weight loss in geriatric horses is a common concern and may be a primary reason to consider euthanasia if owners are unable to provide adequate management. Aside from medical conditions that result in weight loss, veterinarians should consider and query owners about the adequacy of the horse’s diet (both the amount and the type of feed are important), potential issues with feed access such as changes in group hierarchy or chronic pain, (9) and environmental conditions such as the need to maintain a horse outside during the winter. A complete dental examination is indicated in every horse presenting for weight loss, but especially in geriatric horses where changes may be “terminal” in nature and require changes to the horse’s feeding regimen in order to maintain adequate feed intake. Investigating and addressing medical conditions in a horse that cannot be maintained on a roughage-only diet based on its dentition, and whose owner is unable to invest in long-term provision of alternative feeds, is unlikely to result in a satisfactory outcome and improved welfare for the patient.

Complete feeds are an excellent choice for senior horses that cannot meet their energy requirements through roughage alone. The ideal complete feed is designed to contain sufficient roughage to allow for exclusive feeding; however, the vast majority of complete feeds on the market does not fit this description. Maintaining a minimum of roughage intake for digestive health can often be achieved through grazing or feeding of soaked hay cubes or beet pulp, even if horses are unable to chew long-stem hay. Choke has been identified as a significant concern in geriatric horses with dental issues by some authors, (6) and care should be taken to allow horses to adjust to a new diet gradually. Gradual accommodation is also needed if fat supplements such as oils (e.g. up to 2 cups of vegetable oil/day, with addition of 100 units Vitamin E/100 mls oil) are used to increase caloric intake. Fat supplementation is probably preferable to grains or other high-carbohydrate feeds in horses suffering from, or at risk for, PPID or equine metabolic syndrome. (7) Importantly, feeding changes for horses in marginal or poor body condition that are housed outside need to be made in a timely fashion that allows for sufficient fat build-up before the winter season. Some geriatric horses may need to be moved inside or at least may need to be blanketed with access to a well-bedded shelter and frequent checks on their condition. In addition to maintaining or improving caloric intake, many authors recommend special attention to vitamin and mineral supplementation of geriatric horses, or recommend the addition of yeast or probiotic products to account for a potential change in the intestinal microflora that may limit feed efficiency. (10) Ensuring free access to good quality water and offering
“flavored” water or warmed water sources for horses with dental pain are other considerations that may aid in maintaining good health of the aging horse.

Common dental disorders in aged horses include malalignment and malocclusion (e.g. wavemouth formation), tooth loss, periodontal disease and development of diastemata, tooth root infections and accompanying sinusitis. Thorough physical examination should precede dental examination to uncover any conditions (e.g. cardiovascular diseases or painful lamenesses) that may affect the horse’s response to sedation or its ability to withstand the examination. In addition to thorough oral examination, imaging modalities such as intraoral endoscopy, radiography or even computed tomography can be useful. With severe abnormalities, several sessions in approximately 3 month intervals may be needed for correction of the problem. Six-monthly evaluation is recommended for horses with malocclusions as they have been shown to recur in this time period.

Once feeding strategies and dental abnormalities have been evaluated and, if needed, corrected, additional diagnostic testing may be indicated in geriatric horses with weight loss. Investigation may be challenging in patients with no additional clinical findings or findings on routine blood work (complete blood count, serum biochemistry), but may include diagnostic tests such as rectal examination, abdominal and chest ultrasound, abdominal fluid evaluation, glucose absorption tests, gastroscopy and testing for PPID. Differential diagnoses include neoplasia, peritonitis including abdominal abscessation, protein-losing enteropathies, chronic renal failure, chronic liver disease, gastric ulceration, severe chronic respiratory disease (equine asthma) to name just a few.

PPID is one of a few distinctly age-related conditions of horses and is a neurodegenerative disease likely based on oxidative stress. Dopaminergic neurodegeneration leads to an overproduction of proopiomelanocortin (POMC)-derived peptides, which are likely responsible for clinical signs although the exact pathophysiology has not been elucidated. A number of diagnostic tests has been evaluated over time and, currently, the thyrotropin-releasing hormone (TRH) stimulation test is considered the most reliable testing method by many clinicians. (11,12) For this test, a plasma sample is collected for baseline ACTH measurement, then 1 mg TRH is injected intravenously and additional samples for ACTH measurement are collected 10 and/or 30 min later. Because of their simplicity and non-invasiveness, baseline ACTH concentrations are often used initially, however, and may be very useful as long as instructions for sample collection and storage are followed and laboratory-specific seasonal reference ranges (13) are used for interpretation. Seasonal differences in test performance probably need to be considered for any of the diagnostic tests for PPID, as do the possibility for false negative and false positive test results. Early diagnosis of PPID is an area of great uncertainty, and veterinarians faced with test results that do not “match” the patient history or the clinical concerns should consider re-testing after a period of time or using another diagnostic test in an attempt to confirm their findings. Because of the potential overlap between PPID an equine metabolic syndrome, and the increased risk of laminitis in horses with insulin dysregulation, testing for insulin dysregulation should also be considered in geriatric patients presenting for suspected PPID.

Treatment of PPID primarily depends on life-long administration of pergolide, a dopamine D2 receptor agonist, along with optimization of management and treatment of any complications such as laminitis or recurrent infections. Dosage must be adjusted to the individual patient but Dosage 2-4 µg/kg/day, in one or divided into 2 doses, is commonly recommended as a starting point. (14) Aside from inappetence, pergolide probably has few side effects in horses; this problem can often be successfully addressed by splitting the total daily dose in 2, reducing the dose temporarily or taking the horse off medication for a period of time and re-starting at a lower dose. Treatment goals may vary among patients and owners should be counseled on the benefits of monitoring baseline ACTH concentrations or other diagnostic test results versus treating based on clinical signs alone.
References


5. Bosy-Westphal A et al. The age-related decline in resting energy expenditure in humans is due to the loss of fat-free mass and to alterations in its metabolically active components. J Nutr 2003; 133: 2356-2362.


Antimicrobial resistance is not on most equine practitioners’ radar, but AMR is emerging as an important issue in equine medicine. We are moving from the antimicrobial era through the antimicrobial resistance era into the era of antimicrobial stewardship. This shifting paradigm in our relationship with antibiotics is part of the global responsibility that all users have to preserve for the long term the rather small range of antimicrobial drugs available to treat bacterial infections.

**Antimicrobial stewardship** refers to an approach that promotes, improves, monitors and evaluates judicious antimicrobial use (AMU) to preserve the future effectiveness of antimicrobials and to promote and protect human and animal health. It is a term that is preferred to the previously used terms judicious or prudent use, since it includes the idea of not using antimicrobials. The general mindset of **good stewardship practice (GSP)** is a “5R” approach: Responsibility, Reduction, Refinement, Replacement, and Review. A 5R stewardship approach is an active, dynamic, process of continuous improvement in AMU, a pragmatic ethic with many steps of different sizes. All veterinary users of antimicrobials are now inevitably forming part of the global “One Health” strategy to address AMR. This strategy, as endorsed in the 2017 pan-Canadian Framework for Action for Tackling Antimicrobial Resistance and Antimicrobial Use, includes surveillance, infection prevention and control, stewardship, and innovation.

Antimicrobial stewardship (AMS) is a rapidly evolving field, with greatest activity and leadership in large tertiary care human hospitals where AMR problems are most visible. Nevertheless, there is increasing focus in both human and veterinary medicine on primary care physicians and veterinarians, since they are the major users of antimicrobials. In Canada, the move to veterinary prescription only for antimicrobials in food animals is a major shift in responsibility for stewardship and GSP. It seems likely that, within a short time, provincial veterinary regulations will require all practices to develop AMS policies and that regulators will monitor and evaluate AMU by veterinarians against agreed benchmarks.

**A practical approach to antimicrobial stewardship in equine medicine**

The concept of AMS is of a dynamic process of continuous improvement in how we use antimicrobials and of reduction in their use to where the benefits are clear and substantial. Figure 1 is an illustration of some of the different elements of GSP. The different elements will be discussed.

**Practice guidelines:** The British Equine Veterinary Association (BEVA) has developed an award-winning approach to AMS, Protect ME, and is the best veterinary stewardship approach currently available: [www.beva.org.uk/protectme](http://www.beva.org.uk/protectme). Two-thirds of British equine practices have adopted the ProtectME approach. The brilliance of the approach is that it requires practices to think through their AMS approaches and to engage the entire veterinary team; it’s adopt an approach rather than adopt a defined practice. Protect ME offers a comprehensive approach for a practice to develop its AMS policies and practices: the acronym stands for Practice policy; Reduce prophylaxis; Other options; Types of drug and bacteria; Employ narrow spectrum; Culture and sensitivity; Treat effectively; Monitor use, compliance and resistance; Educate. For example, under Practice policy it recommends making a list of first-line drugs, with dosages; under monitoring, it suggests assigning functions of resistance reporters, resistance monitors and resistance leaders to engage the whole veterinary team, and provides templates or wall charts where resistance data can be entered. BEVA offers an Equine Antibiotic Resistance Champion plaque for the clinic if you meet certain criteria.
Infection control: The emergence and spread of methicillin-resistant *Staphylococcus aureus* (MRSA), particularly clonal type CMRSA 5, in horses represents a serious example of an AMR problem that is encountered both in equine hospitals and in the community. Infections include soft tissue infection, incision infections, bone and joint infections, as well as disseminated infections. Other clonal types, notably the livestock-associated strain ST398, may also become problematic. MRSA are resistant to all beta-lactam antibiotics. Nasal colonization is common in horses and infection with these strains is common in equine personnel, including veterinarians. In one study, there was a strong correlation between high nasal colonization of equine veterinarians and lack of handwashing. Good infection control is fundamental in reducing the spread of infections, including of resistant bacteria.

Clinical microbiology: Greater use of clinical microbiology data to guide selection of antimicrobials is an obvious approach to GSP, but is currently hampered by delays in obtaining data and cost. There is considerable effort being made to speed the process, particularly through rapid DNA-based approaches. Protect ME recommends such testing before the use of Category 1 antimicrobials, those of critical importance in human medicine (fluoroquinolones, third-generation cephalosporins).

Resistance and use surveillance: On-going monitoring of resistance and use is an important part of the “continuous improvement” mindset of GSP. One equine specific example is of the emergence of macrolide resistance in the foal pneumonia pathogen, *Rhodococcus equi*. On some farms resistance in isolates is as high as 40%, seriously impairing the ability to treat infection. A unique type of macrolide resistance gene, *erm*46, has been spreading in *R. equi* and in soil non-pathogenic rhodococcal species on horse breeding farms around the world. It has been the practice on many farms to use ultrasound to detect incipient lung abscesses, and then to treat foals with azithromycin. Recently it has become clear that many foals will develop small lung abscesses but will recover spontaneously, without treatment, even if abscesses are as large as 10 cm. This is an outstanding example where not treating unnecessarily, despite the temptation, will prevent the development of resistance and preserve antimicrobials. We still need to find out how to
know which foals will go on to developing clinical disease, which is part of the conundrum of *R. equi* infection.

**Pharmacokinetics and pharmacodynamics:** Knowing the difference between concentration-dependent (aminoglycosides, fluoroquinolone) and time-dependent antimicrobial drugs is an important pharmacokinetic consideration both in optimal dosing and in the prevention of the emergence of resistance. One survey found that inappropriate dosing generally was common in equine medicine.

**Regulations:** Regulations should be followed. They are particularly important in relation to human health considerations. Protect ME suggests that use of vancomycin and carbapenems should be avoided in horses.

**Education:** Staff and owner education posters and related materials are available on the Protect ME website.

**Summary**

Many aspects of antimicrobial stewardship, including regulatory monitoring, evaluation and certification are in their infancy. Equine practitioners will however be expected to hold to a high standard of AMS, as part of the broad changes around how we use antimicrobial drugs so as to preserve them for the foreseeable future.

**References**

BEVA offers free access to papers on resistance in equine medicine:

Micronutrient deficiencies are a common differential in diagnosing a variety of herd problems. While a number of trace minerals and vitamins may be considered, those which are typically the focus of interest when investigating problems in western Canada include copper, molybdenum, selenium, zinc, vitamin A and vitamin E. Although it is relatively rare to see animals with serious clinical signs of deficiency, impacts on herd performance are much more common and are more difficult to recognize. This presentation will focus on the investigation of micronutrient deficiencies in beef herds and troubleshooting micronutrient management to minimize the risk of production losses.

Serum, whole blood, and liver are the most common biological samples used to assess individual animal and herd status. Careful collection and sample handling are critical to useful results. Challenges with interpreting the laboratory results will also be discussed. For example, most of the guidelines used in interpretation of micronutrient concentrations in samples from beef cattle are 30 years old, and the evidence for many of the diagnostic thresholds is not well defined. Similarly, the evidence definitively linking micronutrients to specific clinical problems or production losses in beef herds is also sparse.

Several studies have been published from work done in the past 15 years in western Canada to try to determine the extent of micronutrient deficiencies and their impacts. The samples examined have included livers from necropsy studies and serum samples from neonatal calves, replacement heifers, and cows collected as part of a number of surveillance initiatives. Findings of interest include an association between severe drought and low vitamin A levels in neonatal calves that were then linked to an increased risk of calf mortality. Lower vitamin E concentrations in calves were associated with an increased risk of scours, and low selenium concentrations in pregnant cows were associated with an increase of skeletal myopathy in young calves. One of the findings of greatest concern, however, has been the relationship between copper deficiency and reproductive performance in young cows. Copper is also by far the most common deficiency identified in all Saskatchewan studies.

When faced with a suspected micronutrient problem, there are a number of factors that can be considered when evaluating the extent of the herd problem and identifying opportunities for change. Many of the most relevant questions focus on the management of micronutrient supplementation:

1. When during the production cycle is the supplement provided? How much is provided and in what form (free choice, mixed with salt or another supplement, or force fed)?
2. What type of supplement is being provided? What are the benefits of chelated minerals and should they be used?
3. Are other forms of supplementation such as boluses or injectables an option?
4. What are the challenges and issues with interpretation of feed and water testing?
5. Are there issues with the stability and palatability of micronutrient supplements due to storage and feeding management?
6. What factors might alter animal requirements for micronutrients?
7. How is soil type associated with risk of deficiency in Saskatchewan?
8. Is there a link between precipitation and micronutrient status in beef cattle?
9. Has the balance and relationships among micronutrients (e.g. Cu and Mo) been considered?
10. Finally, there is the important reminder that too much can be as dangerous as too little.

The first consideration when any micronutrient deficiency is suspected to rule out/address more direct causes of the herd health or performance problem. For example, the role of body condition is critical to...
reproductive performance. Given there is no strong evidence that other factors are responsible for the production problem, the suggested steps for evaluating micronutrient deficiencies in cow-calf herds under conditions in western Canada, and particularly in Saskatchewan, include:

- **Evaluate supplementation management**
  - Look at how much supplement the cows are consuming.
  - Compare what they need to consume to what they actually have consumed.
  - Recognize that this will be an average value and many cows will still be consuming either too little or too much.

- **Blood testing cows**
  - Spring tests are more useful for herd management than fall samples.
  - Where possible, compare groups of cows with and without the reproductive problem of interest and take enough samples to account for variability in intake among animals.

- **Test liver samples from dead or aborted calves / animals sent to local slaughter.**
  - Caution is warranted as these are typically very small samples and often atypical animals.

- **Collect representative water samples to measure:**
  - Sulfates & iron (tie up copper)
  - TDS (impacts free choice intake)

- **Collect representative feed samples to measure:**
  - Mo concentrations – tie up copper
  - Check for sulfur concentrations especially in some feed types (e.g. canola)
  - Selenium

  *Note copper is deficient in most SK forage – knowing how deficient isn’t that helpful given the low percentage of feed copper taken up by the animal.

**Selected references from Western Canada:**


Reproductive performance is a key driver of profitability in cow-calf operations. The industry is changing and as herd sizes get bigger many producers are moving their breeding dates back to better manage labour requirements during calving season. Calving later can minimize risks to young calves from cold weather and infectious disease associated with crowding. However, producers are also asking their cows to recover after calving, start cycling and become pregnant later in the summer, often after the peak growing season.

One of the questions that has been raised by both producers and veterinarians is whether or not pregnancy rates have been affected by later breeding seasons. A second issue currently raising concern is that when herd size is increased, the risk of exposure to infectious disease also increases. Herd biosecurity is compromised as replacement animals are bought in from a greater number and variety of sources. These and other factors have resulted in a steady stream of investigations related to poor reproductive performance in beef herds.

There have been a number of studies during the past 15 years examining factors that can influence reproductive performance in western Canada. The Western Canadian Cow-Calf Surveillance Network provides a current source of data from which to benchmark reproductive performance and to investigate risk factors for reproductive loss. In this talk, we will examine pregnancy rates as well as abortion and stillbirth losses in herds with a range of breeding and calving dates. We will also review the most current field data on the role of nutrition in reproductive performance.

While trace minerals are often the focus of attention in herds with higher than expected open rates, all investigations should include an evaluation of body condition score. Body condition scoring at pregnancy testing can provide useful insight on the role of energy balance in the face of high open rates. Simple statistical analysis can be used at pregnancy testing to assess the extent to which thin cows were more likely to be open than cows in good body condition. However, condition scoring is best used on a routine basis to proactively manage nutrition and prevent reproductive problems. Particularly in herds with a history of poor reproductive performance, condition should be assessed at least visually on as many cows as is practical three times per year: before calving, before breeding, and at pregnancy testing. Poor body condition at these time points and loss of condition score from one time point to the next are both important risk factors limiting reproductive success. Poor body condition has been associated with failure to become pregnant, increased time to pregnancy, abortions, difficult calvings, and stillbirths under field conditions in western Canada.

In addition to nutrition and breeding management, infectious disease is also an important cause of poor performance. Diseases that continue to raise concerns include BVDV, trichomoniasis, vibrio (*Campylobacter fetus* spp venerealis), neospora and leptospirosis. Two of the most effective tools for control, in addition to good herd biosecurity, are vaccination and testing and removal of infected animals. We will review current vaccination practices from Western Canada as well as available field research data on the benefits of vaccination and approaches to disease screening. In addition to the risks of disease introduction with purchase of cattle, other biosecurity challenges continue to threaten herd performance. One example is the increased risk of being open or aborting for non-vaccinated cattle (BVDV/IBR) exposed to community pastures as demonstrated by a large field study in Saskatchewan and Alberta.
Other infectious agents are less commonly identified but still lead to sporadic problems. The most current available information will be presented for neospora and leptospirosis, and we will briefly review testing and management options for trichomoniasis and vibrio.

Finally, this presentation will include a general framework for investigating reproductive failure. The first and most important step is to clearly define the problem. Management history and reproductive examination of cows and bulls are important in determining the extent and duration of the problem. How does performance compare to other herds in the immediate area? The most common challenges in evaluating records of herd reproductive performance will be discussed. The identification of groups for comparison of differences can be critical in developing and testing a hypothesis. Groups of interest may be based on reproductive status (e.g. pregnant yes or no), age, breeding or management groups, comparison among sire groups, pasture location, recently purchased vs other animals, exposure to specific feed sources, and extent of exposure to other herds. Body condition score data, breeding soundness exams and laboratory testing (e.g biological, feed, water) can also be used to further examine hypotheses regarding the differences in reproductive performance. The combination of laboratory results, clinical findings, and herd records can be used to target the factors most likely responsible and develop an action list to minimize the potential for future losses.

Selected references from Western Canada:


You know more about poultry than you think!!

Canadian Poultry Production Systems

- **Intensive Commercial with traditional poultry species**
  - Marketing Boards regulate production by a quota system (national & provincial boards)
    - Egg, Chicken, Hatching Egg, Turkey
  - Manage domestic markets & imports by allocation of quota
  - Balance the production supply chain
  - Strong government lobby & unified voice of producers

- **Commercial Specialty Birds**
  - # unknown but blossoming niche markets with premiums
  - Active genetic selection to satisfy specialty markets
  - **Specialty Birds**
    - **Product**: organic, nutrient fortified (Ω-3 eggs, ginseng chicken),
    - **Rearing method**: free-run, free-range, enriched cage, welfare-certified
    - **Species**:
      - Silkie, Taiwanese chicken
      - Pigeon squab
      - Duck & goose
      - Gamebirds: pheasants, quail

- **Small farm flocks**
  - # unknown but growing popularity
  - Limited dedicated veterinary resources
  - Questionable internet advice
  - Relies on the commercial industry infrastructure
    - Feed, hatcheries, vaccines, pharmaceuticals

- **Urban chickens**

The Backyard Poultry Flock Philosophy

Understanding the various motivations for keeping small flock poultry is essential in developing a successful VCPR. This sector is unlike the commercial poultry sector which is unified with bottom-line economics being the primary driver. Reasons people keep small flock poultry:

- Cheaper
- Self-sufficiency
- Healthier
- Knowing your food source
- Generate income
- Improved welfare
- Hobby, enjoyment
- Heritage breeds
- Farm tax exemption
- Anti-intensive poultry industry
- Nostalgic lifestyle
- Provide purpose

Recognizing Illness in Birds

- Poultry are a prey species → mask illness or get eaten
- Owners need to know their birds
  - Good record keeping ensures regular observation
Recognize first clues
- reduced feed/water intake
- reduced egg production or weight gain
- lethargy, inactivity, segregation

**Disease Expression** can be influenced by:
- Virulence or infectivity of agent
  - Strain variations
- Inherent susceptibility:
  - Species, age, gender
- Concurrent infection, parasitism
- Poor nutritional status
- Environmental stress, poor management
- Immune status
  - Vaccinated
  - Recovered antibodies

**Latent infections & carrier birds**

Several bird-adapted infectious agents are **not cleared following the development of immunity**

- **Lifetime carriers**, appear clinically healthy
- Stress-induced shedding
- **Become a source of infection for naive birds**

This is a characteristic of the following avian diseases:

- ILT
- Mycoplasma
- Chlamydia
- Salmonella sp.
- Coryza
  - Chickens (*Avibacterium paragallinarum*)
  - Turkeys (*Bordetella avium*)

**Causes of Disease**

- **Metabolic**
  - Obesity, cage layer fatigue, fatty liver
- **Genetic**
  - Heritable predispositions or disorders
    - Silkies & Marek’s Disease
    - Feather-sexing gene
- **Congenital**
  - Developmental abnormalities
    - High incubation temps → beak deformity in chicks
- **Toxicity**
  - Ionophores, mycotoxins, rodenticides, toxic plants
- **Nutritional**
  - Malnutrition
    - Poor feed quality
    - Formulation errors
    - Nutritional availability
    - Need grit source
      - Oyster shell ≠ grit
Vitamin over-supplementation
  ▪ Renal calcification
  ▪ Dehydration
Nutrient deficiency or imbalance
  ▪ Curly Toe Paralysis (riboflavin deficiency)
  ▪ Rickets
    • Vitamin D deficiency or Ca/P imbalance
    • Soft rubbery bones, painful
    • Rapid response to Vit D/Ca supplementation

Mismanagement
  ▪ Temperature extremes
  ▪ Dehydration
    • Lack of water access
    • Vitamin over-supplementation or electrolyte misuse
  ▪ Suffocation, Crowding or Stamping
  ▪ Wet litter/poor ventilation
    • Footpad burns/Bumblefoot
    • Ammonia keratitis
  ▪ Ingested foreign bodies
  ▪ Predation
    • Varies with varmint
      • Neck bite, missing birds, feather piles
  ▪ Feather-picking & Cannibalism
    • May present as “unexpected death”
      • Perineal hemorrhage +/- evisceration
    • Manifestation of stress
      • Overcrowding, Social pecking order
    • Extension of feather-picking
      • Behavioural or nutritional basis
    • Attracted to “red flash” at egg-laying
    • Habitual → “blood lust” (find this bird & eliminate)
  ▪ Treatment
    • Address obvious stressors like overcrowding
    • Adequate nest box space (1 per 4-5 birds)
    • Remove injured birds
    • Red light bulbs?

Neoplastic Disease
  ▪ Spontaneous tumours
    • Ovarian adenocarcinoma etc
  ▪ Leucosis (retrovirus)
    • Eliminated through genetic selection
  ▪ Marek’s Disease (herpesvirus)
    • Ubiquitous virus
    • Controlled by hatchery vaccination
    ▪ PHASE 1: Nerve form ~10-12 weeks
      • Lymphocytic infiltrates in brain, sciatic nerve
      • Range paralysis
      • Histologic diagnosis
    ▪ PHASE 2: point-of-lay
      • Lymphoid tumours in liver, spleen, kidney, heart, intestines, ovary

Diseases of High Production
  ▪ Rare in small flocks
  ▪ Can be eliminated by reducing production pressure
  ▪ Examples: ascites, flip-overs, leg deformity, slipped tendons, cage layer fatigue
- **Infectious Disease: Viruses**
  - Require a living cell to survive & replicate
  - Variable environmental survival times outside host cells
  - Limited treatment options but some viral vaccines are available
  - Can cause primary disease or be complicated with bacteria.
  - **Viruses with “target organs”** → clinical signs related to tissue(s) affected
    - **Infectious Laryngotracheitis (ILT)**
      - Herpesvirus targets tracheal epithelium
      - Hemorrhagic tracheas
    - **Inclusion Body Hepatitis (IBH)**
      - Avian Adenovirus targets hepatocytes
      - Broiler chickens 2-6 weeks
    - **Avian Pox**
      - Proliferative crusty scabs
      - Insect bites can spread it
      - Commercial vaccine available
      - Spillover to wild birds
  - **Viruses without specific “target organs”** → usual outcome is death
    - Highly Pathogenic Avian Influenza (HPAI)
    - Velogenic Newcastle Disease

- **Infectious Disease: Bacteria**
  - Can survive & reproduce away from a host
  - Have protective mechanisms for survival (form spores, live in biofilm)
  - Antibiotic treatment options
  - Poor vaccine candidates
  - **A: Ubiquitous bacteria**
    - In normal “balance”
    - Opportunistic
    - Need “help” to cause disease (MG, viruses, pH)
      - *E. coli* (Colibacillosis)
        - Variety of disease syndromes related to location of infection
      - Staph
      - *Clostridium perfringens* (Necrotic enteritis)
  - **B: Pathogenic bacteria**
    - Introduced therefore can be eliminated with sanitation
    - Prevented with strict biosecurity & sanitation
      - Salmonella
      - Pseudomonas (dirty water)
      - *Pasteurella multocida* (Fowl Cholera)
      - Erysipelas (birds on range after rain)

- **Infectious Disease: Fungi**
  - Includes molds & yeasts
  - Environmental exposure (spore inhalation)
    - Incubator, wet feed or litter
    - Aspergillosis
  - Source of mycotoxins
  - Difficult (impossible) to treat
• **Infectious Disease: Parasites**
  o Characteristics of Parasites
    ▪ Single celled or multi-cellular
    ▪ Have specific survival strategies when away from host (eggs, cysts, tough shells)
    ▪ Little effect by disinfectants
    ▪ Need to consider environmental contamination in a control strategy
    ▪ Life cycle can be direct or indirect
    ▪ Age-related immunity, host specificity
  o Treatment
    ▪ De-wormers, anti-coccidials, insecticides

  o **Internal Parasites**
    ▪ Nematodes
      • Ascaridia (round worms)
      • Heterakis (cecal worms)
      • Capillaria (hair worms)
    ▪ Tapeworms
      • Multiple intermediate hosts
        ▪ Snails, slugs, beetles

  o **External Parasites: Lice**
    ▪ Biting lice feed on dander & scale
    ▪ Irritation may lead to feather loss or over-preening

  o **External Parasites: Mites**
    ▪ *Scaly Leg Mite*
    ▪ *Northern Fowl Mite*
      • Entire life cycle on bird
    ▪ *Roost or Red Mite*
      • Feeds at night
      • Environmental control
    ▪ Presentation
      • Weak birds
      • Poor egg production
      • Pale carcass
      • Mortality (anemia)

**Avian Mycoplasmas**

  o Bacteria with no cell wall
    ▪ Limits antibiotic selection (penicillin won’t work)
    ▪ Fragile, easily destroyed
  o Strong host adaptation (strains are species specific)
    ▪ Chickens vs turkeys vs ducks vs pigs
  o 4 significant avian strains
    ▪ MG (*Mycoplasma gallisepticum*)
    ▪ MS (*Mycoplasma synoviae*)
    ▪ MM (*Mycoplasma meleagridis*)
    ▪ MI (*Mycoplasma iowae*)
  o Strains vary in pathogenicity

  ▪ **Mycoplasma gallisepticum (MG)**
    o Eradicated in commercial flocks but endemic in small flocks
      ▪ “auction market” disease
    o *Recovered carriers are sources*
    o Sustained by multiple ages
Transmission
- bird → bird
- hen → chick
- chick → chick
Sinusitis +/- E. coli complication = mortality

Diagnosis
- PCR
  - Detects presence of organism
  - May not detect latency
- Serology
  - ELISA screening of flock
  - HI confirmation of ELISA reactors
  - May cross react with other antigenic challenges (e.g. vaccines)
- Suggestive microscopic lesions
  - Sinus, trachea, lungs
- Specialized bacterial culture (limited antibiotic sensitivity testing)

Antibiotic Treatment of Avian Mycoplasmas
- Resolves clinical signs but does not eliminate infection & latency
- Reduces egg transmission, improves egg production
  - Tetracycline, tylosin
  - Penicillin ineffective
  - Baytril effective but not approved
- Continuous in feed or 1 week/month
  - May promote resistance!
- Egg transmission
  - Intermittent shedding (1-2%)
  - Narrow window of bacteremia → into egg as immunity wanes
- Egg treatments
  - Antibiotic egg dips/injections/heating
  - Previously used at commercial level to eradicate from breeders in combination with other strategies (no longer needed)
  - Difficult to control necessary conditions/dosages

Control is challenging
- Small flock strategies differ from commercial control strategies

Control as a NEGATIVE closed flock
- Buy TEST NEGATIVE replacements
- Commercial parent flocks → monitored & negative
  - +ve flocks are depopulated or eggs diverted
- All in-All out management
- NO multiple ages or sources
- Biosecurity to prevent introduction
- Active monitoring
  - NPIP oversees MG/MS/MM control program in US breeders
    - Blood testing (x300) pre-lay then every 60-90 days
- Depopulation?
  - Drastic and unnecessary

Control as a POSITIVE closed flock
- Treat clinical signs, then maintain a closed POSITIVE flock without bird/chick movements until full flock replacement with NEGATIVE birds
- Heritage genetics replacement plan
  - Treat breeders → eliminate positives
  - Isolate (off-site?) & test progeny → eliminate positives
• Depopulate parent flock
  • Control using vaccination not practical
    • Reduces clinical signs BUT
      • Variable efficacy
      • Local injection site reactions
      • Doesn’t prevent respiratory colonization & transmission of field strains
      • Live vaccine F-strain virulent to turkeys
      • Ts-11 vaccine needs liquid N

Coccidiosis
  o Pathogenic Eimeria species (9 in chicken, 4 in turkeys)
  o A disease of younger birds
    • gain protective immunity through low level exposure & intestinal cycling
  o Difficult to eradicate → reduce risk through management
  o Maintain good ground or litter conditions – dry
  o All-in / all-out management (allows clean-out)
  o Vaccination allows a “controlled” infection (live attenuated oocysts)
    • Sprayed onto chicks at hatchery or onto feed <1 week
  o Medication
    • Coccidiostat-medicated feed as an early preventative
      • Contraindicated with live vaccines
    • Treatment with Amprol

Egg Production Problems
  • Environment/Management
    o Poor egg production
      • Hens too young or too old
      • Short day length
      • Inadequate nest box space
      • Egg loss (theft, thin-shelled, egg-eater)
      • Excessive heat
      • Crowding & social stress
    o Poor egg quality
      • Egg size (too small, too large, double-yolks)
      • Shell quality (thin or no shells, rough shells, misshapen shells, wrinkles, ridges)
      • Loss of yolk pigmentation (dietary)
      • Soiled shells
  • Nutritional
    o Nutritional imbalance
    o Inadequate calcium or Vitamin D
    o Excessively low or high protein (influences egg size)
  • Metabolic
    o Obesity
    o Cloacal prolapses
    o Cage Layer Fatigue
      • Inadequate dietary calcium for egg shell
      • Depleted femoral medullary bone calcium reserves
  • Infectious
    o Bacteria or viruses that cause fever
      • Fowl Cholera (Pasteurella multocida)
    o Bacteria or viruses that infect the reproductive tract
      • Infectious Bronchitis Virus (IBV)
      • E. coli salpingitis (impacted oviduct)
      • Salmonella pullorum & Salmonella enteriditis
    o Yolk Peritonitis, Internal Lay
    o Any virus causing severe illness
What contributes to the health of poultry?

- Genetic Potential
- Housing
- Veterinary Care
- Husbandry
- Nutrition
- Biosecurity
- Vaccination
- Management Skills
- Clean Water
- Freedom to Express Natural Behaviours

Acquiring Chicks

- Ideally from an accredited hatchery
  - Accountability
  - Quality control programs
  - Salmonella monitored
  - Breeder flock health programs, including vaccination
  - Commercial parent flocks are given an array of vaccinations & boosters that stimulate antibodies for the chicks

- Hatchery Vaccinations (in ovo 18dg or day1):
  - Marek’s Disease (HVT, HVT + SB1, HVT + SB1 + Rispens)
  - Infectious Bronchitis
  - Coccidiosis
  - Vectored vaccines (vHVT + IBV, +ILT, +NDV)

Acquiring New Birds

- Only purchase new birds from reputable suppliers
- Be wary if a supplier trivializes the importance of disease control and disease records.
- If acquiring ready-to-lay pullets, make sure they are vaccinated against common diseases in your area (surveillance?). Determine ILT status.
- Bird swaps: many diseases are spread through trading of birds with unknown or questionable health history

“They will get a little bit of a cold and then get over it”

New Bird Management

- Segregate and monitor new birds for signs of disease before introducing them to the existing flock
- Isolate new birds for at least 21 days
- Isolate birds returning from shows or exhibits for at least 14 days
- Supplemental vitamins?

Coop Design Considerations

- What do the birds need?
  - Protect from adverse weather conditions
  - Adequate ventilation
  - Place to feed and water
  - Place to nest & dust bathe
- Protection from predators

- What do the humans need?
  - Access to feed and water the birds
  - Access to collect eggs
  - Access to check the birds
  - Access and ability to Clean out and Disinfect (C & D)

The FLAWS of good management

- F Feed
- L Light
- L Litter
- A Air
- W Water
- S Space
- S Sanitation
- S Security

Feed and Feeding

- Feed delivery systems
- Adequate feeder space
- Feeders inside coop (restricts access to wild birds)
- Commercial diets highly recommended
  - Will be diluted by scraps, scratch, foraging
- Consider a source with good quality control (HACCP feed mills).
  - Feed properly formulated for type & level of production
  - Accountability, traceability
- Shelf life (~3 months), moisture proof, rodent proof

Feeder Space – Rules of Thumb

- Chickens:
  - Layer Pullets, Broilers 1” (2.5 cm)
  - Layer 3” (7.5 cm)
- Turkey:
  - Poult 1”
  - Grower/Adult 3”
- Ducks: 6”

Light & Dark

- Light
  - Intensity 10 to 30 lux
  - Brighter for brooding to attract chicks to feed and water
  - Avoid shadows
  - Timing important for laying birds (16hr light: 8hr dark)
- Dark
  - Beneficial for all birds during growth, even during brooding
  - Darkness provides a period for rest and helps establish a normal day-night rhythm

Litter

- Type and quality (regional choices)
- Absorbent
  - Maintains proper moisture
- Wet litter
  - Ammonia
  - Promotes coccidiosis
  - Foot problems (erosions)
- Dry litter
  - Dust
• **Tips for Good Litter**
  - **New Litter:**
    - Dry but not necessarily kiln dried
    - Shavings or sawdust – tends to be damp, so allow time to preheat and dry if brooding chicks
    - Careful with straw → mold growth
  - **Ongoing maintenance:**
    - Keep it dry with good ventilation plus heat as necessary
    - If crusty, remove top layer and top dress new litter
    - Remove wet litter from water spills or leaks

**Air Quality**
- **Temperature**
  - Add heat or ventilate as necessary
  - Brooding – 30°C (86-90°F) air temperature at bird height
  - Rearing – drop by ~2.75°C (5°F) per week to 21°C (70°F) until fully feathered
  - Maintenance – 15-18" (60° to 65° F)
  - These are guidelines only!
  - WATCH YOUR BIRDS!
- **Ventilation, ventilation, ventilation**
  - *Add heat and ventilate rather than tightening up to conserve heat if ammonia is detectable or litter or manure is wet.*
- **Ammonia**
  - Detectable at 20 ppm; but even this or lower levels can have adverse health impacts
  - Ammonia eye burns
  - *If you can detect it, it is already too high*
- **Dust**
  - Promotes respiratory disease

**Water**
- Test regularly (at least once per year)
- Clean drinkers when necessary
- Clean and sanitize the water system at least after each flock
- Make sure all the birds can get to the water
  - Dominant hens may patrol
- Consider nipple drinkers
  - + heat tape wrap

**Space**
- Usually small flocks will have more than enough space
- In housed birds, lots of room also means more heat needs to be added
- Rule of thumb for chickens: 2 square feet per bird
- Always consider and evaluate functional space
- Housing for range birds during unfavourable weather – need enough for all the birds

**Sanitation**
- Sanitation physically reduces the amount of infectious agent
- Many methods of cleaning and disinfection
  - All require work and attention to detail
- Disinfectants: all have pros & cons
### Security
- Control rodents
- Predators
  - From air or ground
  - Fencing will help keep out larger predators

### Biosecurity
- Biosecurity is a collection of procedures and rules that are designed to minimize the introduction and transmission of disease-causing organisms.
  - Principles of Biosecurity
    - **ISOLATION** (physical separation)
      - *GOAL: To prevent contact with disease causing organisms*

---

<table>
<thead>
<tr>
<th>Compound</th>
<th>Disadvantage</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alcohols</td>
<td>Evaporates, no residual activity</td>
<td>BioSecur, RelyOn</td>
</tr>
<tr>
<td>Aldehydes</td>
<td>Toxic, irritating</td>
<td>Virocid, Formaline, ProFilm</td>
</tr>
<tr>
<td>Bleach (50ml/4 litres water)</td>
<td>Inactivated by organic material, stains</td>
<td>Biosentry</td>
</tr>
<tr>
<td>Iodine</td>
<td>Corrosive, irritating</td>
<td>Biodine, Barn Storm</td>
</tr>
<tr>
<td>Oxidizing Agents</td>
<td>Corrosive</td>
<td>HyperOx, Virkon</td>
</tr>
<tr>
<td>Phenols</td>
<td>Poor biodegradability, corrosive</td>
<td>1-Stroke Environ, TekTrol, Lysol</td>
</tr>
<tr>
<td>Quaternary Ammonium</td>
<td>Variable effectiveness, easily inactivated</td>
<td>ProQuat, Germex, Coverage 256</td>
</tr>
</tbody>
</table>
- **SANITATION** (disinfection)
  
  GOAL: To reduce the level of disease causing organisms

- **HEALTH MANAGEMENT**
  
  GOAL: promotion of disease resistance and the early detection & management of disease

### Treatment Concepts for Small Flock Poultry

- The principals of treatment of poultry are the same as those for any other species
- Strategies are the same but there will be differences in tactics, delivery systems and therapeutic availability
- For poultry, information gained from a diagnostic workup has value for subsequent flocks
  - The economics of treatment objectives determine where the real value lies
    - individual bird?
    - the flock?
    - future flocks?
- An accurate diagnosis is critical for the design of an appropriate treatment (and prevention) strategy
- Bacterial culture and antibiotic sensitivity testing provides guidance
- *For food producing birds, especially non-traditional birds, antibiotic selections are limited*

### Individual Medication

- Delivery
  - Oral
    - Gavage
    - Pills
  - Injectable
  - Topical
- Ensures appropriate dosage received

### Currently Available Antimicrobials – Poultry

<table>
<thead>
<tr>
<th>Drug Class</th>
<th>Products</th>
<th>Distribution</th>
<th>Spectrum</th>
<th>Human Use Category</th>
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</thead>
<tbody>
<tr>
<td>Penicillins</td>
<td>Penicillin</td>
<td>OTC</td>
<td>Gm +ve</td>
<td>II</td>
</tr>
<tr>
<td>Penicillins</td>
<td>Amoxicillin</td>
<td>Pr</td>
<td>Broad</td>
<td>II</td>
</tr>
<tr>
<td>Bacitracin</td>
<td>Bacitracin</td>
<td>OTC</td>
<td>Gm +ve</td>
<td>III</td>
</tr>
<tr>
<td>Flavenoid</td>
<td>Bambermycins (Flavomycin)</td>
<td>OTC</td>
<td>Gm +ve</td>
<td>IV</td>
</tr>
<tr>
<td>Cephalosporin</td>
<td>Ceftriaxone</td>
<td>Pr</td>
<td>Broad</td>
<td>I</td>
</tr>
<tr>
<td>Tetracyclines</td>
<td>Tetracycline, Chlorotet, Oxytet</td>
<td>OTC</td>
<td>Broad</td>
<td>III</td>
</tr>
<tr>
<td>Macrolides</td>
<td>Erythromycin, Tylosin</td>
<td>OTC</td>
<td>Mod. Broad</td>
<td>II</td>
</tr>
<tr>
<td>Phenicol</td>
<td>Florfenicol</td>
<td>Pr</td>
<td>Broad</td>
<td>III</td>
</tr>
<tr>
<td>Aminoglycosides</td>
<td>Gentamicin</td>
<td>Pr</td>
<td>Broad</td>
<td>II</td>
</tr>
<tr>
<td>Streptomycin</td>
<td>Streptomycin</td>
<td>OTC</td>
<td>Broad</td>
<td>II</td>
</tr>
<tr>
<td>Spectinomycin</td>
<td>Spectinomycin</td>
<td>OTC</td>
<td>Broad</td>
<td>?</td>
</tr>
<tr>
<td>Sulphas</td>
<td>Triple sulfa, sulfamethazine, sulfaquinax</td>
<td>OTC</td>
<td>Broad</td>
<td>III</td>
</tr>
<tr>
<td>Streptogramin</td>
<td>Virginiamycin</td>
<td>OTC</td>
<td>Gm +ve</td>
<td>II</td>
</tr>
<tr>
<td>Ionophores</td>
<td>Monensin, Salinomycin, Narasin, etc</td>
<td>OTC</td>
<td>Gm +ve poor</td>
<td>IV</td>
</tr>
</tbody>
</table>
• Mass medication
  ▪ Always consider flock-wide treatment
    ■ Helps birds in incubation period
  o Water
    ▪ Sick birds more likely to drink than to eat
    ▪ Can be influenced by pH & hardness
    ▪ Water consumption rate required to calculate dosage
    ▪ Some OTC formulations available
      ■ Fresh solution should be made at least daily and preferably twice daily
  o Feed
    ▪ Medicated feed must be ordered, vet script only
    ▪ Formulated or hand-mixed in
  o Extra-label or Off-label
    ▪ Any approved drug that is NOT used in accordance with the label directions
    ▪ Requires a prescription or be under the direction of a vet

• Medications & Withdrawal Times
  o Treatment options may be limited due to unknown safety, unknown efficacy, and unknown withdrawal times.
  o Minimizes drug residues in food
  o Withdrawal times are only for label indications & dose
  o Very few drugs with established withdrawal times for layers and specialty birds
  o Extra-label use +/-or combinations may alter withdrawal times
  o Vets are bound by “prudent use” guidelines
    ■ Can get withdrawal recommendations from CgFARAD (literature review)

• Special Consideration for Organics
  o Limited or restricted use of medications, rodenticides, pesticides & disinfectants
  o However, if there is a significant disease outbreak there are usually* provisions to allow treatment and extended withdrawal times may be necessary.

Vaccinating Poultry
  o To protect from field challenge and the negative effects of disease
  o To confer maternal immunity to chicks
  o Displace pathogenic field strains (Salmonella, Marek’s Disease)
  o Types of vaccines (strength & length of immunity varies)
    ▪ Attenuated live
    ▪ Killed with adjuvant
    ▪ Engineered or vectored
  o Vaccine delivery systems (individual → flock)
    ▪ Drinking water
    ▪ Aerosol Spray
    ▪ Wing web (pox)
    ▪ SQ injection
    ▪ Eye drop (ILT)
    ▪ In ovo (Marek’s Disease, Infectious Bronchitis)

• Small Flock Vaccination Challenges
  o Nothing is “routine”
  o 1,000 to 10,000 dose vials
  o Proper storage conditions
    ▪ Liquid N
    ▪ short shelf life following reconstitution
  o Live coccidial oocysts can be killed by medicated feed
  o Live vaccine virus can spread and attenuate
Once start → can’t stop (ILT), yearly boosters

- Opt for hatchery vaccinated chicks!
  - Marek’s Disease & coccidiosis

Disease Investigation: Diagnostics

- History: ask questions!!
  - Contact information & signalment
  - What? When? & describe
  - Recent bird movements?
  - “What do you think this is?”

- Physical Exam
  - Posture, demeanour
  - Temperature, RR (listen to breathing)
  - Assess coloring, weight for bird type & age, body condition, hydration
  - Feathering, presence of mites/lice
  - Check corneas, crop fill, palpate abdomen

- Blood Samples
  - Avian hematology & biochem panels
    - Most useful for inflammation
  - Serology for antibody levels
    - Weak tool for individual birds
    - Vaccine response or field exposure?
    - Results must be interpreted in association with vaccination history

Humane Euthanasia of Small Flock Poultry

- If an animal’s life is to be taken it is our responsibility to ensure it is done with the highest degree of respect, with emphasis on making the death as painless and distress free as possible
  - Euthanasia for slaughter may differ from euthanasia for culling

- Acceptable Methods
  - Pharmaceuticals (injected)
    - Barbiturates, chloral hydrate, T-61
  - Inhalant anesthetics
    - Ether, halothane, isofluorane
  - Carbon Monoxide
    - Highly effective but user danger (insidious)
    - Considered humane when pure (i.e. not vehicle exhaust)
  - Carbon Dioxide
    - Has rapid anesthetic & respiratory effect
  - Physical
    - Captive bolt, gunshot, electrocution, maceration, stunning, kill traps, exsanguination, pithing,
    - “Blow to head” or thoracic compression is not acceptable
  - Best for small flock poultry
    - Cervical dislocation
      - PRACTICE on a dead bird
      - NOT DUCKS or GEESE
    - Decapitation/guillotine

- Unacceptable methods
  - Suffocation
  - Drowning
  - Chemical poison or irritant
  - Hypothermia (freezing)
  - Euthanasia without stunning
  - Hot gas (tailpipe)
  - “Wringing”
Zoonotic Disease

- A disease of animals that can be transmitted to humans under natural conditions
- An infection or infestation shared in nature by humans and other animals; a disease of humans acquired from an animal source.
  - Despite high exposure rate to animal pathogens the incidence of human disease relatively low
  - Infectious organisms often highly adapted to a host species
  - Significant differences in avian and human physiology.
    - Body temperature
    - Cellular level
  - STRESS of transport, confinement or poor diet can enhance SHEDDING of organisms in birds

Changing Social Dynamics

- Human-animal interactions are more often and more intimate
  - urbanization
  - pet-friendly tenancy laws
  - institutionalization & pet therapy
    - jails, geriatric care, chronic care hospitals
  - captive breeding facilities (fewer imports) for retail pet avian species
  - urban chickens

Zoonotic Contact (birds in general)

- Poultry consumption (food-borne illness)
- Occupational exposure
  - Slaughterhouse workers, pet store
  - Wildlife rehabbers
  - Poultry agriculture
    - Vaccinators, catching crews, feed reps
- Breeding aviraries & pet shops: hand-reared pets
- Feral bird interactions
  - City park pigeons,
  - Backyard bird feeders
  - Hunters

Zoonotic Contact (transmission)

- Ingestion
  - Food-borne
  - Poor post-handling hygiene
- Inhalation (implies close proximity)
  - Aerosol dander, dried feces, respiratory droplets
- Ocular absorption
- Skin abrasions, bites, scratches

Zoonotic Diseases of Poultry

- Bacteria
- *Campylobacter jejuni*
- Salmonella species
- *Chlamydia psittaci*
- *Mycobacterium avium* complex (avian TB)
  - Risk to immunocompromised

- **Viruses**
  - Avian influenza (Influenza A viruses)
    - Direct & heavy exposure to highly pathogenic strains
    - Recently H7N9 LPAI in Asia
  - Newcastle disease
    - Conjunctivitis following ocular exposure

- **Mite Infestations**
  - *Ornithonyssus sylviarum* - northern fowl mite
  - *Dermanyssus gallinae* - red or roost mite
    - poultry houses, sparrow nests
    - biters (bird blood-feeders)
  - infest households, bite but don’t persist
  - irritating to the point of insanity!

**Food-borne Illness**

- **Salmonella & Campylobacter**
  - Account for majority of human food-borne illness related to poultry consumption (eggs, meat)
  - Gastroenteritis, fever, cramps, vomiting
  - Symptoms start 1-3 days post-infection and may last 4-7 days
  - Poultry are usually *asymptomatic* despite colonization of the intestinal tract
    - Co-evolution?

- **Campylobacter jejuni**
  - Poultry are non-clinical carriers; colonizes cecum & gall bladder → carcass contamination at processing
  - unsafe food handling practices → human illness

- **Salmonella sp.**
  - >2500 species of *Salmonella* in birds
  - asymptomatic carriers, stress induced intermittent shedding
  - antibiotic treatment promotes the carrier state
  - *S. typhimurium* - poultry, pigeons, wild birds & hunter cats
  - *S. enteriditis* – colonizes the chicken ovary → *internal* egg contamination leading to vertical transfer to chick

- *Salmonella & Campylobacter Control*

- **Prevention strategies farm to fork (commercial farms)**
  - National On-Farm Food Safety Program “Start Clean-Stay Clean”
  - Licensed egg grading removes cracked eggs, dirty eggs, meat/blood spots
  - Environmental testing
    - Re-direct eggs to breakers
    - Flock eradication
  - On-farm rodent control
  - SE vaccination
  - Carcass wash at processing

- **Prevention strategies for small flock poultry**
- Purchasing chicks from federally registered hatcheries
  - Best chance for Salmonella-free chicks through fluff & breeder testing
- BUT challenges exist
  - LIMITED or INADEQUATE or NONE
    - Egg grading
    - Environmental testing
    - On-farm rodent control
    - Se vaccination
  - VARIABLE
    - Egg sanitation practices
    - Egg storage conditions
    - From “never wash eggs” to inadequate washing practices
    - Use of floor eggs

2015 small flock outbreak of SE (Western Canada)
  - Outbreak disseminated from a mail-order hatchery in Alberta
    - Traced back to a positive breeder farm
    - Notices sent to all clients suggesting testing birds & environment
    - In BC, an estimated 1000 premises received birds
      - Ministry of Agriculture provided free sampling kits and testing to 600 requests;
        ~500 returned → 248 POSITIVE
  - What would you recommend?

- Reducing the Risk of Egg Transmitted Salmonella: EGG HANDLING
  - Make sure that the hens have adequate nest box space and that the nest boxes are kept clean.
  - Discourage hens from roosting in nest boxes
  - Gather eggs twice daily.
  - Floor eggs, especially those laid in litter should be discarded.
  - Eggs for consumption should be clean and free of manure, dirt, or feathers.
  - Do not reuse egg cartons

- Reducing the Risk of Egg Transmitted Salmonella: EGG WASHING
  - Wash only slightly dirtied eggs
  - Light sanding of minor dirt without washing
  - Clean fresh water +/- sanitizer
  - Make sure the wash water is 10˚C warmer than the egg temperature
    - Aim for water temp > 32˚C
    - Colder water causes the egg interior to shrink, drawing bacteria through the shell and into the interior.
  - Don’t let eggs sit in wash water. Pat dry eggs.
  - Use washed eggs first

- Reducing the Risk of Egg Transmitted Salmonella: EGG STORAGE
  - Cracked eggs should be discarded.
    - The risk of contamination of the egg by bacteria is up to 100 times higher in cracked than intact eggs.
  - Refrigerate eggs following a period of tempering and keep them refrigerated <7˚C.
    - Refrigerated eggs last 4X longer (2 months vs 2 weeks)
    - Store blunt end up
  - Candling will highlight eggs with small cracks, allowing the selection of only the best eggs
    - Also helps to discover inclusions that increase the risk for Salmonella
      - Meat spots, blood spots
Reportable Poultry Diseases

- Except for “reportable” or “notifiable” diseases all other diseases are “unregulated” and are a private issue between the vet and client. Disease control often relies on the “good neighbor” policy.
- Federally reportable diseases are those that Canada chooses to eradicate upon detection to maintain a “freedom from” status to PROTECT INTERNATIONAL TRADE. Compensation is available.
  - 4 immediately notifiable diseases in poultry
    - Highly pathogenic Avian Influenza (HPAI)
    - Velogenic Newcastle Disease (vNDV)
      - Catastrophic mortality
    - Salmonella pullorum & Salmonella gallinarum
      - Poor egg production, early chick mortality

Avian Influenza

- Natural hosts are birds
- Extreme variability in individual virus characteristics & pathogenicity
- High variability in how each species reacts to infection (seagulls vs. ducks vs. chickens vs. pigeons all differ) re: clinical signs & lesions
- Influenza viruses are simply... UNPREDICTABLE

- Avian Influenza: virus classifications
  - Subtyped by surface proteins reported as H-N combinations (H1N1, H5N2 etc.)
  - Identified by source information
    - A/CK/Penn/3779/97 (H7N2)
    - A/Seal/Mass/1/80 (H7N7)
    - A/ruddy turnstone/NJ/2000 (H5N3)
    - These are virus “names” and cannot be used to predict biological characteristics such as species susceptibility or pathogenicity
  - Genetic sequencing
    - Allows “relatedness” comparisons between individual viruses
      - Molecular epidemiology
        - North American vs. Eurasian strains
        - Can be used to define pathogenicity

Small Flock Risk of Avian Influenza

- Seasonal shedding of endemic waterfowl LPAI’s can contaminate range
- Waterfowl adapted viruses have “difficulty” infecting chicken cells
- LPAI infections in poultry are usually asymptomatic or mild
  - immunity develops and viral shedding stops
- Limited “mutational opportunity” in small flocks to mutate to HPAI
- Small flock poultry are susceptible to HPAIs
  - “heritage” breeds or organic chickens ARE NOT RESISTANT
  - Usually spillover from a commercial outbreak
  - Recent exception: H5N8/N1/N2/N5 can be transmitted by wild birds and has been reported in small flocks & commercial poultry in Europe, Africa, Middle East, India and
Asia (watch this one closely)

Small Flock Outreach

- Most small flock owners want the best for their poultry
- Frustrated by the lack of dedicated veterinary support
- Difficult to unify
- A communications challenge
- Provincial leads?

Knowing the disease challenges in small flocks can be used as a risk assessment tool for commercial poultry

Best Poultry Resources

- Universities (US & Canada)
  - Poultry Extension Services
- Provincial Ministries of Agriculture
  - Provincial Vet Labs
- Canadian Association of Poultry Veterinarians (CAPV)
- American Association of Avian Pathologists (AAAP)
- American College of Poultry Veterinarians (ACPV)
- Avoid the social media internet!
INTRODUCTION

Chronic bronchitis (CB) is the most common chronic respiratory impairment in dogs. The condition is defined as a chronic cough occurring for two consecutive months during the preceding year that is not attributable to another cause (e.g. neoplasia, congestive heart failure). This definition is based loosely on the definition of chronic bronchitis in humans, which is characterized by a well-defined cascade of clinical and histologic changes. The early changes are typically triggered by an inciting irritant stimulus (usually cigarette smoking), and include increases in airway mucus production, impairment of mucociliary clearance, and alterations in the local immune response. The cascade of events in dogs is similar to that seen in humans, and, if left untreated, results in a cycle of chronic inflammation, chronic cough, copious mucoid airway secretions, and decreased mucociliary clearance. This session will focus on the diagnostic approach and therapeutic management of the patient with chronic bronchitis.

EPIDEMIOLOGY

The average age at the time of diagnosis of chronic bronchitis is eight years of age or older, and many clients will report a several year history of intermittent cough. All breeds of dog can be affected. Higher incidence has been reported in West Highland White Terriers, Poodles, Cocker Spaniels, Pomeranians, and German Shorthair Pointers. Many dogs afflicted with CB are obese, but are typically otherwise healthy.

DIAGNOSTIC EVALUATION

Because chronic bronchitis is a diagnosis of exclusion, it is important to complete a full diagnostic evaluation for any dog presented with a chronic cough. Common differentials for chronic cough in dogs should include congestive heart failure, heartworm disease, pneumonia, neoplasia, infectious tracheobronchitis, and tracheal collapse. Less common conditions include foreign bodies, parasitic bronchitis, and primary ciliary dyskinesis. The initial laboratory evaluation of the chronic bronchitis dog is an important means of characterizing the overall health of the dog, and serves as a screen for other potential aggravating or inciting conditions. Evaluation for all animals should include CBC, serum biochemical profile, urinalysis, fecal flotation, Baermann analysis, and heartworm antigen test. Additional screening tests may include serologic testing for infectious diseases (fungal, viral, Rickettsial) and echocardiography, where indicated. Arterial blood gas analysis in the early stages of disease can be normal or may reveal mild hypoxemia due to ventilation/perfusion mismatch. Severely affected dogs may become hypercapnic due to ventilatory failure.

DIAGNOSTIC IMAGING

Thoracic radiographs typically show bronchial or peribronchial patterns, and can also reveal secondary conditions including pneumonia, bronchiectasis, and right-sided cardiac enlargement secondary to pulmonary hypertension (cor pulmonale).
ENDOSCOPIC ASSESSMENT

Bronchoscopic examination in cases of chronic bronchitis usually reveals non-specific mucosal erythema with a roughened, cobblestone, or granular appearance, and copious amounts of mucus. Large airways may appear relatively normal in dogs with significant small airway collapse and mucus trapping. The airways of severely affected dogs may have a pale appearance as a result of fibrosis. Bronchoscopy is also a valuable tool for evaluation of tracheal and bronchial collapse secondary to chondromalacia, mainstem bronchus collapse secondary to cardiomegaly, and other large airway abnormalities (e.g. bronchoesophageal fistula, foreign bodies, luminal tumors). Bronchomalacia is a particularly important co-morbidity, as the presence of distal airway collapse may be an indication for cough suppression, a treatment modality not typically utilized in chronic bronchitic patients.

DIAGNOSTIC SAMPLING

Bronchopulmonary cytology is typically characterized by non-degenerate neutrophilic inflammation. A smaller yet significant population of dogs will have cytology characterized by eosinophilic inflammation. Eosinophilic airway inflammation has historically been associated with hypersensitivity reactions, and has been erroneously used to make the distinction between “allergic” bronchitis and idiopathic chronic bronchitis. While eosinophils can certainly be recruited in allergic reactions, eosinophils can also be associated with neoplastic disease (e.g. lymphosarcoma), fungal infections, and systemic parasitism. Additionally, studies in humans suggest that acute exacerbations of chronic bronchitis can be associated with a transient inflammatory shift from neutrophils to eosinophils.

Most dogs with chronic bronchitis do not have active infection at the time of diagnosis. However, culture and sensitivity should always be performed on airway samples in the newly diagnosed bronchitic, in acute exacerbations of previously stable disease, or with radiographic evidence of bronchiectasis. Airway samples should be cultured for general aerobic and Mycoplasma culture. Anaerobic culture should also be considered in patients with bronchopneumonia.

THERAPY

CB is a slowly progressive condition for which there is no definitive cure. In the absence of intervention, the cycle of cough-induced inflammation and inflammation-induced cough will self-perpetuate. The three goals of therapeutic intervention in chronic bronchitis are, 1) do no further harm; 2) slow the progression of the histologic changes, and; 3) control the clinical signs. Because the inciting cause in dogs is rarely identified, the primary treatment of CB is based on controlling airway inflammation. Therapeutic tools commonly used in the control of CB include modulators of the inflammatory cascade, bronchodilators, anti-tussives, antimicrobials, environmental manipulation, and weight management. Surgical intervention may be indicated in management of severe secondary changes or exacerbating conditions (e.g. tracheal collapse, bronchiectasis).

Control of airway inflammation is the single most effective means of ameliorating the clinical signs of CB in humans. Most of the clinical signs of CB in humans and dogs (coughing, expiratory dysfunction, mucus hypersecretion) can be primarily or secondarily attributed to airway inflammation.

Oral corticosteroids are successful as a sole therapy in resolving the clinical signs in the majority of canine CB patients, and have historically been considered the mainstay of therapy in veterinary medicine. The short-acting oral corticosteroids (prednisone, prednisolone) should be started at anti-inflammatory doses (1-2mg/kg/day divided BID), and tapered to the lowest effective dose. The dose reductions should initially be every 1-2 weeks until physiologic doses (0.25-0.5mg/kg/day) are attained, at which point the dose should be maintained for 2-4 weeks. In the event of a relapse during the steroid taper, the previous dose at which
signs were controlled should be reinstituted, and the duration at that dose should be doubled prior to tapering.

Side effects of oral corticosteroids are many and known, and can be prohibitive in some cases. These include polydipsia, polyuria, inappropriate urination, lethargy, aggression, polyphagia, weight gain, and corticosteroid withdrawal syndrome (iatrogenic hypoadrenocorticism). In cases where control is achieved but adverse effects are intolerable, combination therapy with bronchodilators or anti-tussives (when appropriate) may provide control at lower steroid doses.

Inhaled corticosteroids are the standard of care in humans with CB. Advantages of inhaled steroids in humans include increased drug delivery to the affected site, significant reduction in systemic absorption, and reduction in prednisone-associated adverse effects. Anecdotal use of inhaled corticosteroids in dogs has been associated with improvement in clinical signs and reduction in prohibitive side effects. Fluticasone propionate (Flovent, GlaxoSmithKline) at 200, 225, or 250g dose can be administered to dogs using tidal breathing with a spacer device and facemask. At a dose of 2 puffs q12h, a single vial lasts approximately 30 days. Limitations to the usage of inhaled corticosteroids in veterinary medicine are many, and include a lack of controlled studies demonstrating efficacy, delivery, and reduced systemic absorption, drug delivery problems, patient compliance, and cost.

Other inflammation modulators that have been historically utilized in CB include antihistamines, mast cell stabilizers, anti-oxidants, Omega-3 fatty acids, NK-1 receptor antagonists, and mucolytics. With limited experimental data available and minimal anecdotal success reported, the regular use of the therapeutics cannot be fully advocated. In addition, some therapies may do more harm than good (e.g. anticholinergic effect of antihistamines).

The role of bronchoconstriction in canine CB remains unclear. Studies supporting the use of bronchodilators in CB have demonstrated improvements in objective data (pulmonary function tests) and subjective assessments (reduction in coughing, improvement in thoracic auscultation findings, owner perception of exercise tolerance). In addition, bronchodilators appear to have a steroid-sparing effect in some cases. Bronchodilators commonly used in canine CB are the methylxanthines and inhaled beta agonists (usually combined with inhaled corticosteroids).

The mechanism of methylxanthine bronchodilation was initially attributed to phosphodiesterase inhibition. It is now believed that both the mechanism of activity and mechanism of adverse effects of this class of drugs are mediated by adenosine inhibition. In addition, the methylxanthines possess additional effects, including anti-inflammatory effects (sPL-A2 inhibition) and stimulation of respiratory musculature (cAMP-mediated). Metabolism of the methylxanthines is variable, and, when combined with limited formulations, can make dosing quite difficult, particularly in small dogs. Because of patient-to-patient variability in therapeutic and toxic dosages, administration should start at the low end of recommended doses, and increase to effect. Theophylline should be administered in slow-release formulations, and should start at 5-10mg/kg PO BID. Frequency and duration can be increased to as high as 20mg/kg BID should clinical signs warrant and adverse effects allow. Because of an increased risk of methylxanthine toxicity, co-administration of theophylline with fluoroquinolones should be avoided when possible.

Studies both in people and in dogs suggest that the long-term use of beta-agonists may worsen airway inflammation, and has been associated with an increased risk of asthma-related death in human asthmatics. For that reason, beta-agonists are rarely used as mono-therapy. However, as mentioned above, some canine chronic bronchitics exhibit better symptom control when combining anti-inflammatory therapy with long-acting bronchodilation. In dogs who exhibit symptom improvement following a short trial of oral bronchodilators, a transition to long-acting inhaled bronchodilators may offer an option for better long-term
control. This can be accomplished by using combination inhaled products such as combination fluticasone propionate + salmeterol (Advair®). Combination products can be administered using the same spacer device and facemask as stand-alone inhaled corticosteroids.

This author typically reserves trials of bronchodilators once anti-inflammatory therapy has been stabilized. My experience is that canine patients exhibiting partial improvements with anti-inflammatory therapy, but still exhibiting cough or wheeze at higher tidal flows (e.g., post-exercise), may experience improvements with additive bronchodilator therapy.

As mentioned earlier, most dogs do not have active airway infections at the time of diagnosis. For this reason, antimicrobial therapy is rarely indicated in cases of CB. If indicated, therapeutic decisions should be based on culture and sensitivity results whenever possible. Empirical therapy selections (while awaiting C/S) should have coverage of common airway pathogens (Mycoplasma, Staphylococcus, Streptococcus, Pasteurella, Bordetella), and may include azalides, tetracyclines, fluoroquinolones, and macrolides. If radiographic evidence of pneumonia or bronchiectasis is present, spectrum of activity should be expanded to cover Gram-negative bacteria, anaerobes, and Mycoplasma.

The cough reflex in cases of CB is usually a protective and therapeutic mechanism. Coughing aids in the clearance of excess viscid secretions, and can minimize aspiration of irritants and organisms in the face of an impaired mucociliary transport system (secondary ciliary dyskinesis). For these reasons, most coughs associated with chronic bronchitis should not be directly suppressed, but should be alleviated by suppression of the inflammatory cascade. Some coughs may require direct suppression, and include dry, hacking paroxysmal coughs (which may also be associated with concurrent tracheal collapse), coughs associated with cough-induced syncope, and night coughing. The most effective anti-tussives for use in canine CB are the narcotic cough suppressants, including hydrocodone (0.25-1.0mg/kg PO q6-12h), codeine (1-2 mg/kg PO q6-12h), and butorphanol (0.25-1.0 mg/kg PO q6-12h). The most common side effect is sedation, which can be beneficial in some cases for alleviation of night coughing. Other side effects are rare at the anti-tussive doses, and include constipation and respiratory depression.

Well-controlled bronchitic patients who experience either acute or progressive worsening should be screened for late-stage complications of chronic bronchitis. Two common complications of chronic bronchitis include bronchiectasis and pulmonary hypertension. Bronchiectasis is a progressive, irreversible destruction of bronchial wall cartilage, leading to persistent dilation of the large airways. Airway wall destruction is caused by persistent inflammation. Leukocyte proteases can initiate airway wall destruction. Bacterial infection, a recurrent consequence of bronchiectasis, can worsen airway wall destruction through the generation of bacterial toxins. Bronchiectasis results in impaired mucociliary clearance, mucus stasis and accumulation, and subsequent airway obstruction. Ultimately, if left untreated, bronchiectasis will lead to ineffective cough and an increase in dead space ventilation. Because bronchiectasis is frequently complicated with bacterial infection, radiographic detection of ectatic airways in a poorly controlled bronchitic likely warrants antibiotic therapy. If possible, antimicrobial therapy should be based on airway culture and susceptibility. If this is not possible, empirical therapy should include spectrum for gram negative enterics, Staphylococcus spp., and Streptococcus spp. Because of the high likelihood of recurrent infections, some patients may require pulse or prophylactic antibiotic therapy, particularly in late stage disease.

Pulmonary hypertension is a multifactorial complication of chronic airway disease. The best understood component of pulmonary hypertension secondary to chronic airway disease is chronic alveolar hypoxia. Airway wall remodeling and airway collapse decrease ventilation in severely affected segments of lung. The resulting alveolar hypoxia causes local pulmonary arteriolar vasoconstriction (hypoxic pulmonary vasoconstriction). If this occurs locally, the result of this vasoconstriction is an improvement in matching of ventilation and blood flow and correction of hypoxemia. However, in severe, global airway disease, the
Vasoconstriction is extensive, resulting in an increase in pulmonary vascular resistance, and eventually, pulmonary hypertension. Symptoms of pulmonary hypertension are similar to those exhibited by bronchitics, including non-productive cough, lethargy, cyanosis, and exercise intolerance. The diagnosis of pulmonary hypertension can be difficult, as the gold standard diagnostic test is right heart catheterization. However, thoracic radiographs reveal right sided cardiomegaly (cor pulmonale) and pulmonary arterial enlargement. Estimates of elevated right sided pressures can occasionally be made using echocardiography. Severe cases may exhibit syncope or post-tussive collapse. Early pulmonary hypertension may be primarily a smooth muscular response, and as a result, may respond to pulmonary vasodilator therapy. Pharmacologic therapy is largely limited to sildenafil (2-5 mg/kg q8-12 hours), although the most effective pulmonary vasodilator is oxygen therapy.

Obesity is a major complicating factor in the management of canine CB cases. Obesity causes decreases in thoracic wall compliance, increased abdominal pressure on the diaphragm, and increases the work of breathing. Common locations for geriatric fat deposition in dogs include pericardium, mediastinum, and the cervical and thoracic subcutaneous space, all of which have the effect of decreasing ventilatory volume. Weight management alone can improve exercise tolerance and oxygenation. The most important factors in creating a successful weight management plan are client education and reasonable goals from the start. A weight-loss plan should try to target 1-3% weight loss per week, with the expectation that this rate will decrease as the dog approaches goal weight.

Environmental modifications can also help to decrease irritant, antigenic, or traumatic stimulation of the airways. Steps to decrease airborne pollutants can include elimination of cigarette smoking in the dog’s environment, elimination of aerosol and powder cleaners or deodorants, and using room or whole-house air filtration systems. Saline nebulization/airway humidification (bathroom “spa” therapy) can help to mobilize airway secretions. Use of harnesses instead of collars can also reduce direct stimulation of the large airways.

The overall prognosis for canine CB is poor. It is important to emphasize to clients that the goal of management of CB is reduction of clinical signs and slowing the progression of this condition. It is also important to emphasize to clients that no chronic cough is benign, and that earlier intervention can prevent or delay the onset of potentially life-threatening sequelae (e.g. syncope, hypoxemia, pulmonary hypertension) and irreversible structural changes (e.g. bronchiectasis, fibrosis).

REFERENCES:


INTRODUCTION

Canine and feline noses are incredibly important and often underappreciated organs. Normal nasal function is important in maintenance of olfactory function, but also plays a role in appetite and behavior in cats and dogs. Symptoms of nasal disease may be caused by any of a myriad of primary respiratory disorders or non-respiratory causes. Idiopathic chronic rhinitis is one of the most common chronic nasal disorders in dogs and cats. It is a diagnosis made by exclusion of other disorders, and usually requires chronic management. Other causes of chronic nasal symptoms include structural, mechanical, neoplastic, parasitic, infectious, and allergic disorders. The approach to chronic nasal disease should be designed to first identify or rule out primary nasal conditions with specific therapeutic options, then to secondarily manage chronic idiopathic inflammatory nasal conditions. Treatment of secondary infections and symptomatic therapy should be tertiary goals. The purpose of this session will be to review normal nasal structure and function, to use this information to highlight the potential effects of the loss of these functions, to provide the basis for a diagnostic and therapeutic approach to chronic nasal disease that can be largely accomplished without referral, and to provide insights into potential causes of treatment failure or relapses. The focus of these sessions will be on feline nasal disease, but many of these strategies will be applicable for canine chronic rhinitis as well.

OVERVIEW OF FELINE NASAL STRUCTURE AND FUNCTION

The nose is a structurally and functionally complex organ in the upper respiratory tract. It is the primary site of entry for inhaled air in the feline respiratory system, and therefore has many important and diverse functions. The nasal cavity functions to efficiently filter, warm, and humidify inhaled air before it enters the more delicate distal tracheobronchial airways and alveolar parenchyma of the lung. The nose serves as the principal organ for olfaction (the sense of smell). In addition to olfactory sensory function, the nasal cavity also serves as a sensory organ for the detection of irritants and noxious inhaled substances. The goals of therapy for chronic rhinitis are largely aimed at restoring nasal function, so an understanding of normal nasal structure and function is essential to developing therapeutic strategies.

Gross and Functional Anatomy of the Nose

The feline nasal airway is divided into two passages by the nasal septum. Each nasal passage extends from the nostrils to the nasopharynx. The nasopharynx is defined as the airway posterior to the termination of the nasal septum and proximal to the termination of the soft palate. Inhaled air flows through the nostril openings, or nares, into the vestibule, which is a slight dilatation just inside the nares and before the main chamber of the nose. Unlike the more distal main nasal chamber that is surrounded by bone, the nasal vestibule is surrounded primarily by more flexible cartilage. The luminal surface is lined by a squamous epithelium similar to that of external skin.

The rostral main chamber in cats has two turbinates, the maxilloturbinate (ventral nasal concha) and the nasoturbinate (dorsal nasal concha), that emanate medially from the lateral wall of the main chamber. The main chamber is divided by the maxilloturbinate and nasoturbinate into a dorsal, middle, and ventral meatus. These turbinates are lined by mucosa containing abundant capacitance vessels that are under autonomic
control. Dilation of these vessels causes engorgement of the erectile mucosal tissue, leading to nasal congestion. In the caudal main chamber, the ethmoturbinates emanate rostrally from the dorsal septum and the ethmoid bone. These turbinates are primarily lined by olfactory epithelium, and contribute to the acute olfactory capacity of cats. Feline turbinates have complex folding and branching patterns that serve to increase nasal airway surface area for filtration, absorption, conditioning, and clearance. These turbinates also divide the nasal airspace into multiple narrow, tortuous columns that are vulnerable to obstruction.

**Nasal Breathing**

The upper airways provide the majority of the resistance in the respiratory tree (up to 75% of the inspiratory resistance). While cats are technically capable of nasal breathing, many cats will maintain nasal breathing, even in the face of severe nasal obstruction or cardiopulmonary dysfunction. A switch to oral breathing in a cat usually suggests that there is a significant reduction in cardiopulmonary reserve. It is therefore very important that nasal airway patency be preserved in cats presenting with any type of respiratory dysfunction.

After passing through the nasal vestibule, inhaled air courses through the narrowest part of the entire respiratory tract, the nasal valve (ostium internum), into the main nasal chamber. All nasally inspired air passes through the main chamber into the nasopharyngeal meatus prior to passage through the laryngopharynx into the lower airways. The cross-sectional area of the nasal airways decreases by 4-5x between the main chamber and the nasopharynx, requiring an increase in flow rate to accommodate bulk flow. Because of this abrupt change in airway caliber at this site, even minor changes in the diameter of the nasopharyngeal airway lumen can have profound effects on inspiratory airflow and respiratory effort.

**Nasal Filtration and Mucociliary Clearance**

Most of the luminal surfaces of the nasal mucosa (with the exception of the most proximal regions of the nasal vestibule) are covered by mucus. Its physical and chemical properties are well suited for its role as an upper airway defense mechanism, filtering the inhaled air by trapping inhaled particles and certain gases or vapors. The mucus is produced by mucous (goblet) cells in the surface respiratory epithelium and subepithelial glands in the lamina propria. The synchronized beating of surface cilia propels the mucus and entrapped particulates from the main nasal chamber caudally to the nasopharyngeal meatus. With normal nasal function, secretions pass through a ring of nasal associated lymphoid tissue (NALT) surrounding the caudal aspect of the nasopharynx. Since this site is one of the first lines of defense against inhaled pathogens, dusts, and irritant gases, compromises in mucociliary clearance could lead to increased nasal infections and increased susceptibility to lower respiratory tract diseases. From this site, nasopharyngeal contents are cleared to the oropharynx, where they can be swallowed into the esophagus and cleared through the digestive tract or expectorated.

**Olfactory Function**

The ethmoturbinates lining the dorsal and caudal main chamber of the nasal cavity are lined by olfactory epithelium, a sensory neuroepithelium that is responsible for olfactory function. This epithelium contains bipolar neurons that pass through the cribriform plate and synapse directly in the olfactory bulb of the brain. The vomeronasal organ (VNO), a paired tube-like structure in the ventral nasal cavity, is an important sensory organ of the accessory olfactory system. The VNO is involved in the detection and processing of pheromones, and can influence behavior and appetite in cats.
DIAGNOSTIC APPROACH TO NASAL DISEASES

The initial approach to the patient with suspected nasal disease should be designed to verify that the patient’s clinical signs and symptoms are due to nasal disease, and to localize the problem to a specific region or regions in the respiratory tract. Once the condition has been localized as precisely as possible, specialized diagnostic procedures can be employed to obtain a diagnosis.

For many reasons (cost, time, patient stability, etc.), the typical approach to most patients involves using the least invasive diagnostic tests early, and reserving more invasive diagnostic tests for later in the diagnostic process. When time and resources permit, staging the diagnostic process to rule out differential diagnoses can provide a more complete assessment and facilitate better therapeutic recommendations.

Because many cases of nasal disease are eventually treated empirically or symptomatically, ruling out the conditions that will not respond to routine empirical therapeutic options should occur as early as possible. These include non-respiratory causes of nasal symptoms (alimentary, regurgitation and reflux, tooth root abscesses, hypertension, coagulopathies), and structural obstructive abnormalities (choanal atresia, choanal strictures, nasopharyngeal polyps, nasopharyngeal stenosis, caudal aberrant turbinates, nasal foreign bodies). Neoplastic causes are also important to rule out as early as possible, as these may be life-threatening or time-sensitive. Clients may be more inclined to treat nasal tumors if they’re diagnosed early in the course of disease. After structural and neoplastic differentials are ruled out or considered, evaluation for differentials for which specific treatments (curative or palliative) exist should occur. These include parasitic causes (mites, nematodes, Cuterebra), infectious causes (viral rhinitis, fungal rhinitis), and allergic rhinitis. The goal of this approach is to arrive at a diagnosis of idiopathic chronic rhinitis with the knowledge that non-respiratory, anatomic, neoplastic, and potentially curable causes have been ruled out as much as possible, in order to maximize the likelihood of treatment success for a condition that is difficult and frustrating to manage.

Minimum Database

The diagnostic approach to nasal disease starts with a CBC, serum chemistry, urinalysis, coagulation profile (in cases involving epistaxis), and a blood pressure measurement. In young cats for whom viral causes of rhinosinusitis +/- conjunctivitis are likely, collection of deep conjunctival, nasal, and tonsillar swabs for detection of Feline Herpesvirus, Feline Calicivirus, and Chlamyphila felis by PCR should be considered early in the diagnostic process. These tests are highly sensitive, particularly during active outbreaks, but may not detect latent viral infection during quiescent periods. Knowledge of this diagnosis early in cats can offer important prognostic information to clients.

Diagnostic Imaging

The three-dimensional evaluation offered by advanced imaging modalities (CT, MRI) is extremely valuable in the assessment of space-occupying or obstructive nasal diseases. In many cases, however, useful, and even diagnostic information can be obtained from a single, straight, intra-oral, dorso-ventral or ventro-dorsal radiograph. This view allows for the assessment of symmetry or asymmetry between the left and right nasal cavities, turbinate loss, mass effect, and nasal foreign bodies, and can help to limit differential diagnoses.

Rhinoscopy

Nasal endoscopy provides a detailed visual assessment of the nasal airspace and mucosal surfaces. Because of the strong nasal irritant reflex, rhinoscopy should only be performed under general anesthesia. If imaging studies are planned (CT, radiographs), they should be performed prior to rhinoscopy, as the presence of the endoscope causes hemorrhage, which can interfere with the interpretation of nasal imaging studies. Retroflex nasopharyngoscopy is typically performed with a flexible endoscope placed in the oropharynx and
flexed 180° over the soft palate, or using a rigid endoscope with a reverse offset (e.g., 120°), providing a visual assessment of the walls and airspace of the nasopharyngeal meatus, and the choanae. Endoscopic views of the nasopharynx and caudal nasal cavity can be enhanced by retracting the soft palate rostrally with a spay hook and directing the endoscope dorsally and rostrally into the nasopharynx. Anterior rhinoscopy is best performed with a rigid arthroscope or cystoscope directed through the nares into the left and right nasal cavities.

In addition to providing a direct visual assessment of the nasal cavity, rhinoscopy can also be used to guide diagnostic sampling of the nasal cavity (samples for cytology, histopathology), and can also be used for therapeutic intervention (e.g., nasal flushing).

**Diagnostic Nasal Sampling**

Because of the risk of potential complications, a lack of specialized equipment, and uncertainty about indications and interpretation of results, many practitioners consider diagnostic sampling of the respiratory system to be a daunting task. However, for many causes of nasal disease, there exist no pathognomonic hematologic or radiographic findings, making cytologic or histopathologic evidence of the condition the gold standard for diagnosis. With potential risks (chronic antibiotic therapy, steroidal or non-steroidal anti-inflammatory, immunosuppressives) and potential costs (inhalational therapy) associated with empirical and symptomatic therapy, a specific diagnosis should be sought whenever possible. There are several techniques available that will allow the safe and successful collection of samples for cytologic, histopathologic, and microbiological analysis in a general practice setting.

Diagnostic samples from the nasal cavity and nasopharyngeal meatus should always be collected from anesthetized patients. Patients should be intubated with a cuffed endotracheal tube. The oropharynx should be packed with gauze to prevent aspiration of nasal contents during sampling. Patients should be positioned with the head level, or with the nose tipped slightly downward to facilitate collection of flush samples. Sampling devices should not be inserted caudal to the level of the medial canthus of the eye, to prevent possible trauma to the cribriform plate.

Cytologic samples from the airway surface can be collected using nasal flushing, cytology brushes, swabs, or impression smears from harvested tissue samples. Since collection of surface samples is safe and relatively easy, one could argue that they are indicated in the evaluation of any case of airway obstruction, nasal discharge, sneezing, or reverse sneezing. The trap in collecting cytologic samples is the risk of overinterpreting results. In general, nasal cytology is poorly correlated with histopathology, and surface samples should not be submitted for microbiological culture. However, cytologic results can be reliable for certain conditions, including fungal rhinitis, allergic inflammation, and lymphoma [1,2].

Flushing can be performed in cats using a 5 Fr or 8 Fr red rubber catheter, or by inserting the luer tip of a 20 cc syringe directly into the nostril. In dogs, flushing should be performed with larger catheters, or can be performed with luer or catheter tip syringes. Each lavage should be performed using 5-10 ml of room temperature buffered saline. Lavage fluid should be collected from both nostrils. Intact pieces of tissue or debris can be gently squashed between two slides, while fluid samples can be submitted for direct and cytospin preparation. Samples collected using nasal swabs or cytology brushes can be gently rolled onto microscope slides, or dispersed in EDTA (purple top tube).

Tissue samples for histopathology and macerated tissue culture can be collected by several techniques. A coagulation profile, platelet count, and blood pressure should be obtained prior to collecting nasal biopsies. Because of the small size of the nostrils and nasal cavity, most nasal biopsies are collected blindly, but rhinoscopic and diagnostic imaging studies can be used to estimate the intranasal location of lesions. Arthroscopic biopsy forceps can be used to collect turbinate biopsies and biopsies of focal lesions. Traumatic flushing or traumatic catheterization techniques can yield tissue fragments that are of suitable size and quality for histopathology and culture. Samples are collected using a 5-7 Fr polypropylene catheter with the
tip cut at a 45° angle. Small, staggered notches can be cut into the length of larger catheters using a scalpel blade. 5 ml aliquots of buffered saline are repeatedly flushed into and aspirated from the nose while the catheter is raked along the nasal mucosa. Saline hydropulsion [3] is a less traumatic technique that can be useful for collecting samples from friable masses (e.g., necrotic tumors, fungal granulomas). A 20 cc Luer tip syringe filled with saline is placed directly into one nostril, while the contralateral nostril is digitally occluded. Saline is forcefully pulsed into the nasal cavity to dislodge tissue fragments, which can be collected in the draining lavage fluid or cleared from the oropharynx after removal of the gauze packing.

HOW I TREAT (AND RE-TREAT) FELINE IDIOPATHIC CHRONIC RHINITIS

Once structural and anatomic abnormalities have been addressed, and other treatable causes of chronic nasal disease have been ruled out, a management plan for chronic idiopathic rhinitis should be developed. When possible, nasal biopsies should be collected for histopathology and culture. In cases of idiopathic chronic rhinitis, nasal biopsies will exhibit a combination of lymphocytic, plasmacytic, and neutrophilic inflammation in the nasal mucosa, with no identified etiologic agents. In some cases, biopsies may also identify superficial bacterial colonizing the nasal mucosa, often associated with neutrophilic inflammation. After nasal biopsies have been completed, the nasal cavity should be vigorously flushed with room temperature saline to stop post-biopsy hemorrhage and to clear the nasal cavity of mucus and debris. This clearance of the nasal cavity helps to maximize the opportunity for a successful therapeutic plan, which should be treated in stages. In cases where nasal biopsies are not obtained, a therapeutic nasal flush should still be performed under general anesthesia prior to starting empirical therapy in order to help maximize the likelihood of treatment success.

Secondary bacterial infections should first be treated, ideally with antibiotic selection based on tissue culture and sensitivity profiles. Lipid soluble antibiotics that achieve high concentrations in airway lining fluid are good first choices. When antibiotic selection is not based on culture results, empirical choices should be broad spectrum, including activity against common nasal opportunistic pathogens including *Mycoplasma spp* and *Bordetella*. Macrolides and azalides (e.g., azithromycin), fluoroquinolones, and tetracyclines are good empirical choices, and should be employed for three weeks.

Once secondary infections have been treated, I determine whether or not the rhinitis is corticosteroid responsive with a trial of anti-inflammatory prednisone or prednisolone. It has been my experience that most cases of chronic rhinitis are in fact corticosteroid responsive if therapy is started in a properly prepared nasal airway (i.e., after nasal flush and antibiotic therapy). I typically start at 1-2 mg/kg/day in dogs, and 2-3 mg/kg/day in cats, for 14 days, with regular communication with the client during this time. Ideally, if patients are corticosteroid responsive, I recommend starting a training period with a facemask and spacer device, and implementing inhaled corticosteroid therapy as soon as possible. I start at high inhaled doses of fluticasone propionate (220 µg metered dose inhaler), 1 puff twice daily with a facemask and spacer device. Once inhalation therapy has comfortably been implemented, I start 25% dose reductions of the oral corticosteroid therapy every two weeks, with regular monitoring of clinical signs during the dose reduction. For patients who are prednisolone-responsive, but may not be candidates for inhalation therapy, I recommend a longer course of therapy at 1-2 mg/kg/day (up to 1 month), followed by a more gradual dose reduction (25% reduction every 3-4 weeks), with frequent monitoring for corticosteroid-induced side effects.

For cats with lymphoplasmacytic rhinitis who are not prednisolone-responsive, or for those patients who are prednisolone-intolerant or poor candidates for corticosteroid therapy, my second choice for anti-lymphocyte therapy is to implement lymphotoxic therapy with an alkylating agent. Chlorambucil is well tolerated in cats, and has anecdotally been associated with improvements in clinical signs in some cases of prednisone-unresponsive chronic rhinitis. I start at 2 mg orally per cat every 48 hours. Monitoring should include a CBC prior to the start of therapy, with follow-up CBCs at 7 days, 1 month, and then every 3 months.
A modality that may show promise as an option for anti-inflammatory therapy is the use of low-dose radiation therapy. The radiation sensitivity of B-cells, T-helper cells, and cytotoxic T-cells makes low dose radiation therapy a potential option for patients exhibiting either corticosteroid resistance or corticosteroid intolerance [4]. Protocols currently being employed on an experimental basis in dogs typically involve low daily doses (3-4 Gy) and low total doses (15-30 Gy). Anecdotal reports suggest that these protocols are associated with a low rate of acute and late toxicity, and partial or complete resolution of clinical signs for periods of over one year in some cases. To date, most of the evidence in support of radiation therapy for chronic rhinitis has been anecdotal and testimonial. Therefore, well-designed, controlled studies need to be conducted in this area before widespread recommendations can be made.

In cats with ongoing inflammation or severe turbinate loss, recurrent bacterial infections are an unfortunate but expected complication. These will typically manifest as an acute change in the volume and quality of nasal discharge, and a new onset of sneezing in a previously controlled patient. Since it may not be practical to biopsy and culture the nasal cavity with each flare, many recurrent infections will require empirical therapy. These infections should be treated based on the frequency of their recurrence. Infrequent infections can be treated as they occur. More frequent infections may respond to prophylactic antibiotic therapy (1 week per month). As a last result for frequently recurring infections, chronic antibiotic therapy protocols can be employed. Macrolide/azalide antibiotics can be employed chronically using every-other-day or every-third-day protocols (e.g., azithromycin, 5-10 mg/kg every 48-72 hours).

Symptomatic therapy may also be an important management component of idiopathic chronic rhinitis. Most techniques are designed to facilitate nasal mucociliary clearance. Commercially available pediatric saline drops can be directly instilled in the nasal cavity to keep nasal secretions fluid and enhance clearance to the nasopharynx. Owners can instill 1 drop in each nostril once daily, using a dropper or syringe. Topical decongestants are vasoconstrictors that act on the capacitance vessels in the turbinates. These can shrink the nasal mucosa, open the ostia to the frontal sinuses, and facilitate sinus and nasal cavity drainage. Phenylephrine (0.125%) or oxymetazoline (diluted to 0.025%) can be administered at a rate of 1 drop in each nostril once daily. Topical decongestants should not be used for more than three consecutive days, as this can cause a rebound vasodilation and nasal congestion. For cats experiencing severe nasal airway obstruction, intermittent nasal flushing under anesthesia can help to clear the nasal airways, facilitate mucociliary clearance, and enhance the efficacy of anti-inflammatory and antimicrobial therapy. Neurokinin-1 (NK-1) receptor agonists (e.g., substance P) are believed to contribute to nasal inflammation and nasal symptoms through neutrally-mediated pathways. While no controlled studies have conclusively supported a role for NK-1 receptor antagonists, maropitant (Cerenia) at 1 mg/kg/day has been anecdotally associated with improvement in nasal-localizing symptoms (e.g., sneezing). Chronic use of NK-1 receptor antagonists should be avoided, as this can contribute to accumulation of substance P, leading to neurologic symptoms in cats. Finally, analgesics should be considered in cases with bony involvement (invasive nasal tumors, rhinitis with osteomyelitis), or to ameliorate post-biopsy pain. Maropitant can be used to provide a mild analgesic effect at the dose listed above. The injectable form of buprenorphine can be administered sublingually at 5-10 µg/kg up to every 6 hours. Tramadol can also be used in cats for nasal or bone pain at 2-4 mg/kg BID.

SUMMARY

While referral to specialty practice will always be an option, thorough diagnostic evaluation for chronic nasal disease can be done in most practice settings, and may not always require specialized diagnostics. Practitioners should be comfortable recognizing opportunities to provide definitive therapy, empirical therapy, and symptomatic therapy for these patients.

REFERENCES

OVERVIEW

An important step in the pathogenesis of Canine Infectious Respiratory Disease (CIRD) involves the colonization of the upper airway mucosa by primary respiratory pathogens. In the susceptible host and the proper environment, these primary respiratory pathogens are capable of bypassing the mechanical barriers, evading the innate immune response, and disrupting mucociliary clearance, thereby allowing both primary and secondary bacterial and viral pathogens to colonize and infect the upper and lower respiratory tract. An understanding of the complex relationship between these primary respiratory pathogens and the respiratory immune system is crucial to the development of strategies to effectively treat and prevent CIRD. The objective of this presentation is to provide an update on our current understanding of the interactions between the canine immune system and the classical and emerging respiratory pathogens underlying this disease complex. We will first review the components of the intact mechanical, innate, and adaptive canine respiratory immune system in health. We will discuss the mechanisms by which primary respiratory pathogens, like *Bordetella bronchiseptica*, and *Mycoplasma cynos*, can evade or bypass the immune system. We will discuss the potential role of emerging pathogens (e.g., Canine Influenza Virus, Canine Respiratory Coronavirus) in disease pathogenesis. Finally, we will discuss both immunologic (vaccination, natural immunity) and non-immunologic (premise control, environmental management) strategies to effectively prevent CIRD.

RESPIRATORY DEFENSE SYSTEM

The normal, intact respiratory defense system can be divided into three distinct levels. The first level consists of mechanical barriers, including the mucus and epithelial lining fluid that overlies the airway epithelium. These mechanical barriers serve to prevent inhaled pathogens from attaching to the epithelial surface, thereby inhibiting their ability to infect the host. If inhaled pathogens are able to bypass the mechanical barriers and engage the epithelial surface, the second level of the respiratory defense system, the innate immune system, can become activated. The innate immune response is triggered by binding of pathogen-associated-molecular-patterns (PAMPs) on the surface of the pathogen by receptors on the epithelial cell surface. Binding of these receptors stimulates the release of preformed mediators, including interferons, enzymes, and chemoattractant molecules, which function to inhibit infection and prime or amplify adaptive immunity. Thus, the innate immune response serves as an important bridge toward the development of the adaptive immune response. The adaptive immune system involves antigen presentation to T-helper lymphocytes in mucosal-associated lymphoid tissues (MALTs), which subsequently drive development of local IgA and systemic IgG-producing plasma cells and antigen-specific cytotoxic T-lymphocytes (CTL). It is the adaptive immune response that provides both immunologic specificity and long-term immunologic memory.

Mechanical barriers and the innate immune response are present in all patients, but may vary in their efficacy from patient to patient as a result of concurrent respiratory disease processes. As an example, dogs with chronic bronchitis may have airway epithelial hyperplasia and squamous metaplasia, along with alterations of mucus production and mucus quality that together impair normal mucociliary clearance functions. While these functional aspects of the respiratory immune system do not confer immunologic specificity or memory, they both help to stimulate or amplify the adaptive immune system. The barrier lining the nasal airways, the trachea, and the first several generations of bronchi is a ciliated respiratory epithelium. This airway surface...
possesses motile cilia that effectively drag the overlying mucus blanket, along with any trapped particles or organisms from the inhaled air, directly in contact with mucosal-associated lymphoid tissues (MALTs) in the caudal aspect of the nasal cavity and in the upper portions of the tracheobronchial tree. Activation of the innate immune response recruits antigen presenting cells to the site of initial pathogen contact, which can mediate the early steps necessary for immunoglobulin production and CTL generation.

The nasal cavity, trachea, and bronchi are the principal sites for pathogen colonization and infection in dogs with CIRD. Activation of MALTs in these same regions is an important first step in the generation of an adaptive immune response. These MALTs possess follicles of B-cells surrounded by parafollicular T-cell zones, and are populated by antigen presenting cells (APCs). The APCs on the airway present CIRD antigens to T-cells, which then direct the B-cells to produce immunoglobulin A (IgA) and immunoglobulin G (IgG). IgA and IgG confer mucosal immunity through the processes of immune exclusion and immune elimination. IgA produced locally by MALTs is translocated through epithelial cells and resides on the airway surface in the epithelial lining fluid. IgA is very effective in binding airborne pathogens and inhibiting pathogen attachment to the airway surface (immune exclusion), but is not an effective opsonin, does not activate complement, and is relatively short-lived. IgG is principally a circulating (or humoral) immunoglobulin, but can be recruited to the airway mucosal surfaces during following pathogen colonization. IgG is a potent opsonin, is a potent activator of complement (immune elimination), and is produced by plasma cells with longer half-lives. Both IgA and IgG are important in mediating immune responses against extracellular pathogens. CTLs, which recognize processed intracellular antigens in the context of MHC-I, are important in mediating immunity against intracellular pathogens. All three work together in order to provide variable degrees of protection against CIRD infection in either naturally infected or effectively vaccinated patients.

**PATHOGENESIS OF CIRD**

CIRD, or “kennel cough,” or “canine shipping fever,” is a complex, highly contagious respiratory infection that is spread primarily through aerosolized respiratory secretions. Aerosolized viral and bacterial pathogens in the complex initially colonize the respiratory epithelium lining the nasal cavity, trachea, and bronchi. Because most dogs are infected through exposure to aerosolized secretions, there is often a predictable temporal relationship between exposure to other dogs and the onset of clinical symptoms (anywhere from 3-10 days in most cases). The communicable nature of CIRD makes it a frequent cause of morbidity in shelters, kennels, boarding facilities, “doggie” day care centers, and veterinary clinics. While most dogs are infected through direct exposure to aerosolized respiratory secretions, dogs can also be exposed to CIRD pathogens indirectly through fomites. Potential fomites in CIRD transmission include improperly sanitized hospital surfaces (exam tables, cages, scales, waiting areas), toys, endotracheal tubes, medical equipment, and hospital personnel or personal protective equipment (e.g., contaminated scrubs).

The primary pathogens in the CIRD complex colonize the ciliated respiratory epithelium in the upper airway. Once they gain access to the tissues, many of these pathogens possess virulence factors that allow them to disrupt mucociliary clearance, often by altering ciliary function or by causing injury to ciliated cells. Mucociliary clearance dysfunction subsequently allows other pathogenic or opportunistic bacteria and viruses to colonize the airway surface, maintain longer residence time, and complicate the infection. Many viral and bacterial principal pathogens are known to disrupt ciliary function or morphology (e.g., Canine parainfluenza virus), and many others are suspected to do so on the basis of their possession of known virulence factors (e.g., *Mycoplasma cynos*).

“Old Friends”--Established Pathogens in CIRD

**Canine parainfluenza virus and canine adenovirus.**

Canine parainfluenza virus is the most commonly isolated pathogen in the CIRD complex. Several vaccines are available for prevention of clinical infection. Appropriately vaccinated dogs will develop high titers against canine parainfluenza virus that may persist for at least three years, and vaccination is effective in minimizing or preventing the clinical signs associated with infection. However, vaccinated dogs subsequently
exposed to canine parainfluenza virus may transiently shed virus, possibly leading to false positive results in PCR panels. Another viral pathogen, canine adenovirus-2, is occasionally isolated as a co-factor in multiple pathogen infections, but rarely causes overt respiratory symptoms as a single agent infection. Vaccination for canine adenovirus-2 is included as a core vaccine for prevention of canine infectious hepatitis (CAV-1). Both canine adenovirus and canine parainfluenza virus typically cause mild, self-limiting respiratory infections exhibiting minimal systemic symptoms[1].

**Canine distemper virus.**

Canine distemper virus is another important cause of CIRD. Initial symptoms in infected dogs are frequently localized to the upper and lower respiratory tract, and in many cases, symptoms are limited to the respiratory tract (cough, nasal discharge, fever). In systemically infected dogs, multiple organs may become infected. Characteristic lesions in systemically infected dogs include ocular (periocular dermatitis, conjunctivitis, keratitis), dermal (footpad and nasal hyperkeratosis), and neurologic (acute and/or progressive myeloencephalitis) manifestations. Canine distemper virus is unique among viral causes of CIRD due to its longer incubation period. Dogs infected with canine distemper may exhibit symptoms several weeks after exposure, while dogs infected with canine parainfluenza virus of canine adenovirus may exhibit symptoms within a few days after exposure[2].

**Bordetella bronchiseptica.**

*Bordetella bronchiseptica*, an aerobic, gram negative bacterium, is the most commonly isolated bacterial cause of CIRD. There are hundreds of isolates of *Bordetella bronchiseptica* in the environment with variable virulence, pathogenicity, and host distribution. Unlike many of the other causes of CIRD, which tend to be relatively host-adapted, *Bordetella bronchiseptica* is uniquely able to infect dogs, people, as well as other mammals. Because of this, dogs naturally infected with *Bordetella bronchiseptica* or recently vaccinated with a modified live Bordetella bronchiseptica vaccine may pose a zoonotic risk to immunocompromised people, although the risk is likely very low, and evidence supporting dog-to-human transmission is very weak and largely circumstantial.

Among the virulence factors possessed by *Bordetella bronchiseptica* are filamentous hemagglutinin and fimbriae, which facilitate bacterial attachment to cilia on respiratory epithelial cells, and a type-3 secretion mechanism that allows for translocation of cytotoxins that mediate ciliary stasis into the cytosol of airway epithelial cells[3, 4].

**Emerging Pathogens in CIRD**

**Canine respiratory coronavirus.**

Canine respiratory coronavirus is an enveloped RNA virus that was first associated with acute respiratory infections in shelter dogs in England over ten years ago. Canine respiratory coronavirus is distinct from the canine enteric coronavirus, and immunity to the enteric form does not provide cross-protection against the respiratory virus. Canine respiratory coronavirus infection is usually associated with mild, self-limiting respiratory disease, but has been associated with outbreaks of severe respiratory disease in shelters and boarding facilities. Exposure of dogs to canine respiratory coronavirus is widespread in North America, with >50% of dogs demonstrating serum antibodies reactive against the virus. Canine respiratory coronavirus may facilitate infection with other CIRD pathogen, as up to 12% of dogs infected with this virus are co-infected with canine parainfluenza virus, canine influenza virus, *Mycoplasma spp.*, and *Bordetella bronchiseptica*[5, 6].
Canine influenza virus.

Up until December 2014, Canine Influenza Virus infection was due to a H3N8 Canine Influenza A virus that emerged in the canine population following a mutation of the equine influenza virus. This mutation allowed horse-to-dog transmission of the virus. Infections in dogs have now been documented in over 40 states, as well as in Canada, Australia and England. In 2015, the first cases of infection by a novel H3N2 Canine Influenza Virus were reported in Chicago. Since those initial reports, cases of H3N2 Canine Influenza have now been reported in over 30 states and Canada, with “hotspots” of ongoing infection occurring in Chicago, Atlanta, and the northeastern United States. To date, the H3N8 and H3N2 Canine Influenza Viruses are the only two known to be capable of infecting and causing disease in dogs and capable of being transmitted from an infected dog to a vulnerable dog. The H3N2 virus has also been reported to be transmitted from dogs to cats, and may cause clinical disease in vulnerable cats.

Like canine respiratory coronavirus, canine influenza viruses exhibit a short latency period of ~24 hours. Viral replication and shedding occurs within one day of exposure in infected dogs. The incubation period of canine influenza virus is longer (2-5 days), resulting in 1-4 day period during which dogs shedding virus may be asymptomatic. For this reason, the viruses are very effectively transmitted between infected and vulnerable dogs in overcrowded environments. The period of peak viral shedding of H3N8 canine influenza virus is short for clinically affected dogs (<7 days). For that reason, PCR testing may yield false negative results for H3N8 in dogs exhibiting symptoms for several weeks. In these patients, paired serologic antibody titers against canine influenza are a more appropriate diagnostic method [7, 8]. Conversely, the period of viral shedding for dogs infected with the H3N2 Canine Influenza virus can be as long as 4 weeks.

Mycoplasma cynos

Mycoplasmas are among the primary pathogens implicated in Atypical Respiratory Infections in people. These refer to bacterial respiratory infections associated with milder, chronic respiratory disease and prolonged infections. Mycoplasma canis and M. felis are common inhabitants of the laryngeal mucosa and nasopharynx of healthy dogs and cats. Their role as pathogens in CIRD is unclear. However, Mycoplasma cynos has been associated with pneumonia and lower respiratory infections in puppies, and has been documented to experimentally induce respiratory disease in dogs. Mycoplasmas are fastidious organisms and are very difficult to culture, but PCR techniques can be very useful to document Mycoplasma cynos infections if appropriate samples are collected[9].

Streptococcus equi subspecies zooepidemicus.

Streptococcus equi subsp. zooepidemicus, or “strep zoo”, is a β-hemolytic Streptococcal species that has been recently associated with widespread outbreaks of severe, often fatal, respiratory disease in shelter dogs. Dogs infected with Streptococcus equi initially resemble dogs infected with other components of the CIRD complex. However, many infected dogs will progress to develop hemorrhagic pneumonia. The later manifestations of disease are similar to those exhibited by people suffering from toxic shock syndrome, suggesting that the clinical aspects of disease may be associated with Streptococcal toxins. Reported mortality rates in infected dogs during outbreaks are as high as 50%. The risk of infection in dogs in communal settings is considerable, while reports of Streptococcus equi infections in individual pet dogs are rare. Streptococcus equi can be isolated from infected, affected dogs as well as from healthy horses, and both species pose a potential zoonotic risk to people. Cases of both dog-to-human and horse-to-human transmission of Streptococcus equi have been reported[10-12].

Canine herpesvirus

Canine herpesvirus is one causative agent of a rapidly progressive, often fatal condition in neonatal dogs known as “fading puppy syndrome.” In young puppies, canine herperviral infections can cause ocular, dermal, and genital lesions along with a severe interstitial pneumonia. Affected puppies may die within 24 hours of the onset of symptoms, and some dogs may die acutely with no overt symptoms. The mortality rate in
puppies 1-3 months old has been reported as high as 100%, while deaths are rare in puppies older than six months old. Latently infected adult female dogs are believed to be the primary source of infection in young puppies.

Canine herpessviral infections in adult dogs may manifest as symptoms consistent with CIRD, including nasal discharge and a non-productive cough, along with ocular and genital lesions similar to those exhibited in infected puppies. Symptoms in adult dogs are usually self-limiting, requiring no intervention or general supportive care. Canine herpesvirus can enter a latency period in infected adult dogs, and may exhibit recrudescence later in life, although the these events are typically less severe[13].

Other emerging pathogens potentially associated with CIRD

Mammalian reoviruses, canine minute viruses, and canine pneumoviruses have all been isolated from dogs exhibiting upper and lower respiratory symptoms. Mammalian reoviruses are capable of infecting all mammals, and have been isolated from the respiratory and gastrointestinal tracts of dogs exhibiting co-morbid respiratory and gastrointestinal symptoms. Canine minute viruses are another etiologic agent in “fading puppy syndrome,” and can cause manifestations in puppies similar to those caused by canine herpesvirus. Surviving littermates may exhibit upper respiratory localizing symptoms. Canine pneumovirus has been isolated from pharyngeal and nasal specimens of CIRD-affected dogs, predominantly as co-infections with canine parainfluenza virus, canine influenza virus, and canine respiratory coronavirus. While each of these agents has at least been temporally associated with respiratory disease in dogs, their roles as primary pathogens in the CIRD complex remains uncertain [14, 15].

CLINICAL PRESENTATION OF CIRD

Most dogs infected with CIRD pathogens exhibit “uncomplicated” infections, characterized by mild to moderate, upper respiratory localizing symptoms. Many will experience sneezing and nasal discharge as an early symptom, followed often by an acute onset of a non-productive, “honking” cough. Coughing is usually paroxysmal, with episodes lasting for minutes in severe cases. There is often a known or suspected exposure event associated with direct contact with other dogs or indirect contact with fomites within 3-10 days of the onset of symptoms. Despite what can often be severe upper airway localizing signs, these dogs are typically otherwise healthy, and infections are usually self-limiting (with the notable exceptions of canine distemper virus and canine influenza virus). In a subset of these dogs, an initially “uncomplicated” infection can become “complicated”, and associated with more mucoid or purulent nasal and pulmonary secretions, fever, anorexia, productive coughing, and occasionally respiratory distress. “Complicated” infections are often seen in puppies, immunocompromised patients (either through concurrent disease processes or as a result of drug therapy), and dogs with concurrent respiratory disease (e.g., chronic bronchitis with bronchiectasis). Severely affected patients can die as a result of bronchopneumonia secondary to CIRD infections, demonstrating the importance of airway imaging early in the course of complicated infections.

DIAGNOSIS

Dogs with uncomplicated cases of CIRD can often be managed without extensive diagnostic testing, on the basis of their signalment, history, and physical examination findings. Documentation of the presence of the pathogen along with an immune response to the pathogen are necessary to confirm diagnosis, however, this is rarely performed in uncomplicated cases. Diagnostic testing may be indicated in uncomplicated cases which fail to respond to appropriate therapy, or in patients in whom the risk of progression to a complicated case is high (e.g., dogs with chronic lower airway disease).

Bacterial components can be documented via airway cytology and culture of respiratory secretions obtained via trans-tracheal or endotracheal lavage. Both bacterial and viral components of the complex can be documented through the use of respiratory polymerase chain reaction (PCR) panels. Pathogens in these
panels include *Bordetella bronchiseptica*, *Mycoplasma cynos*, *Streptococcus equi*, canine adenovirus, canine distemper virus, canine herpes virus, canine parainfluenza virus, canine pneumovirus, canine respiratory coronavirus, and H1N1 influenza virus. It is important to note that not all pathogens are offered by all vendors. Collection of samples for PCR should include DEEP nasal, pharyngeal, and tonsillar swabs, as well as fluid or tissue samples from respiratory tract sampling, and serum samples. Interpretation of PCR results requires some degree of scrutiny, and should be considered in light of the patient.

Dogs with complicated cases should have respiratory sampling done when possible, as these dogs may be infected with CIRD pathogens or opportunistic pathogens. Systemic assessments should include complete blood counts and thoracic imaging.

**TREATMENT AND PREVENTION OF CIRD**

Dogs with uncomplicated cases of CIRD may require no therapy at all, as many cases are self-limiting within a few days to a couple of weeks. Dogs presenting with non-productive coughing may respond favorably to cough suppression both for patient comfort and to prevent cough-induced airway damage. Antibiotic therapy can be useful in cases caused or complicated by bacterial CIRD pathogens, both by shortening the duration of illness and decreasing the shedding of bacteria in the environment. In these cases, antibiotic selection should include drugs with an antimicrobial spectrum including gram negative bacteria (*Bordetella bronchiseptica*) and mollicutes (*Mycoplasma cynos*), and should be maintained for two weeks. Examples of these include tetracyclines, fluoroquinolones, azalides, and macrolides.

Dogs with CIRD cases complicated with bronchopneumonia should be treated with broad spectrum, lipid soluble antibiotics to cover both for CIRD pathogens as well as opportunistic bacteria. The typical duration of therapy for dogs with complicated infections is 6-8 weeks, or 2 weeks beyond radiographic resolution of alveolar infiltrates. Intravenous fluids, saline nebulization, couple, and low-intensity exercise should all be employed where possible in order to generate and maintain a productive cough. Cough suppressants should not be employed in dogs with bronchopneumonia, as coughing is an important clearance mechanism for the lower airways.

Maternal antibodies provide an important source of protection for young puppies against CIRD pathogens during the first 6-8 weeks of life, after which point, antibody titers begin to wane gradually. Circulating maternal antibodies directed against canine parainfluenza and canine adenovirus may interfere with the efficacy of parenterally-administered vaccines in young puppies. For this reason, core vaccination for canine parainfluenza virus and canine adenovirus should begin near the expected time of maternal antibody waning (6-8 weeks), and be boosted until ~12 weeks of age or beyond in order to ensure vaccine efficacy.

Vaccines are currently available for only six of the potential pathogens in the CIRD complex (canine parainfluenza virus, canine adenovirus, canine distemper virus, canine influenza viruses H3N8 and H3N2, and *Bordetella bronchiseptica*). Vaccination against canine parainfluenza virus, canine distemper virus, and canine adenovirus are considered part of the core canine vaccine protocols. While it is not technically considered a “core” vaccine, this author routinely recommends vaccination of puppies and adults to provide protection against *Bordetella bronchiseptica*. Vaccination against canine influenza virus is frequently recommend for “high risk” dogs (e.g., frequent boarders, dogs participating in day care, participants in dog shows, travelers), but as the reports of canine influenza become more widespread, recommendations for vaccination against canine influenza virus may be considered for any dog spending time commingled with other dogs.
References


Antimicrobial stewardship in companion animals: Welcome to a whole new era

Untreatable bacterial infections (“the post-antibiotic era”) are far more likely to emerge in companion animals than in food animals, and have already arrived in the form of the multi-drug resistant (MDR) ESKAPE pathogens, the scourge of hospitals. These highly resistant, and in some cases hospital-adapted pathogens, are *Enterococcus faecium*, methicillin-resistant *Staphylococcus pseudintermedius* (and *S. aureus*), *Klebsiella pneumoniae*, *Acinetobacter baumanii*, *Pseudomonas aeruginosa*, and *Enterobacter* species. There has however been a general increase in extended-spectrum beta-lactamase (ESBL) producing *E. coli* in companion animals which, like the ESKAPE pathogens, can be shared with their owners and escape into the veterinary clinic and hospital. We are moving rapidly from the antimicrobial era through the antimicrobial resistance (AMR) era into the era of antimicrobial stewardship (AMS). This shifting paradigm in our relationship with antibiotics is part of the global responsibility that all users have to preserve for the long term the rather small range of antimicrobial drugs available to treat bacterial infections.

We are unlikely to get the resistance genie back into the bottle but a stewardship approach will buy time until we eventually have new antibiotics and new approaches to controlling bacterial infections. **Antimicrobial stewardship** refers to an approach that promotes, improves, monitors and evaluates judicious antimicrobial use (AMU) to preserve the future effectiveness of antimicrobials and to promote and protect human and animal health. It is a term that is preferred to the previously used terms judicious or prudent use, since it includes the idea of not using antimicrobials. The general mindset of **good stewardship practice (GSP)** is a “5R” approach: Responsibility, Reduction, Refinement, Replacement, and Review. A 5R stewardship approach is an active, dynamic, process of continuous improvement in AMU, a pragmatic ethic with many steps of different sizes. A combination of the multiple interventions and approaches embraced in the stewardship model has the potential to have a cumulative impact that will help control AMR. All veterinary users of antimicrobials are now inevitably part of the global “One Health” strategy to address AMR. This strategy, as endorsed in the 2017 pan-Canadian Framework for Action for Tackling Antimicrobial Resistance and Antimicrobial Use, includes surveillance, infection prevention and control, stewardship, and innovation.

**Antimicrobial stewardship is a rapidly developing and evolving field**, with greatest activity and leadership in large tertiary care human hospitals where AMR problems are most visible but with increasing engagement by national and international veterinary organizations and specialties. There is increasing focus in both human and veterinary medicine on primary care physicians and veterinarians, since they are the major users of antimicrobials. There are an increasing array and depth of resources and it is hard to keep up with their pace of development. In Canada, the move to veterinary prescription only for antimicrobials in food animals is a major shift in responsibility for stewardship and GSP. It seems likely that, within a short time, provincial veterinary regulations will require all practices, including companion animal practices, to develop AMS policies and that regulators will monitor and evaluate AMU by veterinarians against agreed benchmarks. A program of AMS Certification could become required.

**A practical approach to antimicrobial stewardship in companion animal medicine**
The concept of AMS is of a dynamic process of continuous improvement in how we use antimicrobials and of reduction in their use to where the benefits are clear and substantial. Figure 1 is an illustration of some of the different elements of GSP.

![Figure 1: An approach to antimicrobial stewardship for companion animal practitioners. The inner circles are important major areas, and the outer circles are additional aspects. Some circles are deliberately blank so users can add their own ideas.](image)

**Practice guidelines:** The British Small Animal Veterinary Association (BSAVA) has developed a practical and accessible approach to AMS, called PROTECT: [https://www.bsava.com/Resources/Veterinary-resources/PROTECT](https://www.bsava.com/Resources/Veterinary-resources/PROTECT).

PROTECT offers a comprehensive approach for a practice to develop its AMS policies and practices: the acronym stands for Practice policy; Reduce prophylaxis; Other options (eg, lavage, topical use); Types of drug and bacteria (drug properties, likely bacterial agents); Employ narrow spectrum; Cytology and culture; Treat effectively; For example, under Practice policy it recommends making a list of first-line, second-line and third-line drugs, where culture and sensitivity is used for second- and third-line drugs, and the latter are only used for life-threatening infections where first- and second-line drugs are not appropriate. Using downloadable PROTECT templates, practices can develop policies on empirical antibacterial use for commonly encountered bacterial infections, for surgical prophylaxis, and for when not to use antibacterial drugs. A Guide to PROTECT is available on the website. I don’t think the PROTECT site has the brilliance of the British Equine Veterinary Association PROTECT ME approach since, although it requires practices to think through their AMU approaches, it doesn’t engage the entire veterinary team in the same way and misses out the important ME (Monitor use, compliance and resistance; Educate) aspect. Assignment of responsibility in the clinic for developing, implementing, monitoring and evaluating an AMS program is essential for success. Nevertheless, PROTECT is a well-organized, well-supported, systematic and thoughtful way on which to build.

An excellent downloadable guideline resource is the Danish Small Animal Veterinary Association’s Antibiotic Use Guidelines for Companion Animal Practice. Other relevant guidelines (superficial bacterial folliculitis, urinary tract infections) are referenced.

**Benchmarking:** “Benchmarking”, the quantitative determination of norms for antibiotic use by veterinarians or at the farm level has been a powerful driver in reduction of antibiotic use in agriculture in countries which has significantly reduced AMU. There are increasing reports surveying AMU in companion animals, some through capture of digital records through commonly used practice software. Not unexpectedly, the majority
of AMU in dogs and cats is with AMs classified as “critically important” for humans using the World Health Organization (WHO) criteria, and up to about one-third of cats are with AMs of the WHO “highest importance” classification. In Canada, Murphy and others (2012) found that there was overuse of cefovecin and of fluoroquinolones for the treatment of cat and dog diseases for which antibiotics were either not indicated or for which first line antimicrobials were quite appropriate. A focus on use is important since reducing AMU will be one of the drivers of reduced AMR. Thus reliable “benchmark” data fed back to the prescribers has great potential future value in AMS.

**Infection control:** There are many reports of the remarkable clonal spread of resistant pathogens in companion animals, both ESBL-producing *Enterobacteriaceae* in Europe and, most importantly, of methicillin-resistant *S. pseudintermedius* (MRSP) globally. The increasing prevalence of canine MRSP globally, including in Canada, is well documented, as is the higher frequency of MRSP infections in cats and dogs that have been hospitalized or have visited veterinary clinics. The reason for the dramatic emergence in the last decade of MRSP, of which there are five major lineages globally, and which are often MDR, is unclear but they demonstrate why good infection control is an important part of AMS. Human infections or colonization with MDR MRSP, the majority of which are apparently transmitted from dogs, have been identified in Canada. The close proximity of dogs and cats with people is one reason for the spread of different MDR bacteria between them, and veterinary clinics or hospitals are recognized to be a potential source for further dissemination between animals (and from them to people).

Companion animal practitioners are sources of MRSA or MRSP and hand and other hygiene practices in companion animal medicine often leaves a lot to be desired. In a video observational study in Canada, median contact time for soap and water handwashing in companion animal practices was just two seconds (Anderson and others, 2014). As noted by others, resolving the issue of MDR endemic bacterial infections will not be through development of new antibiotics if current hygiene practices remain and if we don’t undertake good stewardship practices to preserve our existing drugs. In Ontario, no companion animal practices surveyed in 2009 had an infection control program (Murphy and others 2010). Good infection control is fundamental in reducing the spread of infections, including of resistant bacteria.

**Clinical microbiology:** Greater use of clinical microbiology data to guide selection of antimicrobials is an obvious approach to GSP, but is currently hampered by delays in obtaining data and cost. There is considerable effort being made to speed the process, particularly through rapid DNA-based approaches. PROTECT suggests such testing before the use of Category 1 antimicrobials, those of critical importance in human medicine (fluoroquinolones, third-generation cephalosporins).

**Resistance and use surveillance:** On-going monitoring of resistance and use is an important part of the “continuous improvement” mindset of GSP. Resistance data can be misleading as a measure of population resistance since it is often derived from antibiotic-treated but unresponsive infections but will help identify if there is an infection control problem at the veterinary clinic level (as well as at the country, continental, or global level). Development of practice policies based around PROTECT provides an excellent framework to integrating resistance data into practice policy around stewardship.

**Pharmacokinetics and pharmacodynamics:** Knowing the difference between concentration-(aminoglycosides, fluoroquinolone) and time-dependent antimicrobial drugs is an important pharmacokinetic consideration both in optimal dosing and in the prevention of the emergence of resistance. One survey found that inappropriate dosing generally was common in equine medicine.

**Regulations:** Regulations should be followed. They are particularly important in relation to human health considerations. It may eventually become illegal to prescribe the “last resort drug” vancomycin and carbapenems for animals.

**Education:** Client education posters around antibiotic use in companion animals are readily available for download and display. As society increasingly expects responsible antibiotic use, clients might expect to see such posters in the clinic, including those promoting compliance.

https://www.avma.org/PracticeManagement/ClientMaterials/Pages/clinic-posters-be-careful-antibiotics.aspx
Summary: Companion animal practitioners are on the front line of the fight against AMR and need to engage now before it is too late. Many aspects of AMS, including regulatory monitoring, evaluation and certification are in their infancy and concepts and practices are evolving rapidly.

References


Anesthesia and analgesia for birds, reptiles, and exotic small mammals has many similarities to that of other companion animals. There are a few key differences that should be addressed to provide the safest, most effective anesthesia for these animals. The Bain’s or non-rebreathing circuit is used for most of these species because of their smaller tidal volume. This type of system uses higher flows of oxygen such as 200-800mL/kg/min when the patient is entubated and 1L/kg/min when using a mask. There are a variety of systems available that vary in length, weight, and location of ventilator valve. Care must be taken with these systems as they often weigh more than the patient and can extubate or pull a patient off the table if moved too quickly. A sandbag works well to hold the circuit in position to prevent this from occurring.

Except for the ferret, all these mammals and the pet birds are prey species and the stress physiology associated with prey species is significantly different from that of predatory species such as dogs and cats. In prey species, stress must be kept at an absolute minimum because of what I call their fright or flight response. If prey animals are subjected to stressful situations they will attempt to escape, perhaps harming themselves, and if they cannot escape they will release large quantities of catecholamines. Excessive catecholamines can predispose these animals to increased heart and respiratory rate, increased blood pressure, resulting in myocardial hypoxia and arrhythmias. Stress can be reduced by the choice of anesthetics, particularly premedications, and the manner in which the animal is anesthetized. Rabbits and rodents should be given premedications that cause relaxation and have a calming effect. These include the benzodiazepines and opioids. These animals should be induced and recovered in very quiet, calm areas away from noise, especially barking dogs. Using familiar smells can be calming and minimizing visual and auditory stimuli while the premedicants are taking effect is helpful. Pain can have a similar effect and must be managed pre-emptively. Analgesics given before surgery (and thus pain) are much more effective because the formation of substance P is inhibited and blood concentrations are appropriate before the pain occurs. Analgesics used in all these species include buprenorphine and butorphanol, as well as meloxicam and tramadol (see doses later). Gabapentin (3-10mg/kg bid for most species) has been used for neurologic pain. Ketamine (20-30mcg/kg/min) and lidocaine (20mcg/kg/min) given as low-level constant rate infusions have also been used for bone and GI pain. Multimodal pain relief is very beneficial in these situations so local treatment with lidocaine/bupivacaine combinations is also used frequently.

The surface to body mass ratio in these species is much greater than dogs and cats and because of this, hypothermia can occur very quickly. Heated water blankets, warm water gloves, and heated air blankets can all be used to maintain body temperature. The use of conductive heating pads also seems to work well in these species. These animals should be recovered in an incubator until they have reached normal body temperature. Be careful when heating the animals up after surgery as hyperthermia can occur much more easily because of the vasodilatory effects of most anesthetics. Digital thermometers should be used by inserting into the cloaca or rectum during surgery to monitor body temperature during prolonged anesthetic periods (>15 minutes).

Anesthetic monitoring of these animals depends primarily on their size, in that the smaller the patient the more difficult it can be to monitor anesthesia. The anatomy of reptiles and birds also makes many monitoring devices difficult to use. For birds and reptiles, the ECG and Doppler are the most common monitoring devices used. For ferrets and rabbits, the majority of monitoring devices used in dogs and cats can be used. Ultrasonic Dopplers can be used to monitor heart rate, rhythm, and blood pressure. In birds, the Doppler crystal is placed on the ventral surface of the proximal ulna and radius or the medial aspect of the hock. For reptiles the crystal is most commonly placed over the heart or in the thoracic inlet aimed towards the heart. For
ferrets and rabbits, the crystal is placed over the metatarsal or metacarpal bones. The brachial artery can also be used on the medial surface of the distal humerus. For ferrets and rabbits, the cuff is placed just proximal to the probe to determine blood pressure. Small size and high heart rate can make blood pressure monitoring difficult and the Doppler is often used simply to monitor pulse rate and character. It can be placed on any peripheral artery that can be palpated, such as the auricular artery in rabbits and the femoral artery in many species. In very small mammal species the probe can simply be placed on the chest over the heart.

EKGs can be used in most patients to give information on heart rate, rhythm, and contractility. The leads are placed in a manner similar to other pets. In birds the leads are placed at the patagium and cranial thigh folds. For all species the teeth of the leads should be blunted or flattened to reduce tissue trauma. Alternatively, small gauge needles or wire suture can be passed through the skin and the leads clamped to the metal. Be judicious in your use of alcohol on the leads to avoid any hypothermic effect. Small squares of alcohol-soaked gauze can also be helpful in protecting the skin and limiting the amount of alcohol placed on the animal.

Esophageal stethoscopes can be used in large birds, ferrets, rabbits, and reptiles. The smallest size stethoscope head is passed down the esophagus until the heart sounds are heard. This method does not have any advantage over the Doppler except that they are less expensive.

Pulse oximetry uses a probe and a light source to continuously measure oxygen saturation of the hemoglobin as it passes through the tissue. For mammals, when oxygen saturation gets below 90% the animal is experiencing tissue hypoxia and steps should be made to correct this, such as intubation, lowering the anesthetic concentration, repositioning the animal, and assisted respiration. These instruments are not calibrated for the nucleated red blood cell of birds and mammals and so they are less useful in these species. The pulse oximeter also gives a heart rate as part of the read out. The probe should be placed on a non-pigmented, hairless tissue such as the tongue, lip, rectum, ears, and toes.

Visual examination is perhaps the most important method of monitoring patients, although this can be limited by drapes, size, and other factors. The depth of anesthesia can be monitored by the amount of muscle relaxation, reflex response, and response to stimulation (toe pinch, surgery). The palpebral response is lost early in a surgical plane of anesthesia; so more painful stimuli may elicit a response. Rabbits may maintain a palpebral response throughout surgery, even at appropriate depths of anesthesia. If possible, always maintain a clear view of the chest so that respiratory rate and depth can be monitored. Respiratory rate will decrease with deeper anesthesia and will increase with painful stimuli if the anesthetic plane is too light during surgery. Some drugs may cause apnea, such as propofol, and intubation is recommended with such drugs. Heart rate will decrease with deeper planes of anesthesia and increase in response to painful stimuli in animals that are too light.

Emergency drugs should be available and doses calculated for animals undergoing anesthesia. A chart with common doses for common species or varying weights can be made and placed within an emergency box containing syringes, needles, and emergency drugs.

**BIRDS**

Pre-anesthetic fasting is recommended as follows: 1-2 hours for small birds(<100g), 2-3 hours for medium sized birds(100-500g), and 4-6 hours for large birds(>500g). Birds should be induced in a quiet, calm area away from the sights and sounds of predators. Once induction drugs are given, the bird should be placed back into a small cage that is well-padded for their protection. A low perch can be provided if it will minimize stress, but keep in mind many drugs (especially benzodiazepines) can reduce the perching reflex and birds may fall from the perch.

Premedicants used in birds vary with species, temperament, procedure to be performed and personal preference. For anxious or easily stressed birds, which include macaws, African greys, raptors and many wild birds, midazolam at 1.0mg/kg IM is used. This will cause mild sedation and relaxation. Doses as high as 6mg/kg have been reported resulting in considerable sedation. Butorphanol has been shown to be most effective in parrot species and is used for sedation and analgesia. The dose range is 1.0-2.0mg/kg depending
on the depth of anesthesia required and the pain associated with the procedure. Butorphanol and midazolam together produce good sedation for pre-anesthesia and for non-painful procedures such as radiographs and bandage changes. For longer abdominal procedures or orthopedic surgeries, ketamine can be added at 5-10mg/kg to the butorphanol and midazolam combination.

For short procedures where no premedicant is used, birds should be held comfortably and a face mask placed over the head. The bird should be kept in an upright, perching position. This reduces stress and maximizes air sac volume. A towel may be used to restrain the wings in fractious patients. Pre-oxygenating birds is debatable as high oxygen levels will reduce respirations. I recommend starting the oxygen and placing the inhalant on the lowest possible setting for 20 seconds, then turning the anesthetic flow rate up slightly every 15 seconds. This allows the bird to accommodate to the mask and the smell of the inhalant. It also gives finer control of anesthetic depth. As the anesthetic depth increases, the patient can be moved to recumbent position and intubated if desired.

Vascular access is an important part of successful avian anesthesia. Intravenous catheters can be placed in the median metatarsal vein in birds over 300g and work especially well in galliformes and anseriformes. Cathers can also be placed in the superficial ulnar or basilica veins of the wing but are more difficult to maintain, especially after surgery. The jugular vein in birds is quiet mobile and may result in extravasation of fluids if the vein moves away from the catheter with head and neck movement. IV catheters can be taped or sutured in place depending on location and duration of placement. Intraosseous catheters are fast and convenient in birds, especially small birds. The proximal ulna is the most common site of placement. The proximal tibiotarsus can also be used but will cause lameness following placement. Fluids should be given at a rate of 10ml/kg/hour for the first hour and 5ml/kg/hr after that. The bird should be making urine during the procedure, so check the cloaca for urine production and adjust the fluid rate accordingly. The fluids should be warmed to help maintain body temperature.

Intubation in birds is generally very easy. The avian glottis is easily visualized at the base of the tongue in moderate to large parrots and raptors. The beak can be held open by hand or straps of tape or gauze and a pen light or room light used to visualize the glottis. In chickens and water birds the glottis may rest ventrally further in the mouth and can be more difficult to visualize. Gentle pressure with a fingertip under the chin can help elevate it for easier intubation. In smaller parrots, the tongue may be large enough to limit the view of the glottis and will need to be pulled out of the mouth using forceps. The beak may be held open with small gauze strips or paper clips for better viewing.

The endotracheal tube size will vary according to species as well as size. Large parrots usually require 3.5-4.0mmID tubes, while smaller species such as cockatiels and lovebirds require a 1.0 – 2.0mmID tube. 18-22G intravenous catheters can be used in very small birds. Any tube below 2.0mm is at a greater risk of being plugged from tracheal secretions and should be monitored closely. Soft, uncuffed tubes should be used when possible. Cuffed tubes can be used when the risk of aspiration is high but should be inflated gently to avoid excessive pressure on the trachea. The trachea of many large birds (macaws, cockatoos, egrets, cranes) can narrow abruptly so narrow tubes or Cole tubes should be used to avoid pressure on the tracheal mucosa. The area around the glottis and ET tube should be swabbed just before extubation to prevent aspiration of any mucus that has accumulated in the area.

In small birds or birds undergoing surgery that interferes with the upper airway, an air sac cannula may be placed for respiration. This is placed in the caudal thoracic air sac and bypasses the upper respiratory system. A skin incision is made in the triangle formed by the last rib, cranial thigh muscles and vertebrae. Blunt dissection through the muscles is performed with a hemostat. The air sac membrane is gently punctured and the hemostats opened to pass a tracheal tube or red rubber tube to be used as the cannula. A cuffed endotracheal tube is ideal because condensation from respirations is easily visualized and the cuff can be inflated to help maintain the cannula. The tube is sutured in placed with a purse-string then finger-knot suture. The tube should be cut short to decrease resistance of respiration.
Isoflurane or sevoflurane are the inhalant anesthetics of choice and are often used as the sole anesthetic agent for short procedures. These drugs can be given via mask alone for short procedures (<10 minutes). The mean alveolar concentration for these drugs is 1.3% and 2.2% respectively. Birds are typically maintained at 2-2.5% isoflurane and 2.5-3.5% sevoflurane in my experience. All anesthetics delivered via oxygen cause respiratory depression in birds because of the effect of the oxygen on the carotid bodies and intrapulmonary chemoreceptors. Because of this, assisted ventilation is required on all birds under anesthesia for more than 10 minutes. Two to six breaths per minute is usually adequate depending on the anesthetized bird’s respiratory rate and depth. There are several ventilators that can be used in birds. These machines allow breath volume and frequency to be set at the variation needed in avian practice. The bellows are often visible, making monitoring of the respirations easier. Inhalant anesthetic levels may be decreased gradually towards the end of a procedure to allow more spontaneous respiration and smoother recovery.

Analgesia in birds is best provided with butorphanol (1-2mg/kg q6h) which is often given during or after surgery to prevent respiratory depression. Buprenorphine (0.03-0.06mg/kg q12h) has also been used but may not provide as much analgesia. NSAIDS can also be used with meloxicam (0.5-1.0mg/kg q12-24h) being used most commonly. Ketoprofen or carprofen (1-2mg/kg q12h) are also given, especially in larger birds but have been linked to subclinical renal damage in one study. Tramadol has been anecdotally reported to be helpful in birds. A published dose of 4-11mg/kg has produced effective concentrations in bald eagles for 10 hours. A multimodal approach to pain relief should be provided using opioids and NSAIDs especially before or early into a painful procedure. Topical anesthetics of mixed lidocaine and bupivacaine (0.5-1mg/kg of each) can be applied directly to nerves or as a block to provide further analgesia.

**REPTILES**

Reptiles should be maintained at the mid to high end of the preferred optimum temperature zone (POTZ) for better metabolism of the anesthetic drugs, better recoveries and fewer peri-anesthetic problems. These animals should be recovered in an incubator set at the mid-portion of the POTZ. Digital thermometers can be inserted into the cloaca during surgery to monitor body temperature during anesthetic periods.

The respiratory anatomy varies between classes of reptiles. The glottis is normally in a closed position and you may have you slide the endotracheal tube in gently to open the glottis. The trachea is incomplete in snakes and lizards but complete rings in chelonians (turtles and tortoises). The lungs of snakes and most lizards are large and sac-like with an avascular air sac on the caudal end. The epithelium is very thin and overuse of pressure can cause rupture of the lungs so don’t inflate the lungs to more than 10-12mmHg. Air movement in reptiles generally involves skeletal muscle movement of the limbs or ribs, so anesthesia requires assisted ventilation to inflate the lungs. Additionally, respiration is triggered by decreasing oxygen tension, thus 100% oxygen is a respiratory suppressant in reptiles. For the anesthetist, that means switching to room air as soon as possible during recovery to stimulate spontaneous respirations.

Pre-anesthetic fasting in reptiles is generally indicated and the length of time depends somewhat on the metabolism of the animal. Medium to large animals are usually fasted for 8-12 hours, while smaller species may only be fasted for 2-4 hours. Another way to look at it is to skip one meal, so animals that eat once daily are fasted for 24 hours, animals that eat twice daily are fasted for 12 and animals that eat weekly or less are anesthetized 24-48 hours after the last meal. Pre-medications are routinely given in these species because breath-holding is common if mask induction is attempted. Ketamine doses vary widely between species so check the dose with an exotic animal formulary. Ketamine can be used alone (10-80mg/kg IM) or in combination with dexmedetomidine (0.15mg/kg IM) or midazolam (1-2mg/kg). Ketamine causes prolonged recovery, especially when used alone so be prepared for more intense recovery monitoring. When using the drugs in combination, use the lower doses of ketamine to help avoid the prolonged recovery. Also, dexmedetomidine and midazolam can be reversed using atipamazole (0.75mg/kg) and flumazenil (0.1mg/kg) to help speed recovery. I occasionally use ketamine (10-20mg/kg) with dexmedetomidine (0.1-0.15mg/kg)
intramuscularly in lizards and turtles. This gives sufficient anesthesia to easily intubate the animal so that anesthesia can be maintained using isoflurane. Tiletamine/zolazepam (Telazol: 4-40mg/kg) can also be used but also causes prolonged recovery. The ketamine/medetomidine combination and Telazol often provide enough anesthesia for minor procedures, provided analgesia is used. Respiratory suppression may occur with these drugs therefore ventilation with room air may be required. Propofol (5-10mg/kg IV) is safe and easy to use in many species and provides approximately 20-30 minutes of anesthesia with a single dose. Apnea is common with this drug and intubation and ventilation with room air is recommended.

Vascular access is important for longer procedures (>30 minutes). Intraosseous catheters are generally placed in the distal femur of lizards. Short-term butterfly catheters can be placed in the ventral tail vein of larger lizards but can be difficult to maintain. A cut-down can be performed for a cephalic catheter in lizards as well. Chelonians require an IV catheter in the jugular vein. For snakes, a catheter can be placed in the palantine vein or jugular vein via cut-down. Warmed fluids are given at 10ml/kg/hr.

Inhalant anesthetics are, such as isoflurane and sevoflurane, are commonly used in reptiles. Assisted ventilation is required whenever inhalant drugs are used. Generally 1-4 breaths per minute is adequate. If using Injectable anesthesia alone and during recovery, always ventilate the patient with room air to prevent the respiratory suppression seen with oxygen. The anesthetic gas should be turned off about 15 minutes before the end of longer surgeries and the patient switched to room air at this time. This will speed recovery and return of spontaneous respiration.

Analgesia in reptiles can be provided with NSAIDS or opioids. The data for opioids in reptiles varies considerably between species. In iguanas, morphine at 1mg/kg IM or butorphanol at 1.5-8mg/kg IM can be used. For the bearded dragon, morphine at 10-20mg/kg can be used. For the corn snake, buprenorphine at 0.1mg/kg IM or butorphanol at 20mg/kg appear to be efficacious. NSAIDS are often used predominantly because of this varied data. Meloxicam at 0.3-0.5mg/kg q 24h is used for most species. Ketoprofen and carprofen can be given at 1-2mg/kg once daily. The use of topical anesthetics such as lidocaine and bupivacaine can be given at 0.5-1.0mg/kg topically or as a block.

FERRETS

Anatomically and physiologically ferrets are the most similar to dogs and cats. Ferrets are generally induced in a calm, quiet environment and recovered in an incubator. Overheating is very easy in these animals so monitor post-op temperatures frequently. Hyperthermia has also been seen with opioids in this species.

Vascular access is warranted in ferrets for any procedure lasting more than 15 minutes. Short, small catheters (24-26G) can be placed in the cephalic or lateral saphenous veins. Numb the area with topical lidocaine cream or sedate the ferret first to make catheterization easier. Nick the skin over the vein with a needle so the small catheters don’t burr when being pushed through their thick skin. Tape the catheter in place securely and wrap with cast padding and vet-wrap. Fluids are given at 10ml/kg/hr and can be dropped in half after 1-2 hours if body temperature and blood pressure are maintained.

Short procedures, such as radiographs, venipuncture, catheter placement, and very short surgeries may be performed using isoflurane via facemask. This can be irritating to the ferret so always start with the gas at the lowest setting for 30 seconds to allow the animal to get used to the smell, and then slowly turn up the gas incrementally every 15 seconds. Also, keep in mind that isoflurane can cause artifactual decrease in many red blood cell parameters so if blood is taken using isoflurane is should be done as soon as possible after induction of anesthesia. Sedation can also be achieved for short procedures with midazolam at 0.5mg/kg IM or butorphanol at 0.3-0.5mg/kg IM. The two combined produce heavy sedation.

For longer surgeries, induction should be performed using any of several drugs, the choice of which depends on the health status of the patient and the clinician’s choice. Injections are given in the thigh or epaxial muscles. My favorite protocol for most ferrets is ketamine (5-7mg/kg IM), midazolam (0.5mg/kg IM),
atropine (0.04mg/kg), and buprenorphine (0.03-0.06mg/kg SC). Other drugs that can be used include acepromazine (0.1-0.3mg/kg IM), xylazine (1mg/kg SC), diazepam (2mg/kg IM), and butorphanol (0.1-0.5mg/kg SC,IM). Acepromazine and xylazine can cause hypotension and should be used with another agent that counteracts these effects. These drugs also have a longer recovery time than other drugs.

Intubation of ferrets is generally performed with a 2.0 or 2.5 uncuffed endotracheal tube. Topical application of lidocaine is helpful. Spontaneous respirations are usually maintained throughout surgery in ferrets, although periodical full ventilations can be helpful. Isoflurane or sevoflurane should be used in ferrets as the gas anesthetics of choice.

Hypotension is a common occurrence in ferrets during anesthesia. Prevention is the key, this can be done by maintaining body temperature and with fluid administration and judicious use of pre-medications. Changing isoflurane levels and fluid administration during surgery should also be done. The use of dopamine in ferrets has caused severe renal dysfunction in my experience, and I recommend avoiding it. Dobutamine does not seem to cause this problem and can be given at standard cat doses. Colloids are also helpful to maintain blood pressure during surgery at 5-10ml/kg boluses.

RABBITS

As discussed earlier it’s very important that rabbits are kept calm and quiet before anesthesia and during induction and recovery. Many hospitals have special areas set aside for these species. You must also be careful when handling rabbits, especially for injections, so that they do not cause themselves harm. Intramuscular injections can be given in the cranial or caudal thigh and lumbar muscles. Gastrointestinal stasis is common in rabbits following general anesthesia, especially abdominal procedures. Fecal output and appetite should be monitored closely following an anesthetic event. Offering food or force feeding soon after surgery, especially food high in fiber, can help keep GI tract motility normal. Motility modifiers such as cisapride or metoclopramide can be used if rabbits are showing signs of stasis and are not eating well. Rabbits do not vomit and pre-anesthetic fasting is only recommended for abdominal procedures to reduce the size of the stomach. A short period of fasting can also help to keep the mouth clean to make intubation easier. Rabbits, and other hind-gut fermentors such as guinea pigs and chinchillas, have large abdominal cavities and relatively small thoracic cavities. Because of this, positioning the animal with the head and thorax tilted above the abdomen can increase lung volume and decrease respiratory effort.

Intubation rabbits is difficult because of their long soft palate, small oral opening, large incisors and thick, muscular tongue. Rabbits also have a tendency for laryngeal spasm so topical lidocaine spray should always be used. Many facilities do not intubate rabbits, even for abdominal procedures because of the problems associated with this procedure. Intubation should always be performed for oral, facial, and thoracic surgery. Intubation can be performed by several methods; direct visualization, blind intubation, and the use of a tracheal catheter. Direct visualization is best done by inserting the laryngoscope on the side of the mouth and then pushing the base of the tongue down and soft palate up to reveal the glottis. Intubation in this situation is similar to other species. Intubation can be performed blinding by positioning the head looking straight up, pulling the tongue out over the lower incisors then passing the endotracheal tube along the tongue until the glottis is encountered. Condensation can be seen in the tube with each exhalation when the tube is positioned appropriately. Once the tube is in place, wait until the beginning of inspiration and attempt to pass the endotracheal tube through the glottis. A coughing response and continued condensation on the inside surface of the tube indicates success. The final method involves passing a jugular catheter into the tracheal lumen through the skin of the neck and passing the soft catheter portion into the trachea and out the mouth. An endotracheal tube is then placed over the catheter and passed along the catheter into the trachea. The catheter is then removed from the neck. An important thing to remember about tracheal intubation in rabbits is that repeated attempts may cause trauma to the larynx resulting in laryngospasm and respiratory arrest following extubation. For this reason, direct visualization of the glottis is the recommended
method. If using one of the blind techniques be extremely gentle and do not attempt intubation more than 2 or 3 times.

Vascular access is important for longer procedures. Small, short catheters (24-26G) can be placed in the cephalic or lateral saphenous veins using the technique outlined for the ferret previously. The marginal ear vein can also be used during surgery using a small catheter. This catheter is more difficult to maintain after surgery but can be capped and covered with Tegaderm® for intermittent use after surgery.

Sedation in rabbits can be performed using IM or IV injectable medications. Midazolam, given IM at 0.5-1.0mg/kg works well for light sedation for radiographs, ultrasound and non-painful dental procedures. Midazolam can also be combined with dexmedetomidine (0.1-0.15 mg/kg IM). Dexmedetomidine has the benefit of being reversible using atipamazole (0.5mg/kg IM). Ketamine (10mg/kg) and diazepam (0.5mg/kg) given in combination intravenously also gives sedation for short procedures such as tooth trims, skull radiographs, and treating minor wounds. Some drugs have been shown to be efficacious in rabbits if given intranasally, such as xylazine (3mg/kg) and ketamine (10mg/kg). Glycopyrrolate can be used at 0.01-0.1mg/kg Isoflurane or sevoflurane delivered via mask may be used in rabbits as the sole anesthetic agent for short procedures. There is recent evidence that isoflurane alone can cause severe hypotension and increased catacholamine release so the addition of anxiolytics like midazolam may be warranted. My preferred protocol uses midazolam (0.5-1mg/kg IM), ketamine (7-10mg/kg IM), and butorphanol (0.5mg/kg IM) for induction and then gas anesthetic via mask or intubation for maintenance.

Analgesia in rabbits is done with buprenorphine at 0.3-0.6mg/kg SC. This causes minimal opiod-related GI stasis. Butorphanol can also be used at 0.1-0.5mg/kg but may cause more sedation and GI effects. Meloxicam at 0.3-0.5mg/kg twice daily is my preferred NSAID although carprofen, ketoprofen, and ibuprofen can also be used at 1-2mg/kg q12-24h. Tramadol can be added orally at 5-10mg/kg q12h.

GUINEA PIGS AND CHINCHILLAS

These animals are very similar to rabbits except that they are smaller, making certain monitoring devices more difficult to use. These animals are only occasionally intubated because of the difficulty associated with viewing the glottis and the small size of the oral opening. Guinea pigs often have gastric reflux with anesthesia and should be positioned so that the head and chest are raised above the abdomen, this will also make breathing easier. GI stasis is also a concern in these species and should be treated as in the rabbit. Pre-anesthetic fasting for short time periods, such as an hour, may be helpful in reducing reflux and keeping the mouth clean.

Injectable medications are commonly used in these species as part of the anesthetic protocol so that the levels of isoflurane can be minimized. High levels of isoflurane for long periods can cause hypotension and respiratory depression. Injectable medications used in guinea pigs and chinchillas include midazolam, ketamine, medetomidine, and buprenorphine. Doses are similar to rabbits.

RATS, MICE, AND OTHER SMALL RODENTS

Anesthesia in these animals is similar to that of guinea pigs and chinchillas although their smaller size can make monitoring anesthesia more difficult. Often, simply viewing the patient once the drapes have been placed can be difficult. Clear drapes can be very helpful for this reason. These pets do not vomit and so pre-anesthetic fasting is not recommended. Also, their high metabolic rate can make hypoglycemia a problem if they are fasted for any length of time. Heat loss is also faster in these species because of their small size. Fluid loss during anesthesia can be an important point and it is often helpful to give them SQ fluids during or before a procedure. Because of their higher metabolic rate, drug dosages are considerably higher in these species. Ketamine doses as high as 100mg/kg have been recommended either alone or in combination with dexmedetomidine.
(10mg/kg IP). I don’t tend to give doses that high but wanted to report what is possible. Midazolam can be used in these species at a dose of 1-2mg/kg IM or IP. Many drugs are given IP in these animals because of their small size, this can cause a more rapid induction and slightly prolonged recovery.

Isoflurane or sevoflurane can both be used and are often used as the sole anesthetic. I usually recommend using an injectable drug along with the inhalant to smooth the anesthetic event. Midazolam at 1mg/kg and butorphanol at 0.5-1.0mg/kg are my choices for this. Commercially made face masks are often too large for these animals and the anesthetist will often have to find innovative ways to deliver the anesthetic gases, such as using small syringe cases as face masks.

ANESTHETIC RECOVERY

The length of recovery obviously depends on the drug(s) used and the metabolism of the animal. It is important that the patient be kept warm and hydrated during recovery. A dark, quiet place is often preferred, especially for animals that burrow. Birds should be held gently using a towel to prevent injury during the initial phases of recovery. Be careful, especially when using heat lamps, not to burn or overheat the patients. Analgesics should be given before surgery, but may need to be repeated every 4-12 hours after surgery, depending on the drug used. Pain relief is particularly important in the prey species. It is important not to move or lift these small animals quickly during the recovery period as orthostatic hypotension is a serious concern and can cause cerebral ischemia and result in peri-anesthetic complications.
Emergency medicine in exotic pets uses the same principals as in other species. Triage parameters are also very similar with priority being given to bleeding animals, animals in respiratory distress and animals in shock. It’s important to be prepared for the emergency; have a designated area that with emergency supplies, drug dosages, and equipment that might be needed. Train your personnel in emergency procedures; from the front office staff to the techs to the animal caretakers. They should all know what signs constitute an emergency in exotic pets and where to bring the critical patient.

BIRDS

Trauma/bite wounds

Fractures of the extremities should be bandaged until the patient is stabilized adequately for radiographs and definitive treatment of the fracture. Wing fractures may be stabilized in figure of 8 bandages for fractures distal to the elbow. Fractures of the humerus should be wrapped in a light figure of 8 bandage and then secured to the body to prevent should movement. Fractures of the pelvic limbs can usually be stabilized using an external splint. Femoral fractures need to be surgically repaired or allowed to heal by cage rest. Open fractures should be cleaned and flushed (if not pneumatic) and fixation should be attempted as soon as possible to minimize osteomyelitis and septicemia. Fracture repair methods will depend on several factors such as the location of the fracture, size of the patient, owner compliance, and clinician’s preference. Stabilization of the patient will be determined by the animal’s condition, but may include fluid support, analgesics, and antibiotics in open fractures.

Treatment of lacerations depends on the severity of the wound, time since occurrence, and the condition of the patient. Minor lacerations treated within 12 hours may be cleaned and closed surgically. Lacerations older than 24 hours should be cleaned and debrided and allowed to heal by second intention.

Birds sustaining bite wounds from dogs, cats, or other birds are frequently encountered. These wounds are usually of the crushing and tearing type and may require surgical repair or debridement. Mammal bites, especially cat bites, are true emergencies because of the pathogenic organisms, such as *Pasteurella multocida*, introduced into the tissues. Bite wounds should be flushed with warm saline or saline with chlorhexadine. Birds should be placed on systemic antibiotics with activity against *P. multocida*. The penicillins (clavamox 125mg/kg po bid) or fluoroquinolones (enrofloxacin 20mg/kg po sid) are often used. Birds suffering from bite wounds may present in shock and should be treated as such with IV fluids, colloids and warmth. It may be difficult to locate bites wounds within the feathers. For this reason, it is recommended to place any bird with potential bite wounds on systemic antibiotics.

Crop burns occur most often in birds being hand fed formula that is too hot. The formula is often heated by a microwave oven, producing small pockets of formula that are very hot but not noticed when the food is tested. Cases of crop burn have also been seen in adult birds fed coffee and other hot foods. The burn affects the crop wall and overlying skin and causes initial erythema, which may not be noticed by the owner. As the crop wall and skin fuse and necrosis occurs, a fistula is formed. The first sign noted by most owners is the formula passing through the newly formed fistula. Crop fistulas are not true emergencies unless the fistula is very large. Smaller, more frequent feeding may be used initially to maintain the bird’s weight until the fistula is fully developed. If the fistula is very large
and oral feeding causes extensive leakage, the food may be placed directly into the proventriculus through the fistula using a soft red rubber catheter. Smaller amounts should be used (approximately 1/2 of the volume of the crop). If the bird is thin, weak or appears otherwise malnourished, supportive care should be initiated until the bird is capable of undergoing surgery. The surgical procedure involves initial separation of the crop from the skin then incorporates a two-layered closure of the crop. The skin is closed with a simple continuous pattern.

Blood feathers are actively growing feathers that still maintain a blood supply as they grow. If the portion of the feather that is still growing is damaged it will bleed and can bleed profusely. This is often a frightening problem for the owner but birds rarely bleed to death from these. Classic treatment is to remove the feather at the base to stop the bleeding. The feather is grasped at the base and pulled sharply in the direction it is growing. This should stop the bleeding but may also damage the feather follicle, making new feathers more prone to malformation. Another option is to stop the bleeding at the point of trauma using hemostatic agents and direct pressure. This usually maintains the viability of the follicle and stops the bleeding.

Unilateral lameness, especially in budgies and other small birds, is often the result of a renal or gonadal tumor placing pressure on the sciatic nerve as the nerve passes between the kidney and the pelvis. This change can often appear spontaneously but is the result of a chronic problem. The bird still maintains use of the femoral nerve so has an active withdrawal but is unable to move the rest of the leg or perch with the foot. Diagnosis is often based on history and PE findings but radiographs can be helpful. Treatment is supportive and the prognosis is poor.

**Egg-binding**

Dystocia can be a serious event in birds. A bird presented for egg binding should be placed in a warm, humid environment while a history is taken. Pertinent information includes: age, previous egg laying activity, history of egg binding, and diet. Many birds presenting for egg binding are chronic egg-layers that have insufficient calcium stores necessary for uterine contractions. The calcium deficiency may be the result of continual egg production and/or poor dietary calcium intake. Once a history is taken, the patient should be examined quickly to establish the presence and location of the egg, as well as general condition. If ascites is present, peritonitis or neoplasia should be suspected. If an egg is palpable within the pelvic canal, supportive therapy may be initiated. This includes calcium gluconate at 50-100mg/kg IM, SQ fluids, and tube feeding. Before using oxytocin, the cloaca should be examined for strictures or other problems and lubricated to facilitate egg passage. Most cases of egg binding can be resolved with calcium therapy and supportive care.

If no egg is passed in a few hours, then radiographs are recommended to check for multiple eggs, egg size, and other factors that could change the treatment plan. Anesthetizing birds for radiographs also gives an opportunity to palpate the relaxed abdomen. If the bird is stable under anesthesia, this opportunity can be taken to remove the egg manually or via ovocentesis to collapse the egg. Manual expression involves gentle, slow manipulations of the egg through the oviduct and out the cloaca. Manual pressure should be intermittent to allow the bird to breathe normally between pushes.

Ovocentesis should be done by directly visualizing the egg through the cloaca and oviductal opening. If pressure is applied on the egg towards the cloaca, direct visualization can usually be achieved. If the egg can be visualized through the oviduct, the contents can then be aspirated and the egg collapsed. A 23-20 gauge needle on a 6-12cc syringe is used to aspirate the egg contents. If the egg cannot be collapsed, larger needles can be used. Small cotton swabs should be used to lubricate around the egg before any attempt is made to remove it. If the egg cannot be directly visualized, the egg contents can be aspirated percutaneously. There is more risk with this method, therefore is should only be used if absolutely necessary. The egg should collapse after aspiration of the contents, if not, gentle pressure applied to two sides of the egg should cause it to collapse. There is some risk associated with the sharp edges created by ovocentesis, therefore, every effort should be made to remove the egg whole before attempting this procedure.
If the egg is collapsed, but unable to be removed, the bird can be recovered and allowed 12-24 hours to pass the collapsed egg. If the egg is still not passed, abdominal surgery and salpingohysterectomy should be performed.

Respiratory Disease

Respiratory emergencies are common in avian practice and may be caused by a primary respiratory disease or secondary to other diseases. Primary respiratory diseases include infectious disease, inflammatory (allergic) problems, toxic insults, foreign bodies and neoplasia. Secondary respiratory disease is usually caused by abdominal distension, abdominal masses, and thyroid enlargement. Upper respiratory diseases may occasionally present as a respiratory emergency because of open-mouthed breathing and increased respiratory rate. In these cases, remove any nasal plugs that occur and submit them for bacterial and fungal culture/sensitivity and histology. Cytology slides can be made for quick analysis of the type of bacteria and/or fungus present. Treat the bird with topical and systemic antibiotics and antifungals (if warranted) until the results of the cultures are obtained. Nasal flushes may be therapeutically and diagnostically beneficial. Collect the flushed solution in a sterile container for cytology and culture. Birds with respiratory signs from abdominal distension usually show tachypnea with short, shallow respirations because of the loss of air sac volume. The distension may be due to abdominal masses, ascites or an egg. Treatment should include removing any fluid present for cytology and culture. This will often greatly ease the respiratory burden on the patient. The bird should be placed in an oxygen cage and kept quiet and calm. If an egg is present, stabilize the patient and remove it when the bird is calm. If a mass is present then surgical removal is often the best choice to relieve the respiratory distress. Large air way disease is usually caused by tracheal foreign bodies, granulomas or thyroid enlargement. These birds usually present with a history of voice change, exaggerated respiratory click and open-mouthed breathing. These birds should be placed in oxygen as soon as possible until they are calm. Terbutaline (0.1mg/kg IM BID) may be of some use but often an air sac cannula must be placed. Tracheal or coelomic endoscopy and radiographs should be used to identify the problem and develop a treatment plan. Small air way disease is usually caused by inhaled toxins, such as smoke or Teflon, or allergies. These birds are difficult to manage. They present open-mouthed breathing with a wide-based stance, wings abducted and an expiratory squeak. Oxygen and IM terbutaline should be administered. If these do not relieve the distress, intubation and nebulization with terbutaline (01mg/kg in 9cc NaCl) via IPPV has been beneficial in some cases. Radiographs may show hyperinflated air sacs but are often normal. Parenchymal disease can be caused by infections, pulmonary neoplasia or congestive heart failure. These birds present with increased respiratory rate and effort (as evidenced by tail bob) but are rarely open-mouthed breathing. Treat these birds with oxygen and other supportive care as deemed by a physical examination. A thorough history is important in determining the cause of the disease but radiographs and endoscopy with air sac and lung biopsies are usually warranted.

FERRETS

Insulinomas

Functional tumors of the pancreatic beta-cells in ferrets are common. These insulinomas may cause ferrets to present on emergency either at initial diagnosis or when there is an acute episode of tumor growth that results in an insulin surge. The resulting hypoglycemia causes ferrets to become weak, glassy-eyed, drool, exercise intolerant or in hypoglycemic seizures. The presumptive diagnosis can be made with a point-of-care glucometer. Less than 60mg/dl is highly suggestive of this problem, especially when no other cause of the hypoglycemia can be found. A definitive diagnosis requires concurrent insulin levels. Treatment is with prednisolone (0.25-1mg/kg initially) either IM or PO depending on the patient. Feeding the ferret a high fat/high protein meal will also help to improve the glucose level without an insulin spike that occurs when oral dextrose is given. Severe hypoglycemia may require intravenous fluids with dextrose (2.5-5%). If the ferret is currently on medication then I usually recommend increasing by 25%, if the ferret is not currently on medication, I start with 0.25-0.5mg/kg PO BID.
Urinary Tract Disease

Ferrets with adrenal tumors may also present on emergency because of prostatic enlargement causing partial to complete urinary obstruction. The ferrets have a history of straining to urinate, frequent, small urinations that leads to lethargy and depression as azotemia develops. The onset is usually gradual if the owners are astute. Other signs of adrenal disease include symmetrical hair loss and aggression. A turgid, painful bladder may be palpated and the enlarged prostate is rarely palpable in the caudodorsal abdomen. Presumptive diagnosis is based on history and clinical signs. Ultrasound examination will show the enlarged prostate and enlarged adrenal gland. Prostatic cysts or abscesses can also occur with this disease. Initial treatment is to place a urinary catheter using a 3.5Fr red rubber catheter (use a sterile guitar string to pass this catheter by the os penis) or a Slippery Sam® catheter. The ferret should then be given intravenous or subcutaneous fluids depending on the azotemia and antibiotics if an abscess is present. Treatment of the adrenal disease with luprolide acetate (Lupron® 100ug IM monthly), deslorelin (Suprelorin F® implant yearly) or surgery.

Stranguria or dysuria with blood in the urine is more likely to be from a urinary tract infection or stones. Urinary tract infections can occur with adrenal disease or stones and occasionally as a discrete entity. Gram negative bacteria are most common. Stones occur in ferrets from using plant-based protein diets and are composed of struvite. Diagnosis is with palpation, radiographs or ultrasound and urinalysis. Treatment of bacterial cystitis is based on culture and urolithiasis is treated with surgery. Urinary acidifiers may work if there are only crystals and high pH.

Anorexia and Vomiting

Gastric foreign bodies are common in ferrets because of their inquisitive nature. Foreign bodies are usually soft, rubber or plastic items, especially in young animals but trichobezoars are more common in older ferrets. Clinical signs include anorexia, bruxism, hypersalivation, cranial abdominal pain, diarrhea, and melena. Vomiting is more common with gastritis than with foreign bodies but can occur when there is complete obstruction by the foreign body. Diagnosis is with plain or contrast radiography. Treatment is with surgical or endoscopic removal. Treatment for gastritis is recommended following removal of the foreign body as outlined below.

Gastritis and gastric ulcers are common in ferrets with a variety of diseases causing them to present on emergency. Ulcers may be secondary in a foreign body, due to infectious disease, toxin ingestion, and azotemia or induced by anti-inflammatories or stress. Any cause of anorexia in ferrets may cause gastritis as well. Clinical signs include lethargy, anorexia, bruxism, hypersalivation and melena. Ferrets with gastritis are actually more likely to vomit than ferrets with foreign bodies. The diagnosis is based on clinical signs and finding one of the causes listed above. Baseline diagnostics, such as radiographs, CBC and chemistry panel can help to elucidate many of these causes. Definitive diagnosis requires endoscopy or surgical biopsy, but this disease is usually treated based on clinical suspicion. Treatment is aimed at relieving the hyperacidity with ranitidine (3.5mg/kg q12h), famotidine (0.5mg/kg q24h), cimetidine (10mg/kg q8h) or omeprazole (2.5-5mg/kg q24h). Gastric protectants, such as bismuth subsalicylate (1ml/kg q8h) or sucralfate (100-200mg/kg q12h) are also used. Supportive care, such as intravenous fluids and assist-feeding are also helpful. If ferrets are vomiting, they can be held off food for 12-24 hours. These ferrets should be kept on IV fluids with dextrose in case of concurrent insulinoma. Broad spectrum antibiotics (especially those that are effective against Helicobacter) are also used.

Diarrhea and Cachexia

Chronic or acute diarrhea may cause ferrets to present on emergency because of weakness and lethargy associated with electrolyte disturbances. Chronic disease can result in weight loss and cachexia that will cause a patient to present for emergent treatment. Acute diarrhea can be from bacterial enteritis caused by Salmonella spp., and Campylobacter jejuni. Clostridial organisms are often seen in low numbers in feces but have been associated with enteritis and colitis when present in large numbers and are sporulated.
Salmonellosis is a contagious disease in ferrets characterized by fever, bloody diarrhea, and lethargy. The incidence in pet ferrets is low and the infection maybe associated with feeding uncooked meats. *C. jejuni* has been associated with diarrhea and enterocolitis in many species, but can be found in the feces of normal ferrets, so the significance as a pathogen in ferrets is unknown. Diagnosis is with fecal cultures. Treatment is with supportive care and broad-spectrum antibiotics.

Chronic diarrhea is usually the results of inflammatory bowel disease or viral infections. Inflammatory bowel disease is relatively common in adult ferrets and can progress to cause severe dehydration and lethargy. Clinical signs include loose stools, intermittent nausea, weight loss and occasionally vomiting. The cause is unknown but may be related to dietary intolerance, hypersensitivity reactions or aberrant immune response. The inflammation is usually lymphoplasmacytic but can also be eosinophilic enteritis. Definitive diagnosis requires gastric and intestinal biopsies, but a suspected diagnosis can be made based on clinical signs and history to rule out exposure to ECE, toxins, etc. Abdominal ultrasound may show thickened bowel and enlarged lymph nodes. Aspirates of these lymph nodes reveal inflammation. Routine blood work may reveal lymphocytosis, elevated liver enzymes and hyperglobulinemia. Treatment is aimed at controlling the immune response and dietary management. Prednisolone and azathiaprine are treatment options. Hypoallergenic diets used in cats are also indicated.

Coronavirus can cause severe diarrhea, especially in younger and older ferrets. This disease is highly transmissible and is often brought into a group of ferrets by an asymptomatic juvenile animal. Clinical signs begin 2-14 days after introduction of the new ferret or exposure through fomites and include anorexia, vomiting, green or mucoid diarrhea, melena, dehydration, lethargy and weight loss. The virus causes blunting of the intestinal villi and consequent maldigestion and malabsorption. Definitive diagnosis is difficult but a presumptive diagnosis can be made from history and clinical signs. Treatment is supportive with fluids, nutritional support, broad spectrum antibiotics, and gastrointestinal protectants.

*Lawsonia intracellularis* can cause a proliferative bowel disease, especially in younger ferrets. Signs include diarrhea, weight loss, and rectal prolapse. Treatment is with chloramphenicol (25mg/kg po q12h) for 14-21 days.

**RABBITS**

**Anorexia**

Dental disease is a common cause of anorexia in rabbits. All teeth in rabbits are open-rooted and grow 10-12cm/year. Incisor malocclusion is usually noted by the owners earlier and is not often an emergency presentation but malocclusion of the cheek teeth is harder to recognize. These rabbits may appear hungry but drop food or stop chewing quickly. Points occur most commonly on the buccal surface of the upper arcades and lingual surface of the lower arcades because of the jaw alignment. The cheek teeth may be examined using an otoscope or speculum in many awake rabbits but sedation or anesthesia may be required for a thorough view. Treatment is by occlusal adjustment with a low-speed dental drill or hand files. Points may be removed with ronguers. The rabbits are then supported with meloxicam, fluids and assist-feeding until eating on their own.

The most common problem associated with anorexia in rabbits is gastric stasis which is often related to diets low in indigestible fiber or high in carbohydrates. Rabbits may also develop stasis secondary to stresses such as changes in the diet or environment or dental disease. Other clinical signs include dehydration and the formation of small, hard fecal pellets. The rabbits are typically bright and alert despite these problems, unless the stasis has been prolonged. A firm, dough-like stomach may be palpable on physical examination. Radiographs usually show a soft tissue mass in the stomach surrounded by a halo of gas and excess gas in the intestines.
Treatment for gastric stasis is rehydration, force-feeding, and motility modifying drugs. Rehydration of the patients as well as the stomach contents is important in the treatment of gastric stasis. Subcutaneous fluids (50-100 ml/kg/d) can be administered in the hospital or at home by the owner. Oral fluids, such as water or electrolyte replacement mixture, should also be given using a syringe. This helps to rehydrate the stomach material for easier passage. Force-feeding the patient a high-fiber diet, such as Critical Care Formula (Oxbow Products), mixed vegetable baby food, canned pumpkin, or soaked alfalfa pellets will help to stimulate gastric motility and place the animal in a positive nitrogen balance. Additionally, fresh greens and timothy or grass hay should be available for the rabbit to eat on its own. Metaclopramide (0.5 mg/kg SC q 8 hr) or cisapride (0.5 mg/kg PO q 12 hr) are used to stimulate gastric and intestinal motility. Radiographs are recommended before using these drugs to rule-out gastric obstruction. Most rabbits respond to this medical care in 1-2 days, although occasionally rabbits may take longer to return to normal food intake. Surgical treatment for gastric stasis is rarely indicated.

Acute gastric obstruction can occur in rabbits and can be caused by a hairball, foreign body, or pyloric disease. These rabbits usually present extremely depressed, weak, and may be in shock. Treatment of the metabolic condition and decompression of the stomach are necessary if these rabbits are to be saved. The stomach tube is passed to remove excess gas and fluid and then flush the stomach. A large red rubber tube or other large bore tube works well for this. The rabbit should be sedated or anesthetized (usually via mask with isoflurane or sevoflurane) unless the animal is severely recumbent. Be prepared for a drop in heart rate while passing the tube over the glottis, this usually resolves once you get past this area. Once the tube enters the stomach, remove as much gas and fluid as possible with gentle palpation and gentle suction. Then flush the stomach with warm (100°F) water until it is clean or blood is noted. This often dislodges the pyloric/duodenal blockage and no further treatment of the GI tract is needed. Metabolic disturbances caused by the blockage still need to be treated aggressively with IV fluids, colloids and monitoring and correcting blood gas and electrolyte abnormalities and blood pressure changes. Radiographs are useful in making this diagnosis and determining the presence of foreign body or pyloric disease. Keep the rabbit NPO for 12-24 hours, but on IV fluids with dextrose, then slowly start to feed blenderized pellets or Critical Care Diet. The prognosis is guarded with 50-60% survival. Surgical treatment with removal of the foreign body have been used after gastric flushing with about 50% overall success rate. A more recent paper used subcutaneous fluids, subcutaneous metoclopramide and feeding 2-3ml every two hours with an 80% success rate. We have tried this on a few rabbits with success.

Torticollis

Acute head tilt in rabbits can be caused by peripheral or central vestibular disease. Peripheral disease is usually caused by head trauma or otitis media from bacteria such as Pastuerrella, Staphylococcus and Streptococcus that travel through the Eustachian tube. Central disease can be caused by E. cuniculi, cerebral larval migrans, and trauma. Clinical signs are similar and include head tilt, circling, rolling and nystagamus. The nystagamus is generally vertical with central disease and horizontal with peripheral disease. Diagnosis of peripheral disease can be with radiographs or CT scan. Central disease is more difficult to definitively diagnose although a negative E. cuniculi titer can help rule that disease out. Treatment is with antibiotics for otitis, usually enrofloxacin (20mg/kg PO SID) or penicillin (60,000 units SC EOD). E.cuniculi is treated with albendazole or oxybendazole (20mg/kg PO SID). Cerebral larval migrans are treated with ivermectin. Anti-inflammatories are helpful in all these cases. Usually meloxicam (1mg/kg PO SID) but in severe cases prednisolone (1mg/kg PO BID, tapering dose) has been used. Meclizine (12.5-25mg per rabbit PO BID) can help with rolling and nausea. A solid flooring with good traction and padding on the sides of the cage can help rabbits that are rolling. Syringe feeding may be necessary initially. Prognosis is guarded if rolling continues. Many rabbits will have a permanent head tilt but learn to walk normally.

Respiratory Disease

Rabbits with respiratory disease may present on emergency with either upper or lower respiratory disease. URD is commonly called “snuffles” because of the sonorous respirations heard over the nasal cavity and trachea. Affected rabbits may be lethargic and inappetant. Mucopurulent discharge may also be found on
the nose or medial aspect of the forearm as the rabbit grooms its face with the forepaws. Conjunctivitis may occur if the organism travels up through the nasal-lacrimal duct. A serous nasal discharge can occur with allergies and irritations but is not usually mucopurulent. Treatment is based on culture and sensitivity with topical and systemic antibiotics. Ophthalmic drops can be used intra-nasally or placed in the eye to travel through the nasolacrimal duct into the nasal passages. Nebulization of antibiotics and mucolytics may be helpful in severe cases.

Pneumonia has few distinct clinical signs in the early stages of the disease. Typically, affected rabbits exhibit non-specific signs such as weight loss, anorexia, and lethargy. Dyspnea and tachypnea are not typically seen until there is severe loss of respiratory function. Rabbits showing dyspnea generally have a poor prognosis unless managed intensively. Auscultation may reveal areas in which lung sounds are absent because of lung consolidation or abscesses. Radiographs are diagnostic. Lung aspirates for culture and sensitivity can be performed. Treatment is with systemic antibiotics and nebulization of antibiotics, mucolytics and potentially bronchodilators. Long-term therapy is often required.

**RODENTS**

**Guinea Pigs**

Gastric stasis and dental disease occurs in guinea pigs similarly to rabbits, though lingual entrapment is much more common in guinea pigs because of their dental anatomy. These animals are usually interested in food but drop it from the mouth partially chewed because they can’t swallow. Urinary tract calculi can occur in the kidneys, ureters, bladder or urethra of guinea pigs. Most stones are calcium based, but struvite stones have also been reported. High levels of calcium in the diet (alfalfa hay, alfalfa pellets, etc) may cause increased calcium excretion and result in uroliths. Guinea pigs normally have a basic urine (pH 8) but an acidic urine caused by diet or disease processes may facilitate the precipitation of calcium crystals. Clinical signs of urolithiasis include hematuria, dysuria, and a hunched posture. Uroliths in the bladder and ureter can often be palpated and are typically visible radiographically or ultrasonographically. Treatment is by surgical removal of the stones; however, owners should be warned that recurrence is common. Treatment with sodium or potassium citrate (30mg/kg PO BID) to increase urinary pH and inhibit calcium accumulation may reduce recurrence or be helpful with small stones in the kidneys or ureters. Systemic anti-inflammatories before and after surgery or medical management are helpful (meloxicam 0.5-1mg/kg PO SID).

Urinary tract infections may be caused by crystaluria or stress. These animals will have pollakiuria and hematuria but no stones will be visible on radiographs or ultrasound examination. Urine cultures are warranted and systemic antibiotics and anti-inflammatories are helpful. Sterile cystitis has been seen and responds well to NSAIDs.

Dystocia is also a common emergency presenting complaint for guinea pigs. Dystocia can also be caused by obesity, uterine inertia, and large fetal size (small litter size). Clinical signs of dystocia are prolonged contractions and straining. Pups are normally born quickly within approximately 30 minutes with a 5 minute resting period between pups. Therefore, a sow seen to be straining for over 60 minutes should be considered abnormal. Sows may become depressed after continued straining. A greenish brown or bloody mucoid discharge is often present. A partially extruded fetus may be seen in severe cases. Diagnosis is by history and clinical signs.

Treatment depends somewhat on the cause of dystocia. The pelvic symphysis should be palpated to determine the amount of separation. If the space is less than 25mm then immediate Cesarean-section should be performed. If the symphysis is wider than 25mm and uterine inertia is suspected then oxytocin should be given at 0.2-3.0 units/kg IM. If oxytocin fails to stimulate uterine contractions in 15 to 20 minutes then a C-section is indicated. Inhalants should be used as the only anesthetic agent and should be delivered by tank induction and maintained by a face mask. Preparation and surgical time should be minimized to increase the chance of survival of the sow and pups. The surgical procedure and pup care is similar to other species. If
the sow is stable, an ovariohysterectomy should be performed to prevent recurrence. Guinea pigs may not tolerate anesthesia and surgery and the owners should be given a poor prognosis for the sow and the pups.

**Chinchillas**

Chinchillas are adapted to a mountainous environment with a typical ambient temperature of 65-80°F; therefore, exposures to temperatures above this range for even small periods of time may result in heat stroke. High humidity is also a contributing factor. Chinchillas with heat stroke are often laterally recumbent, tachypneic, and have a body temperature above 103°F. The mucosa may be brick red and a thick, ropy saliva, diarrhea or hematochezia may also occur. Treatment is by slowly cooling the animal using lukewarm water bottles or tepid baths. It is important not to cool the animal too quickly or hypothermia and seizures may result. Intravenous or intraosseous fluids are beneficial both for the cooling effect and to treat the hypovolemic shock common with heat stroke. Acute renal failure, brain damage, and sloughing of the intestinal mucosa can occur within 24-48 hours of heat stroke, therefore animals should be hospitalized and monitored closely during that time.
As a veterinary professional, you change the lives of pets and their people for the better by easing pain, relieving the suffering and even saving lives. You take on enormous responsibility and face ethical dilemmas, often with life changing consequences. You work hard physically and emotionally to make a difference every day. While the rewards for such work are great, there is a personal cost to this level of caring that needs to be thoughtfully managed – keeping a mindful balance between our heart and our head.

Several perspectives, communication skills, personal awareness tips and exercises are presented in this discussion. What resonates with each individual will vary - focus on what is relevant for you at this particular time. A different skill or tip may be fitting at a future point.

The first premise: expect shifts in balance within your world, after all life is a continuum of adjustment and correction. Don’t fear being out of balance however it is important to recognize the shift, understand the cause and have strategies in place to manage these shifts before the pressure becomes too great. The key is constant adjustment and keep moving forward.

Often, our stress is due to the reactions of the heart (our fears, worries, what we imagine, emotional responses to differences or the unexpected) versus the reality and objective view of the situation. You have control over both and more control than you may think.

We are wired to take our inherent social styles, preferences, experiences, perspectives and all other factors that create our world and impose this perspective on others which can lead to false assumptions, explanations that make sense only to us or concerns as we see them. We see the world not as it is but as we are. We may even go as far as viewing the situation as our way or the wrong way! So the first step is to gather enough information to recognize and appreciate the difference between our perception, the view of others and/or reality.

Communication Skill: When faced with a difference (conflict) – be curious! Make it a habit to ask more questions than stating your views. Tread lightly until you have a more complete understanding – seek first to understand and then to be understood. Use open-ended questions or statements (encourages more than a yes or no response) to hear the other person’s story. With more information, you may find that what is obvious to you is not even seen by others or what you think are concerns for others is not accurate, etc. Once you have an understanding of the difference, clearly state the two positions and define a common objective. Use “and” instead of “but” to express the two positions: “You are concerned that Smokey has an allergy that is causing his hair loss and I would like to make sure it is not due to a hormonal imbalance. We both want to find the appropriate treatment for Smokey so let’s talk about how we can accurately diagnose what is causing his hair loss.” Now, replace the “and” with “but” to see how this can diminish the owner’s view.

Another common source of stress is the “icky” conversation that is usually more about managing the emotions (yours and those of others) rather than dealing with the facts. We are wired to think rationally or react emotionally but typically we are not able to do both at the same time. Recognize the physical signs that indicate you are moving into the emotional (heart) capacity instead of your logic (head) abilities. If you
experience some of the common emotional signals such as accelerated heart rate, trembling, sweating, tension in stomach then your goal is to find a way to stay in your rational mindset.

Communication Skill: To keep your emotions at bay here are a few suggestions to stay factual: write down the facts of the other person’s story while they are speaking; ask fact based questions; imagine that an audience is watching your interaction; or keep in mind that you will have to paraphrase your understanding of the issue back to the other person. Take away the emotional component, often the answer becomes clearer.

You also have to try to move the other person into a rational thought process. Start by acknowledging the emotion (frustration, anger, disappointment, etc) of the other person before getting into the facts. Look for facts in their stories, ask fact based questions, speak to specific events or behaviours, remain goal-oriented to help avoid defensiveness. Remember that anger is a secondary emotion – it is the result of the person feeling another emotion such as fear, embarrassment, guilt, anxiety or feeling judged.

Personal Awareness tip: You are not perfect! Our medical training and work environment often feed our compulsive, perfectionistic tendencies. Less than perfect is NOT a failure. Set a realistic goal for yourself and share this with colleagues and clients – this removes the fear of letting people down. It is impossible to know everything and our own personal resources are limited however continual improvement and complementing your resources with others is a healthy expectation. Asking for assistance is not a weakness or a threat to your independence. Repeating the same error and persisting in an unhelpful manner is a weakness. Often you know what you want or need but there is a fear about expressing this – focus on what is stopping you from asking.

Personal Awareness tip: Do you take on the work and/or emotional burden of others (pet owners, colleagues, employees, family, friends)? Being in a helping profession, caring is what we do. It is unhealthy caring when it is done at the expense of yourself. Recognize the boundaries of your responsibility and control. Ask yourself: What can I control and what is beyond my control? Am I solely responsible for this situation or not? Issues are much smaller when you don’t take on someone else’s burden. When an issue is beyond your control – let it go!

Learn to say “no.” We often worry that saying no is being selfish, others will get upset with us, it goes against teamwork or you may simply have a strong desire to please others. Realistically, how effective are you when you have spread yourself too thin? You can say no without being rude especially when it is a request that keeps you from doing what you should be doing or what is a priority. Acknowledge the person’s request and their reasons for asking. Say no with an honest, brief reason for declining. Offer to come up with other solutions to help.

Personal Awareness tip: Do you suffer from the Good Guy or Good Gal complex? Do you feel pressure to provide free goods or services, discounts, regularly provide service to clients by phone or personally outside of your scheduled hours, partake in considerable charitable or welfare work that interferes with family or personal time? When you are devoting so much time to your profession, what other areas are suffering? If you excel in all areas – great but be careful not to base your self-acceptance on how others view you or whether your clients accept your recommendation. Be careful that you are not handing over your happiness to someone else that is, solely basing your happiness on how happy you make others.

Communication Skill: Learn to deal with criticism or you will always be victim to the approval of others. Be clear on your own set of personal values or principles as the foundation of all your decisions. Example: I will always be an advocate for the pet and consider what is in the pet’s best interest given family circumstances? You can still empathize with your client’s situation and hold a different approach. Unfortunately, some may criticize or demean you. You have NO control over this side of the conversation however you CAN control how your react to these comments. The reality is that not everyone will agree with you or like you. It is often more important that they respect you.
Personal Awareness tip: Reframe your thinking away from you and focus on what you are doing for the client and the pet. How will they both benefit from your recommendation versus what the client thinks about you. Instead of thinking that you are taking money away from a client, think of the benefits and the value you are providing for them.

Personal Awareness tip: Be clear on your own personal values. Values are lasting beliefs or ideas about what is good or bad and desirable or undesirable. Value are the standards for behaviour and attitude. A solid consistent application of personal values brings clarity to decision-making, helps to prioritize time and resources, and can resolve tension in conflictual situations.

Personal Awareness tip: We all make mistakes or do something we are sorry for. Know the difference between regret and guilt. Regret is a result of knowing that something could have been done in a better way from the way it was actually done. You are sorry and don’t intend to repeat the same action. Guilt arises from doing some action that we know was not the right thing to do ‘at that particular time’. This can be an emotion that does not go away and may immobilizes you unless you identify the specific reason and commit to make a positive change by resetting your moral compass. In both cases, it is best to accept the you did something wrong, apologize, make changes in the future and move on.

“To achieve balance, you must become un-balanced!
Growth in one area of life may require another area to slide slightly ... temporarily.”

Larry Wenger (“Shut Up, Stop Whining and Get A Life”)
ESTABLISHING DIAGNOSIS IN CATTLE:
PHYSICAL EXAM
Allen J. Roussel, Jr., DVM, MS, Diplomate, ACVIM, ECBHM
Department of Large Animal Clinical Sciences, College of Veterinary Medicine & Biomedical Sciences, Texas A&M University, College Station, Texas

OVERVIEW OF THE PRESENTATION

I fear that the physical examination is becoming a lost art. Perhaps every generation of veterinarians has had the same feeling as they see more and more technology enhancing our ability to reach a diagnosis, but at the same time replacing some of the time-tested techniques of the physical examination. I am certainly not against technological advances-most of us at academic institutions are drawn there because of the advanced diagnostic equipment available to us. I am, however, chagrined by the growing dependence upon imaging, laboratory evaluation, and other sophisticated techniques to make a diagnosis when often a physical examination and a very simple confirmatory test would reach the same conclusion in less time and for less cost. My goal in this presentation is to review the techniques of physical examination both for the part-time bovine veterinarian as well as the experienced bovine veterinarian. There is no question that the combination of excellent physical examination and rational use of sophisticated diagnostic equipment will achieve the optimal results.

We often hear the term “complete physical examination,” but how often do we perform one? The truth of the matter is that we do not need to perform a complete physical examination on every patient, nor do we have the time. We routinely perform what might be called a “standard physical examination” which includes a brief review of all important body systems. Based on the history and the results of the standard physical examination, we then perform one or more focused physical examinations. If we performed every one of the focused physical examinations that we knew, we would then perform a “complete physical examination.” But let’s not argue over semantics. Let’s try to learn how to efficiently evaluate an animal by use of the standard physical examination and how to focus on particular areas to gain the most information possible from a physical examination.

There are many ways to approach a physical examination; many correct ways. The approach that I will use in this paper is to begin with observation at a distance and then examination of the restrained animal. I’ll then discuss the acquisition of vital signs and basic auscultation, concluding with regional focused examinations beginning at the head. Because neurological examination is frequently difficult and confusing, I’ll spend a bit more time on that aspect.

THE EXAM AT A DISTANCE

I believe that physical examination of cattle should begin with observation of the animal from a distance. This is particularly important when one suspects neurological or musculoskeletal disease. The animal should be observed at rest for several minutes and then in motion. Note the general condition of the animal and the breed, as some neurological diseases are heritable. When the animal is at rest, pay particular attention to the animal’s awareness of its surroundings which reflects cerebral function. Note if the animal is depressed, hyper-excited, or otherwise responsive to external stimuli, if it is head pressing, wandering aimlessly, vocalizing abnormally, behaving abnormally or aggressively. Diseases such as polioencephalomalacia, lead poisoning, nervous ketosis, bovine spongiform encephalopathy, rabies, brain or pituitary abscess, nervous coccidiosis, and salt poisoning/water deprivation cause these signs. Before the animal is disturbed, observe the character and rate of respiration, look for a jugular pulse (indicative of right heart failure), and for signs of abdominal pain like bruxism, restlessness, kicking at the belly, or straining. Also look carefully for muscle fasciculation, twitching of the ears or eyelids, tail position and switching and abnormal attempts at
swallowing which may indicate nervous system or metabolic disease such as hypomagnesemia, lead toxicity, tetanus, or rabies. Lameness is often detectable in cattle at rest by observing how the animal bears or shifts weight on the limbs. An easy way to assess weight bearing is to observe how far the dewclaws are from the ground. If the dewclaws are higher on one side, the animal is not bearing full weight on that side. Abdominal contour should also be assessed at a distance and from behind the animal. While the animal is in the open and not confined in a chute, careful attention should be paid to the muscle mass, particularly over the rump and hindquarters. In unilateral neurological disease, as well as chronic upper limb lameness, atrophy of the muscles will occur, and asymmetry of the muscles will be obvious.

If the animal is recumbent, observe if and how it rises. It is best to observe an animal in motion as it moves away from and towards the examiner, as well as from each side. To optimally evaluate gait, it should move at its own pace with only slight prompting from an assistant. It should be driven and not led (unless it is very well halter broken) so that the head and neck are free to move. The carriage of the head and neck sometimes give important clues about neurological disease. Observation should be carried out from directly behind the animal and then from each side, with particular attention being paid to the carriage and placement of the legs, to ability of the animal to walk in a straight line, to knuckling, and to other signs of weakness. If hind limb ataxia is suspected, the animal should be pulled from side to side by the tail so that the examiner can assess if the animal is able to place its back feet under itself correctly. Another important observation to make when the animal is moving is to assess its vision. The menace response can be misleading in cattle, particularly young cattle. Therefore, cattle suspected of blindness should be moved through a maze or an obstacle course where they will have to turn to avoid running into objects. In this way their visual capacity can be properly assessed.

THE EXAM UP CLOSE

The next part of the physical examination is conducted with the animal restrained in a head chute. The order in which most of the examination is conducted is not important except that the rectal exam should be conducted at or near the end of the examination. The following description is the sequence that I usually follow. In dairy cattle particularly, it is often important to collect urine to check for ketonuria. This can most easily be accomplished without catheterization if it is done before the cow is “disturbed” by the physical examination. Stroke the vulva or perineum without touching any other part of the cow. Bulls will often urinate if their sheath is grasped at the orifice and “shaken” vigorously for 30 seconds. If the animal is lying in a stall and rises when the examiner approaches, it will frequently urinate and defecate spontaneously. Rectal temperature, pulse and respiration should always be measured. If the examiner stands on the left side of the animal while taking the temperature, rumen motility can be assessed simultaneously. After measuring and recording the temperature and assessing rumen motility, auscult the heart for rate, rhythm and murmurs. Remember that in order to auscult the heart, the head of the stethoscope must be pushed cranially behind the elbow and humerus. This is especially true in heavily muscled beef cattle. Next, listen to the lung fields and record the respiratory rate. Reference ranges for mature cattle are as follows: RR (12-36 bpm); HR (50-80 bpm); rectal temp (100.5-102.5°F, 38-39°C); Rumen contractions (2-3 in 2 minutes). For calves, these values are: RR (20-50 bpm); HR (90-112 bpm); rectal temp (101.4-103.4°F, 38.5-39.5°C) It is important to remember that lung sounds in cattle are usually quieter than they are in horses and small ruminants. Therefore, careful attention must be paid to detect abnormalities. The most frequent change in the lung sounds of cattle (except feedlot cattle perhaps) is simply an increase in the normal breath sounds which is caused by tachypnea. Heart failure, pulmonary disease, excitement, exertion, or elevated body temperature (which may be due to infection, exertion or high environmental temperature) may cause tachypnea. Except for pulmonary disease and pulmonary edema secondary to left heart failure, all of these other conditions will cause a simple elevation in respiratory rate and effort which is accompanied by louder-than-normal sounds, but which is not accompanied by crackles, wheezes, increased bronchial sounds or areas of dullness. In my experience the most frequent abnormal lung sound is increased large airway or bronchial sounds which are indicative of lung consolidation. It is a misconception that consolidated bovine lungs produce areas of dullness on auscultation. Often severe pneumonia in cattle is accompanied simply by increased large airway sounds but not crackles and wheezes. If areas of diminished or absent lung sounds are noted, one should
suspect pleural effusion or lung abscess. It is critical to differentiate between true lung sounds and upper airway (nasal, laryngeal, pharyngeal and tracheal) sounds. Referred upper airway sounds can be heard loudly in the thorax, but if one listens over the trachea and pharyngeal area, the sounds are louder. Also, most sounds associated with breathing that are audible without a stethoscope are associated with the upper airway. Inspiratory sounds are almost always associated with a narrowing of the lumen of the upper airway. Audible grunts are occasionally heard, and these are consciously made sounds that usually reflect pain or severe disease that may not involve the respiratory tract. In young cattle, percussion of the thorax may help detect lung consolidation or pleural fluid, but this technique has been of limited value to me in older cattle, particularly beef cattle. After ausculting the thoracic cavity, move to the abdominal cavity and perform simultaneous auscultation and percussion (pinging) on both sides of the abdomen. Tests for abdominal pain can be conducted at this point. These include the withers pinch test and the xyphoid pressure test. The withers pinch test is performed by abruptly and firmly squeezing the animal rights dorsal midline over the withers. The interpretation of the test is as follows: the animal ventro-flexes and grunts-positive for cranial abdominal pain; the animal ventro-flexes and does not grunt-negative for cranial abdominal pain; animal neither ventro-flexes or grunts or shows signs of discomfort- inconclusive results.

### Table 1. Assessing hydration in calves (from Walker and Constable, JAVMA, 1998)

<table>
<thead>
<tr>
<th>% dehydration</th>
<th>0</th>
<th>2</th>
<th>4</th>
<th>6</th>
<th>8</th>
<th>10</th>
<th>12</th>
<th>14</th>
</tr>
</thead>
<tbody>
<tr>
<td>eyeball recession (mm)</td>
<td>0</td>
<td>1</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>6</td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td>skin tent duration (secs)</td>
<td>2</td>
<td>3</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
<td>8</td>
<td>9</td>
</tr>
</tbody>
</table>

After completion of examination of the thoracic and abdominal cavities, one moves to the head of the animal. Hydration is best assessed by measuring eyeball recession and tenting of the skin of the neck. The values for assessing dehydration (Table 1) have been validated for calves by Constable, et al, but not for mature cattle. Anecdotally, I feel that the values for skin tent are probably similar in calves and cattle.

In suspected neurological cases, it is very important to do a thorough examination of the head, mouth and neck region. Begin by observing the animal from directly in front. One can observe the positions of the ears, eyelids, lips and eyeballs. After observing the animal’s head, the examination of the head in neck begins by noting the temperature of the ears. Cold ears indicate hypocalcemia or shock. Look in the ears for otitis externa. The oral examination follows. One should always be mindful of rabies before examining the mouth of cattle, especially those suspected to have neurological disease, choke, or bloat. Gloves and protective clothing should be worn before examining the mouth of any animal with central nervous system disease. Look at the lips, gums, dental pad, hard palate and tongue for color, vesicles and ulcerations. Gingival mucous membrane color and capillary refill time are much more difficult to assess and interpret in cattle than in horses. Vulvar mucous membrane pallor is usually easier to detect (except in bulls & steers!). While examining the gums, check the incisors for eruption, color, wear and soundness. Grasp the tongue (a towel helps) and pull it to one side assessing consistency and muscle tone as you do. Look for ulceration, foreign body or ranulae on the underside of the tongue. Examine the cheek teeth for wear, points, attrition or overgrowth and the buccal mucosa for lacerations, ulceration or blunting of the papillae. "Impacted cud" may be in the cheek or under the tongue. Pull the tongue to the other side and repeat. Smell the breath and oral cavity for a necrotic odor, ammonia or ketones. Visual examination of the oropharynx can sometimes be accomplished with the use of a speculum (like a Drinkwater gag) and a flashlight. The torus lingua makes
visualization of the pharynx difficult in some cases. Be ready to catch brief glimpses, especially when the animal bellows. Retropharyngeal masses, perforations, ulceration, and laryngeal lesions may be observed in this manner. Optimal visualization of the pharynx, larynx and esophagus is obtained by endoscopy. Traumatic pharyngitis (usually iatrogenic), necrotic laryngitis, chondritis, etc. can be visualized by nasal endoscopy. (Note: bovine nasal passages are smaller relative to body weight, than equine.) The esophagus can be examined for ulceration, laceration, choke, etc. Unlike the equine stomach, the ruminant forestomachs and abomasum cannot be examined by easily endoscopy. To examine the throat manually, insert the hand into the mouth while pushing the tongue between the cheek teeth nearest you. Do not keep your arm in the mouth for too long as the animal cannot breathe and may struggle and bite.

CRANIAL NERVE EXAM

Proceed with a systematic evaluation of the cranial nerves, beginning with the second cranial nerve. A menace response can be elicited in cattle, as with other species, by moving a hand or other object toward the eye. A positive response is blinking of the eye with or without an attempt to move. One must be very careful when examining cattle to not create wind with the hand or other object toward the eye. A positive response is blinking of the eye with or without an attempt to move. One must be very careful when examining cattle to not create wind with the hand or other object, as this may give a false menace response. Animals that cannot see can still perceive the movement of the hand and may react to the air movement on the eyelashes. Also, in young calves, many normally visual calves will not have a menace response. The menace response is a “learned” response and they don’t perceive the need to flinch yet. The menace response assesses the optic nerve (II), the cerebral cortex and the facial (VII) nerve. The optic and oculomotor (III) nerves are involved in the pupillary light reflex (PLR). To evaluate the PLR, with the animal in a dark place, shine a bright light into each pupil and observe that pupil, as well as the pupil in the other eye. If the pupil into which the light is shined constricts, then the direct PLR is intact. This means that cranial nerves II and III, as well as the retina, on that side are intact. If the pupil in the other eye also constricts, then the consensual PLR is intact. This means that in addition to cranial nerves II and III on the first side, cranial nerve III on the opposite side is also intact. The same procedure is repeated for the other side. If the PLR is intact, but there is no menace response, the lesion is in the cerebral cortex. This occurs in polioencephalomalacia and lead poisoning. The position and movement of the eyeball is under the control of the oculomotor, trochlear (IV), and abducens (VI) nerves. Dysfunction of these nerves results in strabismus. The most common and important clinical strabismus in cattle is probably the dorso-medial strabismus associated with some cases of polioencephalomalacia.

The trigeminal nerve (V) provides sensation to the face and motor function to the muscles of the jaw. Pulling the jaws apart and assessing the strength of the muscles assesses the function of the masseter muscle. In order to assess sensation of the head and face, place a piece of straw into the nasal cavity or gently touch the eyelashes. When the lashes are touched, the animal should blink her eye; this is the palpebral reflex. Another cause of an absent or delayed palpebral reflex is lactic acidosis in diarrheic calves. The mechanism for this is unknown. The following table is a guide to interpreting the menace response, PLR and palpebral reflex.

<table>
<thead>
<tr>
<th>Reflexes involving the eye</th>
<th>Absent</th>
<th>Absent</th>
<th>Absent</th>
<th>Present</th>
</tr>
</thead>
<tbody>
<tr>
<td>Menace</td>
<td>Absent</td>
<td>Absent</td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td>PLR</td>
<td>Absent</td>
<td>Present</td>
<td>Present</td>
<td>Present</td>
</tr>
<tr>
<td>Palpebral reflex</td>
<td>Present</td>
<td>Present</td>
<td>Absent</td>
<td></td>
</tr>
</tbody>
</table>

**INTERPRETATION**

- II or retinal deficit
- Cerebral cortical deficit
- VII or orbicularis oculi muscle deficit
- V deficit

Cattle with facial nerve paralysis will have a drooped ear, ptosis, and atonic lips. Occasionally saliva will drip out of the affected side of the mouth. Facial paralysis is seen most often with listeriosis and ear infections.
The vestibular system is composed of the auditory nerves and ganglia, and the vestibular apparatuses in the middle ear. Clinical signs associated with dysfunction of the vestibular system include head tilt, circling and loss of balance. Cattle with unilateral lesions tilt their heads and lean toward the lesion; recumbent cattle lie with the lesion side down. If the lesion is peripheral (such as an otitis interna), horizontal nystagmus will be present, and the slow phase is toward the lesion. Central lesions are associated with a vertical or rotary nystagmus. A combination of dysfunction of cranial nerves VII and VIII can be seen in either brainstem disease (listeriosis) or peripheral disease (ear infection) because the facial nerve passes through the petrous bone via the internal acoustic meatus with the auditory nerve. Cattle with deficits of the glossopharngeal, (IX) vagus (X) and spinal accessory nerves (XI) usually present with abnormal vocalization, dysphagia (trouble swallowing or regurgitating out of the nose) or abnormal breathing sounds. Pharyngeal paralysis can usually be assessed best by allowing the cow or animal to eat or drink. Sometimes this is difficult because sick cattle will not eat, especially in a strange environment. Alternatively, water can be administered via a dose syringe and the cow’s ability to swallow can be assessed. Endoscopic examination is helpful in determining if nerve dysfunction exists. The hypoglossal nerve supplies motor innervation to the tongue. The tongue’s function is assessed by grasping it and pulling it out and to each side. Unilateral lesions may result in the tongue sticking out one side of the mouth.

OTHER TIPS

After the completion of the examination of the head, one should palpate the neck concentrating on the submandibular lymph nodes, the skin and musculature of the neck and trunk, and finally the superficial cervical or prescapular lymph nodes. These lymph nodes should be approximately as big as one’s index finger. When examining the rear legs, the prefemoral lymph nodes should be palpated. Dependent edema, indicating circulatory failure or hypoproteinemia, will be noted when the ventral part of the jaw, thorax and abdomen are examined. When moving from the head to the rear of the animal, move your hand over the back to detect subcutaneous emphysema, parasites or neoplasm, and skin lesions. Particular attention should be paid to the joints of the rear legs. Palpation of the stifle joint and hock joint may reveal accumulation of synovial fluid. Palpation of the stifle joint is best accomplished by first locating the middle patellar ligament, then sliding the fingers medially until the next hard structure is encountered. This is the medial patellar ligament. There should be a depression in the space between the ligaments. Similarly, a soft depression should exist between the middle patellar ligament and the lateral patellar ligament. If the ligaments are difficult to palpate because the space between these ligaments is filled and very firm to the touch, then there is substantial distention of the stifle joint. This can be seen in both septic and non-septic conditions such as rupture of the cranial cruciate ligament. One easy way to differentiate between foot lameness and upper leg lameness or neurologic disease of the rear limbs is by lifting the rear legs. Animals with a foot lesion will often kick violently when the back leg is lifted, while those with upper leg lameness and neurological disease will not. Occasionally, lameness and neurological disease can be confused, especially by owners.

While standing behind the animal just prior to rectal exam, the last part of the neurological exam can be conducted. Tail tone can be assessed by picking the tail up and noticing the ability of the animal to clamp down, and by assessing the tone of the anus and sensation around the perineal area.

THE NON-REPRODUCTIVE ABDOMINAL EXAMINATION PER RECTUM

The rectal examination is an extremely valuable diagnostic technique, seldom appreciated until the practitioner is faced with a challenging G.I. case in a 300 pound calf or a sheep. The key to garnering the most from a rectal exam is in knowing which structures are usually palpable and which are not. There is no standard accepted sequence in which the abdomen is examined per rectum, so I will simply discuss each quadrant beginning with the left dorsal and proceeding clockwise. Before beginning, let me point out 3 things that should always precede a rectal exam:
1. Rectal temperature measurement. Cattle often develop pneumorectum following a rectal exam.

2. Pinging. Pneumorectums ping, confusing the examiner.

3. Collection of feces for occult blood (if indicated). Certainly not a routine procedure, so try to think ahead. After a rectal, false positives may occur for 24 hours or more.

Now we are ready:

1. Pelvic exam. The pelvis has many ridges and bumps that become more prominent when you palpate with the suspicion of a pelvic fracture. Remember that cattle have a large prominent symphysis pubis and a step-like sacroiliac junction. Fractures or luxations can best be identified by rocking or walking the animal. Patient compliance is essential. Pay particular attention to crepitus and asymmetry. There are several lymph nodes in the pelvis that may go unnoticed in a healthy cow, but become very enlarged in lymphosarcoma.

2. Left Dorsal Quadrant. The dorsal sac of the rumen is palpable several centimeters cranial to the pelvic brim. Sometimes the rumen extends to the pelvic canal. Size, consistency, gas caps and relative position of the rumen can be assessed. LDA’s are not palpable per rectum, but the rumen may feel displaced medially. The left kidney is usually midline but may be to either side depending on the fullness of the rumen.

3. Right Dorsal Quadrant. The right kidney lies cranial to and to the right of the left kidney and the caudal pole can be felt in some cattle. The aorta and vena cava run along the dorsal midline and can be palpated. Occasionally the cecum in a healthy cow will be in or near the pelvic brim, but usually it cannot be identified. Spiral colon and small intestines are not palpable in a normal cow. In cattle with obstructions, they may be subtly or obviously distended, but any palpable intestine (other than cecum) is abnormal. RDA is seldom palpable but abomasal volvulus is often palpable. Torsed abomasae are palpable at the furthest extent of the reach in the right mid-to-dorsal quadrant.

4. Right Ventral Quadrant. Distended intestines may be palpated here and are interpreted as in the RDQ. A distended, displaced or torsed cecum is usually palpated easily in the right ventral or dorsal quadrant or in the pelvic canal. It is much more caudally situated than an RTA. In vagal indigestion, the right ventral sac of the rumen is prominent. It is occasionally palpable in normal cattle.

5. Left Ventral Quadrant. Usually, only the ventral sac of the rumen is present. The urinary bladder may lie on either side of midline beneath the uterus, but it is usually flaccid and not always easily located.

Other abnormalities that can be detected by rectal examination include pneumorectum which results in the rectum being tightly adhered to the arm like a sleeve, while the examiners arm and hand seem more freely movable than usual. Acites may cause the rumen to float. Adhesions due to peritonitis may give a feeling of roughness to the serosal surfaces, or may create a tearing sensation as immature fibrinous adhesions are broken down, or may severely restrict movement in the abdomen if extensive firm adhesions are present. Of course, a complete physical examination is not warranted in every case but it is important to know how to perform one for those cases in which the diagnosis is not obvious.
One of the most important concepts is that of the "normal or reference range." The reference range is ideally based on a large number of samples from a population similar to the patient population and is calculated to theoretically include 95% of the healthy population. To be truly valid, the reference range should be established using the equipment and techniques that will be used on the patient population. "Book values", while useful for most hematologic parameters, are of limited use for interpretation of serum chemistries. Most references are established for "cattle": not dairy cows in the United States, or calves in Muleshoe, TX. Where possible, we have included some of the important differences that exist among classes of cattle.

Remember that the reference range is intended to include 95% of the healthy population; therefore, 5% of the population will have values outside the range for any single test. When one performs a battery of tests, the probability of at least one of them being outside the reference range is far greater than 5%. Over interpretation of a single value that falls slightly outside the reference range should be avoided. Finally, if a laboratory value just doesn't look reasonable in the context of the case, it probably isn't correct. Call the lab, rerun the test in your office, or submit another sample. Never base a clinical decision on a laboratory value you have trouble believing.

SERUM PROTEIN

Serum contains many different proteins, but the two components of diagnostic significance relative to the chemistry profile are albumin and globulin. Albumin is synthesized in the liver and is the protein primarily responsible for the oncotic pressure of plasma. A large portion of the globulin fraction is made up of immunoglobulins which are synthesized by lymphoid tissue. The ratio of albumin to globulin (A:G ratio) is fairly constant in healthy cattle (reference range 0.84 -0.94). Most chemistry profiles include measured albumin and total serum protein, while the value for globulin is typically derived by subtracting the former from the later. One must remember that if plasma is used instead of serum, fibrinogen will be included in the value for total protein, and hence, the derived globulin value; this may obfuscate the interpretation of the total protein and globulin. The discrepancy cannot be totally rectified by simply subtracting the value for fibrinogen, which is often determined by refractometry, from the value for plasma protein on the profile because of the difference in methods of analysis. Because most reference ranges are established for serum proteins, it is our opinion that serum is the sample of choice when evaluating the blood proteins. More specific and detailed evaluation of the globulin fraction can be achieved using electrophoresis, radial immunoassay and other methods which will not be discussed here.

Hyperproteinemia can result from an increase in albumin, globulin or both. The only cause of hyperalbininemia is dehydration. In dehydration, both albumin and globulin rise, but whether they exceed the reference range is determined by the degree of dehydration and the original protein concentration in the serum. Hyperproteinemia without dehydration is almost always the result of hyperglobulinemia. Globulin increases substantially with age in dairy cows. The difference between two year-olds and five year-olds was about 1.5 g/dl, potentially a clinically relevant difference. Causes of hyperglobulinemia include chronic inflammatory diseases (traumatic reticuloperitonitis, liver abscess, chronic pneumonia) and hepatic disease. In chronic inflammatory disease, the A:G ratio usually decreases because of an increase in globulin which is often accompanied by a small decrease in albumin. In chronic hepatic disease, the decrease in albumin may be more substantial. Serum globulin may be one of the most overlooked values on the routine chemistry profile. Changes in the hemogram are often rather subtle and transient in inflammatory disease of cattle,
compared to other species. Therefore, the evaluation of serum globulin is of great value in chronic inflammatory disease.

In mature cattle, hypoproteinemia is usually the result of hypoalbuminemia or panhypoproteinemia. Hypoalbuminemia occurs when 1) hepatic production is insufficient to meet demand, either as a result of insufficient production or increased consumption or 2) there is excessive loss of albumin. Insufficient production can occur in animals with chronic severe hepatic disease or as a result of inadequate protein intake, digestion, or absorption. Because bovine albumin has a half-life of 16.5 days and the reserve capacity of hepatic tissue is so great, liver disease must be chronic and severe to result in severe hypoalbuminemia. In the authors' experience, cattle with chronic debilitating disease of many causes may be hypoalbuminemic with low or normal total protein. If the A:G ratio is low, chronic inflammatory disease should be suspected. In acute and subacute disease, hypoalbuminemia frequently results from loss of albumin. Avenues of albumin loss include the kidney (particularly the glomerulus), the gastrointestinal tract, hemorrhage and exudation. In many instances, loss of albumin may be accompanied by loss of globulin, resulting in panhypoproteinemia.

Renal amyloidosis can result in severe albumin loss in the urine due to glomerular damage. In one report, 5 of 6 cattle with amyloidosis had hypoglobulinemia along with hypoalbuminemia. Panhypoproteinemia is the rule in protein-losing gastro-enteropathies such as nematode parasitism, paratuberculosis and salmonellosis. Because digestion and absorption may be impaired in these diseases, decreased production due to amino acid deficiency may contribute to the hypoproteinemia in chronic cases. Acute hemorrhage results in panhypoproteinemia accompanied by anemia.

Hypoglobulinemia is infrequent in cattle except neonates, either as a result of failure of passive transfer of maternal antibody, or severe infection when transferred antibodies are consumed rapidly prior to the efficient production of endogenous antibody by the young calf.

**HEPATIC TESTS**

The leakage enzymes aspartate transaminase (AST, formerly SGOT), L-iditol (formerly sorbitol) dehydrogenase(IDH), ornithine carbamoyltransferase (OCT), glutamate dehydrogenase (GDH) and lactate dehydrogenase (LDH) have been used to evaluate the liver. Of these, AST, LDH and IDH are the most popular in the United States. Both AST and LDH are found in a wide variety of tissues, the most important of which are liver and muscle. Muscle damage, especially due to recumbency in cattle, may result in marked increases of both; hence, AST and LDH should be interpreted in conjunction with a liver-specific enzyme (such as GGT), or a muscle-specific enzyme such as creatine kinase (CK) to determine the source of the tissue insult. Usually, high AST or LDH and normal CK indicates liver disease. If serum is allowed to remain on the clot too long or the sample is hemolyzed, the AST and LDH may be falsely elevated because both enzymes are found in red blood cells. Because concentrations of these enzymes are high in serum when damaged cell membranes allow their escape from hepatic cytosol, they indicate cell damage, not abnormal hepatic function. In fact, in chronic or slowly progressive hepatic disease, these enzymes may be within or below reference ranges because few hepatocytes are being damaged at one time, or because hepatocellular mass is substantially reduced. Consequently, these enzymes may be more sensitive indicators of acute disease such as some toxicities and infectious hepatitis. They may also be high in cattle with hepatic lipidosis, passive venous congestion and diseases that cause distension of the forestomachs and abomasum. IDH is a sensitive and specific indicator of hepatocellular damage. Unfortunately, its usefulness is limited by its relatively instability in vitro.

The "cholestatic", enzymes gamma glutamyltransferase (GGT) and serum alkaline phosphatase (SAP), are more sensitive to biliary obstruction caused by conditions such as fascioliasis or cholelithiasis. The cholestatic enzymes are more likely to be high in chronic hepatic disease than are the leakage enzymes because fibrosis constricts and blocks some bile ducts.

Although GGT is found in many tissues, the source of essentially all of the GGT in the serum is the biliary and hepatocellular membranes. Therefore, it is one of the most liver-specific tests available to veterinarians. Serum GGT rises principally in cholestatic disease, although hepatocellular diseases in which cholestasis is a
secondary feature, also causes an increase in GGT. Because it tends to decrease less rapidly than the other leakage enzymes, it may be of more value in identifying cattle with chronic hepatic disease. Serum GGT of precolostral calves is similar to that of mature cattle, but serum concentrations rise sharply following consumption of colostrum, which is rich in GGT. By 24 hours after colostral intake, serum GGT concentration is 50 to 100 times that of colostrum-deprived calves. In fact, serum GGT can be used to estimate the success of passive transfer, but not to detect hepatic disease in neonates.

Serum alkaline phosphatase, a useful indicator of hepatic or cholestatic disease of dogs, is often included in chemistry profiles of cattle. Several isoenzymes from different tissues have been identified, and almost all of the SAP in healthy cattle is of osseous origin. In cattle with hepatic disease, SAP of hepatic origin increases, but the increase is not large in magnitude. Therefore, SAP is of limited diagnostic value for hepatic disease of cattle. Interestingly, though not clearly explainable, SAP was found to be useful as a prognostic indicator in cattle with abomasal volvulus.

Bilirubin is a breakdown product of hemoglobin that is conjugated and excreted by the liver. Unconjugated (or direct) bilirubin is the result of rapid breakdown of hemoglobin which occurs in acute hemolysis. Conjugated (or indirect bilirubin) accumulates in plasma when there is intra- or extrahepatic biliary obstruction. The plasma concentration of bilirubin in healthy cattle is very low compared to that of the other species; and the magnitude of increase is relatively small, even in severe liver disease. Severe bilirubinemia and icterus in cattle is almost always a result of hemolysis, and hence, is primarily due to unconjugated bilirubin.

Though usually considered an index of renal function, blood or serum urea nitrogen (BUN or SUN) is also an indicator of hepatic function. In the liver, ammonia is converted to urea. In severe hepatic failure or partial vascular anomaly, SUN is low while ammonia is high. However, low SUN is not associated only with hepatic disease. Because rumen microbes use urea to synthesize protein, the rumen acts as a "sponge" for urea in cattle that are anorectic or protein-deprived.

Laboratory reference ranges for mature cattle are invalid for neonatal calves, especially those under a week of age. Neonatal calves have somewhat higher concentrations of bilirubin, AST, SAP, and SBA, and markedly higher concentrations of GGT than do mature ruminants.

**ELECTROLYTES**

The serum electrolyte profile typically includes sodium (Na), potassium (K), chloride (Cl), and total carbon dioxide (TCO₂) or bicarbonate (HCO₃). From these values, the anion gap (AG) can be calculated. Although there is a nominal difference between the TCO₂ and HCO₃, the HCO₃ usually being slightly smaller, we will consider them equivalent in this paper. Serum electrolytes are useful in the evaluation of several body systems, as well as for the formulation and monitoring of fluid and electrolyte therapy. Due to the abundance of K and scarcity of Na in erythrocytic fluid relative to serum, hemolysis can falsely increase serum K and decrease serum Na in cattle.

Because their concentrations change in concert in a number of conditions, the electrolytes will be discussed together. Sodium is the major extracellular cation, while Cl and HCO₃ are the major extracellular anions. Chloride and HCO₃ often maintain a reciprocal relationship in extracellular fluid. Because the majority of the exchangeable Na and Cl are found in the extracellular fluid, measuring serum Na and Cl provides an accurate assessment of the total body status of these electrolytes. Serum potassium, on the other hand, provides a less reliable and sometimes paradoxical reflection of total body K status because only a small portion (approximately 5%) of the animal's K is in the extracellular fluid. Changes in blood pH greatly affect serum K by causing the movement of K across cell membranes; K moves into cells during alkalosis and out of cells during acidosis. Therefore, serum K should be interpreted along with serum HCO₃. Serum HCO₃ is a measure of metabolic acid-base balance; concentrations above the reference range indicating metabolic alkalosis and those below indicating metabolic acidosis.
Hypernatremia and hyperchloremia occur in salt toxicity/water deprivation, but are not commonly present in cases of dehydration because typically fluid loss in cattle occurs with concurrent loss of electrolytes. Cattle with clinical "salt toxicity" may have normal serum Na because clinical signs often do not occur until after the cattle drink, and serum Na concentration and osmolality return to normal. Hyperkalemia is almost always secondary to acidosis as K moves out of the intracellular fluid into the extracellular fluid. Therefore, serum K is an unreliable index of total body K. For example, diarrheic calves often are acidic and hyperkalemic, but they have total-body K depletion because of fecal K loss. In these cases, as in most cases where serum K is increased secondarily to acidosis, K supplementation may be indicated during or immediately following correction of acidosis. Hypochloremia, hypokalemia, metabolic alkalosis, and, to a lesser degree, hyponatremia, are typical findings in obstructive gastrointestinal diseases including abomasal volvulus, displaced abomasum, vagal indigestion, intussusception and cecal torsion. In these diseases, HCl is sequestered in the abomasum, causing hypochloremia, metabolic alkalosis, and secondary hypokalemia. In general, the more orad the lesion (abomasal impaction vs jejunal intussusception), and the more complete the obstruction (abomasal volvulus vs LDA), the more severe the alkalosis and hypochloremia. Hypochloremia and metabolic alkalosis are fairly non-specific abnormalities in sick cattle however. In a study of over 500 mature cattle in the authors' hospital, over 40% of the dehydrated cattle were hypochloremic and/or alkalotic.

Serum Na and Cl are consistently low in uroperitoneum, and often are low in diarrhea and renal failure, while serum HCO₃ is variable in these conditions. Urinary obstruction and uroperitoneum are associated with hyperkalemia in non-ruminant species, but not in cattle.

RENAL TESTS

Elimination of nitrogenous wastes, such as urea and creatinine (Cr), and concentration of urine to conserve body water are two of the many vital functions performed by the kidney. Evaluation of these functions is exploited in the diagnosis of renal disease. Serum or blood urea nitrogen (SUN or BUN) and serum creatinine (Cr) are rough indices of glomerular filtration rate. The generous reserve capacity of the kidney makes SUN and Cr insensitive indicators of renal function; 75% loss of functional renal mass is required for azotemia to occur. Slightly more sensitive than SUN and Cr, the urinary specific gravity (USG) can detect about a 67% loss of functional renal tissue. The USG is most easily estimated by refractometry. By convention, USG of > 1.025 is considered indicative of appropriate concentrating ability in the face of dehydration or azotemia. It is quite common, however, for normally hydrated cattle, especially dairy cattle, to have USG < 1.025.

Azotemia, the accumulation of nitrogenous wastes in the blood, is reflected in the serum chemistry profile as high SUN and Cr. Remember - AZOTEMIA DOES NOT EQUAL RENAL DISEASE! Azotemia can be classified as renal (due to renal disease), prerenal (due to sluggish renal blood flow, as in shock or dehydration), or postrenal (due to obstruction of urine outflow, as in urolithiasis). Though not without exception, the simplest way to distinguish among the three is by measuring the USG. In azotemic cattle, if the USG is > 1.025, the azotemia is prerenal; if the USG is < 1.025, the azotemia is renal. In postrenal azotemia, urine is often difficult or impossible to obtain, and the diagnosis is based on physical examination. In our experience, cattle with prerenal azotemia eliminate urea and Cr rapidly when rehydrated, often returning to or near the reference range in 24-48 hours if appropriate fluid therapy and correction of the primary problem is accomplished.

Urea is formed in the liver by the detoxification of ammonia, a by product of protein metabolism. Therefore SUN is influenced by diet and hepatic function. Urea is recycled in a functional rumen, a process which may tend to moderate the rise in SUN in renal disease and result in a low SUN/Cr ratio. Although Cr, a product of energy metabolism in muscle, can be very low in emaciated cattle with little muscle mass, it tends to be less influenced by extraneous factors than SUN. For this reason, Cr is the test of choice over SUN.

While SUN, Cr and USG can identify renal disease, the final diagnosis cannot be obtained from this information. For example, acorn toxicity, pyelonephritis, and amyloidosis are diseases which cause renal failure in cattle. These diseases cannot be distinguished from one another simply based on the results of the chemistry profile.
However, the characteristics of the urine in each of these diseases is very different. Whenever renal disease is suspected, a complete urinalysis should be performed, as well as, rectal palpation of the kidneys. Ultrasonography and renal biopsy may also be informative.

GLUCOSE

Glucose metabolism is unique in ruminants because they absorb essentially no pre-formed glucose from the gut. The reference range for serum glucose in adult cattle is lower than for calves and non-ruminant species. Erythrocytes metabolize glucose in vitro in a blood tube at a rate of about 10% per hour at room temperature. Serum should be separated from the clot within 30 minutes, or sodium fluoride-containing tubes should be used if timely separation is not possible. Hyperglycemia occurs in stress, milk fever, and following administration of dextrose solution, xylazine, or corticosteroids. It is interesting that most milk fever remedies contain dextrose, even though hypocalcemia prevents the release of insulin from the islet cells of the pancreas resulting in hyperglycemia. Endogenous and exogenous steroids increase gluconeogenesis and increase serum glucose. Xylazine causes a dose dependant hyperglycemia that persists for over 6 hours. Diabetes mellitus although uncommon in cattle, causes permanent hyperglycemia if untreated.

MUSCLE ENZYMES

As previously mentioned, LDH and AST are released into plasma as a result of muscle damage, but they are not muscle-specific enzymes. Serum CK, on the other hand, is a very sensitive and specific indicator of muscle damage. Subtle increases can occur due to intramuscular injection, exercise or struggling. Recumbent mature cattle may have >100-fold increases due to the secondary pressure damage that is a part of the downer cow syndrome. Very high concentrations of CK in the absence of recumbency, or in young recumbent cattle suggest primary myopathy, such as white muscle disease or Senna toxicity. The half-life of CK in serum is short, and CK concentrations fall rapidly in recumbent animals even if they remain recumbent. Because AST concentration rises and falls more slowly, it can be used in combination with CK to stage muscle damage. In recumbent cattle, if the AST is very high and the CK is not, the damage is likely several days old. In an attempt to use laboratory tests for prognosis, New Zealand investigators found that fewer than 5% of cows with an AST value > 7.4 times the upper limit of the reference range survived. For CK, the "critical" value above the reference range was related to the duration of recumbency.
Cattle caregivers have exciting obligations, responsibilities, and opportunities to contribute to cattle well being. Shifting caregiver priorities from disease detection to performance enhancement results in new levels of cattle welfare.

Webster defines welfare as health, happiness, and general well being. We have a responsibility to provide cattle with physical comfort, disease protection, nutritional needs, and emotional stability. Veterinarians understand that physical and psychological stress play important roles in cattle disease resistance and performance levels. Caretakers can be trained to realize that all human contact with cattle impacts animal well being. Human contact can either create a very positive impact or can create an impact that can devastate cattle health, performance and cattle and human safety.

Veterinarians and managers must improve their abilities to train caretakers to encourage cattle to communicate their true state of health. Understanding predator-prey relationships is the foundation for successful cattle handling and the development of communication with cattle - which enables early detection of disease.

Cattle exhibit very strong prey animal instincts. Prey animals have survived in nature aware that predators select the lame, depressed, and weak to harvest. If caretakers behave like predators, cattle will hide signs of depression and disease from these people as long as possible. Understanding more about the visual, auditory and sensory abilities of cattle encourages handlers to override their predator tendencies to chase and yell.

Handlers that reward cattle motion with release of pressure can quickly train cattle. This creates mutual respect and trust between themselves and the cattle. Understanding that cattle like to see what is pressuring them and like to see where they can go is fundamental to low stress handling. Cattle that trust handlers, volunteer to move away from handlers and will walk straight away and move as directed. This attitude of willingness has a positive effect on herd social interaction. Sensitive cattle are more content and timid cattle are more willing to compete for feed and water. Consistent handler communication that creates voluntary cattle movement and an attitude to train cattle reduces stress levels in cattle and people.

Caretakers can have a positive impact on cattle health, performance, and well-being through effective low stress handling at key interventions like calving, tagging, grazing, weaning, processing and shipping. Caretakers that concentrate on low-stress handling skills recognize abnormal behavior and attitude and develop the confidence and skill to manipulate behavior to improve levels of animal welfare.

BRD CASE DEFINITION
Tom Noffsinger, DVM

Notes not available
Overview of the Issue

Otitis externa (OE) is defined as inflammation of the external ear canal and is a common condition in small animals.\textsuperscript{1-2} OE is due to primary, predisposing and perpetuating factors. Primary factors account for the underlying etiology e.g. allergic skin disease. Predisposing factors are present prior to the development of otitis and perpetuating factors occur as a result of inflammation and include secondary infections.\textsuperscript{1} The most commonly isolated pathogens in cases of infectious canine OE are \textit{Staphylococcus pseudintermedius}, \textit{Pseudomonas aeruginosa} and \textit{Malassezia pachydermatis}.\textsuperscript{1}

Diagnosis of Otitis Externa

Diagnosis of OE is based on history, clinical signs, otoscopic examination, cytology and possibly cultures from the external ear canal. In any case of OE, cytology must be performed to diagnose any secondary infections.\textsuperscript{1} There is discrepancy between studies as to how many organisms constitute an infection versus normal flora. For gram-negative rods, even small numbers constitute an infection as they are not normal flora of the ear canal.\textsuperscript{3-4} When inflammatory cells are noted on cytology, this is a significant finding and the number of organisms is irrelevant.\textsuperscript{1} In cases where gram-negative rods are visualized, a bacterial culture maybe recommended; as well as in cases where previous antibiotics have been ineffective.\textsuperscript{3,5} If an oral antibiotic is selected, for cases of otitis media (OM), these culture results will guide the choice of oral antibiotic. With OE, there is debate as to whether systemically administered antibiotics will reach the desired concentration within the external ear canal. Unless the ear canal epithelium is ulcerated, systemic antimicrobials are unlikely to reach therapeutic concentrations and should be reserved for cases of suspected OM.\textsuperscript{6} For most cases of OE, topical therapy will be used. Response to topical medication does not often correlate with susceptibility testing results due to the fact that topical medications will reach much higher concentrations within the ear. Therefore the choice of topical antimicrobial should not be based on the culture results.\textsuperscript{6} There is some discordance noted between otic cytology and culture results so always interpret findings in light of cytological findings and clinical signs.\textsuperscript{7} A thorough otoscopic examination is an important diagnostic tool.

Treatment of Otitis Externa

Topical medications must be able to reach the surface of the ear canal. In cases with excessive debris, flushing of the ear canal is warranted.\textsuperscript{8} Ear cleaners can be prescribed with instructions to properly clean the ear canal. There are many products available including ceruminolytics, anti-inflammatories and antimicrobials, some of which may be the only treatment required.\textsuperscript{9}
Multiple otic medications are available and many contain antibiotics, antifungals and anti-inflammatory agents. Antimicrobials for canine OE are often selected empirically based on cytology. Current debate over the application of first and second line antimicrobials is ongoing. When cocci are noted on cytology, antimicrobials with action against coccoid bacteria are appropriate. When rods are noted on cytology, otic medications including gentamicin, polymyxin B and enrofloxacin can be considered for treatment. A topical ticarcillin preparation was found to successfully treat *Pseudomonas* otitis in a small numbers of cases.  

Tromethamine edetate disodium dihydrate (Triz-EDTA® Aqueous flush, Dechra Veterinary Products, Kansas, US) is commonly used as an adjunct therapy for dogs with *Pseudomonas* otitis. It has been documented that flushing the canal with Triz-EDTA® 15 minutes prior to instilling a topical antimicrobial agent is beneficial in resolving gram negative infections. Studies suggest that a chelating agent may be beneficial in cases of *Malassezia* OE. Neomycin and gentamicin may not always be successful in clearing OE as they are inactivated in purulent material, so are best selected when there is scarce pus within the ear canal. For acute *Pseudomonas* OE, polymixin B is an appropriate choice. For chronic cases, topical fluoroquinolones or aminoglycosides may be required. For treatment of *Malassezia* otitis, clotrimazole, miconazole and nystatin are commonly found in otic medications. Resistance to antifungals has only rarely been reported and there is little evidence for *in vitro* resistance.  

Antifungal medications can be compounded to contain solely an antifungal. For inflammatory OE, topical glucocorticoids can aid in decreasing inflammation. In one study a 0.1% Tacrolimus solution was instilled into the ears of dogs without otitis and was well tolerated. With further research this could be an option for inflammatory otitis. For cases of chronic proliferative OE, consider Triamcinolone injections as a salvage procedure prior to total ear canal ablation.

**Bacterial biofilms and otitis**

A bacterial biofilm is a community of bacteria that form layers of bacterial cells and then become irreversibly attached to a surface. The biofilm produces a matrix made of carbohydrates, proteins and DNA. This protects the bacteria from dessication, host immune response and antimicrobials, serving to increase antimicrobial resistance and immune system evasion. A previous study shows 40% of *Pseudomonas* otic isolates form biofilms and once a biofilm has formed, this increases the MIC against certain antimicrobials. As with planktonic cells, studies have shown that Triz-EDTA® used in combination with an antimicrobial, can reduce the MIC and minimum bactericidal concentration (MBC) for certain antibiotics against biofilm embedded bacteria.

**5 KEY treatment tips:**

1. In cases of OE with stenotic ear canals, anti-inflammatory therapy with oral glucocorticoids maybe needed for 7-14 days before otoscopic examination or treatment with topical medication is successful. Most cases of OE will benefit from either topical or oral anti-inflammatory treatment with glucocorticoids.
2. Ceruminolytic ear cleaners should be used for cases with excessive cerumen accumulation.
3. Demonstrate to clients how to clean the ear and apply medication. Rechecks every 2-4 weeks depending on length/treatment selected. At these rechecks repeat cytology to determine whether treatment is working. Continue treatment until clinical and cytological resolution.
4. Often owners apply 1-2 drops into ears, must be stressed that this volume is not enough to coat ear canal. Consider using ml volume instead of drops.
5. Always address underlying etiology.
References/Suggested Reading


Overview of the issue

Food allergies (cutaneous adverse food reactions; CAFR) can manifest as pruritus, recurrent secondary infections, alopecia, erythema and up to 32% of patients will present with concurrent gastrointestinal signs. The majority of animals developing cutaneous adverse food reactions have been on a diet containing the offending allergen for years. This can make diagnosis difficult. To date, there is a lack of correlation between clinical food allergy and laboratory tests for food allergies. Confirmation of food allergy can only be determined by a novel protein/hydrolyzed restriction diet trial.

Diagnosis:

There is currently no single test that can be run to diagnose CAFR in companion animals. Diagnosis is based on history, clinical signs and exclusion of other causes of pruritus and dermatologic disease such as ectoparasites. There appears to be no sex predilection for CAFRs in dogs and cats. Top breeds associated with food allergy include Labrador retrievers and cocker spaniels, along with others described in the literature including, Soft-Coated Wheaton terrier, dalmatian, West-Highland white terrier, Bichon Frisee, collie, Shar Pei, Lhasa apso, Dachshund, miniature schnauzer, boxer, springer spaniel, Cairn Terrier, Irish / English setter, Golden retriever, German shepherd dogs, along with Siamese and Birman cats. Age at presentation is typically less than one year (2 months to 16 years; 33–52%) in dogs. In cats, typical age at presentation is less than 2 years of age (4 months to 15 years; 38.5%).

The most common clinical sign of food allergy is non-seasonal pruritus. In rare cases there may be a direct correlation of onset of pruritus with a dietary change or indiscretion, but this is most often an exception to the rule. Food allergic patients may respond temporarily to antimicrobial therapy as secondary infections are treated, but pruritus will return, +/- lesions, at the termination of therapy. Greater than 60% of patients with food allergies have a minimal to absent response to anti-inflammatory doses of glucocorticoids.

If a patient is thought to have food allergies, part of the history taking should include questions regarding previous diets fed, treats and table scraps given, flavoured medications such as monthly flea preventatives, flavoured toys/chews, pill vehicles and flavoured toothpastes. Questions should also be directed to any gastrointestinal disturbances occurring such as stool consistency, flatulence, number of bowel movements, etc.

In dogs with CAFR, the ears are most consistently involved (80%) followed by the feet (61%) and the inguinal/ perineal region (53%) (think ears, feet and rears). In 24% of dogs, the ear may be the only affected region of the body. Many dogs will also have dorsal pruritus and if this pruritus extends past the thoracolumbar region, this will increase the suspicion of an CAFR in this author’s opinion.

CAFR in cats can present similarly to dogs. However, cats may also present with manifestations of the eosinophilic granuloma complex, symmetrical alopecia and miliary dermatitis. Studies have previously
tried to document regions on the cat most commonly affected by AFR and it does appear that the head and neck are more commonly affected than the ears, feet and rear end.

Up to 32% of patients with AFR will present with concurrent gastrointestinal signs, as well as cutaneous signs. These include vomiting, changes in the stool consistency, increased frequency of bowel movements, halitosis, borborygmus, flatulence, tenesmus, eosinophilic or lymphocytic-plasmacytic colitis / IBD, anal gland impaction & scooting, pica and/or coprophagia.

**Treatment:**

Confirmation of food allergy can only be determined by a restriction diet trial. The diet is changed to one with a combination of ingredients to which the animal has no previous exposure. The diet is then restricted to exclude ALL OTHER treats, table scraps, pilling vehicles, flavoured medications and toothpaste and flavoured toys. Protein sources are more often to blame than grains for CAFR, so selecting a novel protein with no history of exposure is paramount. Food items most commonly causing food allergy include beef, milk, lamb, wheat, corn, chicken egg, soy, chicken in dogs, adding tuna and salmon to the list in cats. The gold standard of performing a restricted diet trial is to use a home-prepared diet. Diet trials should be undertaken for a period of 8–12 weeks to determine response. Veterinary diets are preferable over diets obtainable from a pet store based on concerns regarding ingredient contamination and the cleaning process. Whether to select a novel protein diet or a hydrolyzed diet is a common question. In multiple studies analyzed, up to 50% of dogs with CAFR enrolled exhibited increases in clinical signs after ingesting partial hydrolysates from foods to which they were hypersensitive. Although limited studies are available there is evidence to suggest reduced immunological and clinical allergenicity of hydrolysate-based commercial diets. However, a proportion of dogs with CAFR will exhibit a worsening of clinical signs when fed partial hydrolysates. After 8-12 weeks on the diet, when improvement is noted, dietary challenges can be undertaken to introduce food items back into the diet if wished.

**5 KEY “TAKE HOME” POINTS:**

1. An underlying CAFR should be considered in any pet that develops pruritus / clinical signs prior to 6 months and after 6 years of age, with no previous history of skin disease.
2. The majority of dogs who develop an CAFR have been on a diet containing the offending allergen for years.
3. Greater than 60% of patients with food allergies have a minimal to absent response to anti-inflammatory doses of glucocorticoids.
4. A restricted diet trial with either a novel protein diet (commercial or home cooked) or a hydrolyzed diet are the best way to diagnose an underlying CAFR.
5. Foods can be reintroduced one at a time during dietary challenges.

**References/Suggested Reading**


Overview of the Issue

Frustration can arise when treatment for a particular disease fails to lead to clinical improvement. Other cases may initially improve, but then show a decline. Secondary infections, development of another disease or adverse drug reactions can all lead to presumed treatment failure. In these cases of “treatment failure”, diagnostic steps should be revisited and further diagnostics may be needed.

Key Diagnostic steps:

When faced with a patient not responding to appropriate therapy, there are multiple steps to take to find out why therapy is not working. First and foremost, client compliance must be checked. The correct dose and frequency of administration must be verified. If the treatment failure involves topical therapy, the owner should be asked how they have been applying or using the topical to make sure application is correct.

In allergic patients with previously controlled pruritus, who re-present for pruritus +/- dermatologic lesions, cytology should always be performed to check for the presence of a secondary infection. Secondary infections (bacterial pyoderma and Malassezia dermatitis) are common reasons for perceived treatment failure as they mask the effects of anti-inflammatory therapy. Demodecosis or other ectoparasites must also be ruled out via skin scrapings as they can lead to a worsening of clinical signs in a previously stable patient. An animal receiving high doses of glucocorticoids can develop demodecosis. Antibiotic resistance has received much attention over the past few years with the incidence of MRSP increasing dramatically. Any animal with a bacterial pyoderma not responding to an appropriate dose and selection of antibiotic, should have an aerobic bacterial culture performed.

Doses of medications and length of treatment should also be revised when dealing with a case of treatment failure. For example, an animal receiving glucocorticoids for atopic dermatitis presents for non-pruritic hair loss. This hair loss may, in fact, be due to the long-term use of glucocorticoids as opposed to the atopic disease.

Approach to treatment failure:

Treatment failure is most often reported when there is a lack of response to glucocorticoids, cyclosporine or oclacitinib in a presumed atopic dog. If this occurs, there could be an underlying food allergy and therefore it is imperative to perform an adequate novel protein (or hydrolyzed diet) restricted diet trial. The owner must be informed that no treats, table scraps, pilling vehicles or flavoured medications can be used during the diet trial. The diet trial should involve a diet with a novel protein that the animal has not been exposed to previously and should last for 8-12 weeks to determine whether an improvement is noted.
Cutaneous adverse drug eruptions can lead to a worsening of clinical signs. For example, a dog with a secondary bacterial pyoderma due to allergic skin disease, receives a cephalosporin antibiotic and then develops further dermatologic lesions consisting of erythema, alopecia etc. This animal could be exhibiting signs of a cutaneous adverse drug reaction as opposed to a flare-up of allergies. If pruritus occurs primarily in one location where topical products are applied, one must also consider a contact dermatitis. In an animal previously responsive to glucocorticoids but now the beneficial effect is waning, consider steroid tachyphylaxis (“steroid fatigue”). The steroid can be switched to another glucocorticoid e.g. oral dexamethasone, Methylprednisolone. If there is a lack of response to a correct dose of cyclosporine, cyclosporine levels can be checked. However, in atopic dogs there does not appear to be a correlation between blood concentrations and clinical response. If the lack of response to glucocorticoids, cyclosporine and oclacitinib, in a patient with atopic dermatitis, is definitively proved and cytology, skin scrapings etc are all negative, one must consider either a case of pruritus refractory to conventional therapy or an incorrect diagnosis. At this point diagnostic steps should be retraced and revised if necessary. Skin biopsies sent for histopathology can also be a great way to help eliminate and identify a diagnosis. Immune mediated disease can usually be diagnosed via histopathology.

Other dermatological diseases, such as cutaneous epitheliotropic T cell lymphoma, can present with clinical signs similar to atopic dermatitis but are non responsive to conventional anti-inflammatory therapy. If histopathology is consistent with atopic dermatitis then more aggressive immunosuppressive therapy may be needed in these refractory cases.

Prior to beginning systemic immunosuppressive therapy, all patients should have full bloodwork performed (complete bloodcount and serum biochemistry) as well as a urinalysis. Azathioprine has been used in certain cases to treat refractory canine atopic dermatitis at a dose of 2-2.5 mg/Kg once daily. Further bloodwork is also recommended every 2-4 weeks after starting therapy due to the potential adverse effects such as myelosuppression and hepatic toxicity (increase in ALT, ALP).

Pruritus can originate from regions other than the skin. Pruritus maybe associated with pain or can also be neuropathic. Studies are lacking to document efficacy of the following medications for treatment of pruritus but could be considered in specific cases. Gabapentin is used in humans to treat neuropathic and uremic pruritus. Side effects include sedation and ataxia. Maropitant is a neurokinin-1 receptor antagonist that inhibits substance P. This medication can have an anti-pruritic effect. Maropitant is licensed to prevent and treat vomiting in dogs as a dose of 2 mg/kg once daily. This dose has also been suggested to decrease pruritus.

**5 KEY “TAKE HOME” POINTS:**

1. In any case where treatment is not efficacious, cytology and skin scrapings should be performed and client compliance should be verified.
2. Consider potential antibiotic resistance in a patient with bacterial pyoderma, confirmed on cytology, which is unresponsive to antibiotics.
3. Taking skin biopsies and sending for dermatohistopathology can provide more information in cases of treatment failure.
4. In certain pruritic cases consider non-conventional therapy or changing the type of glucocorticoid prescribed.
5. In a pruritic patient non responsive to therapy for atopic dermatitis, consider food allergies or cutaneous epitheliotropic T cell lymphoma as an underlying etiology.
References/Suggested Reading


Pain Review

American College of Veterinary Anesthesia and Analgesia (ACVAA)

- Useful website: acva.org
- Has a position statement regarding the treatment of pain in animals:
  - "believes that animal pain and suffering are clinically important conditions that adversely affect an animal’s quality of life, either in the short or long term."
  - "endorses a philosophy that promotes prevention and alleviation of animal pain and suffering as an important and tenable therapeutic goal."
- What do these statements mean?
  - Pain matters
    - It is clinically important, whether it is physical pain, or emotional suffering
  - Prevention of pain when possible, and alleviation of pain once it is present, is a viable goal for veterinarians

Pain Physiology

- The basic nociceptive pathway is a three neuron chain
  1. First order neuron
     - Originates peripherally and projects to the spinal cord
     - Consists of a primary afferent fibre that is activated by a noxious stimulus
  2. Second order neuron
     - Ascends the spinal cord
  3. Third order neuron
     - Projects into the higher brain structures (somatosensory cortex)

There are four key physiologic components to the pain pathway:

1. Transduction
2. Transmission
3. Modulation
4. Perception

**Transduction**

- Nociceptors in the periphery change mechanical, chemical, or thermal energy into electrical impulses
- Nociceptors are the free nerve endings of primary afferent neurons
  - Signal actual or potential tissue injury

**Transmission**

- Electrical impulses are carried to the dorsal horn of the spinal cord by A alpha (Aα), A beta (Aβ), A delta (Aδ), and C fibres
- Transmission by Aδ and C fibres are most important from the pain perspective
- Transmission by Aδ fibres, which are relatively large diameter and myelinated fibres, is very rapid
  - Results in sharp “first pain”
- Transmission by C fibres, which are small diameter and unmyelinated fibres, is much slower
  - Results in dull “second pain”
- Both Aδ and C fibres are afferent nerve fibres found throughout skin, peritoneum, pleura, periosteum, subchondral bone, joint capsules, blood vessels, muscles, tendons, fascia, and viscera

**Modulation**

- Nociception is first amplified or suppressed at the level of the dorsal horn of the spinal cord
- Secondary amplification or suppression occurs at the level of the brain
- Amplification is facilitated by:
  - Excitatory amino acids and peptides
    - Aspartate, glutamate, substance P
  - Excitatory receptors
    - NMDA (N-methyl D-aspartate), AMPA (α-amino-3-hydroxy-5-methyl-4-isoxazole-propionic acid), and KAI (kainate)
- Suppression is facilitated by:
  - Endogenous opioids
    - Endorphin, dynorphin, encephalin
  - Inhibitory receptors
    - GABA (gamma amino butyric acid), glycine
  - Exogenous opioids and other analgesic drugs (local anesthetics for example)

**Perception**

- Involves integration, processing, and recognition of sensory input
  - Occurs at the level of the brain, via several neural pathways – this redundancy is called parallel processing
Strategies for pain management:

1. Pre-emptive analgesia
   - Providing analgesics prior to the painful stimulus
   - Decreases pain intensity
   - Decreases duration of pain
   - Minimizes the likelihood that chronic pain and sensitization will develop
   - Adjunctive to post operative pain management, not a replacement for post operative pain management

2. Multimodal analgesia
   - Administering two or more classes of analgesics to address pain at more than one site of action
   - Different classes of analgesics administered together often act synergistically
     - Improves analgesia with lower doses of drugs – less chance of side effects
   - Minimizes the likelihood that chronic pain and sensitization will develop
   - Decreases neuroendocrine responses to pain
   - Decreases healing time
     - Fewer stress responses to surgical injury results in less tissue catabolism and maintenance of immune function
     - Less pain results in improved patient mobility

Local Anesthetics

Overview
- Local anesthetics (LAs) reversibly block the generation and propagation of electrical impulses in nerve endings or fibres
  - May result in some or all:
    - Autonomic nervous system blockade
    - Anesthesia and analgesia
    - Loss of muscle motor control
- A basic understanding of nerve physiology and drug pharmacology facilitates appropriate use of local anesthetics

Peripheral Nerve Anatomy
- Peripheral nerves are fibres (axons) grouped together in bundles (fascicles within an outer sheath)
- Myelinated or non-myelinated
  - Myelinated:
    - Schwann cells form multiple myelin layers around each axon
    - Ion channels are concentrated at the nodes of Ranvier = periodic interruptions in the myelin sheath
  - Non-myelinated:
    - Schwann cells only form a single membrane layer around each axon
    - Ion channels that support propagation of the action potential are distributed along the axon

From: biomedical-engineering-online.com
## Classification of Nerve Fibres

<table>
<thead>
<tr>
<th>Classification</th>
<th>Function</th>
<th>Diameter (µm)</th>
<th>Myelination</th>
<th>Sensitivity to blockade</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Type A</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Alpha (α)</td>
<td>Proprioception, motor</td>
<td>12 – 20</td>
<td>Heavy</td>
<td>+</td>
</tr>
<tr>
<td>Beta (β)</td>
<td>Touch, pressure</td>
<td>5 – 12</td>
<td>Heavy</td>
<td>++</td>
</tr>
<tr>
<td>Gamma (γ)</td>
<td>Muscle tone</td>
<td>3 – 6</td>
<td>Heavy</td>
<td>+++</td>
</tr>
<tr>
<td>Delta (δ)</td>
<td>Pain, temperature</td>
<td>2 – 5</td>
<td>Heavy</td>
<td>++++</td>
</tr>
<tr>
<td><strong>Type B</strong></td>
<td>Preganglionic autonomic</td>
<td>&lt; 3</td>
<td>Light</td>
<td>+++++</td>
</tr>
<tr>
<td><strong>Type C</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dorsal root</td>
<td>Pain</td>
<td>0.4 – 1.2</td>
<td>None</td>
<td>+++++</td>
</tr>
<tr>
<td>Sympathetic</td>
<td>Postganglionic</td>
<td>0.3 – 1.3</td>
<td>None</td>
<td>+++++</td>
</tr>
</tbody>
</table>

### Order of Nerve Fibre Blockade when using Local Anesthetics

1. Autonomic nervous system
   - B fibres
2. Sensory (pain, warmth, touch, pressure)
   - Aδ and C fibres
3. Muscle tone
   - Aγ fibres
4. Motor function, touch, pressure
   - Aβ fibres
5. Motor and proprioception
   - Aα fibres

- **Exceptions:**
  - Large peripheral nerves, where motor axons are located on the outer circumference of the nerve
    - These are exposed to local anesthetic first and hence motor block will occur before sensory blockade
    - The outer circumference of peripheral nerves contains sensory innervation to the proximal aspect of a limb, whereas the core contains distal sensory innervation
    - This means anesthesia develops proximally before distal areas become desensitized

### Site of Action of Local Anesthetics

- LAAs inhibit generation and conduction of nerve impulses by blocking sodium channels in the nerve membrane
- LAAs bind to a receptor site in the sodium channel, which prevents the movement of sodium ions required for electrical conduction
- LAAs also physically block the ion-conduction pore
- If sodium movement is blocked over a critical length of the nerve, electrical conduction is blocked for that length of the nerve – hence local anesthesia
**Nerve Ion Channels**

![Image of nerve ion channels](https://via.placeholder.com/150)

**Basic Pharmacology**

- Grouped as Amides or Esters based on molecular structure
  - Structure determines site of metabolism and potential for allergic reaction
- Most LAs used in current practice are Amide local anesthetics
  - Weak bases, ionized or cationic form (active) in solution
- **Ionized** (charged, dissociated, active) form:
  - Responsible for the effects – reversible loss of sensation and blocked nociception
- **Unionized** (uncharged, non-dissociated, inactive) form:
  - Important for penetration and diffusion through biological membranes
- Amount of drug in base (active, **ionized**) form at physiologic pH strongly influences the **onset** of drug action and the **potency** of the drugs
- Degree of lipid **solubility** has been correlated with **potency**
  - Bupivacaine is highly lipid soluble, and very potent
- Degree of **protein binding** has been correlated with **duration** of action
  - Bupivacaine is highly protein bound, and has a long duration of action

**Factors Influencing Local Anesthetic Activity**

- More rapid onset is facilitated by a greater volume of LA and a more concentrated solution
- Addition of epinephrine to local anesthetics delays absorption and prolongs action
- What about mixing?
  - When lidocaine and bupivacaine are mixed a median onset and duration occurs
  - Therefore the rapid onset of the lidocaine is lost as is the longer duration of the bupivacaine

**Effects**

- Reversible loss of sensation
  - Nociception is blocked
<table>
<thead>
<tr>
<th>Drug</th>
<th>Onset</th>
<th>Duration (minutes)</th>
<th>Clinical Use</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Esters</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Procaine</td>
<td>Slow</td>
<td>45 – 60</td>
<td>Infiltration, nerve block</td>
</tr>
<tr>
<td>Tetracaine</td>
<td>Slow</td>
<td>60 – 180</td>
<td>Topical</td>
</tr>
<tr>
<td></td>
<td>Amides</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lidocaine</td>
<td>Rapid (5 mins)</td>
<td>60 – 120</td>
<td>Topical, infiltration, nerve block, interpleural, epidural, intrathecal, IV</td>
</tr>
<tr>
<td>Mepivacaine</td>
<td>Intermediate</td>
<td>90 – 180</td>
<td>Infiltration, nerve block, interpleural, epidural, intrathecal, intra-articular</td>
</tr>
<tr>
<td>Bupivacaine</td>
<td>Intermediate (40 mins)</td>
<td>180 – 480</td>
<td>Infiltration, nerve block, interpleural, epidural, intrathecal</td>
</tr>
<tr>
<td>Ropivacaine</td>
<td>Intermediate</td>
<td>180 - 480</td>
<td>Infiltration, nerve block, interpleural, epidural, intrathecal</td>
</tr>
</tbody>
</table>

**Other Uses for Lidocaine**

- Class Ib antiarrhythmic
- Reduces inhalant requirements
- Analgesic when administered systemically
  - Action at Na\(^{+}\), Ca\(^{2+}\), K\(^{+}\) channels, NMDA receptor
- Anti-inflammatory
- May improve intestinal motility and prevent post operative ileus in horses

**Toxicity**

<table>
<thead>
<tr>
<th>Animal</th>
<th>Toxic Dose mg/kg</th>
<th>Working dose mg/kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cat</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lidocaine</td>
<td>6</td>
<td>1 - 2</td>
</tr>
<tr>
<td>Bupivacaine</td>
<td>2</td>
<td>0.5 - 1</td>
</tr>
<tr>
<td>Dog, Small</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ruminants</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lidocaine</td>
<td>10</td>
<td>2 - 3</td>
</tr>
<tr>
<td>Bupivacaine</td>
<td>3</td>
<td>1 - 2</td>
</tr>
</tbody>
</table>

- Bupivacaine should **not** be administered intravenously (IV)
- This “working dose” above is simply my preference – many use higher doses
  - These doses allow me to be free with local blocks, while allowing “extra” room in case of emergency or unforeseen requirements for other LA (intra-articular...)
- When administered at appropriate doses LAs are relatively free of harmful side effects
Unlike esters – allergic reactions were common
Most harmful reactions occur after accidental IV administration, or following vascular absorption of large amounts of anesthetic after regional administration
Cats metabolize local anesthetics more slowly than dogs and are more susceptible to overdose with these drugs
  • Lower doses are used, and caution should be observed
  • Larger species with larger body weight are less likely to be overdosed
    • Use the above doses for dogs and small ruminants as a guide when working with larger species

Central Nervous System (CNS)
  • CNS excitement, followed by depression
  • Seizures
    • Dogs - relative toxicity of bupivacaine: lidocaine = 4:1
      o Bupivacaine is much more lipid soluble than lidocaine
        ▪ This means it crosses the blood-brain-barrier in the CNS more quickly at a given dose
        ▪ Much more potential for toxicity than lidocaine

Cardiovascular System (CVS)
  • Decrease heart rate and cardiac output
  • Vasodilation, hypotension
  • Cardiac arrest
  • Epidural/intrathecal cranial spread of local anesthetics to the thoracic spine will block sympathetic outflow to the heart and may result in cardiovascular collapse

Local Anesthetic Techniques

When are local blocks administered?
  • Small Animals:
    • Once the animal is anesthetized
    • Before the procedure is to begin – remember onset time for the particular drug!
    • Bupivacaine is used most often for local and regional blocks, as its duration of action means its analgesic effect will persist into recovery from anesthesia
    • Lidocaine is used for procedures where long lasting analgesia is not required
  • Large Animals:
    • Horses:
      • After sedation and before the procedure is to begin (epidural, joint blocks)
      • After anesthesia (many dental blocks)
    • Cattle:
      • After restraint and before the procedure is to begin
      • Sedation is usually dependent on the particular animal and the procedure being performed

What equipment is required?
  • Local blocks:
    • 22-25 gauge needle
• 2.5-3.8 cm length for many
• 7.5 cm length for some
• 1-10 ml syringe
• Local anesthetic

• Epidurals:
  • Epidural needle
    • 20-22 gauge, 3.8-8.9 cm long
  • NEW bottle of sterile saline
  • NEW bottle of bupivacaine
  • NEW vial of preservative-free morphine
  • Needles and syringes
  • Surgical (NOT EXAM) gloves

• Local anesthetics may need to be diluted with sterile saline if the animal is small
  • Lidocaine is typically 2% (20 mg/ml) but may be diluted to 1% (10 mg/ml)
  • Bupivacaine is typically 0.5% (5 mg/ml) but may be diluted to 0.25% (2.5 mg/ml)

Dose reminder:

• Bupivacaine:
  • Usual dog, small ruminant dose 1 mg/kg (range 1-2 mg/kg)
  • Usual cat dose 0.5 mg/kg (range 0.5-1 mg/kg)

• Lidocaine
  • Usual dog dose 2 mg/kg (range 1-5 mg/kg)
  • Usual cat dose 1 mg/kg (range 1-3 mg/kg)

• Sometimes the dose calculated will be too much volume to be placed locally without excessive pressure
  • ALWAYS calculate the usual dose
  • You may not administer the entire calculated dose
  • Usual volumes listed below are simply guidelines – DO NOT forget to calculate actual dose every time!

Techniques

Topical anesthesia

• Effective for mucous membranes
  • Short term analgesia – lidocaine 2% applied topically lasts for approximately 30 minutes
  • Most formulations of local anesthetics will not penetrate intact skin
  • EMLA cream is the exception, but must be applied, then covered with an occlusive dressing, and left for 60 minutes

• Used to desensitize cat arytenoids immediately prior to intubation
  • The glottis of cats is narrow, is very easily traumatized, and spasms very easily

• Options:
  ▪ Apply a small amount of lidocaine per arytenoid
    • Lidocaine in a 1 ml syringe – usually 0.1 ml per arytenoid, but ALWAYS calculate 2 mg/kg to ensure 0.1 ml per arytenoid does not exceed 2 mg/kg
    • SECURELY place a 22 gauge catheter on the end of the syringe
    • Drip the lidocaine on each arytenoid – do not touch the arytenoids
  ▪ Spray applicators
    • Easy, convenient
• Some applicators dispense > maximum recommended dose in 1 spray if the animal is small
  • Wait 30 seconds for lidocaine to take effect
• Used prior to nasal oxygen line placement
• DO NOT use benzocaine gel applied to endotracheal tubes (ETT’s) prior to intubation
  • Causes methemoglobinemia
    • Methemoglobin cannot bind O₂ or CO₂

Infiltration anesthesia
• Lidocaine or bupivacaine used
• Intradermal or subcutaneous injection of local anesthetic
• Used to desensitize a small area of dermis and subcutaneous tissue prior to diagnostic tests or minor procedures (skin biopsy)
• Still must calculate the maximum recommended dose for each patient prior to beginning
  • Only practical for small areas
  • Remember the total calculated dose must be distributed over as many sites or as large an area as necessary
• Aspirate prior to injecting, and after repositioning needle
  • If blood is aspirated, change needles and try again
• Usual volume 0.2-1 ml/site (see above regarding calculation)

Intra-articular anesthesia
• Used less commonly in small animals than in large animals
• Used most commonly during surgery, at the end of a procedure involving a joint
• Bupivacaine most commonly used
  • Volume that can be injected into a joint is small, but remember to calculate the total maximum dose for the animal, as this dose must be distributed among all of the sites to be blocked
    • For example, a 20 kg dog can safely receive 1-2 mg/kg total dose = 20-40 mg = 4-8 ml
    • So if you perform a brachial plexus block and use 15 mg/3 ml, you still have 25 mg/5 ml to use for an intra-articular (or other) block

Local nerve blocks
• Bupivacaine is usually used
  • Long onset (20-30 minutes)
  • Intermediate duration of action (4-6 hours)
  • Remember usual dose
    • Dogs: 1-2 mg/kg
    • Cats 0.5-1 mg/kg
• Lidocaine may be used
  • Fast onset (5 minutes)
  • Short duration of action (1-2 hours)
  • Remember usual dose
    • Dogs 2-3 mg/kg
    • Cats 1-2 mg/kg
Dental Procedures

• What nerves are involved?

1. Maxillary branch of the trigeminal nerve (cranial nerve V)
   • Specifically the infraorbital branch (sensory) of the maxillary nerve
   • Rostral branch (rostral maxillary nerve) – blocked with the infraorbital nerve block
     • Supplies sensation to the upper incisor teeth
     • Branches off the infraorbital nerve just before the nerve exits the infraorbital foramen
   • Middle and caudal branches (middle and caudal maxillary alveolar nerve) – blocked with the maxillary nerve block
     • Middle branch supplies sensation to the middle maxillary teeth
     • Caudal branch supplies sensation to the caudal maxillary teeth
   • Maxillary nerve and its branches also supply sensation to the soft and hard palates, the nose, and the upper lips

2. Mandibular nerve, with left and right branches
   • Or inferior alveolar nerve
   • Also a branch of the trigeminal nerve
   • Enters the mandibular foramen on its medial side just caudal to the last molar
   • Supplies sensation to the mandibular teeth, as well as the tongue and chin
   • Mental nerve is a rostral branch of the mandibular nerve, and supplies the lower incisor teeth

3. Auriculopalpebral nerve (or auriculotemporal)
   • Branch of the facial nerve (cranial nerve VII)
   • Located cranial to the pinna, coursing from ventral to dorsal along the cranial edge of the pinna

4. Greater auricular nerve
   • Branch of the optic nerve (cranial nerve II)
   • Located caudal to the pinna, close to the palpable edge of the wing of the atlas

Infraorbital Nerve Block

• Indications: anesthesia of the ipsilateral middle maxillary teeth and upper incisors
• Technique:
  • The gingiva is lifted to show the buccal mucosa
  • The infraorbital foramen is palpated with an index finger – it is located dorsal to the upper 3rd premolar
  • The needle is inserted just inside the foramen so as to not inject into the nerve
  • Aspirate prior to injection and whenever needle position is adjusted
    • Remember the importance of ensuring NEVER to inject into a vessel
Maxillary Nerve Block

- Indications: anesthesia of the entire ipsilateral upper arcade
- Technique:
  - Infraorbital foramen approach:
    - Described in 2013
    - Easy, minimizes chance of side effects
    - 22-24 gauge catheter is measured so catheter hub is at level of foramen and tip is at level of PM4
    - Catheter is introduced into infraorbital foramen
    - Stylet is removed once catheter is inserted
    - Catheter is fed to pre-measured distance, so tip is at level of PM4
    - Aspirate prior to injection
    - Remember the importance of ensuring NEVER to inject into a vessel
  - Extra-oral approach:
    - The zygomatic arch is palpated to locate its most dorsal point
    - The infraorbital foramen is palpated with the index finger of the other hand
    - The needle is inserted through the skin at a point just ventral to the most dorsal point of the zygomatic arch, aiming toward the infraorbital foramen
    - Remember the middle and caudal maxillary alveolar nerves are blocked with this technique, from the caudal aspect of the infraorbital canal, at the level of the maxillary foramen
    - Aspirate prior to injection and whenever needle position is adjusted
      - Remember the importance of ensuring NEVER to inject into a vessel
• Intra-oral approach:
  • The middle and caudal maxillary alveolar nerves are approached from the mouth, caudal to the last upper molar
  • BEWARE of the palatine artery!!

Mandibular Nerve Block

• Indications: anesthesia of the entire ipsilateral lower arcade
• Technique:
  • The notch in the ventral mandible is palpated transcutaneously approximately 1 cm rostral to the angular process of the mandible with one hand
  • The other hand is inserted into the mouth, and the mandibular foramen (or sometimes the actual nerve) is palpated on the medial aspect of the mandible
  • The needle is inserted through the skin at the notch, and advanced along the medial aspect of the mandible until the needle can be felt with the hand inside the mouth
  • Be very careful to stay as lateral as possible, by being firmly against the medial aspect of the mandible
    • Drifting medially while performing this block will block the lingual nerve, leading to self-trauma post anesthesia
    • Some anesthetists only block the mandibular nerve unilaterally, for this reason
  • Aspirate prior to injection and whenever needle position is adjusted
    • Remember the importance of ensuring NEVER to inject into a vessel

Mental Nerve Block

• Indications: anesthesia of the lower incisors (and chin)
• Technique:
  • The lower rostral gingiva is exposed
  • The mental foramen is palpated at the level of the second lower premolar
  • The needle is inserted just inside the foramen so as to not inject into the nerve
  • Aspirate prior to injection and whenever needle position is adjusted
    • Remember the importance of ensuring NEVER to inject into a vessel

From: Pain Management for the Small Animal Practitioner
Auriculotemporal and Greater Auricular Nerve Blocks

- Indications: anesthesia of the pinna and ear canal
- Technique:
  - Clipping and preparing the site is mandatory for this block
  - The auriculotemporal nerve is block rostral to the ear, at the base of the intersection between the caudal aspect of the zygomatic arch and the rostral edge of the palpable vertical ear canal
  - The greater auricular nerve is blocked caudal to the ear canal, at the point just ventral to the wing of the atlas and immediately caudal to the vertical ear canal
  - A line block is done in a V-shape to “connect” the two blocks and provide additional anesthesia of the ear canal and surrounding tissues
  - Aspirate prior to injection and whenever needle position is adjusted
    - Remember the importance of ensuring NEVER to inject into a vessel

From: BSAVA Manual of Canine and Feline Anaesthesia and Analgesia

Brachial plexus block

- Indications:
  - Traditional technique – procedures distal to the elbow
  - Paravertebral technique – procedures of the shoulder and distal
  - All techniques require landmarks to be palpated
    - If the animal is obese and the landmarks cannot be palpated do not perform this block
- Complications:
  - Pneumothorax if needle is placed or adjusted too medially
  - Intravascular injection if aspiration is not performed prior to injection
  - Blockade of the phrenic nerve
    - This is why this procedure is only ever done unilaterally
• Traditional technique:
  • Equipment:
    • 22 gauge needle, 7.5 cm
    • Sterile syringe
  • Landmarks:
    • Point of the shoulder, 1st rib, transverse processes of cervical vertebrae
  • Technique:
    i. Needle is inserted just proximal to the point of the shoulder (scapulohumeral joint)
    ii. Needle is inserted at a point halfway between the point of the shoulder and the dorsal crest of the scapula
  • Both techniques: Needle is inserted medial to the scapulohumeral joint, and advanced toward the costrochondral junction of the first rib, staying lateral to the thorax (don’t enter the thorax!)
  • Usual volume:
    • 1 ml increments as needle is adjusted (see above regarding calculation)
  • Aspirate prior to injection and whenever needle position is adjusted
    • Remember the importance of ensuring NEVER to inject into a vessel
  • Once the needle is located appropriately, aspirate, then inject a small amount of the calculated dose
  • The rest of the dose is administered as the needle is withdrawn toward the skin, always aspirating after adjustment

From Veterinary Anaesthesia: Principles to Practice. Dugdale

• Modified Paravertebral technique:
  • Equipment:
    • 22 gauge needle, 2.5-3.8 cm
    • Sterile syringe
  • Landmarks:
    • Transverse process of C₆, costrochondral junction of first rib
  • Technique:
    • A helper shifts the scapula caudally to expose the first rib for palpation
• Be sure the whole scapula and leg shifts caudally and is not simply rotated around the scapulohumeral joint to swing the paw caudally and the scapulohumeral joint ventrally
• Three injection sites are involved:
  • The transverse process of C₆ is palpated, and the index finger is left on the process
    • Anesthetic is injected:
      1. Cranial and slightly dorsal to the transverse process of C₆
      2. Caudal to the transverse process of C₆
        • This blocks the ventral branches of C₆ and C₇
  • The index finger is placed on the costrochondral junction of the first rib
    • Anesthetic is injected:
      3. Cranial to the costrochondral junction of the first rib
        • This blocks the ventral branches of C₈ and T₁
  • Remember the total calculated dose must be divided over the three injection sites
• Usual volume:
  • 0.5-1.5 ml per site (see above regarding calculation)
• Aspirate prior to injection and whenever needle position is adjusted
  • Remember the importance of ensuring NEVER to inject into a vessel

From: BSAVA Manual of Canine and Feline Anaesthesia and Analgesia

Radial, ulnar, median, musculocutaneous (RUMM) block

• Indications:
  • Procedures distal to the mid-humerus
• Complications:
  • Accidental intravascular/intra-arterial injection
    • Brachial artery and vein lie in close proximity to the injection sites
• Landmarks:
  • Medial and lateral humeral epicondyles
• Technique:
  • Radial nerve is blocked from the lateral aspect of the leg
    • Needle is inserted proximal to the lateral humeral epicondyle, between the brachialis and the lateral head of the triceps muscles
  • Ulnar, median, and musculocutaneous nerves are blocked from the medial aspect of the leg
    • Needle is inserted proximal to the medial humeral epicondyle, between the brachialis and the medial head of the triceps muscles
• Remember the total calculated dose must be divided over the two injection sites
• Usual volume:
  • 0.5-1.5 ml per site (see above regarding calculation)
• Aspirate prior to injection and whenever needle position is adjusted
  • Remember the importance of ensuring NEVER to inject into a vessel

From: BSAVA Manual of Canine and Feline Anaesthesia and Analgesia

**Intercostal block**

• Indications:
  • Post thoracotomy
  • Rib fractures
• Complications:
  • Accidental intravascular/intra-arterial injection
    • Intercostal veins, arteries, and nerves lie in close proximity to each other immediately caudal to each rib
  • Pneumothorax if needle is directed too far medially
• Technique:
  • Two to three ribs cranial to and 2-3 ribs caudal to the affected site must be blocked (overlapping nerve supply)
  • Needle is inserted immediately caudal to the rib at the dorsal aspect, close to the epaxial muscles
  • Needle direction is medial
    • Needle should penetrate skin, subcutaneous tissues, and intercostal muscles but NOT enter thorax!

• Usual volume:
  • 0.5-1.5 ml per site (see above regarding calculation)
• Aspirate prior to injection and whenever needle position is adjusted
  • Remember the importance of ensuring NEVER to inject into a vessel
Regional anesthesia/analgesia: Epidural

- Anatomy review:
  - Dogs: Spinal cord ends at L₆, subarachnoid space ends at L₇
    - Very little chance of encountering spinal tissue and CSF with epidural puncture
  - Cats: Spinal cord ends at L₇, subarachnoid space ends at S₁
    - Spinal tissue and CSF may be encountered with epidural puncture
- Epidural vs subarachnoid:
  - Epidural space contains nerves (cauda equina), fat, blood vessels, lymphatics
    - Lies within the dura mater (outermost meningeal layer)
  - Subarachnoid space contains CSF
    - Lies ventral to the arachnoid mater (middle meningeal layer) and dorsal to the pia mater (innermost meningeal layer)
- Indications:
  - Anesthesia (opioid plus local anesthetic)
    - Results in profound MAC reduction, and motor blockade
    - Preservative-free morphine analgesia lasts for up to 24 hours
      - There are reports of histologic changes occurring within the spinal cord after multiple injections of drug containing preservative
    - Local anesthetic effects last for normal duration of local anesthetic used
    - Useful for:
      - Surgical procedures of the hindlimbs and perineal area, when motor blockade is not a problem
  - Analgesia (opioid diluted with sterile saline)
    - Results in mild MAC reduction with no motor blockade
    - Preservative-free morphine analgesia lasts for up to 24 hours
    - Useful for:
      - Surgical procedures of the hindlimbs and perineal area, where motor function needs to be preserved
      - Surgical procedures of the abdominal cavity, for long-lasting analgesia
- Complications:
  - Sterile technique is absolutely mandatory
    - Meningitis is possible with non-sterile technique
  - Contraindications include sepsis, systemic infection, local infection, coagulopathy, shock, pre-existing neurologic disease
- Urinary retention can occur – recommend expressing the bladder or placing a urinary catheter prior to recovery from anesthesia
- Hypotension during anesthesia
- Delayed regrowth of hair at the site
- Pruritis at the site

**Technique:**
- Sternal or lateral recumbency
  - Either is appropriate, but the “hanging drop” technique is only effective in sternal recumbency
  - Techniques are identical except for the hanging drop description
- Sternal: Hind legs positioned cranially, as if the animal is lying sternally while conscious
- Lateral: Normal lateral recumbency, with the pelvis straight and the hind legs aligned
- Surgical clip and scrub
- Epidural is administered in the L-S space (called an L-S epidural)
- **Landmarks:**
  - Dorsal edges of the wings of the ilia, dorsal spinous process of L₆ and L₇, and the sacrum
  - L-S space lies just caudal to an imaginary line connecting the dorsal edges of the wings of the ilium
  - Using the non-dominant hand, the thumb and middle finger palpate the imaginary line, and the index finger feels for an indentation between the dorsal spinous process of L₇ and the cranial rim of the sacrum
  - The landmarks are difficult to palpate in obese animals
- Epidural needle is inserted at midline in the indentation, with the bevel facing cranially
- Immediately after the skin is punctured, the stylet is removed
- Sterile saline is dripped into the hub of the epidural needle, until a meniscus is formed
  - This is the “hanging drop”
- The needle is slowly advanced until it “pops” through the ligamentum flavum
- The needle is advanced slightly further, until there is a lack of resistance, and the hanging drop is aspirated into the epidural space
- Proper placement is confirmed by injecting a small volume of sterile saline into the epidural space
  - This should feel like injecting nothing, with absolutely no resistance
  - If any resistance to injection is felt, the needle is not within the epidural space and the needle should be withdrawn and the procedure begun again
- If placement is confirmed, the hub of the needle is watched to ensure that no blood or CSF appears
  - If either is seen, the needle should be withdrawn and the procedure begun again
- To inject drugs: mix opioid and local anesthetic or opioid and sterile saline, and give the injection slowly over 1 minute
  - A bubble of air is left in the syringe during injection – with proper placement there is no compression of the air bubble during injection
  - The drug is given, and the injection is stopped just before the air is injected
- The syringe and needle are withdrawn together as a unit
- If local anesthetic is used, the affected side is placed ventrally for 5 minutes to facilitate the gravity-affected movement of local anesthetic
  - The local anesthetic pools ventrally
- If no local anesthetic is used, positioning is irrelevant to the effectiveness of the epidural
  - The opioid stays within the epidural space, and slowly bathes the entire epidural space
Dosing:
- Remember a NEW bottle/vial of drug, including saline MUST be used for each epidural
- Preservative-free morphine: 0.1 mg/kg
- Plus either:
  - Bupivacaine 1-2 mg/kg
  - Cats 0.5-1 mg/kg diluted with saline to give volume of 1 ml/5kg
  - Or:
    - Sterile saline 1 ml/5 kg
  - Or:
    - Lidocaine 2 mg/kg diluted with saline to give volume of 1 ml/5 kg
      - Cats 1 mg/kg diluted with saline to give volume of 1 ml/5kg
- To a maximum combined drug volume of 6 - 8 ml
  - If more volume of saline or local is calculated, the volume is reduced to give a combined morphine/saline or bupivacaine volume of 6 - 8 ml

From: Small Animal Anesthesia and Analgesia. Carroll GL

**Horses**

- **Supraorbital, infratrochlear, lacrimal,** and **zygomatic** blocks for complete anesthesia of the eyelids – the ophthalmic and maxillary branches of the trigeminal nerve are blocked
- **Auriculopalpebral** block to immobilize the eyelids (no analgesia with this block) – blocks a branch of the facial nerve
- **Infraorbital** nerve block to desensitize the ipsilateral upper lip, nostril, dorsal nasal cavity, and teeth and gingivae from the incisors to the 3rd premolar – blocks a terminal branch of the maxillary division of the trigeminal nerve
- **Maxillary** block to desensitize the upper lip, nostril, skin, teeth of the maxilla, paranasal sinuses, and nasal cavity – blocks the maxillary division of the trigeminal nerve
- **Mandibular** block (or mandibular alveolar block) to desensitize the ipsilateral mandible and all associated structures – blocks the mandibular branch of the trigeminal nerve
- **Mental** block to desensitize the ipsilateral lower lip and skin, and if the needle is inserted into the foramen to desensitize the above plus the incisors, canine, and first 3 cheek teeth and gingivae – blocks the terminal branch of the mandibular nerve
- **Intra-testicular** block for castration
• **Epidural** anesthesia:
  - At inter-coccygeal site (Co1-Co2) for procedures or analgesia of the perineal area
  - 0.2 mg/kg, with the caveat below:
  - Volume **MUST** be limited to 3 – 6 ml in a 500 kg horse to avoid motor paralysis and recumbency, which is **NOT** tolerated in conscious horses

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Carroll GL. *Small Animal Anesthesia and Analgesia*. Blackwell Publishing, Ames IA. 2008:


Dohoo Se, Dohoo IR. Factors influencing the postoperative use of analgesics in dogs and cats by Canadian veterinarians. Can vet J 1996; 37 (9):552-556.

Gaynor JS, Muir WW, eds. *Handbook of Veterinary Pain Management*. Mosby Elsivier, St. Louis, MO, 2009:


Tranquilli WJ, Lamont LA, Grimm KA. Pain Management for the Small Animal Practitioner. 2nd ed. Teton New Media, Jackson WO, 2004:
   1. Section 1: Pain Terminology, Physiology, Recognition, and Clinical Strategies, pp 2 – 12.
   2. Section 3: Analgesic Techniques, pp 35 – 75.


Useful resources:

International Association for the Study of Pain (IASP) website: http://www.iasp-pain.org/AM/Template.cfm?Section=Pain_Definitions&MenuID=5

American College of Veterinary Anesthesiologists (ACVA) website: http://www.acva.org/docs/Pain_Treatment
PAIN PHYSIOLOGY AND RECOGNITION
Cate Creighton, DVM, MSc, DACVAA

Pain

Useful organizations and websites

- American College of Veterinary Anesthesia and Analgesia (ACVAA)
  - Website: acva.org
  - Has a position statement regarding treatment of pain in animals
    - Updated periodically, last version 2009:
      - “believes that animal pain and suffering are clinically important conditions that adversely affect an animal’s quality of life, either in the short or long term.”
      - “endorses a philosophy that promotes prevention and alleviation of animal pain and suffering as an important and tenable therapeutic goal.”
  - What do these statements mean?
    - Pain matters
      - It is clinically important, whether it is physical pain, or emotional suffering
    - Prevention of pain when possible, and alleviation of pain once it is present, is a viable goal for veterinarians

- International Association for the Study of Pain (IASP)
  - Website: iasp-pain.org
  - Has a definition of pain:
    - “an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage. Note: The inability to communicate verbally does not negate the possibility that an individual is experiencing pain and is in need of appropriate pain-relieving treatment”.
  - What do these statements mean?
    - Pain can be physical or emotional
    - The damage doesn’t have to be actual, physical damage to the body; potential damaging encounters matter also
    - Pain does not need to be verbalized to matter
      - This seems obvious, but is a really important point!
      - Until 2001 speech WAS considered necessary to feel pain
        - Human infants underwent open-heart surgery with no analgesics until the late 1980’s
        - One mother later found out and made that public knowledge
      - It was thought that pain was needed to keep animals from being active and re-injuring the damaged body part
        - The vocalization exhibited by these animals was considered to be an effect of the anesthesia itself, not pain

Do animals feel pain?

- Again this seems obvious, but there is ongoing debate and research...
- Nociceptive pathways are clear
- Pain responses may not be so clear
- Assume that a procedure/disease that causes pain in humans also causes pain in animals
- Do not assume that lack of an obvious pain response means the animal is not painful
Why treat pain?

- Society demands it
- Pain management improves clinical outcome
- Morbidity and mortality lessen with adequate analgesia
- Veterinarians are well equipped to recognize and treat pain
- Veterinarians are ethically obligated to eliminate or manage pain
- Humans often report fearing pain more than death
- Pain management is a relatively new concept (1990’s)
- Anesthesia textbooks published before the 1980’s rarely contained sections on pain or analgesia
- Now pain and analgesia are major components of most anesthesia texts
- A survey conducted in 1996 found that veterinarian’s knowledge regarding analgesia was not learned in veterinary school – no analgesia was incorporated in the curriculum

Nociception

- Detection, transduction, and transmission of noxious stimuli
  - These terms are discussed below
- Nociceptors: free nerve endings
  - Job of nociceptors = send impulses to CNS
  - Stimulated by thermal, mechanical, or chemical tissue damage
    - Mechanical stimulus – injury, surgical or otherwise
  - Send impulses to the CNS for modulation and interpretation
- Nociception versus pain:
  - Nociception is the process
    - Well conserved across species (as far as we know…)
  - Pain is the perception
    - Consists of sensory and emotional components
    - Unique to each individual
    - Affected by many things

Pain physiology

- The basic nociceptive pathway is a three neuron chain
  4. First order neuron
    - Originates peripherally and projects to the spinal cord
    - Consists of a primary afferent fibre that is activated by a noxious stimulus
      - Afferent fibres head towards the spinal cord from the periphery
      - Efferent fibres head away from the spinal cord towards the periphery
  5. Second order neuron
    - Ascends the spinal cord
  6. Third order neuron
    - Projects into the higher brain structures (somatosensory cortex)

From: Tranquilli WJ, Lamont LA, Grimm KA
Pain Management for the Small Animal Practitioner. 2nd ed.
Teton New Media, Jackson WO, 2004
There are four key physiologic components to the pain pathway:

1. Transduction
2. Transmission
3. Modulation
4. Perception

Transduction

- Nociceptors in the periphery change mechanical, chemical, or thermal energy into electrical impulses
- Nociceptors are the free nerve endings of primary afferent neurons
  - Signal actual or potential tissue injury
    - The body can respond even when it only THINKS something will be damaging – you move away from something that MIGHT be painful
    - The body needs to change the insult from mechanical, chemical, or thermal into something it can recognize

Transmission

- Electrical impulses are carried to the dorsal horn of the spinal cord by nerve fibres:
- Function of nerve fibres:
  - **Aα fibres**
    - Motor and proprioception
    - Afferent and efferent for muscles and joints
  - **Aβ fibres**
    - Motor, touch, pressure
    - Afferent sensory, efferent to muscle
  - **Ay fibres**
    - Muscle tone
    - Efferent to muscle spindle
  - **Aδ fibres**
    - Fast pain, touch, temperature
    - Afferent sensory
    - Relatively large diameter, myelinated
  - **β fibres**
    - Autonomic function
    - Preganglionic sympathetic
  - **C fibres**
    - Slow pain, touch, temperature
    - Postganglionic sympathetic
    - Small diameter, unmyelinated
- Both Aδ and C fibres:
  - Afferent nerve fibres found throughout skin, peritoneum, pleura, periosteum, subchondral bone, joint capsules, blood vessels, muscles, tendons, fascia, and viscera
  - Cell bodies are located in the dorsal root ganglia of the spinal cord
    - The cell bodies produce enzymes and neurotransmitters that are involved in nerve signal transmission
    - Axons extend from the cell bodies to synapse with the dorsal horn neurons in the grey matter (nerve cells) of the spinal cord
  - Sub – populations of both Aδ and C fibres are
    - Low threshold – respond to low level stimuli
    - High threshold – respond to high intensity stimuli
Modulation

- Nociception is first amplified or suppressed at the level of the dorsal horn of the spinal cord
  - Dorsal horn is where axons from peripheral neurons synapse with dorsal horn neurons
- Spinal cord receives input from peripheral, local, and descending neurons
- Sensory homeostasis: balance between activity of peripheral nerve inputs and descending excitatory and inhibitory influences
  - Failure of homeostasis can lead to sensitization
- Secondary amplification or suppression occurs at the level of the brain
- Amplification is facilitated by:
  - Excitatory amino acids and peptides
    - Aspartate, glutamate, substance P
  - Excitatory receptors
    - NMDA (N-methyl D-aspartate), AMPA (α-amino-3-hydroxy-5-methyl-4-isoxazole-propionic acid), and KA (kainate)
- Suppression is facilitated by:
  - Endogenous opioids
    - Endorphin, dynorphin, encephalin
  - Inhibitory receptors
    - GABA (gamma amino butyric acid), glycine
  - Exogenous opioids and other analgesic drugs (local anesthetics for example)

Perception

- Last step in process of nociception
- Involves integration, processing, and recognition of sensory input
  - Occurs at the level of the brain, via several neural pathways – this redundancy is called parallel processing
- 2nd order neuron travels to the thalamus
  - Via spinothalamic tract
- At the thalamus, synapses with 3rd order neuron
  - Thalamus is involved in 2 aspects of pain:
    - Sensory discriminative - the intensity, quality, location, and duration of the pain
    - Affective motivational - the unpleasantness that creates the urge to escape
  - 3rd order neuron travels to the cortex
- The cortex interprets the information received from the thalamus
  - The cortex then mediates behavioural patterns
- Nociceptor neurons also exist in the brainstem: medulla, pons, and midbrain
  - Brainstem structures contribute to the reticular system and the periaqueductal grey matter (PAG)
    - PAG integrates homeostatic control
  - Reticular activating system (RAS) mediates motor, autonomic, endocrine, and sensory function
    - RAS is critical in integration of pain as an experience, mediating the affective and motivational aspects of pain

Antinociceptive pathways:

- Modulate all types of sensory input
- Begin supraspinally – cerebral cortex, thalamus, PAG, brainstem
- Descend to the dorsal horn of the spinal cord
- Spinal cord itself contributes to anti-nociception
  - Dense concentrations of substances acting to inhibit nociception exist here: GABA, glycine, serotonin, norepinephrine, and endogenous opioid peptides (enkephalons, dynorphins, endorphins)
- **Neural plasticity**
  - Ability of the nervous system to modify its function in response to environmental stimuli
  - The brain structures responsible for pain perception process noxious stimuli
    - Fear, anxiety, and aggression can result
    - Afferent pathways are activated, resulting in autonomic, neuroendocrine, and motor responses
    - The brain can be conditioned by visual, olfactory, auditory, somatic, or visceral stimuli to prepare for fearful or painful events
      - This can result in sensitization

**Sensitization:**

1. **Peripheral**
   - Occurs as a consequence of tissue trauma and inflammation
   - Damaged cells, inflammatory cells, nerve terminals, and nerve fibres release intracellular components
     - Produces a “sensitizing soup” of chemical mediators:
       - Direct tissue damage causes the release of: ATP, H⁺, K⁺, prostaglandins, bradykinin, nerve growth factors
       - Inflammatory cells release cytokines – interleukin 1, interleukin 6, tumour necrosis factor
       - Mast cell degranulation releases serotonin and histamine
       - Nerve fibres release inflammatory mediators and the neuropeptides substance P and CGRP
     - “Sensitizing soup” of inflammatory mediators changes high threshold nociceptors to low threshold nociceptors
     - Peripheral nociceptors develop increased sensitivity
   - Results in primary hyperalgesia at the site of injury
     - Increased response to a noxious stimulus at the site of injury
     - This amplifies the pain response

![Sensitizing Soup Diagram](image)


2. **Central**
   - Occurs as a consequence of severe or chronic noxious stimuli
   - More Aδ and C fibres are activated
     - Cause the release of excitatory mediators
       - Glutamate, substance P, and brain derived neurotrophic factor (BDNF)
   - Prolonged activation of dorsal horn neurons activates receptors which alter gene expression and further sensitize the CNS to further input
Once central sensitization is present:
- Secondary hyperalgesia develops
  - Increased pain response to noxious stimulus outside the injured area
- Allodynia is likely present
  - Pain is perceived in response to a stimulus that is normally not painful
- All stimuli are capable of causing pain
- Pain is refractory to analgesics

In summary:
- Nociception processing involves a three neuron chain with excitatory and inhibitory input at each level
- Nociceptive and anti-nociceptive pathways exist at each level of neuron
- Nociceptors transduct energy into electrical impulses
- Impulses are transmitted to the dorsal horn of the spinal cord
- The spinal cord modulates impulses via nociceptive and anti-nociceptive pathways
- Perception of pain occurs in the brain, as a result of integration, processing, and recognition of sensory input
- Physiologic pain is protective, while pathologic pain often results in sensitization
- Neural plasticity allows sensitization to alter the threshold of nociceptors such that all stimuli can cause pain that is refractory to treatment

**Consequences of pain**

- Short term, physiological pain may be beneficial
  - Minimize tissue damage
  - Animals can learn protective behaviours
- Stress response elicited by pain activates CNS
  - Sympathetic changes
    - SNS changes: tachycardia, tachypnea, hypertension, piloerection, midriasis
  - Parasympathetic changes
    - PNS changes: urination, defecation
  - Increases in cortisol, catecholamines, pituitary hormones, adrenal hormones, glucose, cytokines, neutrophils
- Short-term stress helps with survival, no question
- Pain can often be detrimental
  - Can cause stress and suffering
  - Can cause unwanted physiological responses and behaviours
- Long term stress is maladaptive
  - Insulin resistance
  - Immune system impairment

What about anesthesia?

- Remember, anesthesia prevents perception of pain
- Nociception may still occur if insufficient analgesia is provided in the peri-operative period
  - Short-acting analgesics + invasive procedure = intense pain at recovery
- Analgesia is a big part of protocols
  - Pre, intra, post
- Untreated, patients may experience peripheral +/- central sensitization

**Strategies for pain management:**

1 **Pre-emptive analgesia**
   - Providing analgesics prior to the painful stimulus
     - Not always possible…
   - Decreases pain intensity
   - Decreases duration of pain
   - Minimizes the likelihood that chronic pain and sensitization will develop
   - Adjunctive to post operative pain management, not a replacement for post operative pain management

2 **Multimodal analgesia**
   - Administering two or more classes of analgesics to address pain at more than one site of action
   - Different classes of analgesics administered together often act synergistically
     - Improves analgesia with lower doses of drugs – less chance of side effects
   - Minimizes the likelihood that chronic pain and sensitization will develop
   - Decreases neuroendocrine responses to pain
   - Decreases healing time
     - Fewer stress responses to surgical injury results in less tissue catabolism and maintenance of immune function
     - Less pain results in improved patient mobility
Pain Recognition

- Can be difficult – there is significant individual variation in expression of pain
- Prey species and solitary species often do not display signs of pain, while more social species may
- Behavioural indicators of pain vary among species and individuals, and require knowledge of normal values and behaviours
  - Consider the Labrador versus the wild bird
- Good place to start: history and physical examination
- Physiologic indicators of pain may reflect autonomic changes, but assessment can be misleading
  - Significant overlap between "normal" and "elevated due to pain" values for each parameter
  - An individual can be painful and have normal physiologic parameters
- Blood value changes are not specific for pain
  - Glucose, cortisol, catecholamines
- Things that may be seen:
  - Personality or attitude changes
  - Vocalization
  - Attention paid to the affected area
  - Posture or ambulation changes
  - Appetite, thirst, or elimination habit changes
  - Facial expression changes
  - Sweating or salivation
  - Teeth grinding
  - Depends significantly on the species, and the individual
  - You have to know if a particular behaviour is normal or not, and look for signs of pain in each patient
- This is where anthropomorphizing is appropriate, and encouraged
  - Presume that a procedure likely to cause pain in yourself will cause pain in your patients, and treat the pain even if there are no obvious signs of pain
What affects an individual's response to pain?

- **Age**
  - Young animals: less tolerant of pain, but less anticipation
  - May not show “typical” pain behaviours for the species or breed

- **Health status**
  - Ill animals: less tolerant of pain
  - Severely debilitated animals may be physically unable to show pain response

- **Behavioural status**
  - Anxious animals are less tolerant of pain
  - Pain and anxiety seem to fuel each other

- **Species**
  - Prey species hide pain very well
  - Cats often become fractious

- **Breed**
  - Newfoundland may not show pain
  - Toy breeds often become aggressive
  - Labradors simply want to please

Principles of pain assessment

- Evaluate patients early, and often
  - Pain level can change over time

- Uncertain about level of pain?
  - Treat, then reassess

- Response to analgesics = gold standard for pain assessment
  - But remember, you may have simply picked the “wrong” analgesic for the individual or situation...

- No one pain scale is perfect
  - If the one you are using doesn’t seem to apply, use another!
  - You get better at using a particular pain scale over time

Pain Scales

- No single scale has been validated to the exclusion of others
  - Pain scales try to make a subjective thing a bit more objective
  - Huge variability in pain scores among students, anesthesiologists, even between anesthesiologists

- Pick one and **use it**

Visual analog scale (VAS)

- 10 cm line
- Mark made according to animal’s level of pain
- 0 is no pain, 10 is worst pain imaginable
- Very simple, but very subjective
- An example:

```
   Visual Analog Scale (VAS)

No Pain |  | Pain As Bad As It Could Possibly Be
```

physio-pedia.com
**Numeric scale**

- Number assigned to animal’s level of pain
  - VAS with numbers added
- Often out of 10 or 100
- An example:

![0-10 Numeric Pain Rating Scale](capitalrehabofarlington.com)

**Faces scale**

- Face describes level of pain
- Facial coding scales used in non verbal humans to quantify pain
  - Next logical step: identify facial coding scales in animals
- An example:

![Wong-Baker FACES™ Pain Rating Scale](peerj.com)

**Rat grimace scale**

- Mouse grimace scale published in 2010
  - Looked at 5 “facial action units” after nociceptive and surgical procedures
    - Orbital tightening, cheek bulge, nose bulge, ear position, and whisker position
  - Shown to be highly accurate and reliable, and requires minimal training for observers
- Rat grimace scale modified from mouse grimace scale in 2011
  - Same group of researchers
  - Used 3 noxious stimuli, injected while the rats were anesthetized
  - Developed software Rodent Face Finder®
    - Looked at similar facial changes
  - This scale was used to develop an analgesic dose of morphine in the rat, to improve research conditions
  - What it looks like:
Horse pain face

- Published in 2015
- Looked at six horses exposed to two different noxious stimuli
  - Tournequet on antebrachium, topical capsaicin
- Described the horse pain face as having low or asymmetrical ears, angled eyes, tense stare, mediolaterally dilated nostrils, and tension of the lip, chip, and facial muscles
- What it looks like:

![Horse Pain Face Image](image)

Gleerup KB, Forkman B, Lindegaard C et al. An equine pain face. VAA 2015;42;103-114.

Glasgow composite measures pain scale short form

- Single page
- Behaviour – based scale to assess acute pain in dogs
  - Six behavioural categories used to assign overall pain score
- Designed to prompt intervention (provide analgesics) if overall score is above 6/24 or 5/20, depending on how much assessment is done

Colorado State University pain score

- Designed for dogs and cats
- Used to assess acute surgical pain
- Has a reminder to assess if animal can be aroused
- Assesses behavioural response, response to palpation of affected site, and overall body tension
- Designed to prompt intervention (provide analgesics) if overall score is at or above 2/4

Take-Home Message

- Think about pain physiology
  - Will help you to develop analgesic plans
- Anthropomorphize
- Look for pain
- Work at recognizing pain
- Think about ways to address the pain pathway
  - Pre emptive
  - Multimodal

References:

Dohoo Se, Dohoo IR. Factors influencing the postoperative use of analgesics in dogs and cats by Canadian veterinarians. Can vet J 1996; 37 (9):552-556.


Tranquilli WJ, Lamont LA, Grimm KA. Pain Management for the Small Animal Practitioner. 2nd ed. Teton New Media, Jackson WO, 2004:
3. Section 1: Pain Terminology, Physiology, Recognition, and Clinical Strategies, pp 2 – 12.


Useful resources:

International Association for the Study of Pain (IASP) website: http://www.iasp-pain.org/AM/Template.cfm?Section=Pain_Defi...isplay.cfm&ContentID=1728

American College of Veterinary Anesthesiologists (ACVA) website: http://www.acva.org/docs/Pain_Treatment
Rationale for including CRIs as part of an anesthetic protocol

- This technique is also called balanced anesthesia or partial intravenous anesthesia (PIVA)
  - A constant rate infusion (CRI) is used during anesthesia, in addition to an inhalant anesthesia maintenance
- MAC reduction
- Analgesia
  - Pre-emptive?
    - Not always possible, with trauma or pre-existing disease
  - Multimodal
- CRI technique achieves steady-state plasma concentrations of drug
  - This avoids peaks of drug levels, associated with potential toxicity, as well as troughs, associated with potential reduction of effect
  - Allows titration of dose to achieve the desired effect
  - May use less drug over time when compared to repeated bolus administration

Strategies for pain management:

Pre-emptive analgesia

- Providing analgesics prior to the painful stimulus
- Decreases pain intensity
- Decreases duration of pain
- Minimizes the likelihood that chronic pain and sensitization will develop
- Adjunctive to post operative pain management, not a replacement for post operative pain management
• Supporting evidence:
  • Review article from 1993 by Woolf and Chong showing that providing pre-emptive and intra-op analgesia dramatically reduce the pain response and the development of central sensitization in human patients
    ▪ Found that “pain should be continuously anticipated and preempted by persisting with preemptive therapy for as long as the abnormal afferent barrage from the wound and surrounding site is present, by using analgesic techniques targeted at three sites: the periphery, sensory inflow in nerves, and cells in the central nervous system”.

Multimodal analgesia

• Also called balanced analgesia
• Technique of administering two or more classes of analgesic drugs or two or more analgesic techniques
• Different classes of analgesics administered together often act synergistically
  • Improves analgesia with lower doses of drugs – less chance of side effects
• Minimizes the likelihood that chronic pain and sensitization will develop
• Decreases neuroendocrine responses to pain
• Decreases healing time
  • Fewer stress responses to surgical injury results in less tissue catabolism and maintenance of immune function
  • Less pain results in improved patient mobility
• Supporting evidence:
  • Pair of review articles from 2001 by Kelly et al. showing that drugs used to provide pre-emptive and multi-modal analgesia act synergistically to decrease post-op pain and healing time
    ▪ Very important to act pre-emptively whenever possible, and continue analgesia into the post-operative period, to minimize chronic pain and central sensitization
    ▪ Multi-modal analgesia is much more effective than using a single drug to provide analgesia
• Different analgesic drugs often work at different points along the pain pathway:
Drugs used in CRIs

Appropriate drugs:

- Drugs with a relatively short duration of action are preferred
- Opioids: fentanyl, morphine, hydromorphone, butorphanol
  - Small animals and horses
- Local anesthetics: lidocaine
  - Dogs and horses
- NMDA antagonists: ketamine
  - Small animals and horses
- Alpha-2 agonists: dexmedetomidine
  - Small animals and horses

Traditional Analgesics

Opioids

- **Controlled drugs:** Must be recorded – total amount (in ml AND mg) signed out to the patient, used, and discarded
- **Receptors:**
  - Located in the brain, spinal cord, gastrointestinal tract, heart, kidney, adrenal gland, joint capsule
  - Identified as mu (\(\mu\)), kappa (\(\kappa\)), and delta (\(\delta\))
- **Site of action:**
  - **Central:**
    - Dorsal horn of the spinal cord – inhibit pain transmission, increase pain threshold
    - Brain – inhibit pain perception, increase pain threshold
    - Descending inhibitory pathways
  - **Peripheral:**
    - Synovial membranes of joints
    - Produce local analgesic effects
- **Effects:**
  - Analgesia, sedation, euphoria
  - Side effects include dysphoria, excitement, bradycardia, respiratory depression, vomiting, panting, initial increase in GI motility followed by decrease in motility
  - Horses: can also produce excitement, decreased gastrointestinal motility, and urinary retention
    - Excitement is uncommon in painful horses unless high doses or very frequent administration are used
    - Untreated pain also causes decreased gastrointestinal motility, so this is not a valid reason to withhold opioids from a painful horse
    - Excitement, analgesia not clear cut in horses
- **Useful for:** Acute pain; best drugs to treat severe pain
- **Examples:**
• Full (mu) agonist
  • Exerts action at all receptors, and fully activates the receptor
  • Capable of providing profound analgesia
  • Most potential for side effects
  • Morphine, hydromorphone, fentanyl, methadone
• Partial (mu) agonist
  • Exerts action at mu receptors, and does not fully activate the receptor
  • Capable of providing moderate analgesia
  • Fewer side effects
  • Buprenorphine
    • Note buprenorphine has a very high affinity for the receptor, and is very difficult to antagonize once administered
• Agonist/antagonist
  • Agonist at kappa receptors, antagonist at mu receptors
  • Capable of providing mild analgesia, with few side effects
  • Butorphanol
• Doses:
  • Fentanyl:
    • Small animals: 1-5 µg/kg IV loading dose, followed by 5 - 20 µg/kg/hr
    • Horses: 1 µg/kg IV loading dose, followed by 1 – 5 µg/kg/hr
  • Morphine:
    • Small animals: 0.3-0.5 mg/kg IV over 5 minutes, followed by 0.2 mg/kg/hour (cats lower)
  • Hydromorphone:
    • Small animals: 0.05 mg/kg IV loading dose, followed by 0.01 mg/kg/hour
  • Butorphanol:
    • Small animals: 0.1 mg/kg IV loading dose followed by 0.1-0.2 mg/kg/hour
    • Horses: 0.02 mg/kg/hr after a bolus loading dose of 0.01 – 0.04 mg/kg

Local anesthetics (Lidocaine)
• Act at sodium channels in nerve membranes to provide analgesia and minimum alveolar concentration (MAC) reduction
• Block the conduction of nerve impulses by preventing sodium from entering cells, thus preventing the generation and propagation of action potentials
• Lidocaine also acts at many other sites to provide analgesia and MAC reduction:
  • Blocks:
    • Calcium and potassium channels
    • Pre-synaptic muscarinic receptors, NMDA receptors, TRPV-1 receptors
  • Activates:
    • GABA and glycine receptors
• Lidocaine also is an effective anti-inflammatory drug by inhibiting the sequestration, migration, and activation of polymorphonuclear cells, suppression of histamine release from mast cells, and decreasing cytokine release (and many other effects)
• Effects (IV use):
  • Analgesia, MAC reduction
• CRI use: LIDOCAINE ONLY!!
• Remember bupivacaine and the other local anesthetics should **NEVER** be administered IV
• Not recommended in cats – toxicity even at low doses
  • Useful in dogs and horses
• Used intravenously to provide MAC reduction and analgesia
• Used intra- and post-operatively, and to provide analgesia for painful conditions such as colic and laminitis
• Also used as a prokinetic to increase GI motility in colics

Doses:

Lidocaine:
• Dogs: 2 mg/kg IV loading dose over 5 minutes, followed by 50-100 µg/kg/minute
  • I usually decrease over anesthesia time, so I am only using 30-50 µg/kg/minute by end of surgery
  • I usually stop this CRI during surgical closure, so I have a true picture of patient’s level of analgesia in recovery
• Horses: 1 – 2 mg/kg IV **slowly** over 20 - 30 minutes, followed by 25 – 50 µg/kg/minute
  • Stop CRI 30 minutes prior to end of anesthesia
  • Associated with poor recoveries if continued until end of anesthesia

Side effects:
• Usually not encountered when local anesthetics are used at appropriate doses
• Seen with accidental IV administration of bupivacaine (VERY cardiotoxic), or if the rate of a lidocaine CRI is inadvertently increased
• CNS effects are seen first
  • But remember these are not always recognised in anesthetized animals
• Cardiovascular effects follow CNS effects – cardiac arrhythmias or cardiac arrest

**Adjunctive Analgesics**

• These are drugs with a primary indication other than pain, but that provide analgesia in certain painful conditions
  • This is different from traditional analgesics with a primary indication of pain – NSAIDs, opioids, local anaesthetics
• Often co-administered with traditional analgesics
• Administered intra-operatively, post-operatively, or to conscious painful animals with a non-surgical condition
• **Useful for:** Acute and chronic pain, especially pain that is non-responsive to traditional analgesics; often the first line of analgesics chosen for neuropathic pain
• To be used safely, the following information must be known:
  1. Approved indications for the drug
  2. Unapproved but accepted indications for the drug
    - Many of our veterinary drugs fall into this category
  3. Common side effects, as well as uncommon but severe side effects
  4. Pharmacokinetics and pharmacodynamics of the drug
  5. Dosing guidelines for pain
NMDA Antagonists (Ketamine)

Site of action:
- Dissociative anesthetic – good somatic analgesia, short duration
- N-Methyl-D-Aspartate glutamate receptor
- NMDA activity contributes to central nervous system sensitization and hyperalgesia
  - NMDA receptors are important in the process leading to central sensitization
- Phencyclidine derivative, dissociative anesthetic agent

**Controlled drug**
- Must be recorded – total amount (in ml AND mg) signed out to the patient, used, and discarded

**Effects:**
- Low doses administered as a constant rate infusion (CRI) during anesthesia provide MAC reduction and analgesia
- Acts to decrease post-operative pain, and to decrease opioid requirements
- Antagonism of NMDA receptors can minimize “wind up” of dorsal horn neurons, preventing (or minimizing) sensitization and chronic pain

**Sub-anesthetic doses** are used:
- Horses: 0.2 mg/kg IV loading dose, followed by 0.4-0.6 mg/kg/hour
- Dogs and cats: 0.5 mg/kg IV loading dose, followed by 0.6 mg/kg/hour (10 mcg/kg/minute)
- The “micro-doses” used to manage pain cause no or minimal behavioural effects, and no cardiovascular effects
- If side effects are seen: muscle fasciculations, increased locomotor activity, excitement

Alpha 2 agonists

**Site of action:**
- Alpha-2-adrenergic receptors in the dorsal horn of the spinal cord and the locus ceruleus in the brain stem
- Analgesia results from spinal and supra-spinal effects, and is mediated by serotonin and the descending endogenous analgesia system

**Effects:**
- Sedation, analgesia, muscle relaxation
- Side effects include significant cardiovascular depression, even at very low doses
- Synergistic with opioids
  - The analgesia that results from the combination of an alpha-2 agonist and an opioid is more profound than that resulting from either drug alone

**Also useful to manage pain in horses**
- Xylazine was shown to be more potent in horses for treating experimentally induced colic than butorphanol or meperidine/pethidine
  - Muir + Robertson AJVR 1985
- Alpha-2 agonists do cause decreased gut motility, however
- Caution if signs of pain are used as a prognostic indicator, or to decide if referral is needed

**Remember** the significant cardiovascular side effects of alpha-2 agonists
- Even the smallest of doses carries these side effects – it is not true that small doses are free of cardiovascular side effects
  - Carter at al found in 2010 that even 1-3 mcg/kg/hour caused CV depression in dogs
• Patient selection is **always** the key with these drugs, even though horses tolerate the cardiovascular side effects better than small animals do
• I often use dexmedetomidine as a CRI during equine anesthesia, but I almost never use an alpha-2 agonist CRI in small animals during anesthesia
• Examples:
  • Dexmedetomidine
    • Low doses administered as a CRI during anesthesia provide MAC reduction and analgesia
    • Horses: 3 \( \mu \)g/kg/hour
    • Dogs and cats: 1 \( \mu \)g/kg IV loading dose followed by 0.5-1 \( \mu \)g/kg/hour
  • Xylazine: 0.3 – 0.5 mg/kg IV
    • CRI: 0.2 – 0.5 mg/kg/hr after bolus
  • Detomidine: 5 – 10 \( \mu \)g/kg IV
    • CRI: 0.6 \( \mu \)g/kg/minute, decreasing by ½ in 15 minutes
  • Romifidine: 50 –80 \( \mu \)g/kg IV
    • CRI: 80 \( \mu \)g/kg/hour

**Techniques for administering CRIs**
• Low dose of drug is given intravenously in a continuous, instead of bolus, manner
• Often a bolus of drug is given as a loading dose at the start of the CRI to facilitate development of therapeutic plasma concentrations
• Equipment required
  • Syringe pump
    • Most accurate method
    • Most expensive method
  • Buretrol
    • Inexpensive
    • Small chamber that holds up to 100 ml fluid
    • Usually put one hour’s worth of drug into chamber, dilute to 60 ml with IVF, and run at 1 drop/second
    • Place drug label on buretrol chamber, to indicate what drug has been added
    • What it looks like:

• Drug added directly to IV fluid bag
• Very inexpensive
• MUST place a “medication added” label to the IV fluid bag
  • Bright orange or yellow label
  • Indicates base drug or fluid (e.g. LRS), drug added (e.g. ketamine), time and date drug was added, name of person who added the drug
• Must have another bag of IV fluids with no drug added, in case a fluid bolus is required (you don’t want to inadvertently bolus your analgesic drug...)

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