Scientific Proceedings

2018 CVMA Convention
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Overview

Millennials are creating a complex cultural shift in veterinary hospitals. By 2025, they will make up well over 50% of your hospital staff. As you may have discovered, millennials don’t respond to the same kind of training practices that have been used for years in the veterinary field. In How to Train Your Millennial, we will discuss the ways millennials learn, how it is different from prior generations, the most efficient ways to help them receive and retain information, and how to communicate progress (or lack thereof) to trainees. Lastly, we will discuss an innovative way of keeping your employees engaged with the practice well after they are past the new hire phase, while providing relief and added time to your practice manager’s week.

Key Objectives

- Understand how millennials learn differently and why
- Learn about the most efficient, cutting edge ways to help them learn
- Discuss the importance of communicating progress
- Identify quick ways of changing training immediately to increase results and engagement

Notes

- Millennials have a different way of learning than prior generations.
  - They need their information to be free-flowing and organically presented to them.
  - They learn best when they are allowed to choose when they learn different topics.
  - The classic checklist training system does not work for them.
- 51% of employees of hospitals starting with iVET360 feel training program is not effective.
  - 71% of millennials feel their training program is not effective.
- By 2025, millennials will be 75% of workforce 1, and Gen Z is already starting working (Gen Z: Those born after 2000)
- Millennials had a variety of mediums when learning in school:
  - Lecture
  - Individual research time
  - Videos
  - Group learning
  - Group projects
  - More accurate to call them the “We” generation
- Millennials want:
  - A leader who cares, acts like a mentor
  - Team-based training
  - Understanding the “why”
  - Flexible training system
· “Overworked” is a state of mind, not work. Teams with excellent training can sustaining periods of short staffing without feeling overworked.
· Always ask new hires (and even the current staff) how they learn best, and alter your training style to match it.
· Checklists are most effective when the employee oversees determining what to learn next
  o Give them timeframes and a list of people to learn from.
  o Check in with new hires every week!
· Implement microlearning, such as:
  ▪ videos,
  ▪ articles,
  ▪ podcasts,
  ▪ flashcards
  ▪ quizzes,
  o Teaches relevant information in short periods of time. This caters to the new, lower attention span of employees.
  o Use microlearning for long-term engaged learning for all team members.
· Sit down with new hires at least weekly to discuss progress and their experience in your practice, and then move to monthly once they are through the new hire phase. Use a form to track these conversations.
· Gamify your training.
  o Use a combination of:
    ▪ levels,
    ▪ challenges,
    ▪ badges,
    ▪ social status (most important part!)
  o Improves engagement and creates excitement around learning.
  o Make sure participation is voluntary!
Don’t forget to recognize your team every day. Use an app to make it more convenient and fun.

For handouts and forms, please visit https://ivet360.com/checklists/.

References:
Show Me the Money!
Heather Romano
Managing Director of Staff Development/ Co-Founder: iVET360

Summary

As millennials have entered the workforce, there has been a surprising shift in the compensation expectations of veterinary employees. As millennials prefer certain perks over salaries, veterinary practices are struggling to provide compensation packages that are enticing talent to stay in our practices. However, by understanding their motivations, and how they have affected other generations in the workforce, we can create an atmosphere of excitement surrounding your practice’s benefits, which will make your hospital the employer of choice in your area. Included will be a discussion of salary and benefit expectations and aspirations for veterinarian and a new way of looking at benefits.

Notes

- Veterinary practice benefits typically make up 8% of an employee’s total pay. This is on par with businesses in 1970.
- We choose to compete with other hospitals instead of other industries, resulting in a staff turnaround average of 30-51%
- When benefits are increased, turnover falls:
  - 1970’s: 35% average turnover
  - 2015: 16.4% average turnover
- Turnover costs- CLASP/ CEPR calculation method
  - Average practice turnover expense: $46,250
  - Rough average of $5000 per employee
  - Does not include secondary cost, like lost clients
- Average turnover in other industries:
  - Human healthcare: 19%
  - Hospitality: 38%
  - Retail: 67%
  - Non-profit: 18%
- Veterinary field has the worst turnover in professional industries
  - 3 main reasons:
    - What we pay our employees
    - What benefits we offer them
    - Overall lack of focus on culture
- Improving pay and benefits can boost morale long enough to work on culture.
- Millennials want a boss who will look out for their best interests. Want them to care about:
  - The personally
  - Their individual needs
  - Helping them understand available options
- Perks Vs Benefits
  - Benefits: Part of the employee’s salary package
    - Health/dental coverage, PTO, 401K, commute stipends, CE
    - Benefits set you apart from competition
  - Perks: Auxiliary items to make work more enjoyable
• Career development, snacks, paid lunches, etc
• Perks are for staff retention

• For millennials, pay is still #15
• What should I pay?
  o Depends on where you live
  o Higher pay & benefits, lower turnover, higher loyalty
  o General rule: Pay $1 more than local competition
  o How to find out competition?
    ▪ Local and state VMA surveys
    ▪ Practice manager group meetings
    ▪ Payscale.com, Salary.com, etc
    ▪ Glassdoor.com
    ▪ Phone call!
• Doctors: Expect base pay + production (PROSAL)
  o Base expectations depend on location:
    ▪ Some areas: $50K for new grads
    ▪ California: $70,000-80,000 minimum
    ▪ Alma mater can make a difference in expectations
  o EXPECT NEGOTIATION!
  o Production expectations:
    ▪ 5-10 years ago: 20% was normal
    ▪ Some private and most corporate practices have a “negative accrual” method
    ▪ 20% appropriate for newer grads
    ▪ Experienced doctors expect 22-26%
    ▪ Specialists? Up to 35%
    ▪ Research shows doctors who work on higher percentage work harder to achieve production
• Raises
  o 1-4% is not enough anymore, millennials find this insulting
  o Don’t recommend associating raises with evaluations
  o iVET360 Research: After performance evals and “token” raises, increase in employees quitting
  o Pay for potential:
    ▪ 9 box model
    ▪ Raises based on potential dramatically increase rock star loyalty
    ▪ Ratio of how high someone’s potential is to how soon they could reach it: higher or sooner potential means larger raises
    ▪ Not everyone is a rock star, more like 10-20% f your team
      ▪ Everyone else goes into the other categories
      ▪ Those who aren’t at least meeting expectations with good potential should be managed out
  o Doctor raises
    ▪ Doctors want an increase in their production percent
    ▪ Make benchmarks!
• Benefits
  o 55% of millennials feel the benefits and perks offered are an important reason they came to work for their employer 6
  o 63% say benefits are a critical reason they stay with their employer 6
  o Business world: Benefits = 30% of compensation
  o Veterinary field: 8%, before discounts
  o Top benefits:
    ▪ Health insurance
      • Be sure you survey the staff periodically to make sure your plan is meeting their needs
    ▪ Paid time off
      • US average: 13 vacation days and 8 holidays, plus sick time
      • Average veterinary: 10 vacation days, 3 holidays, little (if any) sick time
      • Encourage the team to take time off to improve well being
    ▪ Paid sick days
    ▪ 401K/ retirement with match
    ▪ Performance bonuses
      • Most prefer bonuses paid on performance to token holiday bonuses
    ▪ Other benefits preferred to a raise:
      • Flexible schedule
      • Additional perks (lunch, etc)
      • Employee development programs
      • Tuition reimbursement
      • Employee discounts
      • Gym memberships
      • Wellness programs and stock options
      • Paid parental leave and childcare assistance
      • Commuter assistance
  o How to boost benefits without breaking the bank?
    ▪ Benefits menu program
      • Choose 3 main benefits (such as healthcare, 401K, PTO)
      • Give an employee a dollar amount and a menu of other benefits, each with a monthly “cost” they can use their allowance on
      • 50% of millennials prefer this
      • 80% of workers would prefer a new/ additional benefit to a raise
      • Can add a benefit for less money than a raise would cost

• Perks
  o #1 most valuable perk: training and development 7
    ▪ Improve new hire training
    ▪ Ongoing career development
      • Career focused
      • Personal focused
  o Other perks:
    ▪ Extra PTO at the holidays
    ▪ On-site massage/ spa services
- Drink fridge
- Ping pong/ fuseball table
- Company sponsored sports teams
- Off-site events
- Paid birthday off
- FOOD!
- Paid community service time
- Clubs

- HOW TO IMPLEMENT:
  - Evaluate salary
  - Evaluate and adjust wage increase structure
  - Evaluate benefits
  - Benefits menu system?
  - Pick a few perks to offer
  - **Be employee focused without breaking the bank!**

For handouts and forms, please visit https://ivet360.com/checklists/

References:
Don’t Fear the Feedback
Heather Romano
Managing Director of Staff Development/ Co-Founder: iVET360

Overview

As veterinary professionals, we typically hate one specific aspect of our jobs—managing underperforming staff and doctors. Unfortunately, by overlooking this critical aspect of staff leadership, we allow underperforming, and often toxic employees, to destroy our culture and procedures, leading to outstanding employees quitting while the under performers linger. In Don’t Fear the Feedback, we will discuss how our staff feels about feedback, how we can provide more effective feedback at reviews, compassionate staff leadership strategies, and the best ways to discuss performance and attitude deficits with different types of people. Finally, we will discuss a feedback and accountability structure that allows your team members to hold themselves accountable to their own jobs, being fully aware of the rules and consequences, while creating less confrontation for the managerial staff.

Notes

• Our brains tell us we are right, even when we are wrong. Criticism is a threat to survival, so people perceive it as more aggressive than it actually is.
• New workplace:
  o Emphasis on collaborative work environments
  o Shouting is no longer acceptable
  o Punitive consequences instill anger and resentment, not compliance
  o Annual performance evaluations are not enough feedback
• Why millennials are different
  o Grew up with constant praise
  o Played multiplayer video games, ranking and winning is paramount
  o Hear, “you aren’t as great as you think you are,” for the first time at work.
• Think of feedback like tweets: done frequently, on the spot, and kept brief
• Coach, don’t boss
  o Coaches teach, bosses tell
  o Gain their trust
  o “The boss” doesn’t garner respect anymore
  o Millennials don’t see the boss as a “boss,” authority must be earned
  o Tell them about your experiences and your failures!
• Accountability
  o All employees want a culture of accountability
  o They want rules, and to know that everyone will be held accountable to them
  o Utilize a points system to help improve accountability
    ▪ Easily tracked
    ▪ “Fair,” not subjective
    ▪ Less confrontational and constant sit downs
• Treat people the same, but talk to them differently:
  o Feedback Fanatics (most millennials)1
    ▪ Constantly seek approval
    ▪ Prefer positive feedback
• Demand a LOT of time and attention
  o How to manage feedback
    ▪ VERY frequent feedback
    ▪ 3-5 positive comments for every constructive
    ▪ PUSH yourself to recognize their achievements/performance

• Persecution Complex 1
  o Characteristics
    ▪ Argue into segues
    ▪ Never at fault, “Why do you pick on me?”
    ▪ Frustrating to coach
  o How to manage feedback
    ▪ Keep bringing them back on point
    ▪ Don’t dismiss their concerns, assure you’ll come back to them later
    ▪ 3-5 positive comments for every constructive
    ▪ PUSH yourself to recognize their achievements/performance

• Intimidators 1
  o Characteristics
    ▪ Show intimidating behavior: walking around the room, ignoring you, avoiding eye contact, interrupting, etc
    ▪ Know it causes managers to avoid providing them feedback
  o How to manage feedback
    ▪ Be sure you provide VERY frequent feedback
    ▪ Keep calm, speak slowly and softly
    ▪ If that doesn’t work, be silent

• Alibiers 1
  o Characteristics
    ▪ Always ready with an excuse
    ▪ Never, ever to blame
    ▪ Usually have compelling arguments
  o How to manage feedback
    ▪ Focus on the problem
    ▪ Don’t ignore the problem, but don’t allow it to soften the consequences or conversation
    ▪ Concentrate on what the employee CAN control: their actions

• Hostiles 1
  o Characteristics
    ▪ Common response to hurt feelings, but often a personality trait
    ▪ They know it prevents some managers from providing feedback
    ▪ Need to hear positive feedback, but they don’t always acknowledge it.
  o How to manage feedback
    ▪ No personal criticism, keep it fact-based
    ▪ Get to source of hostility, ask probing questions: “You seem to struggle with this often, can you be more specific?”
    ▪ Be authoritative, but empathetic

• Insubordinates 1
  o Characteristics
    ▪ Reject your authority
    ▪ Break rules on purpose
- Find no validation in any feedback you provide
  - How to manage feedback
    - Find out why they reject you, repair relationships where you can
    - Emphasize job goals and responsibilities, focus on performance deficits
    - Discipline for blatant neglect/disrespect

- Thin Skinned 1
  - Characteristics
    - Shaky self-confidence, take everything personally
    - Tend to cry when confronted, use tears to avoid conflict
    - Managers fearful of breaking the little existing self-confidence
  - How to manage feedback
    - Concentrate on the facts
    - Be solutions-focused, give them the tools to improve
    - Don’t allow tears to stop discussion

- Newbies! 1
  - Characteristics
    - Need LOTS of feedback, all the time
    - Nervous, want to impress
    - Don’t know if they are doing poorly if you don’t tell them (they assume they are doing well)
  - How to manage feedback
    - Start feedback early, do it often
    - Don’t hand-hold, but provide feedback to prevent “watch and learn”
    - Check-ins daily for 2 weeks, then weekly for 2 months, “formal” 30, 60, 90 day evaluations, then monthly check-ins

- Overly-Cautious 1
  - Characteristics
    - Diligent, but SLOW
    - Terrified of making a mistake, perfectionist
    - Creates bottlenecks and decreased efficiency
    - Not doing anything WRONG...
  - How to manage feedback
    - Find out WHY, then tailor feedback
    - May need more training, follow-up
    - Assign detail-oriented tasks without timeframes

- Give praise!!
Performance evaluations
- Annual performance evaluations should be a thing of the past
- People need more frequent feedback
- Perception: performance evaluation = raise
- Best Practice: Monthly Meetings/ Brief Meetings (BM’s!)
  - Performance evaluations should be summary of the years BM’s
  - Nothing should be a surprise

Positive language for positive results
- Feedback should ALWAYS come from a place of caring
- Can’t be caring now? Then wait to provide feedback
- Avoid negativity: “I don’t think,” “You shouldn’t”
- Use positive verbiage:
  - “Have you considered”
  - “What I need from you”
  - “What is the best way to improve here”
  - “How would you feel if we”
- Be empathetic: What went wrong, what to do differently
  - Remember, they are humans with feelings
  - “I’ve been in this situation before.”
  - “I know this can be hard to hear”
- Offer solutions
- Constant, specific, positive feedback is KEY
  - This is the hardest part of feedback
  - It makes it easier for employees to accept constructive feedback if they hear they are doing well, too.

IDEALS Mark Murphy, Hundred Percenters 2
- Invite them to partner
  - “Would you be willing to have a conversation about...”
  - “Is now a good time to talk?”
- Disarm yourself
  - Make yourself an ally, not an enemy
  - “I’d like to review the situation with you to make sure we are on the same page.”
  - “I want to be sure I understand everything that happened with Mrs. X.”
- Eliminate blame
  - Focus on solutions, not fault
  - “We seem to have different opinions about this.”
  - “That’s OK, let’s work together to find a solution.”
  - “I know you don’t agree with me here, what do you think can be done to change the perception in this case?”
- Affirm their control
  - Reaffirm they have some level of control in the situation
  - “Does this sound OK?”
- “Does this sound fair to you?”
- “What are your thoughts about that?”
  - List corrective action
    - Keep it specific and understandable
    - “What seemed to happen in this case was X, what I need to see in the future is Y.”
    - “It sounds like you were trying to accomplish X, it would be more helpful next time to try it this way.”
  - Synchronize your understanding
    - Keep the dialogue open
    - “Tell me how we can work together to prevent this from happening again.”
    - “How can we help you be more comfortable with this?”

- Follow-up!
  - Checking back and providing feedback is critical to gaining respect, trust, and credibility from the employee
  - Put a reminder on your calendar, block off some time. DO whatever you have to do to remember this important step!

- Things to remember:
  - Genuinely care, even for your “worst” employee
  - Listen. Bosses talk, coaches listen
  - Be honest, but kind
  - Be specific, provide examples for both positive and constructive feedback
  - Be inescapably clear, check for understanding
  - Lead by example
  - Sit next to them or on a corner. Don’t sit across from them
  - Until feedback becomes routine, it is a threat. Understand if they are scared, and treat them appropriately

For handouts and forms, please visit https://ivet360.com/checklists/.

References:
Managing Director of Staff Development/ Co-Founder: iVET360

Overview

Often, hospitals call their group of workers a “family,” as though this is a good thing. But let’s face it; families are full of strife, discord, and frustration. Many of us would never want to work with our families, so why would we want to recreate that feeling at work? In It’s All in the Family, we will take a fun look at what a “family” feel can do to a practice’s culture, and how taking a high-performance sports team mentality into your practice can make us more efficient and more effective at providing stellar patient and client care. Attendees will come away with a checklist of team do’s and don’ts to implement in their practice when they return to change the family mentality into a strong team focus.

Key Objectives

- Why a family environment can be toxic to the staff work environment
- Learn about the key identifiers of a team environment
- Discuss how to turn a family environment into a team environment
- Identify ways to break free of the family feel in a practice

Summary

1. Family environments are great for home life, but bad for work. They allow us to tolerate poor behavior out of a sense of loyalty and even displaced affection. This creates a toxic environment.
2. Staff members fall into family hierarchy based on the dynamic and not based on their strengths
3. Team means rules, structure, and expectations
4. In a team, strengths are utilized, weaknesses are worked on or compensated for by other team member strengths. Accountability is key.
5. Agape love is important in a team environment. Simply put, agape love is treating others with kindness and respect always.
6. To create a team environment, create rules, evaluate your hiring, build trust, clarify roles, communicate openly and effectively, create and appreciate diversity, balance the team’s focus, and provide opportunities for learning and development.
7. To remove the family element, remember that you are not their parent, and don’t accept poor work from team members. Don’t let loyalty cloud your judgement, encourage the team to be their best and hold them accountable. Make the team work out their own problems, and establish the “right to play” rules.
Summary

Often, leaders in the veterinary field feel they must be dictatorial to get the results they want from their staff. This heavy-handed approach, however, is seen by younger generations as “old-school,” abrasive, and even hostile. Leading this way now drives away some of our brightest talent, leaving us with individuals who have learned to “put up” with this kind of direction. In Leading with Love, we discuss how today’s veterinary professionals prefer to be communicated to, how the same principles that make our personal relationships work are also appropriate for our work relationships. Based on Joel Manby’s Love Works, we will discuss seven key principles of leadership, translating them into actionable concepts that you can use in your practice the moment you return. We will wrap up with a discussion of simple Rules of Engagement for Supervisors, a list of rules to help ensure a harmonious, yet productive and mutually accountable work environment.

Notes

- Gallup poll of 2 million employees: Most rate a caring boss higher than how much money they make
- Millennials:
  - 12% prioritize “being wealthy” as a goal
  - 79% want a boss who is a coach or mentor
  - 88% prefer a collaborative environment
  - 90% feel their voice and opinion should matter
- All people want to be respected at work, fewer and fewer will tolerate a disrespectful environment
- When asked, “How long would you tolerate a poor supervisor before quitting?”
  - 1995: 5-10 years
  - 2014: 5-10 months
- Supervisors and managers are #1 reason people love or hate their jobs
- Leading with love will help you be the premier “employer of choice” in your area
- 4 Greek words for love:
  - Eros: lusty love
  - Philos: brotherly love
  - Storge: familial love
  - Agape love: loving behavior, not a “feeling”
- Agape love:
  - Foundation for the best relationships
  - Deliberate and unconditional
  - Can exist in most difficult environments, especially work
  - You can treat someone with agape love, even when you don’t like them personally
- It’s hard to talk about. We speak openly about business strategy, harder to talk about how to treat people
- 2 key things to remember:
When you don’t like someone, that is when you need to BEHAVE like you love them.

Treat all people with love at all times.

- Joel Manby: Love Works: Discusses 7 key words to remember for working with others.

- Patient: Self-control in difficult situations
  - Be genuine. False praise kills credibility
  - Praise without specifics is worse than no praise at all
  - Don’t “compliment sandwich” Be upfront, honest, and kind in your criticisms
  - Don’t be patient with poor performance, be patient in how you RESPOND to it
  - Think before you speak, wait for the right moment and the right words
  - How can I be more PATIENT:
    - Wait until you can admonish in private. Respect their dignity, they are human, too.
    - Get to the point, be specific, get them back on track, and then never mention it again
    - Stop. Breathe. Plan, then discuss
    - Praise 3 times more than you admonish

- Kind: Show encouragement and enthusiasm
  - Every interaction, you can make their day better or worse. It is your obligation to make it better, and never worse
  - “Making their day better” is contagious and increases energy, effectiveness, and productivity
  - The enthusiasm of the client experience can never rise above the enthusiasm of your employees
  - Kindness is about intentionally creating and maintaining the right environment so the team can deliver an enthusiastic client experience.
  - How would employees rate you on kindness factors like enthusiasm, passion, and encouragement?
  - How can I be more KIND:
    - Meaningful thank-you notes to staff. Take the time to encourage
    - Meaningful thank-you notes to staff’s family
    - CEO: Chief Encouragement Office: How many employees can be CEO?

- Trusting: Place confidence in people
  - Trusting your team is critical to building a climate of positive morale and performance
  - Taking over projects, hoarding information, shooting down ideas, and interrupting others are signs of distrust. Tells the they are not important; that you are better than they are
  - Interruption isn’t efficient, and it isn’t just rude, it’s degrading.
  - Treating yourself differently shows disrespect and lack of trust
  - Trustful leaders must let people do their own jobs, find their own solutions, and even make occasional mistakes to learn.
  - How can I be more TRUSTING:
    - Let your team make some decisions:
• Responsible: who is responsible, “owns” the recommendation
• Approve: Who has to approve, “owns” the decision
• Consult the people directly affected
• Inform the rest of the team
  ▪ Trust people until proven wrong
  ▪ Be a listener, always

• Unselfish: Think of yourself less
  o Selfish leaders need to remind people they are in power, like to hold on to power
    instead of giving it away
  o Giving time and talent to develop leaders is an excellent example if being unselfish in
    the organization
  o Don’t become “numb” to the needs, emotions, and personal issues in employee’s
    lives. Listen and care.
  o Wait until you can admonish in private. Respect their dignity in your words and
    actions
  o Decide to decide less. If you are becoming involved in someone’s work, you have lost
    faith in their judgement.
  o How can I be more UNSELFISH:
    ▪ Give your skills to make the company and world better. Give your time to
      others
    ▪ Be unselfish with making decisions. Strong leaders of strong companies make
      fewer decisions
    ▪ Ask more questions, without your own opinions already formed, to make
      better decisions

• Truthful: Define reality
  o Create rules in meetings to get to the truth:
    ▪ Don’t shoot the messenger
    ▪ Don’t confuse disagreement with conflict (conflict happens when people take
      disagreement personally)
    ▪ Don’t assume others will see it your way
    ▪ See the truth
    ▪ Speak up
  o If we love our team, it is critical their voices are heard, opinions considered.
  o Getting the truth out ensures real decisions are made with the company and
    employee’s best interest
  o Leaders must be held accountable for their actions, their truth.
  o Firing someone is when truth is critical, as well as love and care and compassion.
    Protect their dignity.
  o How can I be more TRUTHFUL:
    ▪ Be honest, not soft, and truthful with employees
    ▪ Firing someone should never be a surprise. If it is, you have not done your job
      and have not been honest
• Speak last so as not to influence the thinking of the team. Easier to find the corporate truth when you get out of the way.

• Forgiving: Release the grip of the grudge
  o What was done to you doesn’t matter in the end. All that matters is how you respond
  o Giving someone a second chance doesn’t always work out, but consider it anyway
  o Forgiving those who wronged you or the organization releases you to focus on relationships and not anger
  o Forgiveness has a positive ripple effect that often extends far beyond our comprehension
  o How can I be more FORGIVING:
    ▪ Forgive those who have wronged you or the practice
    ▪ Be slow to fire and quick to forgive
    ▪ Never say anything negative about a prior client or employee

• Dedicated: Stick to your values in all circumstances
  o Leader primary role: give and inspire hope. Achieved through clarity of vision, mission and values; creates financial success
  o Great leaders need to use both love and power
  o Measure BE goals and DO goals:
    ▪ DO goals: career choice, reaching a revenue goal, growth plans, etc.
    ▪ BE goals: What kind of person do I want to be? What kind of values do I uphold? What is my integrity when no one is watching?
  o Leaders must make difficult decisions, how they handle them separates who does and doesn’t lead with love
  o It is possibly to lead with love during the tough times, but it takes dedication.
  o How can I be more DEDICATED:
    ▪ BE goals for leaders: What kind of leaders do we want? What kind of behaviors and attitudes are expected? How will they balance love with DO goals?
    ▪ Stick to your values in all circumstances
    ▪ Remember, love works, not EVEN in hard times, but ESPECIALLY in hard times

• How can I implement?
  o Supervisor Rules of Engagement
  o Keep the rules handy (How can I be more...)
  o Evaluate with love
  o Same as/ More of/ Less of
  o DO goals/ BE goals

• Only 10% of companies have clearly defined values. Be one of the 10%
• Leading with Love is a higher testament to one’s leadership ability than fear-based, power-hungry, or back seat management
MAKE THEIR DAY BETTER. ALWAYS.

For handouts and forms, please visit https://ivet360.com/checklists/

References:
Introduction
It is impossible to describe all the dental and oral pathology and pathophysiology in a single set of notes. Instead, the lecture and notes will focus on a visual tour of common dental and oral pathology and the subtle visual and examination clues indicating hidden subgingival disease which will be captured with intraoral radiography.

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

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

The occlusion should be evaluated in the non-anesthetized patient so that the relationships of the teeth and bones can be determined before an endotracheal tube is placed. The normal canine mesocephalic skull has anisognathic mandibles. With orthocclusion, the mandibular incisors occlude on the cingulums of the maxillary incisors, the mandibular canines interdigitate, without touching, between the maxillary third incisors and canine teeth. The tips of the mandibular 4th premolars will point directly upward between the maxillary 3rd and 4th premolars. The mandibular and maxillary premolars interdigitate, and the tips of the upper and lower second premolars are at the same horizontal level.
Appropriate pre-anesthetic blood testing is obtained based on patient signalment and history. No patient is anesthetized without minimal blood work (packed cell volume, BUN, glucose, and total protein). However, most dentistry patients are older and more extensive bloodwork such CBC / Chemistry Panel / Urinalysis are necessary and recommended for anesthetic planning.

**Anesthesia and The Oral Examination**

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

**Pathology**

*Hidden palatal maxillary canine oronasal fistula*

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

Diagnosis of maxillary canine tooth palatal oronasal fistulas can be missed on intraoral radiographs due to summation of anatomical structures. Assessment with radiographs and clinical examination probing is necessary. Surgical extraction and appropriate tension free closure with mucoperiosteal flaps is necessary to correctly treat the oronasal fistula during the surgical procedure. This is an example of always correlating intraoral radiograph findings with the complete oral examination.

*Parulis*

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

*Maxillary 1st and 2nd molars Hidden Periodontal Disease*

The maxillary 1st and 2nd molars in dogs may have minimal clinical probing depths but may be mobile during clinical examination. The intraoral radiographs may identify a very wide or absent periodontal ligament space/large palatal root periapical lucency. Likewise, the superimposed buccal roots may have 50-100% bone loss (Periodontal Disease Stage 4) and without careful evaluation of
the intraoral radiograph the bone loss is easily missed. These clinical and radiographic findings are consistent with severe periodontal disease and surgical extraction and closure of the extraction site is required.

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

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

*Feline mandibular 1st molar periodontal disease*
Many domestic cats have a tight occlusion and with little translation movement of the temporomandibular joint the resulting maxillary 4th premolar cusp can traumatize the buccal aspect of the mandibular first molar. Likewise, brachycephalic cats often have a scissors or level occlusion of the incisors. However, the mandibles have bowed laterally during growth. As a result, the central cusp of the maxillary 4th premolar contacts the mesial/buccal tooth and periodontium of the mandibular 1st molars resulting in periodontal dehiscence and disease.

Likewise, the veterinarian may extract the mandibular 1st molar and identify the surgical site is not healing and/or identify a mass pre- or post-extraction that has a histological description such as pyogenic granuloma, lymphoplasmacytic gingivitis, etc. secondary to the trauma created by the maxillary 4th premolars.

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

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

Assessment, intraoral radiographs, and surgical extraction or endodontic treatment, if not periodontal disease, of the maxillary canine tooth is necessary. However, permanent damage may preclude complete resolution of the epiphora.
**Drug related gingival enlargement/hyperplasia**

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

**Feline sublingual squamous cell carcinoma**

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

**Maxillofacial swellings and draining tracts**

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

**Periocular Swellings**

The dentition is closely related to maxillofacial structures and the orbit/eyes. Periorbital swelling, chemosis, swelling closed of the palpebral fissure can all occur secondary to odontogenic infection. Hence, not only an ocular examination warranted but close evaluation of the oral cavity is necessary. Many patients end up being treated and/or referred to ophthalmologists only to then be referred for odontogenic infection.

**Deciduous tooth fractures**

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

**Uncomplicated crown fractures**

Dentin contains 45 000 – 70 000 tubules/mm² allowing oral bacteria to translocate into the endodontic system and result in pulpitis and death of the tooth. The clinical point is that exposed pulp (complicated crown fracture) always leads to endodontic disease but ALSO uncomplicated crown fractures and enamel fractures exposing dentin tubules can lead to endodontic disease.

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

*Dentigerous Cysts*

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

*Complicated Crown Fractures*

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

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

*Non-vital Teeth*

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

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

Likewise, many examples of full mouth intraoral radiographs during a periodontal cleaning have documented and published identification of hidden non-vital teeth without any color or morphological changes in the teeth.
Apoquel (Oclacitinib)

Please read the package insert! Please read the label and approved usages. It has a good approved usage in veterinary medicine. But do not let it be a honey moon drug. Please read the contraindications and when not to use it. Please understand it is not to be used in face of infections, cancer, and pre-cancerous conditions. It ideally is not to be used long-term and not studied long-term with other immunosuppressant such as cyclosporine, another immunosuppressant at this time. Understand that periodontal and endodontic disease are chronic infections and inflammatory conditions in constant battle with the immune system. Understand it may be possible to unmask and cause acute on chronic exacerbations and acute on chronic infections in geriatric patients, in particular, when using off label (e.g. for “rhinitis”), long-term, and/or concurrently with other immunosuppressant medications. Understand the relationship between the tooth and nose relationship. First do no harm! Regardless, this drug is not to be used in precancerous conditions so another reason not to use it without a diagnosis. I have never prescribed the medication it but many of my patients have presented on it urgently through referrals and the emergency department for acute on chronic maxillofacial infections.

I only have anecdotal observations in several patients and cannot draw any causal relationship or an association but keep an open mind in your own patients and think twice about the geriatric patient with severe periodontal disease or the Labrador with the chronically fractured maxillary 4th premolar before you reach for the prescription pad as you are about to prescribe a drug where the manufacture warns you not to use in the face of infection.
Ophthalmological and Maxillofacial Manifestations of Dental Disease
Kevin S. Stepaniuk, B.Sc., DVM, FAVD

Introduction
Patients often present with maxillofacial swellings, periorbital swellings, periocular swellings, and difficulty and/or pain opening and closing the mouth. There are many causes including odontogenic (dental), ophthalmological, skeletal and soft tissue pathologies that must be considered. Obvious causes may or may not be clinically evident and what may seem the “obvious”, may be hidden odontogenic infection. Particularly when the patient presents with acute ophthalmological manifestations. With acute maxillofacial and ocular swellings and inflammation the patient pain and temperament often precludes an obvious diagnosis without sedation or general anesthesia. Obtaining a correct diagnosis is required to plan and begin appropriate treatment.

Ocular Manifestations
Chemosis may occur with orbital and periorbital inflammation drawing attention to ocular pathology as the primary cause. However, the swelling may be secondary to odontogenic infection resulting in the periorbital/orbital pathology. Exophthalmus, which may have orbital causes, must be differentiated from buphthalmia from primarily intraocular causes (e.g. glaucoma). Draining tracts and ventral periocular mass effects can occur secondary to odontogenic infection. Likewise, a chronic smoldering retrobulbar cellulitis/abscess may lead to many ocular changes.

Ocular Complications Related to Anesthetic and Dental Procedures
Regional nerve blocks (i.e. local anesthesia) in the maxillofacial skeleton may result in periocular and intraocular damage and loss of an eye if performed incorrectly. Both the maxillary and infraorbital nerve blocks can be incriminating. Placing the needle too deep in the dorsal direction for an intraoral maxillary block, too dorsal over the zygomatic arch for an intraoral infraorbital block, and/or entering the infraorbital canal and not remaining parallel to the dental arcade can lead to ocular penetration. Additionally, regional hematoma is a common and anticipated finding of nerve blocks. A hematoma secondary to the maxillary nerve block may lead to a temporary exophthalmus.

Spring loaded mouth gags or any mouth gag resulting in the feline oral cavity being widely opened for any extended period of time diminishes the only perfusion, maxillary blood flow, to the brain in the cat. Death, central nervous system, and/or neurological blindness may result.

Unfortunately retained tooth roots are a very common finding in veterinary medicine. Infected/inflamed retained tooth roots of the caudal maxillary dentition can result in regional inflammation. Likewise, hidden periodontal disease of the maxillary molars, particularly smaller breeds and brachycephalic dogs, can be a source of regional infection/inflammation.

Orbital Disease Differential Diagnosis
The clinician must have a complete differential diagnosis list and plan when evaluating patients in order to differentiate orbital disease from odontogenic or ophthalmological causes. Ophthalmological and non-odontogenic causes including, but not limited to, retrobulbar neoplasia or neoplasia invading from juxtaposed anatomy, scleritis, pseudotumors, extraocular myositis, foreign body abscess/cellulitis, trauma, zygomatic sialoadenitis, and coagulopathies are considered. An ocular and dental examination are necessary.
Maxillofacial Swellings and Draining Tracts
Odontogenic infection (e.g. periodontal disease, endodontic disease) in dogs and cats are chronic, insidious, yet very common. Most patients suffer silently with chronic infection, chronic pain, and chronic systemic inflammation until end-stages of the disease. Chronicity, immunosenescence, co-morbidity of systemic diseases (e.g., diabetes mellitus, hyperadrenocorticism) and administration of medications for other medical conditions (e.g., corticosteroids, cyclosporine, oclacitinib) may unmask the chronic infection and result an acute on chronic infection resulting in an emergency presentation.

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

Differentiation and Diagnosis
In many cases, periodontal probing and intraoral radiographs during anesthesia are necessary. Intraoral radiographs are also more detailed and useful even when the necessary computed tomography (CT) and/or magnetic resonance imaging (MRI) are performed. Viewing CT in a window not specific for the teeth and bone and/or having slices that are too large to identify very small hidden periodontal and endodontic changes will miss what can be picked up with a periodontal probing examination and intraoral radiographs of the distal maxillary dentition. MRI is useful for presumptive retrobulbar tumors, non-responding retrobulbar cellulitis, foreign bodies, and zygomatic salivary pathology. CT is more helpful with bone and teeth but a more accessible faster alternative with contrast enhancement to additionally evaluate for inflammation and tumors.

Urgent Medical Management
Having an accurate diagnosis is necessary for a targeted long-term plan. However, urgent treatment to protect the cornea with a temporary tarsorrhaphy and sterile ophthalmic lubricant may be necessary with exophthalmus. Antibiotic coverage for retrobulbar infection should include anaerobic bacteria arising from the environment or oral cavity. Hence, amoxicillin/clavulanic acid or clindamycin is often a first choice. In the case of retrobulbar cellulitis secondary to an oropharyngeal puncture or foreign body, it may be necessary for several weeks of antibiotic treatment and the addition of additional spectrums of activity in some cases. If no contraindication exists, the use of systemic non-steroidal anti-inflammatories is helpful for inflammation and pain control. Likewise, additional analgesia from opioids or gabapentin should be considered pending work up for a definitive diagnosis. Systemic corticosteroids should be avoided until a diagnosis is made as inappropriate use can lead to worsening of infection resulting in severe maxillofacial cellulitis and death.
**Clinical Signs of Pain and/or Difficulty Opening the Mouth**

**Differentials:**

1. Retrobulbar Abscess/Cellulitis (Odontogenic and Non-odontogenic)
2. Masticatory Muscle Myositis
3. Zygomatic Salivary Mucocele/sialadenitis
4. Neoplasia
5. Maxillary Fracture
6. Craniomandibular Osteopathy (e.g. Terrier Breeds)
7. TMJ Ankylosis (Previous Trauma)
8. Zygomatic Arch Fracture/Healing (Previous Trauma)
9. Tetanus (Clostridium tetani)
10. Osteoarthritis of TMJ (uncommon)

**Zygomatic mucocele/sialadenitis:**

Inflammation or trauma of the zygomatic salivary gland may lead to leakage of saliva resulting in pressure and inflammation in the retrobulbar tissues. Thick, tenacious saliva/blood tinged saliva may be identified if ruptured in the pterygopalatine fossa. MRI is helpful in challenging cases. Treatment may include medical (NSAIDS and appropriate antibiotics). In chronic cases, sialoadenectomy of the gland may be necessary. Sialoadenectomy of the zygomatic salivary gland is challenging due to the regional anatomy of the eye, zygomatic arch, and friable salivary gland tissue.

Retrobulbar Abscess/Cellulitis (Infection) vs. Masticatory Myositis (Autoimmune) – Conditions that Require Rapid and Accurate Differentiation

**Retrobulbar/Maxillofacial Abscess/Cellulitis:**

Infection and inflammation in the retrobulbar and caudal maxillofacial region are common. Puncture wounds from sticks, plant material, and oral foreign bodies traumatize and inoculate the soft tissues of the caudal oropharyngeal region. Regional hidden periodontal and endodontic disease (odontogenic infection) can lead to inflammation and infection in the retrobulbar space due to the close proximity (millimeters) and incomplete ventral orbit of dogs and cats. Ubiquitous environmental bacteria such as Actinomyces and Nocardia cause chronic pyogranulomatous inflammation resulting in swellings and draining tracts of the head and neck.

The clinical signs of acute injury are often missed (e.g., pain and ptyalism). Chronically, pain on opening the mouth and resistance to opening during the examination are common. Retropulsion of the ipsilateral eye is painful and an intraoral swelling or region of trauma may be identified in the pterygopalatine fossa. The ipsilateral eye may have chemosis and prolapse of the third eyelid. In severe cases, maxillofacial swelling and exophthalmus on the ipsilateral side occurs.

Diagnosis includes intraoral dental radiographs and advanced imaging as indicated. Exploration of the pterygopalatine fossa and caudal oropharynx may be both diagnostic and therapeutic. However, the tissue must be carefully and minimally explored to look for regions of abscess, sialocele, and cellulitis if indicated. However, blind stab incisions are NOT recommended as the vital maxillary
artery and its branches, maxillary and optic nerves, and the eye are in close proximity (millimeters). Instead, a superficial mucosal incision can be made, if chosen, and the area bluntly (only once or twice) evaluated with blunt hemostatic forceps. Advanced imaging (e.g. CT, MRI) are very helpful to quickly rule out differential diagnosis. Intraoral radiographs are necessary to assess the maxillary dentition. Close clinical examination of the dentition is necessary to find hidden endodontic or periodontal disease.

Treatment involves surgical extraction of the offending tooth, if it is related to an odontogenic infection (intraoral radiographs are required to make the diagnosis). If it is not odontogenic infection or a zygomatic mucocele, then the patient is treated with antibiotics that cover the aforementioned pathogens, NSAIDS, and additional analgesics as indicated. The owners are advised if the condition resolves, then reoccurs following cessation of medical therapy, that investigation for a hidden plant/wood foreign body may be necessary (ocular/periocular ultrasound) and/or prolonged treatment (4-6 weeks) for Actinomyces and Nocardia may be necessary. If imaging has ruled out differential diagnosis and pathology, in some cases, treatment with amoxicillin/clavulanic acid and a fluoroquinolone antibiotic for 4-6 weeks is necessary.

**Masticatory Myositis (MM):**

In the acute presentation, the patient and the clinician cannot open the mouth. Even with heavy sedation and general anesthesia the mouth will not open due to severe muscle inflammation in the acute phase. The masticatory muscles (masseter, pterygoids, and temporalis muscles) are swollen and firm. It may be unilateral or bilateral. In the chronic presentation, the muscles are severely atrophied and fibrosed making opening the mouth problematic. An autoimmune attack of the type-2M muscle fibers results in inflammation, destruction, and replacement with fibrous tissue. The diagnosis is confirmed by ruling out other differentials (advanced imaging), type-2M antibody titer, and masticatory muscle biopsy while anesthetized. Presumptive treatment may start following collection of blood for type-2M antibody (corticosteroid treatment prior to collection may result in false negatives). If aggressive corticosteroid therapy results in severe drug related side effects, additional immunotherapy with azathioprine, etc. may be added to allow decreasing the corticosteroid dose.

**Clinical note:**

DO NOT place a patient on immunosuppressive corticosteroids for a presumptive diagnosis of masticatory myositis until retrobulbar/maxillofacial abscess/cellulitis is ruled out. Immunosuppression of an infection can lead to severe regional and systemic infection and death in many patients due to a misdiagnosis or inappropriate presumptive diagnosis. Remember many patients with retrobulbar cellulitis/abscess will resist opening the mouth during a conscious examination due to pain. Therefore, placing an intravenous catheter and administering multimodal analgesia and/or anesthesia will allow you to assess the range of motion of the TMJ as well as evaluate the pterygopalatine fossa for evidence of trauma and swelling – the mouth can be opened; in these cases, treat with antibiotics, anti-inflammatory NSAIDS, if not contraindicated, and other analgesics. In an acute masticatory myositis case, the mouth cannot be opened, in the anesthetized patient, due to the extreme inflammation of the
muscles. In these cases, draw blood for a Type 2M antibody titer and obtain a muscle biopsy. Then administer immunosuppressive medications.
Oral Masses in the Dog and Cat: The Eye is not a Microscope
Kevin S. Stepaniuk, B.Sc., DVM, FAVD

Introduction
Neoplasia of the oral cavity represents about 6-7% and 3% of all canine and feline tumors, respectively. Biopsy is required for diagnosis. The biopsy may be incisional or excisional depending on the size, differential diagnosis, and long-term surgical and medical plan. An accurate histological diagnosis is necessary for a treatment plan and prognosis. If the clinical presentation does not correlate with the histological diagnosis, question the pathologist and, if necessary, get a second opinion. A coordinated effort with a veterinary dentist/oral surgeon and an oncologist will provide current treatment options. Management of maxillofacial tumors and cysts requires understanding of tumor biology, diagnosis, and treatment plans.

Embryology
Oral tumors can be separated into odontogenic tumors (historical “epulides”) and non-odontogenic tumors based on the tissues of origin. A review of embryology and the cells responsible for the development of odontogenic tumors is necessary. Odontogenesis is the embryological events in tooth development. Enamel is produced from the enamel organ of ectodermal origin whereas the dentin and pulp is derived from the dental papilla from mesodermal origin. The enamel organ develops through a bud, cap, and bell stage. The mesenchymal cells form a dental sac and papilla adjacent to the enamel epithelium. The dental sac gives rise to the cementum, periodontal ligament, and some alveolar bone. The tooth root forms when the crown has developed but has not completely mineralized. The outer enamel epithelium and inner enamel epithelium form Hertwig’s epithelial root sheath that grows into the underlying connective tissue to form the root. The root sheath will turn back on itself (epithelial diaphragm) and meet to form apices and the furcations of teeth. The dental papilla is stimulated to form odontoblasts, which produce dentin. At this stage, the root sheath breaks up and cementoblasts are formed. Some root sheath cells remain trapped in the periodontal ligament and are known as the epithelial rests of Malassez. Remnants of the dental lamina are known of the epithelial rests of Serres. It is believed odontogenic tumors rise from remnants of odontogenic tissues. The molars arise directly from the dental lamina.

Odontogenic Tumors
By definition, an epulis is any gingival growth. Hence, gingival hyperplasia or any malignant tumor is also, by definition, an epulis. However, historically “epulides” were referred to as benign locally invasive tumors. Epulides compromise 20-30% of oral tumors. Canine acanthomatous ameloblastoma (epithelial origin), peripheral odontogenic fibroma (mesenchymal origin), odontomas, feline inductive odontogenic tumors, amyloid producing odontogenic tumors, cementomas, and giant cell tumors (uncommon) are types of odontogenic tumors. Surgical resection is the treatment. Dentigerous cysts are classified as a type of odontogenic tumor related to unerupted teeth.

Odontogenic Tumors in Cats
By definition, an epulis is any gingival growth. Hence, gingival hyperplasia or any malignant tumor is also, by definition, an epulis. However, historically “epulides” were referred to as benign locally invasive tumors. Odontogenic tumors are less common in cats (2.5% of all oral tumors) compared to dogs. However, odontomas, feline “multiple epulides”, feline inductive odontogenic tumors (FIOT),
Amyloid producing odontogenic tumors (APOT), cementomas, and giant cell tumors (uncommon) are types of odontogenic tumors. Surgical resection is the treatment.

Peripheral Odontogenic Fibromas can occur but are uncommon in cats. Complete remove can be curative.

Feline inductive odontogenic tumors are rare. They are found in the rostral maxilla of young cats. They are locally invasive and early recognition and maxillectomy can be curative. The odontogenic epithelium is organized around a dental pulp-like stroma.

Amyloid producing odontogenic tumors are rare, non-infiltrative, non-metastatic and locally expansive, destroying surrounding structures. Surgery can be curative.

**Odontogenic Tumors in Dogs**

Peripheral canine acanthomatous ameloblastoma (CAA) (a.k.a., acanthomatous epulis) may arise from the epithelial rests of Malessez, adjacent bone, and/or the periodontal ligament (PDL); have no hard dental tissue structures, aggressively invade bone, are benign, have a predilection for the mandibular incisor and premolar regions, and do not metastasize. They rarely transform to squamous cell carcinoma. Radiation can control 90% of ameloblastoma but 5% develop radiation necrosis and 5-20% will undergo malignant transformation at a later date. Therefore, surgical resection is often chosen unless the patient requires the dentition to assist the owner or to perform its duties. Surgical margins should be minimally 1.0-2.0 cm. This tumor is “curable” with resection so 1.0 - 2.0 cm margins are ideal whenever it is functionally possible. However, as a veterinary dentist/oral surgeon, we will utilize symphyseal sparing mandibulectomy and marginal rim excision techniques to preserve the function of the mandibular canine tooth /teeth mandibular symphysis, and ventral mandibular cortex, if possible.

Peripheral odontogenic fibromas (POF) (previously known as fibromatous and ossifying epulides) are benign tumors often found in the premolar and incisor regions. Surgical resection is curative but it requires extraction of the involved teeth and complete excision of the periodontal ligament tissues.

Focal fibrous hyperplasia (a.k.a., fibrous epulis) should not be confused with a peripheral odontogenic fibroma (a.k.a., fibromatous epulis) as the focal fibrous hyperplasia is a “tumorous growth” that is reactive tissue.

Odontomas are of both epithelial and mesenchymal origin and are found in young animals. A complex odontoma has an amorphous mass of poorly differentiated mineralized material, and is rare. A compound odontoma has denticles (tooth like structures) in an unorganized, random, structure throughout the tumor. Odontomas are locally destructive and early recognition and excision is curative. Preservation of the cortical bone will allow functionality of the maxillofacial skeleton long term.

Unerupted teeth (embedded or impacted) can lead to dentigerous cyst formation and destruction of the bone and adjacent teeth. This condition is preventable so all regions of missing teeth should be evaluated with intraoral radiographs. Unerupted teeth should be extracted. Dentigerous cysts need to be surgically debrided and the cystic lining removed with the offending tooth.
Canine Non-Odontogenic Mandibular/Maxillary Tumors
The most common canine oral tumors are: malignant melanoma (30-40%), squamous cell carcinoma (20-30%), fibrosarcoma (10-20% and male dogs predisposed), osteosarcoma (<10%), and others (<5%). The most common feline tumors are squamous cell carcinoma (70-80%) and fibrosarcoma (13-17%). Other tumors have been reported but are rare. The majority of feline and canine oral tumors occur in middle aged and older patients. A complete pathobiology of oral tumors can be found elsewhere.

Canine oral malignant melanoma (MM) tends to occur in older patients and a male predisposition has been suggested. It is the most common oral tumor reported in the canine patient. Breeds with increased oral pigment are overrepresented. Amelanotic melanomas (1/3rd of the melanoma cases) can make the histologically more challenging to diagnose; special staining may be utilized. In general, MM is highly metastatic and is presumed at the time of diagnosis. Tumors with dimensions < 2.0 cm tend to have a better prognosis. However, some well-differentiated oral melanomas with low mitotic indices have a lower metastatic rate may and may be treated with complete surgical resection alone according to some publications. However, assessment by an oncologist is recommended to discuss immunotherapy, radiation therapy, and/or chemotherapy.

Complete surgical resection and adjunctive radiation therapy, immunotherapy, or chemotherapy is often recommended. A melanoma vaccine (xenogenic human tyrosinase plasmid DNA) is available for adjunctive treatment (immunotherapy) following removal of macroscopic disease in order to treat presumed microscopic metastasis. The targeted antigen is a tyrosinase glycoprotein involved in melanin production on melanocytes.

Canine oral squamous cell carcinoma (SCC) is the second most common oral tumor in the dog. It is common in older patients and tends to metastasize late in the course of disease. Therefore, complete surgical resection can have an excellent prognosis if identified early. It is reported tonsillar and lingual SCC have a higher metastatic rate compared to more rostral tumors. Possibly, this may not be true since more rostral tumors are recognized earlier. If early identification and treatment of more caudal tumors can occur, potentially a better prognosis can be achieved. A reason for the recommended annual dental cleaning and ORAL EXAM, in our patients, is to detect oral masses earlier in the disease course.

Canine oral fibrosarcoma is the third most common tumor in the dog. It tends to occur in large breed dogs and male predisposition has been suggested. They also occur in younger dogs. Metastasis is uncommon (<20% to the pulmonary parenchyma). These tumors can be reported as histologically low grade but biologically high grade. They may have a histologically appearing low-grade tumor but the tumor is locally invasive and needs aggressive surgical margins.

Oral osteosarcoma is uncommon but is reported to have better prognosis than appendicular osteosarcoma with complete excision.

Extramedullary Plasmacytoma occurs and can have a very good prognosis with complete surgical resection. They may present as flat mucosal/gingival lesions or may be growing masses.
mastocytoma, hemangiosarcoma, neurofibrosarcoma, lymphoma, osteomas, multilobular osteochondrosarcoma, etc. can occur.

**Feline Non-Odontogenic Mandibular/Maxillary Tumors**
The most common malignant feline oral tumor is squamous cell carcinoma. It is estimated to represent up to 70% of all oral tumors in cats.

Feline oral squamous cell carcinoma (SCC) is the most common tumor in the feline oral cavity. Risk factors include smokers in the house, flea collars, and canned tuna/food. The most common site is the sublingual region. Mucosal ulcerations and secondary inflammation are often associated with SCC. A non-healing extraction site should have a high index of suspicion for SCC in an older cat and be biopsied accordingly. The ventral tissues of the tongue are often involved followed by the mandibles and maxilla. The tumor is invasive and local disease is often the cause of death. Surgery and adjunctive radiation therapy is the best treatment option at this time. The median survival time is 30-45 days without treatment.

Feline oral fibrosarcoma is the second most common malignant tumor in the cat. Wider surgical margins are necessary and is often limited by late diagnosis. Radiation treatment is an option for palliative control. Consultation with an oncologist is recommended. Surgical resection can have reasonable long-term prognosis with a combined surgical and oncological plan.

Oral osteosarcoma is less common but has a better prognosis with complete excision. Surgical resection can have reasonable long-term prognosis with a combined surgical and oncological plan.

Lymphoma, osteomas, multilobular osteochondrosarcoma, etc. can occur.

**Lingual Tumors**
Approximately 4% of all tumors are tongue tumors. Squamous cell carcinoma is the most common (50% of tongue tumors). Other tumors include plasmacytoma, myoblastomas, malignant melanoma, mastocytoma, leiomyosarcoma, hemangiosarcoma, hemangioma, rhabdomyosarcoma, etc. The rostral tongue tumors are generally recognized earlier and are in a more favorable location for resection. Dogs can function with large portions (entire body and leaving root) of the tongue removed. Temporary feeding tubes may be necessary while the patient learns to prehend food. Cats do not function as well and will need assistance with grooming and feeding tubes. Prognosis varies on the type and location of tumor.

**Other Oral Masses**
Any oral mass should be biopsied. In addition to the aforementioned odontogenic and non-odontogenic tumors masses include, but are not limited to, gingival hyperplasia, pyogenic granuloma, eosinophilic granuloma, fungal lesions, Cryptococcus, papillomatosis, etc.

**Oral Tumor Clinical Signs**
Tumors often present with halitosis, ptyalism, oral hemorrhage, maxillofacial disfigurement (if large), and occasionally dysphagia and weight loss; or nothing at all and are found during an annual
periodontal cleaning and oral exam. Hence, detecting them early during annual oral examinations prior to clinical signs gives the patient a better chance a good long term prognosis. Oral tumors may have associated regional lymphadenopathy that represents reactive lymph nodes or metastasis.

**Staging**

As with all tumors, staging is important. A biopsy (incisional or excisional) of the oral mass is necessary, complete CBC/Chemistry Panel/UA/FELV/FIV, as well as 3-view thoracic radiographs or thoracic CT and regional lymphocentrum evaluation is recommended.

When obtaining the incisional biopsy with many oral tumors the biopsy should be obtained from within the clinical margins of the tumor to prevent disruption and violation of clinically normal appearing tissue. There are tumor cells surrounded by a pseudocapsule and reactive zone. Larger tumors may require multiple samples. Be aware that the center of the tumor may be necrotic and the surface of the tissue may be covered with hyperplastic gingiva, so be certain to obtain a deep and representative sample. Disposable punch biopsies are used for most tumors where as a Yamshidi needle be necessary for hard bone tumors. Bone curettes can be used to remove deeper bone through the same site the punch biopsy was utilized and is preferred in my opinion.

It is known that fine needle aspirates and cytology can yield excellent staging results from lymph nodes (dogs). However, the maxillary lymphatics drain primarily to the parotid and medial retropharyngeal lymphocentrum and metastasis may be present WITHOUT any cancer found in the mandibular lymph nodes (the nodes accessible for aspiration). In fact, only 54.5% of cases with metastatic disease to regional lymphocentrum had metastasis including the mandibular lymphocentrum (dogs). Therefore, histological assessment of all regional lymphocentrum should be considered. A single incision can be performed to quickly harvest all regional lymph nodes (parotid, medial retropharyngeal, and mandibular) with minimal to no complications. Alternatively, techniques for ultrasound-guided aspirates of the median retropharyngeal lymph nodes are being utilized. However, the reality in practice is most veterinarians are limited to aspiration of the mandibular lymph nodes only, including specialists.

Intraoral dental radiographs are imperative but advanced imaging (CT and MRI) can prove highly beneficial for surgical planning and CT is necessary for surgical planning in most cases. Advanced imaging can be of great benefit when it comes to achieving clean surgical margins, particularly in the maxilla and caudal mandible. Thereby, extending median survival times and diminishing local recurrence rates. Many of the published studies today lack advanced imaging prior to surgery and therefore, in my opinion, reported local recurrence rates may be much lower if true preoperative surgical margins could be better planned. Advanced imaging is the gold standard prior to surgery. Clinical staging is beneficial for selection of treatment and prognosis.
Prognosis

Prognosis is always changing and being updated. Many textbooks may be outdated but the only source for published information to fairly provide prognosis for owners. Consulting an oncologist following diagnosis and/or resection is indicated.
Feline Oral Inflammation and Treatment Updates
Kevin S. Stepaniuk, B.Sc., DVM, FAVD

Introduction
Until recently there has been no consistent description of inflammatory lesions and patterns in the oral cavity of cats. The historical veterinary literature, textbooks, and lecturers have not used consistent descriptions of oral inflammatory patterns in the oral cavity. The generic description of “stomatitis” often leads to misdiagnosis, prognosis, and treatment planning. Stomatitis is inflammation of the entire mouth and may arise from many etiologies. See www.avdc.org nomenclature for a description of different regions of inflammation in the oral cavity. The following definitions for oropharyngeal inflammation are approved by the American Veterinary Dental College (AVDC) and used with permission from the www.avdc.org website.

Gingivitis: Inflammation of the gingiva
Periodontitis: Inflammation of the non-gingival periodontal tissues (e.g., periodontal ligament and alveolar bone)
Alveolar mucositis: Inflammation of alveolar mucositis: inflammation of alveolar mucosa (i.e., mucosa overlying the alveolar process and extending from the mucogingival junction without obvious demarcation to the vestibular sulcus and to the floor of the mouth)
Sublingual mucositis: Inflammation of mucosa on the floor of the mouth
Labial/buccal mucositis: Inflammation of the lip/cheek mucosa
Caudal mucositis: inflammation of the mucosa of the caudal oral cavity, bordered medially by the palatoglossal folds and fauces, dorsally by the hard and soft palate, and rostrally by alveolar and buccal mucosa
Contact mucositis and contact mucosal ulceration: lesions in susceptible individuals that are secondary to mucosal contact with a tooth surface bearing the responsible irritant, allergen, or antigen. They have been called “contact ulcers” and “kissing ulcers”.
Palatitis: inflammation of mucosa covering the hard and/or soft palate
Glossitis: inflammation of mucosa of the dorsal and/or ventral tongue surface
Cheilitis: inflammation of the lip (including the mucocutaneous junction area and skin of the lip)
Osteomyelitis: inflammation of the bone and bone marrow
Stomatitis: inflammation of the mucous lining of any of the structures in the mouth; in clinical use the term should be reserved to describe wide-spread oral inflammation (beyond gingivitis and periodontitis) that may also extend in submucosal tissues (e.g., marked caudal mucositis extending into submucosal tissues may be termed caudal mucositis)
Tonsillitis: inflammation of the palatine tonsil
Pharyngitis: inflammation of the pharynx

Feline caudal mucositis (a.k.a. “stomatitis”) is a frustrating oral condition for clients and veterinarians. The pathogenesis of the disease is not fully understood but it appears that an immunological mechanism is involved. However, this true immunological “stomatitis” needs to be differentiated from common other conditions such as periodontitis, aggressive periodontitis, pyogenic granuloma and diseases such as epitheliotropic lymphoma, autoimmune conditions, eosinophilic granulomas, etc.
Particularly, aggressive periodontitis and adult onset periodontitis secondary to a plaque and bacterial biofilm and the inflammation, may be mistaken and mistreated as “stomatitis”. It is important to recognize that “stomatitis” involves extension of the inflammation beyond the gingiva and mucogingival junction and involves the caudal pharynx, palatoglossal folds, the palatal mucosa, and the buccal mucosa. In some cases, it just involves the caudal oral mucosa (caudal mucositis). Caudal mucositis is a defining clinical pattern of inflammation for the consideration of “stomatitis”. Severe periodontitis, juvenile gingivitis, juvenile onset periodontitis, and aggressive periodontitis often only involve the gingiva (gingivitis) with or without extension past the mucogingival junction into the mucosa (buccal mucositis). Biopsy of the gingiva in these conditions results in a similar histopathological diagnosis of a cat with immune dysregulated stomatitis. Differentiation of severe periodontitis, juvenile gingivitis, juvenile onset periodontitis, and aggressive periodontitis early in the clinical presentation is necessary for appropriate treatment planning.

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

As the plaque biofilm matures, early bacterial colonizers, gram-positive aerobic cocci, become less predominant. The biofilm shifts to gram-negative anaerobes and spirochetes located more apical in the periodontal pockets. Bacterial products such as ammonia, volatile sulfur compounds, and proteolytic enzymes contribute to the destruction of the periodontium. The host inflammatory response, matrix metalloproteinases that degrade collagen of the periodontal ligament, elastase (break down collagen and elastin), and prostaglandins (PGE2) are directly responsible for tissue damage and/or stimulate osteoclastic bone resorption (PGE2, IL-1β, TNF-α). The calcium carbonate in the saliva of cats combines with the plaque to form calculus. Calculus increases surface area for bacterial attachment and can mechanically disrupt and damage the gingiva.

Variations of Periodontitis
Adult onset periodontitis is a chronic common condition affecting the entire population. There is no specific breed predilection of this chronic disease that is managed with annual periodontal cleanings and home care. However, if left untreated it will lead to significant inflammation in the oral cavity and tooth loss. Juvenile onset and Aggressive periodontitis may occur more commonly in the domestic short hair, Siamese breeds, and Maine Coon breeds. Significant inflammation begins as early 6-9 months of age with abundant plaque and calculus present. This aggressive inflammation leads to gingival recession, bone loss, pocket formation, furcation exposures and rapid tooth loss. Often these cats require full mouth extractions at a young age but can have a very good long-term prognosis with full mouth extractions. Feline juvenile gingivitis reported in Abyssinians, Persians and DSH, begins around 5 to 6 months of age with hyperemic PROLIFERATIVE gingivitis. There is little plaque and calculus present. The proliferative gingiva engulfs the crowns and results in pseudopockets and the start of periodontitis.
Management of Periodontitis
Management of periodontal disease is not a once in a lifetime event for the patient but rather an ongoing program throughout continued life stages of the patient. Gingivitis is the start of the inflammatory cascade and may progress to periodontitis and loss of periodontal attachment. The goal with periodontitis is to stop the disease, minimize further attachment loss, and treat compromised teeth (e.g., periodontal surgery, guided tissue regeneration, and extraction as indicated). Therefore, education and prevention of disease (daily brushing, dentifrices, and frequent professional dental care) are the best defenses.

A professional dental cleaning, to return the tooth to a clean surface, followed by daily home care, to remove the plaque biofilm, is the gold standard to prevent and control periodontal disease. If pockets are eliminated and the plaque biofilm is removed on a daily basis, then the maturation of plaque and further pocket formation can be controlled and minimized. Expired dentition will require surgical extraction.

Feline Pyogenic Granuloma
A region in the caudal mandible that may become inflamed and misdiagnosed, particularly in brachycephalic cats, as well as following surgical extraction of the first mandibular molar and/or secondary to periodontal disease of the first mandibular molar, is a feline pyogenic granuloma. Unless the underlying cause is identified and treated, the clinician can be frustrated, the lesions will re-occur after resection, and the cat will suffer with chronic pain. They often start buccal to the mandibular 1st molar as a result from the occluding maxillary 4th premolar, periodontitis, and the perpetuated traumatic inflammatory cycle. It may continue following extraction of the 1st molar if the surgical site is not sutured closed correctly and atraumatically and/or the maxillary 4th premolar is not treated with odontoplasty and sealant or alternatively surgical extraction in some cases. Congenital or acquired malocclusions may also lead to the development of these lesions. Finally, inappropriate surgical closure and inappropriate transposition of the molar salivary gland into a traumatic occlusion during extraction of the 1st molar, in any cat, may cause these lesions to develop.

Caudal Mucositis
Feline “stomatitis” (gingivitis with buccal mucositis and caudal oral mucositis) is an immune dysregulation of the feline’s local and systemic immune system. The initial trigger of the dysregulation is unknown. There are continued theories, debate, confounding factors, and additional disease entities concurrent with the inflammation, and a lack of standard diagnosis and description of oral inflammatory lesions. A prevalence study concluded approximately 0.7% of cats in British general practices have “gingivostomatitis”.

Pathogenesis
The common dermal-epidermal inflammatory reaction of predominately plasma cells and lymphocytes is suggestive of an immunoreactive condition. The retrovirus status (FeLV and FIV) should be determined in all patients from an overall health management standpoint. However, these immunosuppressive viruses are not the etiology of the immune dysregulation stomatitis. Immunosuppressive conditions can increase periodontitis and secondary oral inflammation due to
lack of control of the plaque biofilm. Once again periodontitis and the associated gingival and mucosal inflammation must be differentiated from a “true stomatitis”.

With a dysregulated immune stomatitis there are changes in cellular and humoral immunity. T-lymphocyte subset ratios are low (CD4+/CD8+) due to high numbers of CD8+ (Suppressor-cytotoxic T-Cells). CD4+ (TH-1 and TH-2 – helper induced T-cells) recognize MHC II on surface antigen presenting cells, other T-cells, B-cells, some mesenchymal cells, and osteoblasts where as CD8+ cells recognize MHC I universally exhibited on all cell types. The levels of salivary IgA are low and IgG and IgM are high in stomatitis patients. It is not known if the low IgA is from the disease, prior to the disease, the result of decreased immunoglobulin secretion, changes in salivary flow, or and/or increased destruction. Additionally, and not surprisingly, there are increased mast cells in the tissues compared to specific pathogen-free cats. Mast cells are also increased in inflamed gingiva secondary to periodontal disease and tooth resorption.

What is the inciting cause of the dysregulated immune system? Bacteria, oral food antigens, environmental antigens, and viruses have all been suggested as inciting causes. Bacterial persistence (plaque) is the most likely inciting cause at this time. It is known that many cats with stomatitis test positive for feline calicivirus and may have increased concurrent shedding of feline calicivirus (recognize this may not indicate active infection but just presence of biological makers such as DNA and RNA). However, other studies have found no association with feline herpes virus, feline calicivirus, and Bartonella species. Do the viruses and/or bacteria disrupt the oral epithelium and subsequently deeper tissues are exposed to oral bacterial, food, and environmental antigens resulting in a dysregulated immune response? Or, are the viruses’ opportunistic colonizers of inflamed tissue and an abnormal functioning immune response? Currently, some Diplomates of the AVDC accept that the shedding of viruses may contribute to the overall clinical morbidity of the patient. To my knowledge, no control patients have been experimentally infected with the feline calicivirus resulting in a stomatitis (Koch’s Postulates). There may be a defect in the patient’s cell mediated immunity resulting in presentation of auto-antigens to infiltrating CD4+ cells and/or damage to mucosa allowing presentation of auto-antigens and subsequent auto-antibody production. Bacteria associated with the plaque biofilm may be the major triggering factor.

It is worth mentioning Bartonella henselae due to its zoonotic significance and the suggestion of its link to “gingivostomatitis”. Positive serological testing for the organism suggests exposure and not active infection. Many normal healthy cats test positive serologically. In cats with “gingivostomatitis” that are positive for Bartonella, appropriate treatment for elimination of the organism does not often result in resolution of the “gingivostomatitis”. Current studies are unable to associate feline herpes virus 1, FeLV, FIV, and Bartonella with chronic “gingivostomatitis” in the cat. Calicivirus was not associated in that study but other studies suggest a comorbidity.

At this time, the elimination of bacteria/plaque from the teeth, sulci, and periodontal ligament space, via surgical extractions, is the treatment that results in the best resolution of clinical signs.

**Diagnosis and Treatment**

Each patient has to be assessed and treated individually. A comprehensive evaluation is necessary. A complete history of signalment, onset, duration, environment, chronic illness, systemic illness,
vaccination, etc. must be obtained. Diagnostic testing includes CBC/Chemistry/UA, T4, FeLV, FIV, +/- Bartonella testing, and +/- calicivirus for many patients. Not every patient is recommended every test initially but each test should be considered on a case-by-case management basis. Although in current practice, Bartonella and calicivirus testing are rarely done, if at all.

Definitive treatment recommendations may differ depending on the consulting specialist and specialty. In many boarded veterinary dentists’ opinion, at this time, full mouth extractions provide the best long-term resolution. However, not every specialist may recommend the same treatment plan for each case. In some cases, every tooth will be extracted whereas in others, every tooth except the canine teeth will be extracted. Intraoral radiographs and clinical disease help determine the plan in each case. Ultimately, most cats with “stomatitis” end up with all the dentition completely extracted and many veterinary dentists will extract all the dentition in one surgical procedure.

Professional periodontal cleanings and home care cannot control the clinical inflammation and pain. Anecdotally, many patients who present refractory to chronic immunosuppression (long-term steroid use) also may have concurrent endocrine disease (diabetes mellitus) and seem to have cross-reacted to additional dietary, environmental, and auto-antigens resulting in poorer surgical outcomes to full mouth extractions. However, a complete assessment and individual treatment plan must be made with each client and patient in order to meet the needs of the patient, the client, and to achieve the best possible outcome for each situation. Not every client can afford or can grasp the concept of removing all the teeth with oral surgery, initially. However long term medical therapy is expensive and may be more expensive, over time, with office visits, blood monitoring, and medications compared to surgery.

Referral for full mouth extractions to a veterinary dentist should be considered due the time it takes to remove all of the teeth and roots if your hospital is not equipped with multimodal analgesia/anesthesia, intraoral radiographs and can complete the procedure in a timely fashion to minimize the duration of general anesthesia. Clients are always advised that the full mouth extractions may not completely resolve all the inflammation and adjunctive therapy may be necessary. Intraoral radiographs and aggressive multimodal pain control are necessary.

Published results from exodontics are favorable:

- 60% complete remission
- 20% no medical treatment necessary other than plaque control
- 13% required continued medical management
- 7% no response

If continued medical management is necessary, fatty acids, appropriate lowest dose possible prednisolone treatment, and possibly cyclosporine, with appropriate clinical, blood, and drug monitoring, has been effective in some cases. After removal of all the teeth, corticosteroids may still be necessary in some cases. However, the dosage and frequency, along with their side effects, is greatly reduced. Veterinary dentists in Europe, and some veterinary dentists with special FDA import permits in the USA and Canada, investigated/investigating the use of Omega-Interferon, which is not currently available in North America with questionable results. Laser ablation therapy to remove
chronically inflamed tissue and create scar formation (hence less blood supply and potential immunological response) with carbon dioxide and Nd:YAG lasers have seen some success. Novel antigen diets and Omega-3 fatty acids may be of benefit in some refractory cases. Certain antimicrobial combinations and/or additional immunosuppressive therapy may be necessary post-extraction for the partial and non-responders. However, chronic long-term therapy with antibiotics is never indicated as a monotherapy for this condition or any condition of the oral cavity.

Currently, UC Davis stem cell treatment holds the most promising future for non-responding cats. However, it is VERY IMPORTANT to understand that the cultured stem cell treatment takes 10-14 days to harvest the appropriate stem cells. This is NOT the weekend stem cell course, teaching harvesting of the stromal fraction in several days, that some “stem cell” veterinarians offer.
Recognizing the Clinical Significance of Malocclusions and Developmental Tooth Defects in the Dog

Kevin S. Stepaniuk, B.Sc., DVM, FAVD

Introduction
The branch of dentistry involved with immature dental structures, immature maxillofacial skeleton, and the eruption of teeth in immature patients is pediatric dentistry. Pediatric dentistry includes malocclusions, tooth developmental abnormalities, maxillofacial developmental anomalies, and pediatric dental and maxillofacial trauma. Familiarity with tooth eruption patterns, tooth numbers, dental anatomy, normal occlusion, and odontogenesis is necessary to identify, intervene, and treat before more serious and permanent dental problems develop. An oral examination should be included with every puppy and kitten visit until adult dentition is identified and the maxillofacial skeleton has completed growth. Early recognition of hidden pediatric and juvenile dental pathology is often overlooked in the busy and informational loaded puppy and kitten examinations. A good opportunity to evaluate the oral cavity, count the dentition, obtain intraoral radiographs of any missing or extra dentition is during the ovariohysterectomy or orchidectomy in the juvenile patient.

Odontogenesis and Maxillofacial Development
The jaws develop independently from the branchial arches. The mandible and maxilla grow in “spurts”. The maxillary and mandibular growth is not continuous or synchronous. Since the mandible is isognathic to the maxilla, the asynchronous growth can lead to abnormal positions of deciduous teeth and dental interlock. An adverse dental interlock can lead to deviation of the jaws and a permanent skeletal malocclusion. The normal occlusion and interdigitation of the deciduous dentition normally allows the mandible and maxilla to maintain a relatively proportionate relationship during growth.

Odontogenesis is the embryological events during tooth development. Enamel is produced from the enamel organ (neuroectoderm origin). Dentin and pulp are derived from the dental papilla (mesoderm origin). The enamel organ develops through a bud, cap, and bell stage. The bud arises from the dental lamina in the regions corresponding to the deciduous dentition. As a concavity develops, the cap stage is present. At this time there is an outer enamel epithelium (OEE), inner enamel epithelium (IEE), and the stellate reticulum. The bell stage begins when the stratum intermedium emerges between the IEE and stellate reticulum. For incisors, canines, and premolars, lingual extensions from the primary dentition form the successional lamina. Therefore, if a deciduous tooth does not form, its adult counterpart will not form. The buds for the molars and the 1st premolars in the dog develop directly from the dental lamina during the same period the deciduous tooth buds are formed.

Tooth eruption begins when the crown enters the oral cavity and ends when the tooth is exfoliated or the patient dies. Deciduous tooth exfoliation is not fully understood. It is believed that the adult tooth bud crown applies pressure to the root of the primary tooth causing resorption and/or the dental follicle of the erupting tooth signals resorption and eventual exfoliation of the deciduous tooth.
**Dental Formula**

Dog:
- Deciduous: $2 \times (3I/3I, 1C/1C, 3PM/3PM) = 28$
- Adult: $2 \times (3I/I, 1C/1C, 4PM/4PM, 2M/3M) = 42$

Cat:
- Deciduous: $2 \times (3I/3I, 1C/1C, 3PM/2PM) = 26$
- Adult: $2 \times (3I/I, 1C/1C, 3PM/2PM, 1M/1M) = 30$

**Approximate tooth eruption times (breed differences):**

<table>
<thead>
<tr>
<th></th>
<th>Deciduous (Weeks)</th>
<th>Adult (Months)</th>
<th>Deciduous (Weeks)</th>
<th>Adult (Months)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Puppy</td>
<td>Adult Dog</td>
<td>Kitten</td>
<td>Adult Cat</td>
</tr>
<tr>
<td>Incisors</td>
<td>3-4</td>
<td>3-5</td>
<td>2-3</td>
<td>3-4</td>
</tr>
<tr>
<td>Canines</td>
<td>3</td>
<td>4-6</td>
<td>3-4</td>
<td>4-5</td>
</tr>
<tr>
<td>Premolars</td>
<td>4-12</td>
<td>4-6</td>
<td>3-6</td>
<td>4-6</td>
</tr>
<tr>
<td>Molars</td>
<td></td>
<td>5-7</td>
<td></td>
<td>4-5</td>
</tr>
</tbody>
</table>

**Delayed Eruption**

Delayed eruption of adult teeth must be recognized early. Intraoral radiographs are required to assess 1) is the adult tooth present? and 2) is the adult tooth unerupted (impacted or embedded)? During the growth phase of tooth eruption, impediments to eruption [extraction of persistent deciduous teeth, removal of bone via osteoplasty, and removal of fibrous gingival tissue (operculectomy)], as indicated, can open the eruption path and allow teeth to move into occlusion. However, recognition of missing and unerupted teeth, well past any eruption window, may be too late and the adult teeth may remain unerupted and result in dentigerous cyst formation.

**Deciduous Dentition**

There are 28 deciduous and 42 adult teeth in the dog. The cat has 26 deciduous and 30 adult teeth. No two teeth of the same type should be in the same place at the same time. That is, a persistent (retained) deciduous canine tooth should be immediately removed if the corresponding adult canine tooth is present, or in an eruptive process, and the deciduous canine tooth is not mobile and exfoliating.

There are no deciduous molar teeth. However, deciduous 3rd premolars look similar too, and function like, adult 4th premolars and deciduous 4th premolars look similar too, and function like, adult 4th premolars.

Persistent (retained) deciduous teeth cause abnormal eruption and position of adult dentition with resulting malocclusions and potentiate periodontal disease. No two teeth of the same type should be in the same place at the same time. Extraction of persistent teeth should occur when recognized.

**Non-Development of Deciduous Teeth**
If deciduous teeth are missing, then the adult counterpart will also be missing. The adult tooth bud develops from the deciduous tooth bud. Missing teeth should be documented and radiographed to be certain no teeth are left unerupted leading to additional dental abnormalities.

**NORMAL OCCLUSION**
The normal mesocephalic skull has anisognathic mandibles. With orthocclusion, the mandibular incisors occlude on the cingulums of the maxillary incisors; the mandibular canines interdigitate, without touching, between the maxillary third incisors and canine teeth. The mandibular and maxillary premolars interdigitate, and the tips of the upper and lower second premolars are at the same horizontal level.

**Malocclusions**
Malocclusions are deviations from the normal occlusion. Considered to be genetic or developmental in origin, malocclusions can be the result of skeletal or tooth abnormalities, respectively. Malocclusions result in trauma from abnormal tooth-to-tooth contact and tooth to soft tissue trauma. Malocclusions are classified as symmetrical skeletal malocclusions, Class 1[(Neutroclusion) abnormalities of teeth position and number but normal jaw relationships], Class 2 (mandibular-distoclusion) with mandibular teeth distal to maxillary teeth, Class 3 (mandibular-mesioclusion) with mandibular teeth mesial to maxillary teeth, and asymmetrical skeletal malocclusions (maxillary-mandibular asymmetry). See www.avdc.org for current accepted veterinary dental nomenclature and classifications.

**Class 1 Malocclusion**
A class 1 malocclusion involves normal jaw relationships but abnormal dental (tooth) relationships (e.g., linguoversed mandibular canines, mesioversed (lance) maxillary canines, rostral crossbites, caudal crossbites)

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

Adult linguoversed canine teeth are a result of retained corresponding deciduous teeth or a developmental defect. This includes a class 2 malocclusion, brachygnathic mandible, excessive anisognathism, or persistent primary mandibular canines. Linguoversion of 304 and 404 can lead to severe damage to the hard palate, oronasal fistulas, periodontal defects, tooth damage to 103, 104, 203, and 204, and inability to close the mouth. Local periodontal disease and oronasal fistulas can develop in the traumatized maxillary arcade. Treatment options include orthodontic movement, crown reduction and partial coronal pulpectomy with a direct pulp cap, crown reduction and total pulpectomy, or extraction of the offending mandibular canine tooth. Surgical extraction of the adult mandibular canine teeth is rarely performed since the teeth are strategic and the procedure is more traumatic and destabilizing compared to an endodontic procedure. Orthodontic movement is successful. Depending on the severity of the linguoversion, the owner’s commitment, and compliance of the patient, various techniques have been discussed for moving 304 and 404. Treatments can consist of removable orthodontic devices (ball), direct acrylic incline planes, indirect acrylic or metal inclined planes, active expansion screws, vital pulpotomy, or extraction.
Mesially displaced (Mesioversed) maxillary canine teeth are encountered mostly in Shelties. A few other breeds such as Italian Greyhounds, Miniature Schnauzers, and some cats have been reported. One maxillary canine tooth is erupting dorsal to the cervix of the ipsilateral third incisor. The crown is pointing straight forward like the lance. Correction of this condition requires orthodontic movement or extraction of the misplaced tooth. If left untreated, mesioversed canine teeth and the adjacent dentition are predisposed to periodontal disease due to crowding. Additionally, the tooth may be in a position of traumatic occlusion with the opposing mandibular canine tooth leading to pulpitis, pain, and pulp necrosis.

A rostral crossbite is a malocclusion where one or more of the incisors is/are in version to the corresponding tooth in the opposite arcade. The malocclusion can lead to endodontic and periodontal compromise with resulting pain and infection. Chronic concussive forces could lead to complicated or uncomplicated crown fractures, pulpitis (reversible or irreversible), root fractures, and periodontal disease.

A level bite is a variation similar to a rostral crossbite where the maxillary and mandibular incisors occlude abnormally on the cusps. Sequelae and treatment are similar to a rostral crossbite.

A caudal crossbite occurs when the mandibular premolars and molar are buccal to their maxillary counterparts. If there is no traumatic contact with hard or soft tissue, then no treatment is necessary. If there is trauma, exodontics or crown reductions and endodontics will be necessary.

Crowded and rotated teeth often present in the brachycephalic breeds predisposed to periodontal disease. Interceptive extractions, following intraoral radiographs, to remove the less strategic premolars to preserve teeth 108, 208, 309, and 409 should be considered on an individual patient basis. Selective extraction of a few teeth can help prevent chronic periodontal infection and loss of several teeth and the strategic teeth.

**Class 2 Malocclusion**
A class 2 malocclusion is a discrepancy of jaw length so that the mandibular incisors are now abnormally distal to the maxillary incisors. This is generally the result of a genetically short mandible. The malocclusion is commonly associated with linguoversed canine teeth causing palatal trauma. Treatment of linguoversion is discussed above.

**Class 3 Malocclusion**
A class 3 malocclusion is commonly seen in brachycephalic breeds due to a genetically short maxilla. It may be considered “normal” for the breed but it is an abnormal skull and jaw conformation with consequences such as brachycephalic airway syndrome, crowded and rotated teeth predisposing to periodontal disease, and unerupted teeth. It is not normal from a dental, oral health, and genetic position.

**Asymmetrical Malocclusion**
Maxillar- mandibular asymmetry is an asymmetry of the maxilla and mandible and can be the result of trauma (i.e. a puppy bit in the face) or genetic. The malocclusion may be rostro-caudal direction
with mandibular mesioclusion or distoclusion on side of the face and the contralateral side is in normal dental alignment. A side-to-side direction is the loss of midline alignment between the maxilla and mandible. A dorso-ventral direction results in an open bite defined as an abnormal vertical space between opposing dental arches when the mouth is closed. A rostral open bite occurs when there is abnormal space between the maxillary and mandibular incisors when the mouth is closed. Treatment is often not needed. A caudal open bite occurs when there is abnormal space between the maxillary and mandibular premolars. Treatment is often not needed.

*Feline Pyogenic Granuloma Secondary to Tight Occlusion (SEE CVMA 2018 FELINE ORAL INFLAMMATION NOTES)*

**DEVELOPMENTAL TOOTH ABNORMALITIES**

Regardless, there are esthetic issues and more importantly, the thin primary and secondary dentin can allow bacteria and toxins into the endodontic system and cause death of the tooth. Sealing of the dentin tubules and removal of unsupported enamel is often necessary.

**Enamel Defects**

Young animals that experience elevated temperatures systemically or locally in the regions where the adult tooth buds are developing can develop enamel defects. The ameloblasts are sensitive to temperature and enamel formation can be affected. Regardless if enamel hypoplasia or enamel hypocalcification, there are esthetic issues and more importantly, the thin primary and secondary dentin can allow bacteria and toxins into the endodontic system and cause death of the tooth. Sealing of the dentin tubules and removal of unsupported enamel is often necessary.

- **Enamel Hypoplasia**
  - Enamel hypoplasia is diminished amounts of normal enamel. The teeth have rough surfaces and often are stained yellow.
- **Enamel Hypocalcification**
  - Enamel hypoplasia is diminished amounts of normal enamel. The teeth have rough surfaces and often are stained yellow. Enamel hypocalcification is normal amounts of poor quality enamel that is easily damaged and flakes off.

- **Amelogenesis imperfecta “like” disorder resulting in enamel defects has been reported in Standard Poodles. Generally, amelogenesis imperfecta is a genetic disorder that will result in small, discolored, pitted, and grooved teeth that wear and break rapidly in people. In Standard Poodles, the condition may be similar but resulting mostly in poor quality enamel that is discolored early in life. Clinically, I have not appreciated the clinical findings often associated in humans.**

**Other Developmental Tooth Defects**

Odontodysplasia can be localized or more generalized. It may be the result of focal abnormalities. Trauma to the developing tooth buds and/or dental lamina is a common cause in dogs. The enamel,
Dentin, and pulp are all affected. Intraoral radiographs are required to determine treatment which may include extraction, endodontic treatments, restoration, and/or serial monitoring.

Dens Invaginatus or dens in dente (tooth within at tooth) is a development anomaly commonly affecting the mandibular 1st molars in dogs resulting in hidden, chronic, endodontic disease and suffering pets. There is an infolding of enamel and dentin commonly in the crown and furcation region of these molars resulting in defects and allowing bacteria into the endodontic system. Surgical extraction is most often necessary. However, endodontics has been reported in some clinical cases.

Other conditions such as macrodontia, microdontia, taurodontia, etc. can occur.

Supernumerary teeth (extra teeth) can be crowded and potentiate periodontal disease. If there is not sufficient space for the crowns and roots, exodontics should be considered.

Some teeth may have supernumerary roots. These teeth with extra roots need to be radiographed to determine if the root is complete and normal. If the root is small, there could be communication between the periodontal and endodontic system leading to infection and loss of the tooth.

Gemini teeth are those in which two crowns are present with one radicular system. Fusion teeth are teeth with fused crowns but have two radicular systems. These teeth should be radiographed to be certain there are no subgingival abnormalities. If there is no crowding or abnormalities predisposing to periodontal disease, no treatment may be necessary.

Abrasion is mechanically wearing of the tooth from abnormal habits and foreign elements.

Attrition is mechanically wearing of the tooth from the opposing teeth.

**ENDODONTIC DISEASE**

Fractured teeth REQUIRE intraoral dental radiographs. All fractured teeth with pulp exposure require endodontic treatment or extraction. “Wait and see” is a negligent and an inappropriate option. This includes deciduous teeth. See www.avdc.org for tooth fracture classification system.

**Fractured Deciduous Teeth**

Complicated crown fractures (pulp exposure) of deciduous teeth result in periapical infections/inflammation adjacent to the developing tooth bud in addition to acute pain. The tooth bud can be damaged, develop enamel defects, or die. Deciduous teeth with complicated crown fractures require extraction.

Complicated Crown Fractures in Young Adult Teeth – Apexogenesis and Apexification

The pulp has good resilient potential at this age and endodontic treatment should be pursued in a timely fashion. If the patient is <12 months of age, a vital pulpotomy (partial coronal pulpectomy and direct pulp capping)/apexogenesis should be performed as soon as possible. If the tooth is not vital, apexification or apexification like procedures are necessary to create an apical portion of the tooth followed by standard endodontic treatment.
**Oral Tumors (very limited discussion)**

Although it is possible for any oral tumor to develop at any time, the most common tumors in growing patients are odontomas (an odontogenic tumor).

Odontomas are of both epithelial and mesenchymal origin and are found in young animals. A complex odontoma has an amorphous mass of poorly differentiated mineralized material. A compound odontoma has denticles (tooth-like structures) in an unorganized, random, structure throughout the tumor (pulp, dentin, cementum, and enamel are present in a normal relation). Odontomas are locally destructive and early recognition and excision is often curative. Odontomas are locally destructive and early recognition and excision is curative. Preservation of the cortical bone will allow functionality of the maxillofacial skeleton long term.

Feline inductive odontogenic tumors are rare. They are found in the rostral maxilla of young cats. They are locally invasive. The odontogenic epithelium is organized around a dental pulp-like stroma.

One additional tumor to consider in a young animal is a papillary squamous cell carcinoma. They tend to be locally aggressive so excision with margins offers a reasonably good prognosis. These may also occur in older animals.

**Odontogenic Dentigerous Cyst**

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

**Cleft Palate**

If a puppy and kitten survives the first few weeks of life, clinical signs become apparent when cleft palates are present. They have difficulty feeding and growing compared to litter mates. Milk often comes out of the nostrils. Aspiration of milk and food leads to aspiration pneumonia.

Cleft palates can be primary if involving the lips and incisive bone or secondary if involving the hard and soft palate. Cleft palates are often genetic in origin but may be congenital secondary to trauma, infectious, or toxic insults during embryological development. The communication between the oral cavity and respiratory system often cause the patients with secondary cleft palates to have poor nutrition and increased respiratory infections. Assisted feeding (tube feeding) is often needed until the patients are 8-16 weeks of age. At this time a surgery can be performed. The actual timing of surgery is debated within the veterinary literature. Scar tissue formation may affect further maxillofacial development, further development may lead to larger defects and early closure may be chosen, the metabolic reserves and stability of the patient for general anesthesia, concurrent respiratory infections, poor nutritional plane, and the technique chosen to repair the defect are all considerations when choosing the timing of the primary closure of the defect. The owners are always advised that pneumonia can occur and additional surgeries may be needed. The first surgery is always the best chance at closure. With each surgical attempt at closure, additional scar tissue forms and vascularization is decreased. Therefore, these surgeries are best performed by veterinarians familiar with different repair techniques and have experience. Regardless, dehiscence is common and additional surgeries to complete the repairs may be necessary. Movement of the
tongue, changes in air pressure during respiration, and tension on surgical flaps contribute to failures.

Microglossia (bird tongue) is a heritable condition that may not be recognized. It is a condition where puppies have small tongues and lack marginal fimbriae necessary for suckling. They often die in the first week of life and may be one of the many unrecognized causes for “fading puppy syndrome”. It may be part of a multiple organ heritable defect condition.
The Senior and Geriatric Dental patient: Age Should Not Preclude Treatment

Kevin S. Stepaniuk, B.Sc., DVM, FAVD

Introduction
To prevent senior and geriatric age pets from suffering with insidious dental and oral disease, preventive professional dental and home care during all life stages is the best medical recommendation. As immunosenescence begins and concurrent systemic and age related disease occurs, the once hidden dental disease may become more obvious, progressive, and require medical intervention to alleviate disease, infection, pain, and suffering. Age categories are arbitrary. However, based on AAHA senior care and life stage guidelines, a senior may be defined as a dog and cat that has lived 75% of a predicted life span. A geriatric patient may be defined at life expectancy and beyond (breed dependent). For cats, the last 25% of their life span (i.e., senior) may defined as 11-14 years of age and a geriatric 15+ years of age.

Concurrent Medical Illness
Anesthesia and dentistry treatment require individualized patient assessment, pre-anesthetic planning, and individualized anesthetic plans. A thorough medical history including all current medical illnesses, past medical illnesses, past dental treatments, and current medications is necessary. A complete physical examination and blood work (complete blood count, chemistry panel, urinalysis, and thyroid evaluation) are necessary for all aged patients. A preoperative blood pressure is recommended. Concurrent disease such as cardiovascular disease (e.g., heart murmurs, cardiac dysfunction), and renal disease are common co-morbidities. Disease such as diabetes mellitus, hyperthyroidism, hyperadrenocorticism, and hepatopathy may be identified. Concurrent morbidities do not preclude general anesthesia. However, assessment with thoracic radiographs, echocardiography, and stabilization of concurrent medical co-morbidities, as necessary, should be recommended for management prior to treatment of chronic dental and oral disease. Anesthesia and dentistry treatment require individualized patient assessment, pre-anesthetic planning, and individualized anesthetic plans.

Oral and Dental Disease
A pain free and infection free oral cavity is a priority. The assessment and treatment requires general anesthesia and intraoral radiographs. The prevalence of periodontal disease increases with age. Periodontal stages (i.e., PD0-PD4) are assigned based on intraoral radiographs and periodontal probing.

Oral tumors represent about 6-7% and 3% of all canine and feline tumors, respectively. Biopsy is required for diagnosis. The majority of feline and canine oral tumors occur in middle aged and older patients. The most common malignant canine oral tumors are malignant melanoma (30-40%), squamous cell carcinoma (20-30%), and fibrosarcoma (10-20% and male dogs predisposed). The most common feline tumors are squamous cell carcinoma (70-80%) and fibrosarcoma (13-17%). Odontogenic tumors (e.g., canine acanthomatous ameloblastoma, peripheral odontogenic fibroma) compromise 20-30% of oral tumors. Endodontic disease is commonly the result of fractured and concussive/luxation injuries of teeth. Fractured teeth, non-vital teeth, caries, developmental anomalies, concussed teeth, luxated teeth, and perio-endo lesions result in inflammation and infection of the endodontic system and periapical
tissues. All fractured teeth with pulp exposure require endodontic treatment or extraction. Classification of tooth fractures can be found at www.avdc.org (nomenclature).

Feline tooth resorption is a common and frustrating dental problem in the feline patient. The prevalence of the disease, in cats, has been reported in the literature as 20-75% and increases with age. The stage of lesions can be classified by the extent of tooth involvement. The lesions can be further divided into radiographic types for treatment planning.

Recently, several publications have evaluated tooth resorption in dogs. Peralta, et al. (2010) identified increased frequency of tooth resorption in older and large-breed dogs with no sex predilection. Tooth resorption was detected in 53.6% of the dogs.

Malocclusions are deviations from the normal occlusion. In some pets, malocclusions have resulted in unrecognized, chronic soft tissue, hard tissue and dental injuries that are not recognized until much later in life. Malocclusions are classified as symmetrical skeletal malocclusions (e.g., Class 1-3) and asymmetrical skeletal malocclusions.

**Anesthetic Considerations**
Anticipated problems in the senior and geriatric patient include but are not limited to, co-morbidities, hypotension, hypothermia, and increased anxiety. Multimodal anesthesia and analgesia is necessary. The use of regional nerve blocks as part of the anesthesia plan is beneficial to decrease the requirements of inhalant anesthetics. Likewise, constant rate infusions of opioids, such as fentanyl, remifentanyl, morphine-lidocaine-ketamine, are commonly used to provided anesthesia and analgesia in senior and geriatric patients with concurrent morbidities so that the depth of anesthesia can be maintained with little impact on the cardiovascular system. Constant rate infusions with dopamine and dobutamine are utilized based on concurrent disease processes such as renal disease and cardiovascular disease to maintain blood pressure as indicated. Thermoregulation with both forced air convection warming devices and conductive polymer fabric heating are both used to maintain normothermia. A dedicated staff member is required to monitor anesthesia. Blood pressure monitoring (e.g., doppler, oscillometric), ECG, end-tidal carbon dioxide, and pulse oximetry are monitored in every patient. A ventilator is beneficial to help control tidal volume and rate of respiration so that anesthetic gas exchange is consistent.
Introduction
Salivary mucoceles, also termed sialoceles, are accumulations of saliva in the subcutaneous tissues and occur most commonly in dogs and rarely in cats. The source of the saliva can be from any of the salivary gland/duct complexes, but most commonly leaks from one of the four major salivary glands (zygomatic, parotid, sublingual, mandibular) with leak from the sublingual gland/duct complex predominating. The cause of a salivary leak is rarely identified but can occur as a result of trauma to the salivary gland and/or duct, foreign material, calculi in the salivary gland/duct or neoplasia.

The most clinical signs associated with a salivary mucocele are dependent on the origin of salivary leak (Table).

<table>
<thead>
<tr>
<th>Origin of salivary leak (gland/duct)</th>
<th>Clinical signs</th>
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<tbody>
<tr>
<td>Zygomatic</td>
<td>Exophthalmos</td>
</tr>
<tr>
<td>Pharyngeal swelling from sublingual/mandibular</td>
<td>Dyspnea, stridor</td>
</tr>
<tr>
<td>Ranula from sublingual/mandibular</td>
<td>Dysphagia,</td>
</tr>
<tr>
<td>Sublingual/mandibular</td>
<td>Cervical swelling**</td>
</tr>
</tbody>
</table>

**most common

Diagnosis
Fine needle aspiration of the swelling is recommended. Salivary mucoceles will contain thick, serosanguinous fluid that resembles saliva. Cytological evaluation of the material should be performed and will reveal variable numbers of nondegenerate nucleated cells and proteinaceous material (mucin). Once a salivary leak is confirmed, medical management can be attempted, however, is not commonly successful in this author’s experience. This is likely because of the continuous leak of saliva in to the subcutaneous tissues. Surgical removal of the gland producing saliva is recommended. In cases of ventral cervical swelling, determining which side (right vs left) sublingual/mandibular gland is leaking can be challenging. To help identify the side, the dog can be placed in dorsal recumbency and particular attention is paid to the side the swelling falls toward as this is generally the side where salivary leaking is occurring. Preoperative cervical ultrasound is can evaluate salivary glad structure and aid in determining the gland (+/- side) to be removed.

Operative Procedure
Because cervical swelling from salivary leak from the sublingual or mandibular salivary gland/duct complexes is most common, the operative procedure for removal of these glands only will be
discussed. These glands share a common duct and, therefore, a removed together as a single structure.

Lateral Approach: The dog is placed in lateral recumbency and the cervical and facial regions are clipped and aseptically prepared for surgery. The mandibular salivary gland will be located at the bifurcation of the external jugular vein, just caudal to the mandibular lymph node. A vertical incision is created through the skin, subcutaneous tissues and platysma muscle centered over the jugular bifurcation. Digital palpation will reveal the capsule of the mandibular salivary gland which is more firmly attached and paler in color compared to the mandibular lymph node. The capsule is sharply incised revealing the mandibular gland. Using a combination of sharp and blunt dissection, the mandibular gland is freed from its capsule. An atraumatic instrument (i.e. Allis tissue forcep) can be used to grasp the gland providing gentle caudal retraction. The cranial extent of the mandibular gland is reached, revealing the duct and portions of the sublingual salivary gland. Maximal visualization is reached by caudal retraction of the mandibular salivary gland and the duct is ligated using monofilament, absorbable suture as proximally as possible. The site is lavaged and closed routinely. Following removal of the salivary gland, mucocele drainage is performed and described below.

Ventral Approach: The dog is placed in dorsal recumbency and the ventral mandibular and cervical region clipped and aseptically prepared for surgery. The angle of the mandible identified and an incision from the cranial third of the mandible to the level of the salivary gland created through the skin, subcutaneous tissues and platysma muscle. The mandibular gland is identified using similar methods as described above for the lateral approach. Once the mandibular gland and distal portion of the salivary gland have been dissected in a similar fashion to the lateral approach, the digastricus muscle is identified and a hemostat is placed from a rostro-medial to caudal underneath the digastricus to clamp the duct of the mandibular/sublingual salivary gland. The salivary tissue distal to the forceps is excised (essentially what can be removed via a lateral approach) and the forceps are withdrawn in a rostro-medial direction. The mylohyoideus muscle is identified and incised which reveals the continuation of the sublingual salivary gland (polystomatic portion) and duct. Careful dissection is performed to the level of the lingual nerve where the salivary duct is ligated using monofilament, absorbable suture and tissue distal to the ligature excised. The site is lavaged and closed routinely. Following removal of the salivary gland, mucocele drainage is performed and described below.

Mucocele Drainage: Once the salivary gland thought to be causing the leak has been removed, the mucocele should be drained. Briefly, the author simply makes a small stab incision into the swelling to allow for drainage of the saliva and a closed suction drain placed. If the mucocele is encountered during the surgical approach (commonly occurs during ventral approach), a separate incision is not required. A closed suction drain or passive drain (e.g. Penrose) should be placed in the mucocele cavity following drainage. If the mucocele is a ranula (peri-lingual mucocele), marsupialization is then performed follllowing removal of the mandibular/sublingual gland/duct complex.

Ventral vs Lateral Approach – What should I do? The obvious benefit of a ventral approach is the additional sublingual salivary tissue that can be removed. This has been thought to reduce recurrence of mucocele, however, a randomized, prospective study is lacking in the veterinary literature. While a lateral approach to the
mandibular/sublingual salivary gland is generally uncomplicated, a ventral approach can definitely become easier with experience. Cadaveric dissection may help the practitioner traverse the learning curve associated with this approach.

References:
Surgical Diseases of the Spleen: How you can Avoid Trouble!

Ameet Singh  DVM, DVSc, Diplomate ACVS

Introduction
The small animal practitioner can face a myriad of splenic diseases and they should be familiar with their diagnosis and surgical treatment. Total splenectomy (via ventral midline laparotomy) is commonly performed for benign or malignant neoplasia, splenic torsion, and infiltrative diseases. To allow for successful surgical treatment, the surgeon should be familiar with splenic anatomy, methods of splenectomy and perioperative management techniques. The basic principles of exploratory laparotomy should be followed such as an appropriate sized ventral midline incision, removal of the falciform ligament and use of Balfour retractors to improve abdominal visualization and thorough exploration of all abdominal organs/structures.

Anatomy
The spleen is purple-red colored organ, located in the left cranial quadrant of the abdomen and has a soft consistency but is less friable than the liver. The head of the spleen is attached to the greater curvature of the stomach with a gastrosplenic ligament whereas the ventrally located tail of the spleen is quite mobile and lies across ventral midline. Occasionally, the practitioner may encounter small (2-4mm diameter) circular pieces of splenic tissue as incidental findings in the abdomen. These have been named accessory spleen fragments and can be the result of congenital and acquired causes.

The surgeon needs to be familiar with splenic vascular anatomy as this is of utmost importance during total splenectomy. The celiac artery is the first unpaired vessel from the abdominal aorta and one of its main branches is the splenic artery. The splenic artery then traverses the pancreas, giving rise to the pancreatic artery (can be several branches from the splenic artery). The splenic artery then enters the splenic hilus with branches heading towards the tail and the head of the spleen. The splenic vein drains the spleen and ultimately enters the portal vein.

Splenic Trauma
Depending on the practice type and location, splenic trauma may be encountered with some frequency as frequent causes include penetrating and blunt trauma such as motor vehicle trauma, bite wounds, gunshot injury, etc. If a patient is presenting following a traumatic injury and hemoabdomen is suspected, abdominocentesis (+/- ultrasound guidance if available) is performed to confirm diagnosis. Standard techniques for patient stabilization are employed including intravenous fluid therapy +/- vasopressor therapy. In most causes of traumatic hemoabdomen, splenic and/or liver trauma is the most common cause and DOES NOT frequently require exploratory laparotomy for hemorrhage control. An abdominal compression bandage applied from the level of the caudal rib cage to the distal limbs should be applied in an attempt to provide pressure for hemostasis. Caution must be exercised when applying a compression bandage in cases with pulmonary dysfunction secondary to trauma.

In cases where hemorrhage is not controlled using conservative management, exploratory laparotomy is indicated for splenectomy (partial or total). Multiple fragments of spleen may be
encountered and in some cases salvage of a portion of the spleen may be possible. Blood transfusion may be required in the perioperative period. Prognosis is variable and depends on concurrent injury.

**Splenic Torsion**
Isolated torsion of the splenic pedicle or primary splenic torsion (PST) is a rare occurrence in dogs and occurs when the spleen rotates around the gastrosplenic ligament. It is most commonly reported in large or giant breed dogs with the German Shepherd and Great Dane overrepresented in the veterinary literature. The most common presenting complaint with PST is acute abdominal pain, weakness and cardiovascular collapse. A torse splenic pedicle results in splenic vein collapse resulting in an engorged spleen with a characteristic “C” shape on abdominal radiographs. Emphysematous changes in the splenic parenchyma can also be seen in some cases with parenchymal necrosis. In equivocal cases, if available, ultrasonography can be performed to confirm reduced blood flow through the main splenic vessels. Many patients with PST have cardiovascular compromise and aggressive intravenous fluid therapy, as well as additional therapy to maintain blood pressure, is required for patient stability prior to general anesthesia.

At time of exploratory laparotomy, the surgeon should NOT derotate the splenic pedicle and simply perform total splenectomy since derotation may result in release of free radiclas, thrombi, and other vasoactive compounds intravascularly. Prophylactic gastropexy following splenectomy for PST is recommended to prevent future gastric dilatation volvulus. In a recent study performed at the authors’ institution, PST is associated with a good prognosis and neoplasia was not identified during histopathological exam of any spleen.

**Splenic Neoplasia**
Splenic neoplasia is common in dogs with most cases not showing clinical signs unless the mass ruptures resulting in hemoabdomen or if the mass grows to a large size causing non-specific gastrointestinal signs such as anorexia and/or lethargy. Recent reports investigating splenic disease in dogs with non-traumatic hemoabdomen requiring a blood transfusion state that approximately 75% of splenic tumors are malignant with hemangiosarcoma (HSA) predominating. In a recent study of dogs undergoing splenectomy for splenic mass, the perioperative mortality rate was 7.6%, however, the challenge arises when counseling dog owners about long term prognosis and whether to pursue surgical intervention. Often these dogs are clinically normal prior to an episode of collapse or acute onset lethargy which prompts the owner to seek veterinary care. A hemoabdomen is diagnosed which is most commonly a result of a ruptured splenic mass and the decision to pursue treatment in the face of the odds of HSA as a final diagnosis are daunting for some dog owners. Long-term prognosis for ruptured or non-ruptured splenic HSA without gross metastatic disease (stage 1 or 2) is median 4-6 weeks without chemotherapy and median 4-6 months with chemotherapy. Cases of hemoabdomen secondary to a ruptured splenic mass are often surgical emergencies and unfortunately this does not provide the dog owner a long time to consider whether they would like to pursue surgical treatment (total splenectomy).

When performing exploratory laparotomy for splenic neoplasia, especially in cases of a ruptured mass, blood transfusion is commonly required and the surgeon should be adequately prepared for this possibility. If this is not possible, referral to a veterinary centre with a blood bank program is recommended. Suction is very helpful as a large volume of blood is commonly present in the abdomen. Often omental adhesions are present to the splenic mass and these should not be pealed.
from the mass. Instead the adhesions should be ligated a few cm proximal to their attachment to the spleen. Total splenectomy is then performed and the spleen submitted for histopathological evaluation. A thorough exploration of the abdomen is then performed to rule out metastatic disease. The liver is a common spot for metastatic disease of HSA and the author routinely performs multiple liver biopsies to rule out microscopic HSA as this likely affects prognosis.

**Total Splenectomy**

Once the decision to perform total splenectomy has been made and exploratory laparotomy performed (including thorough exploration of abdominal contents), several methods for total splenectomy exist in the veterinary literature. Consideration to performing the splenectomy first if there is a large amount of free abdominal fluid (hemorrhage) can be made. As previously discussed, any omental adhesions to the spleen should be ligated and divided without pealing the adhesions from the spleen. The “four-clamp” technique is a well described technique by Dr. Daniel Smeak, and is a safe and efficient procedure for total splenectomy. Briefly, the stomach is visualized and area where the head of the spleen is attached to the stomach isolated to bring the short gastric vessels into view. This “pedicle” can be digitally isolated by puncturing the gastrosplenic ligament with a finger and then three clamps applied. The pedicle can be incised between clamp 2 and 3 which releases the spleen and ligatures placed in the crush of clamps 1 and 2. Three pedicles remain and they are centered on 1) branch of main splenic artery heading to the head of the spleen, 2) main splenic artery and 3) caudal branch of main splenic artery heading to the splenic tail. A three-clamp technique for ligation of each of these pedicles is performed as described for pedicle 1. If active bleeding of the splenic mass is present, these pedicles are created, clamped and incised between clamps 2 and 3 allowing for rapid removal of the spleen. To save time in surgery, only ligate the portion of the pedicle that is remaining in the patient and leave clamps on the spleen side which will be removed.

Partial splenectomy has been discussed in the veterinary literature, however, requires a longer operative time and less efficient hemostasis compared with total splenectomy. Partial splenectomy should NEVER be considered if performing splenectomy for a neoplastic mass as microscopic disease may be present in other areas of the spleen.

**Vessel Sealing Devices**

In recent years, methods for performing splenectomy which reduce operative time compared to suture ligation have been described. Vessel-sealing devices have gained tremendous popularity for both minimally invasive and open surgery. These devices are hand instruments with jaws that are placed around the tissue to be sealed. Once the jaws are compressed, bipolar energy is released between the jaws and then thermal energy induces fusion of the collagen and elastin present in the tissue and vessels creating a seal. One vessel sealing device (Ligasure, Medtronic Inc.) has been shown to safely seal vessels up to 7 mm in diameter. Several advantages to using vessel-sealing devices include reduced operative time, no remaining foreign material and no need for dissection or creation of “pedicles” for ligation.

Consideration should be given by general practitioners who have a large soft tissue surgical caseload to obtain a vessel sealing device as cost has come down considerably. The re-sterilization of single use hand pieces also makes these devices more economical.
**Gastropexy Following Splenectomy?**

There is considerable controversy in the veterinary literature about whether splenectomy increases the risk of GDV with various studies reporting different outcomes. The author does not routinely perform prophylactic gastropexy following splenectomy for splenic masses, but does following splenectomy for PST. There is likely some merit to the idea that with PST the support ligaments of the stomach stretch potential increasing the risk of GDV.

**Postoperative Considerations**

Following splenectomy, the major postoperative complications the surgeon must be weary of include the potential for ventricular arrhythmias and hemorrhage. Cardiac arrhythmias following total splenectomy are not uncommon, however, careful monitoring of heart rate and rhythm is required and anti-arrhythmic therapy considered on a case-by-case basis. Hemorrhage can occur from failure of surgically applied ligatures. Care must be taken when placing ligatures at time of surgery to prevent this complication. Should concern for hemorrhage exist, reoperation must be considered depending on patient cardiovascular status.

**Minimally Invasive Splenectomy**

Laparoscopic surgery has gained tremendous popularity in veterinary medicine and laparoscopic splenectomy (LS) has been described in dogs and cats. In a study where multiport LS (MLS) was performed in healthy dogs without diseased spleens, reduced post-operative pain, wound complications and blood loss was found compared with open splenectomy. However, surgical time was significantly longer in the MLS group. Another study on MLS in 10 dogs with splenic pathology reported a 90% success rate (conversion in 1 dog was required due to omental adhesions and obstruction of splenic visualization from falciform fat). In the same study, median surgical time was 61.5 minutes (range 31-100 minutes) which is significantly longer than what is reported for open splenectomy in dogs.

As with any minimally invasive surgery (MIS), case selection is of paramount importance for successful outcome, since intracorporeal manipulation can result in uncontrollable hemorrhage that could require emergent or elective conversion to open laparotomy. In humans, LS is not recommended for massive splenomegaly since it has been shown that this technique is associated with greater complications related to intracorporeal manipulation of the spleen. Based on this finding, a hand-assisted laparoscopic splenectomy (HALS) technique was developed where an assist incision was created that allowed for insertion of the surgeons’ hand into the abdomen for careful manipulation of large spleens. HALS has been shown to be associated with a shorter learning curve and no difference in surgical time and blood loss compared to LS.

A technique for laparoscopic-assisted splenectomy (LAS) has been described in dogs and cats and, in addition to providing many of the benefits of MIS, allows for safe manipulation of spleens with small to medium sized masses and with moderate to marked splenomegaly with potentially shorter operative times compared with LS. The LAS technique likely expands the capability of MIS for splenectomy since careful case selection (small mass or mild splenomegaly) is required for successful LS.

**References:**
Prophylactic Gastropexy: When? How?
Ameet Singh DVM, DVSc, Diplomate ACVS

Introduction
Because of the morbidity and mortality associated with GDV, prophylactic gastropexy has been recommended for prevention in at-risk breeds. Gastropexy can be performed at the time of sterilization via open laparotomy or using minimally invasive laparoscopy. Prophylactic laparoscopic-assisted gastropexy (LAG) has been described in dogs and has been shown to result in a strong adhesion between the pyloric antrum and right body wall, and is comparable to open laparotomy techniques.

What is the Evidence for Prophylactic Gastropexy in Dogs?
In dogs with GDV, gastropexy reduces recurrence to <5% whereas failure to perform gastropexy results in recurrence rates as high as 80%. These results make the decision to perform gastropexy at time of surgery for the correction of GDV an easy one.

Prophylactic gastropexy has gained considerable interest in recent years in dogs at-risk for GDV. This is commonly done at time of spay or neuter in order to prevent an additional anesthetic episode for the dog. The question associated with a prophylactic gastropexy is what is the risk that a dog will develop GDV? If this risk is low, then prophylactic gastropexy will likely be of little benefit. In a study performed in 2003, five at-risk breeds (Great Dane, Irish Setter, Rottweiler, Standard Poodle and Weimaraner) were studied to evaluate the risk vs benefit of prophylactic gastropexy. After comparing costs associated with treatment of GDV, costs associated with prophylactic gastropexy combined with the probability of death due to GDV, prophylactic gastropexy was deemed appropriate for all five breeds.

Technique - Incisional Gastropexy via Open Laparotomy
Numerous methods exist for gastropexy, however, the author prefers incisional gastropexy due to its speed, technical ease and biomechanical strength. An assistant is of great benefit for this procedure. The pyloric antrum is located, which is 5-7cm orad to the pylorus. The pylorus can be identified as a thick, muscular ring in the right cranial abdominal quadrant and should not be confused as a gastric foreign body. Practitioners should become familiarized with the pylorus, as in cases of GDV it is found in the left cranial abdominal quadrant and can be readily palpated. When derotating the stomach in cases of GDV, it must be grasped and rotated ventrally back to the right (and anatomically) correct side of the abdomen.

A 3-5cm incision is made in the pyloric antrum depending on the size of the dog midway between the greater and lesser curvatures in a parallel plane to the long axis of the stomach. The incision is then created through the seromuscular layer only and the mucosca/submucosa layer will be noted to bulge through this incision. In case of penetration through the mucosa/submucosa layer, the defect is closed primarily using a monofilament, absorbable, long-acting suture prior to continuing with gastropexy. The primary surgeon moves to the left side and an assistant moves to the right hand side of the patient and elevates the body wall using two towel clamps placed through the external rectus fascia. The gastric wall incision is then matched to a location in the body wall caudal to the last rib and in the ventral 1/4th of the body wall. An incision is then created using a #15
scalpel blade through the peritoneum and transversus abdominus muscle in a craniodorsal to caudoventral direction approximating the same length as the gastric seromuscular incision. (The author essential incises parallel to the fibres of the transversus abdominus muscle. The craniodorsal aspect of the body wall and cranial gastric incisions are sutured using a monofilament, absorbable long-acting suture (e.g. PDS, 0 or 2-0). At the medial aspect of the incision, the suture is tied to prevent a purse-string effect and then continued along the caudal aspect of both incisions. Failure of incisional gastropexy is uncommon. Fistula formation has been reported following the use of polypropylene sutures and is not recommended.

Technique - Laparoscopic-assisted Gastropexy
This technique was originally described by Rawlings et al in 2001. The author uses a modified Hasson technique to obtain abdominal access in the sub umbilical region and following establishment of pneumoperitoneum (10-12 mmHg), a 6 mm, smooth or threaded trocar-cannula assembly placed. A 5 mm x 29 cm, 00 or 300 laparoscope is inserted into the abdomen and a superficial exploration is performed. A second portal (12mm-15mm) is created 2-4 cm lateral to the margin of the rectus abdominis and 3-5 cm caudal to the last rib depending on the size of the dog under direct laparoscopic guidance. Ten mm laparoscopic Babcock forceps or DuVall forceps are inserted into the abdomen through the paramedian instrument port and can be used to carefully manipulate cranial abdominal organs to provide a clear view of the antral portion of the stomach. If a clear view can not be obtained, the dog can be placed into reverse Trendelenburg position to shift abdominal viscera caudally away from the stomach.

Once the antral portion of the stomach is clearly visualized, a location 5-7 cm aborad from the pylorus is grasped midway between the greater and lesser curvatures. The surgeon should ensure a secure grasp of the stomach as it can slip out of the forceps. Without placing excessive tension on the stomach, the orientation is maintained and the abdomen desufflated. The skin and subcutaneous tissue incisions are enlarged on either side of the instrument portal incision to create an access incision of ~3 cm in a direction parallel to the last rib. The approach is continued through the external oblique, internal oblique, and tranversus abdominis muscle layers. Each muscle layer should be carefully identified with stay sutures to facilitate accurate closure. Allis tissue forceps are used to grasp the cranial and caudal edges of the tranversus abdominis muscle to maintain clear identification of these muscles for gastropexy. Surgical approach through the muscle layers should be performed parallel to the muscle fibres.

The stomach is carefully exteriorized through the access incision with the Babcock forceps and stay sutures were placed at the orad and aborad extent of the proposed gastropexy to maintain appropriate gastric orientation which is paramount for procedural success. The Babcock forceps are removed and the seromuscular layers of the stomach are incised and the cranial edges of the transversus abdominis muscle and stomach are sutured together followed by the caudal edges in a simple continuous pattern using a monofilament, absorbable suture. The author begins the suture line laterally and progresses medially on the cranial aspect. Once the suture line reaches the medial aspect, 5-6 throws are performed, and the line continued to the other side. Following gastropexy completion, the site is lavaged and the internal and external abdominal oblique musculature, subcutaneous tissues and skin are closed in three separate layers using a simple continuous pattern with monofilament, absorbable suture. Pneumoperitoneum is re-established at 6-8 mmHg through
the sub umbilical port and the gastropexy inspected to ensure appropriate gastric orientation. The laparoscope and cannula are removed and the port site closed routinely.

References:
Introduction
Pain negatively affects quality of life, delays recovery and induces behavioral changes that affect owner-companion animal bond. Pain causes unnecessary fear, anxiety and stress in affected animals and may lead to sympathetic nervous system activation and alter food intake and metabolism (1). For these reasons, the management of animal pain is a significant ethical and economic component in the modern practice of veterinary medicine (2). Pain is now considered as the 4th vital sign, and its assessment should be incorporated into the clinical evaluation of all patients. The veterinary healthcare team needs to be prepared to appropriately assess and treat pain to mitigate animal suffering to the best of our ability (1).

The Somatosensory System and the “Pain Processing Pathway”
The somatosensory system includes all structures allowing the perception of sensory information coming from the skin and the musculoskeletal apparatus. It has a primary function of protection; nevertheless, this system can malfunction and result in several chronic painful conditions. Indeed, the more we learn about pain, the more we see how complex it is. Briefly, the pain processing occurs via four main steps, transduction, transmission, modulation and perception (3,4).

- Transduction occurs via activation of peripheral nociceptors. These are present in the skin, muscles, joints and viscera. The activation of nociceptors results in the opening of membrane ion channels resulting in membrane depolarization and generation of an action potential. Therefore, a mechanical, chemical, electrical or thermal stimulation can generate an action potential at the nociceptor level (periphery).
- Transmission occurs when this impulse travels from the primary afferent fiber (1st order neuron) to the dorsal horn of the spinal cord (2nd order neuron).
- Modulation occurs at the level of the dorsal horn of the spinal cord where the nociceptive message travels via 2nd order neuron to the cerebral cortex (3rd order neuron). During the modulation process, the nociceptive message may be amplified or inhibited depending on the nature of the neurotransmitters. Therefore, these changes could be excitatory (e.g. glutamate, substance P, glycine) or inhibitory (e.g. noradrenaline, serotonin, opioids, GABA).
- Perception occurs when there is ‘translation’ of the stimulus into perceived pain.

Mechanisms of pain inhibition:
- Ascending inhibition (afferent fibers): known as the ‘gate control theory’
- Descending inhibitory system: endogenous control

Note that for perception of pain, there needs to be consciousness. Nevertheless, in animals that are anesthetized and undergoing a surgical procedure, all of the above mechanisms of generation, transmission and modulation of pain are occurring. This means that, although the animal cannot feel pain while it is anesthetized, as soon as it recovers from anesthesia, all of this ‘pain pathway’ will be active and sensitized. Thus, the imperative need of pre-emptive and postoperative multimodal analgesic management.
Mechanisms of Pain

Acute pain, also known as adaptive pain, occurs when inflammation and nociception prevail such as in postoperative pain. It is generally associated with potential or actual tissue damage and serves to avoid or minimize damage during the healing process. It is usually self-limiting (1).

- Nociceptive pain is a process where the peripheral primary afferent neurones are activated by a noxious stimulus which can be chemical, mechanical or thermal. The intensity and duration are proportional to the stimulus which usually produces a protective response such as withdrawal (e.g. pinching of the skin, hot surface) (3,4).
- Inflammatory pain occurs when there is actual tissue injury or immune cell activation. These events result in the release of inflammatory mediators from cells and consequent chemical changes in the tissues around the nociceptors which amplify the nociceptive input to the spinal cord. This occurs by facilitation or direct nociceptor activation (3,4,5).

Chronic pain, also known as maladaptive pain, is characterized by neuropathic, functional and/or chronic inflammatory pain. In chronic pain, the degree of pain does not necessarily correlate with the pathology observed or perceived by the individual and it is not associated with healing. It persists beyond the expected course of an acute disease process and it has no clear end-point (1). Chronic pain should not be considered a symptom, but rather a disease in its own right since it may be present in the absence of a primary cause (5).

- Neuropathic pain occurs due to a primary lesion or disease of the somatosensory system, resulting in complexes mechanisms including sensitisation of neural connective tissues, ectopic excitability, cross-excitation, gliopathy and neuro-immune interactions (6,7). Examples include nerve compression, infiltration by cancers, amputations, intervertebral disk disease, nerve damage during dental extractions, osteoarthritis, among others. Spontaneous pain is a clinical feature of neuropathic pain (5). In humans, neuropathic pain is generally considered to cause more severe and long-lasting pain, and to be less responsive to analgesics. In addition, it causes greater dysfunction and poorer indices of quality of life when compared with other types of chronic pain.
- Functional pain is characterized by a neurophysiological dysfunction with no detectable structural, metabolic or immunological cause. Examples include feline orofacial pain syndrome and interstitial cystitis.
- Chronic inflammatory pain results from the chronic release of nociceptive sensitizers. In some cases, acute inflammatory pain can persist and become pathologic and maladaptive especially when neuropathic pain (e.g. limb amputation) is involved or pain was not addressed properly at the time of initial injury (5). Examples include osteoarthritis, cancer, periodontal disease, pancreatitis, gastritis, inflammatory bowel disease, among others. Indeed, neuropathic pain is now known to be produced by an inflammatory component.

Clinical Relevance and Important Concepts

Patients with acute postoperative pain require aggressive multimodal treatment of pain for decreased morbidity and faster discharge from hospital, not to mention the welfare perspective. Patients with chronic pain may present with widespread sensory sensitivity, low scores of qualities of life, decreased levels of activity and interaction with others, inappropriate elimination habits, etc. The pharmacological treatment of chronic pain should be mechanism-based and is usually
performed on a “trial and error” basis. The importance of non-pharmacological treatment of chronic pain cannot be underestimated.

Central sensitisation is the increased responsiveness of nociceptive neurons in the central nervous system to their normal or subthreshold afferent input. It is expressed as pain hypersensitivity and sustained cerebral nociceptive inputs. These processes translate clinically to hyperalgesia (i.e. increased pain from a stimulus that normally provokes pain) and allodynia (i.e. pain due to a stimulus that does not normally provoke pain) (8).

References:

Introduction
Cancer is the leading cause of mortality in dogs older than two years of age and cancer-associated pain is a major symptom in these patients (1). Cancer pain leads to stress, suffering and low quality of life, and untreated pain in humans is one of the most feared and debilitating symptoms among cancer patients (3). In these patients, pain scores on a Numerical Rating Scale (NRS) from 0 to 10 generally vary from 4 to 6, with exacerbations as high as 7 (3). Furthermore, meta-analysis studies have revealed that 64% of human patients with end-stage cancer experience pain before death (4). Given that dogs and cats frequently present with advanced-stage cancer at initial evaluation and based on similar cancer biology when compared with humans, it is reasonable to presume that canine and feline patients will experience pain at some point in the progression of the disease (1).

Nature of Cancer Pain
The nature of cancer-related pain is multidimensional and can originate from (2):

- Primary tumor due to tumor growth and destruction of adjacent tissues and structures or nerve involvement. Primary tumor pain may affect up to 68% of human patients (3). Indeed, there are now known neuroimmune mechanisms in which the tumor itself can generate pain.
- Diagnostic procedures including surgical biopsies and repeated venipunctures.
- Cancer therapies including surgery, chemotherapy and radiotherapy. These can cause important inflammatory and neuropathic pain.
- Concomitant painful conditions are commonly observed in cancer patients. These might include osteoarthritis, gingivitis, metastatic disease, among others.

Clinical Manifestation of Cancer Pain
Cancer pain may vary greatly in nature and intensity depending on the location of the tumor as each tumor type will behave differently and will cause different types and levels of pain (2). For example, a dog with osteosarcoma will be clinically very distinct from a cat with oral squamous cell carcinoma, and different functions will be affected in each case. Additionally, tumors that affect internal organs result in diffuse visceral pain, whereas skin tumors are more likely to trigger focal pain. With all these in mind, it becomes clear that cancer-related pain is a result of a mixture of different types of pain. An example of that is a tumor causing nerve compression. Tumor necrosis will cause inflammatory/nociceptive pain, and nerve compression will cause neuropathic pain. In this case, central sensitization likely results from the chronic nociceptive input to the spinal cord and patients may develop hyperalgesia or even allodynia. Finally, for all the reasons above discussed, it should be considered that in cancer patients, both acute (clinically manifested as `breakthrough pain`) and chronic pain may be present, but the latter is much more prevalent. Pain is now considered as the 4th vital sign, and its assessment should be incorporated into the clinical evaluation of all patients (6). The clinical signs of pain in cancer patients will greatly vary on a case-by-case basis as they depend on the primary cause of the pain. However, in most cases the signs are related to changes from normal behavioural that are reported by the owner. In this sense, the owners play a crucial role in the assessment of cancer-related pain as well as monitoring of treatment efficacy.

Treatment of Cancer Pain
The treatment of cancer pain comprises a multimodal approach using pharmacological and non-pharmacological options (2,6,7).

**Pharmacological Treatment**

Opioids are the cornerstone of acute pain treatment; nevertheless, in veterinary medicine, oral administration of opioids do not seem to produce consistent and sufficient clinical effects. In addition, opioids are controlled drugs with limited availability world-wide and potential for abuse. Their use for cancer-related pain should be limited to the hospital setting. On an outpatient basis, opioids are not recommended for cancer-related pain (8).

Non-steroidal anti-inflammatory drugs are a viable treatment option for cancer pain, unless there are contra-indications. They exert their effects via inhibition of the expression of cyclooxygenase (COX) enzymes in cell membranes and are excellent analgesics for inflammatory pain. Close monitoring of adverse-effects is imperative (6).

Amitriptyline is a low-cost tricyclic antidepressant largely used for the treatment of human neuropathic pain and has been recommended for use in veterinary patients with chronic cancer pain (1,7). Its analgesic effects result from the inhibition of the reuptake of serotonin and norepinephrine in the central nervous system. Gabapentin is an anticonvulsant drug with analgesic properties that are mediated via calcium channels (8). This drug is commonly recommended as an adjuvant for the treatment of neuropathic pain in humans (2,3).

Tramadol is a centrally acting analgesic that activates µ-opioid receptors and inhibits serotonin and norepinephrine reuptake. Its analgesic effects seem to be superior in cats when compared with dogs due to a higher production of its active metabolite in cats.

Amantadine exerts its analgesic effects by the antagonism of NMDA receptors and by increasing dopamine concentrations in the central nervous system.

All the aforementioned adjuvant analgesics may be considered for the treatment of cancer pain. Since they have different mechanisms of action, different drug associations may be used and chosen depending on the type of pain and treatment response. Nevertheless, little scientific evidence is available for their use in clinical cancer pain in small animals.

Bisphosphonates (pamidronate, zoledronate, etc.) may provide analgesia by inhibition of bone resorption due to osteoclast activity in patients with primary or secondary bone cancer.

Euthanasia comprises a treatment option and should be considered in cases with poor prognosis, impossibility to treat due to owner’s ability, financial constraints, lack of response to therapy, etc.

**Non-pharmacological Treatment**

The benefits of non-pharmacological treatment options should not be underestimated (2). Although there is lacking scientific evidence on the efficacy of these techniques in veterinary patients, their importance in the treatment of chronic pain in humans has been increasingly recognised as it decreases pain, suffering and anxiety in several patients.

Several non-pharmacological options exist and should be considered depending on the clinical case, availability, acceptance by the patient and the owner. These options include acupuncture, physiotherapy, environmental enrichment, massage, natural products, among others.

Finally, continuous ‘tender loving care’ is essential for cancer patients. It will play a role in the patient’s welfare and will strengthen the human-animal bond.
References:
Chronic Pain in Cats: From Recognition to Treatment
Beatriz Monteiro
DVM, Vanier Scholar, 5th year PhD candidate

Introduction
Maladaptive or chronic pain is defined mostly based on its origin and underlying mechanism of disease. It is not associated with healing and persists beyond the expected course of an acute disease process with no clear end-point. Chronic pain should not be considered a symptom, but rather a disease in its own right since it may be present in the absence of a primary cause and the degree of pain does not necessarily correlate with the pathology observed. It infers that neuroplastic changes in the spinal cord and brain will lead to an abnormal sensory processing resulting in persistent pain states and the generation of pain within the central nervous system.

Common Causes of Chronic Pain in Cats
As veterinary care advances and cats live longer, there has been an increased recognition of chronic pain associated with certain conditions. These include osteoarthritis (OA), neoplasia, skin conditions (e.g. otitis, burns, chronic wounds), oral conditions (e.g. gingivitis, stomatitis), ocular conditions (e.g. corneal ulcers, glaucoma), gastrointestinal conditions (e.g. megacolon, constipation, inflammatory bowel disease), urogenital conditions (e.g. interstitial cystitis), persistent post-surgical pain (chronic pain syndrome after feline onychectomy, amputations, thoracotomy), diabetes-induced neuropathy, feline orofacial pain syndrome, among others (1).

Clinical Presentation
In cats with long-term pain, clinical signs will vary according to the origin of pain but may be subtle and unpertinent (1,2). At the clinic, the cat will likely not exhibit chronic pain-induced behaviors that were expressed at home and the cat’s responses to painful stimuli will be affected by where and with whom they are. In this sense, owner assessments are the mainstay of the assessment of chronic pain and quality of life. Common behavior changes include changes in temperament, decreased mobility and easiness of movement, altered eating and drinking, decreased grooming, increased urination and/or defecation directly over the edge of the litter box (3,4).

Clinical Metrology Instruments
Although there are no fully validated pain scales for cats with chronic pain, a few questionnaires have been developed to evaluate cats affected by OA. The Feline Musculoskeletal Pain Index (FMPI) (3) consists of a series of 19 set questions. It has demonstrated its ability to detect cats with musculoskeletal pain and detect treatment effects in correlation with objectively measured activity. The Client Specific Outcome Measures (CSOM) (6) selects 5 activities that are unique to that particular cat. These are chosen by the owner following discussion with the veterinarian and are graded according to the degree of impairment. The Montreal Instrument for Cat Arthritis Testing...
(MI-CAT(V)) (7) was developed to be used by veterinarians. It focuses on mechanical abnormalities and mobility impairments that can be evaluated at a distance.

**Considerations for Long-Term Treatment in Cats**
The environment in which the cat lives should be predictable, provide comfort, mental and physical stimulation, and interaction with other family members (8). Obstacles to long-term treatment exist including the general fear of drug-induced adverse-effects, individual variability, cost and compliance. Realistic expectations must be thoroughly discussed with the owners upon deciding the treatment program, financial constraints and client availability. Chronic administration of medications to cats is usually performed by the oral route which can become a welfare issue in some circumstances.

**Treatment of Chronic Pain**
Treatment of chronic pain often involves a multimodal approach including the administration of analgesic drugs and several non-pharmacological therapies (1,2). Opioids do not seem as effective when administered by the oral route or even the oral transmucosal route (9). NSAIDs are indicated for any type of chronic inflammatory pain (e.g. OA, cancer pain, gingivitis). It is the first-line treatment for cats with OA-related pain, and a strong body of evidence exists for the safe and effective use of NSAIDs in cats with this condition (10-12). The potential for the development of adverse-effects following administration of NSAIDs in cats should not be underestimated. Patients must be screened for pre-existing disease and monitored during treatment (13). Recent evidence shows that NSAIDs can be administered to cats with concomitant painful conditions and chronic kidney disease provided their overall clinical status is stable (1). Tramadol is a central analgesic indicated in conditions characterized by central sensitization. One study evaluated the effects of tramadol and placebo for two weeks in cats with naturally occurring OA. Treated cats showed increased levels of activity and decreased central sensitization when compared with placebo (14). Tramadol should not be administered in association with drugs that affect serotonin reuptake or metabolism due to the risk of serotonin toxicity. Other therapies are currently used in the management of chronic pain in cats including gabapentin, amantadine and amitriptyline. However, it is not clear what dosage regimens and when these therapies are indicated. The analgesic efficacy of anti-nerve growth factor (anti-NGF) monoclonal antibodies (mAbs) for the treatment of OA-relate pain has been recently reported in cats (15). These are species-specific Ab targeting NGF which has an important role in pro-nociception. In addition, recent research has indicated a potential efficacy of the use of autologous mesenchymal stem cell therapy for the treatment of severe refractory gingivostomatitis (16), an important cause of chronic pain in cats. Non-pharmacological treatment options include environmental enrichment for increase in physical activity and mental stimulation as well as weight control, physical therapy, massage, acupuncture, transcutaneous electrical nerve stimulation, among others.

**References:**
Companion Animal: Endocrinology
Cushing’s Disease: Something New, Something Blue
Dr. Anthony P. Carr, DACVIM

Introduction
Hyperadrenocorticism remains one of the most common endocrine disorders diagnosed in the geriatric dog population. It is a disease that is seen in almost every veterinary practice. Unfortunately, the disease tends to be frustrating to deal with, a definitive diagnosis is at times elusive and therapy can have major adverse side effects. Knowing how to diagnose, what problems hyperadrenocorticism can cause in a patient that justify aggressive therapy, and the advantages and disadvantages of the various treatment modalities can be helpful in determining an appropriate diagnostic and therapeutic plan.

Clinical Signs
The most common clinical signs of Cushing’s disease are quite familiar to practicing veterinarians. Commonly PU/PD, pot-bellied appearance, lethargy, polyphagia, obesity, and panting. In addition, many dermatologic manifestations are seen including alopecia (of the trunk), comedones, thin skin, calcinosis cutis, bruising, hyperpigmentation, pyoderma and seborrhea. Less commonly identified signs include muscle weakness or pseudo-myotonia (“frozen” muscles), polyneuropathies, or rupture of the cranial cruciate ligament. Hypertension is relatively common (50% or greater) though with the lack of good blood pressure monitoring devices in many practices it often goes undiagnosed. Pulmonary thromboembolism, recurrent (often asymptomatic) urinary tract infections, proteinuria, pancreatitis, pulmonary mineralization, gallbladder mucoceles and calcium oxalate urolithiasis are also often frequently seen with Cushing’s disease. Recently it has been noted that many dogs with hyperadrenocortism are hypoxic, whether or not they have mineralization of the lung parenchyma. This can be a serious consequence, leading to distress as well as excess strain on the right side of the heart. Some of the clinical problems caused by Cushing’s disease are more bothersome than dangerous. Other clinical problems are life threatening such as thromboembolism or pancreatitis. Still other clinical problems can aggravate other disorders that the patient may have, such as is the case if hypertension is present in a dog with underlying heart disease. This is not an uncommon scenario since older dogs tend to have Cushing’s as well as valvular heart disease. Hypertension in a dog with valvular problems can be a factor that leads to more rapid progression of the heart problem as well as difficulties in treating heart failure if it occurs.

Diagnosis
One of the most frustrating parts of Cushing’s disease is trying to establish a definitive diagnosis. Ideally of course clinical signs should be consistent with hyperadrenocorticism. Basically testing is still divided into screening tests and test to differentiate between pituitary dependent hyperadrenocorticism (PDH) and adrenal tumors (AT). There are differences between labs, so it is advisable to contact them if there are any questions regarding the outcome of testing. A random cortisol level has no diagnostic value in regard to the diagnosis or exclusion of Cushing’s disease. The simplest screening test is a urine cortisol to urine creatinine ratio. False positives occur frequently, however false negatives are rare. As such it is a good test to rule out Cushing’s. Even the stress of a visit to the veterinarian will elevate the values and as such it is advisable to have the
owner collect the urine prior to presentation at the clinic. Because there are so many false positives, a follow-up test such as a low dose dexamethasone suppression test (LDDS) or ACTH stimulation test should be run. These two tests have advantages and disadvantages. The LDDS has some false positives but few false negatives. It can also lead to differentiating between PDH and AT when 4 and 8-hour samples are taken, one of the reasons it is my preferred screening test. If the cortisol levels drop at 4 and escape to above normal range at 8 hours PDH is present. The ACTH stimulation test has few false positives, but false negatives do occur, especially with adrenal tumors. The combined ACTH stimulation /LDDS test is generally not recommended.

Differentiation tests also have their drawbacks. Not all dogs with PDH will suppress on a high dose dexamethasone suppression test. An endogenous ACTH level is a very good test, however the sample needs to be meticulously handled (contact your lab) which often makes it difficult to run. Abdominal ultrasound currently is the most common way to differentiate between adrenal tumors and pituitary dependent disease. At times it is not possible to completely rule out an adrenal tumor.

**Therapy**

There have been some changes in regard to what therapies to consider. Some will probably never become important methods. One method that is interesting but unlikely to be commonly used is surgery for PDH. It is possible to remove the pituitary tumor via transspenoidal surgery. This is of course more likely to be curative since it eliminates the underlying cause of the PDH. Obviously a learning curve exists and it is not widely practiced. The same applies for radiation therapy, which is used when there are indications that the pituitary tumor is actually a large macroadenoma, which would eventually cause neurologic signs. To diagnose this it is necessary to have imaging such as MRI or CT available. Medical therapy still remains the most commonly used form of therapy and will probably remain so long term.

There are 3 major medications to consider for medical therapy; mitotane, L-deprenyl and trilostane. Each of these treatments has advantages and disadvantages. It is however important to know when to treat. Just because Cushing’s has been diagnosed does not mean that therapy is necessary. If clinical signs are absent it is difficult to justify a potentially dangerous and/or expensive therapeutic plan. If problems such as pseudo-myotonia, hypertension, proteinuria, and others are present treatment is clearly indicated and it warrants aggressive management. If the signs are more related to owner noted effects it may be possible to treat less aggressively.

Currently our treatment of choice is trilostane. Trilostane is a reversible treatment of Cushings disease. Side effects can occur but are usually reversible, though rarely adrenal cortical necrosis can occur.

Mitotane is a very effective therapy though side effects are common. Fortunately the side effects can usually be managed, however in rare instances life threatening problems can occur including making the patient a permanent Addisonian dog. Unfortunately, the adverse effects cannot be predicted. It has been helpful to give physiologic doses of prednisone (0.2 mg/kg SID) to dogs during induction; it seems to make this process smoother. ACTH stimulation testing is required to determine efficacy, the lack of response to ACTH is the goal of therapy. Maintenance therapy is needed, however in about 1 year 50% of dogs are Cushingoid again.
L-deprenyl was initially considered a good alternative to mitotane. It has however been shown that although the majority of dogs do better clinically, the objective effects on the Cushing’s disease are only present in 20% of patients.

The currently most common treatment is trilostane. Trilostane is a reversible inhibitor of 3b-hydroxysteroid dehydrogenase-isomerase. It can be given both once or twice daily, whereby in general animals that are treated with BID dosing tend to respond much better. Total daily dose tends to be similar. With trilostane therapy, therapeutic monitoring is indicated. This is generally with an ACTH stimulation test. However it is important to know that clinical response as assessed by the owner may be very discordant in comparison to the results of blood tests. When doing therapeutic monitoring it is important to be consistent with regard to how much time elapses from the time the dog is pilled to when the ACTH stimulation test is started. When dogs tested 2 hours after pill were compared to 4 hours after pill most dogs were lower at 2 than at 4. It also has been found that in dogs with pre and post ACTH cortisols in the addisonian range (<2 ug) had much higher stims 9 to 12 hours post-pill. When doing monitoring of trilostane therapy it is possible to use a much lower dose of ACTH (1 ug/kg) than what is used to diagnose Cushings (5 ug/kg).

Trilostane has highlighted the significant issues with compounded medications. A study published in JAAHA in 2012 looked at compounded trilostane. The medication was purchased from 8 pharmacies, with multiple orders from the same pharmacy. Almost 40% of the compounded drugs were considered unacceptable. Concentration of drug was highly variable, anywhere from 39 to 152% of the amount that should have been present, meaning that if you ordered a 45 mg capsule the actual amount of drug could be anywhere from 18 to 68 mg. Some of compounded products also did not have adequate dissolution meaning they probably would not be absorbed. Differences were seen within pharmacies as well as between pharmacies.

A small proportion of dogs have adrenal tumors. In these cases, therapy via surgery is preferred. If this is however not possible medical therapy can be attempted. Results for mitotane and trilostane are similar (15.6 vs. 14 months median survival). Poorer response is seen in older dogs and in dogs with weakness as a presenting sign.

References:
Available upon request from the author.
Hypoadrenocorticism
Dr. Anthony P. Carr, DACVIM

Problems
- Weakness
- Lethargy
- Vomiting
- Diarrhea
- Dehydration
- Hypothermia
- Melena
- Hyperkalemia
- Hyponatremia
- Hypoalbuminemia
- Anemia (non-regenerative)
- Hypercalcemia
- Azotemia with isosthenuria
- Bradycardia
- Hypotension
- Shock

Overview
- Hypoadrenocorticism (or Addison’s Disease) can be a life threatening emergency.
- Predominantly results from autoimmune destruction of the adrenal glands (primary hypoadrenocorticism) or rarely as a result of insufficient ACTH production (secondary hypoadrenocorticism) by the pituitary gland. It can also be caused iatrogenically by treatment of hyperadrenocorticism.
- Clinical signs develop because of a deficiency of glucocorticoids or mineralocorticoids or both.
- When glucocorticoids production alone is absent, it may also be termed atypical Addison’s disease.
- A marked predisposition for young female dogs and certain breeds such as Standard Poodles, Great Danes, Portuguese Water Dogs, Bearded Collies, Rottweilers, West Highland White Terriers and Wheaten Terriers is present.
- The disease does occur in cats but is relatively rare.

Clinical Signs
- Typical vs. atypical Addison’s: Mineralocorticoids play a vital role in regulating electrolytes and fluid balance. Without aldosterone (main adrenal mineralocorticoid) potassium is no longer excreted adequately while increased loss of sodium occurs. The lack of sodium results in inadequate circulating blood volume. Hypovolemia, hyperkalemia and hyponatremia are all signs of mineralocorticoid deficiency. Glucocorticoid deficiency results in weakness, GI hemorrhage and hypoglycemia (decreased gluconeogenesis in the liver, increased insulin receptor sensitivity). In addition, hyponatremia may occur in this...
situation as well as a result of an impaired ability to excrete free water (ADH release is not counteracted by glucocorticoids).

- General: Because of the varied clinical signs present with hypoadrenocorticism it can be at times difficult to establish a diagnosis. Lethargy is common as are weight loss, anorexia and weakness. Clinical signs often respond to basic supportive care so that there may be a delay in establishing a definitive diagnosis.

- Renal: Without aldosterone, potassium is not excreted and sodium is lost excessively. Hypotension and hypovolemia are common and these result in decreased renal perfusion. Because of this pre-renal azotemia is common. With continued hypoperfusion true renal failure can occur. Urine specific gravity is low, this may be a result of renal medullary washout.

- Cardiovascular: The predominant cardiac manifestations of hypoadrenocorticism are related to hyperkalemia and hypovolemia because of hyponatremia. Hypotension may also be partially a result of decreased vascular catecholamine receptors. A patient with hypoadrenocorticism is often presented hypotensive and in shock. The expected compensatory response would be tachycardia, however many times a bradycardia is detected. The EKG will often show changes consistent with hyperkalemia such as lack of visible P-waves and tall and spiked T-waves.

- Gastrointestinal: Commonly GI problems are a presenting complaint for hypoadrenocorticism. A great majority of dogs have vomiting and/or diarrhea. Hemorrhage into the GI tract can occur. This may be because glucocorticoids are needed to maintain mucosal integrity. Decreased perfusion would also predispose to development of GI ulcers.

- Megaesophagus: This is a relatively rare manifestation of hypoadrenocorticism. It is more significant in dogs with atypical disease where it can be a major presenting complaint and a potential source of severe complications such as aspiration pneumonia.

- Neurological: Occasionally hypoglycemia can be one of the major presenting problems noted. Weakness and trembling can occur, in more severe cases seizures are noted.

Routine laboratory tests
A variety of abnormalities can be detected in hypoadrenocorticism, whereby none are specific enough to allow a definitive diagnosis.

- A non-regenerative, normochromic, normocytic anemia is often present. The anemia can result from GI hemorrhage or may also be because ACTH and adrenocortical hormones influence release of erythropoietin.

- Hypoalbuminemia is common. This may be from increased GI blood loss, protein losing enteropathy or decreased synthesis.

- Hyperkalemia and hyponatremia are common. These can occur with other diseases such as renal failure, chylous effusions, and GI disease (especially whipworm associated).

- Liver enzyme elevations have occasionally been documented, this could be the result of poor perfusion.

- Hypercalcemia is seen on occasion, the etiology is unclear at this time.

Diagnostic imaging
• Diagnostic imaging rarely is of great benefit in this disease since findings are non-specific though changes are common.

Specific tests for diagnosis
• Hypoadrenocorticism is diagnosed by the lack of response to an ACTH stimulation test.

Treatment Recommendations
• Objectives: When dogs are presented in an Addisonian crisis, it is essential that appropriate interventions be instituted rapidly. In severe cases the animals are in shock and can easily go on to develop organ failure, especially renal failure. Long term therapy focuses on maintaining a normal sodium to potassium ratio and preventing problems stemming from glucocorticoid deficiency such as weakness, GI signs and inability to deal with stress.

• Treatment: In the initial management of an Addisonian crisis fluid therapy is needed to reestablish perfusion and treat the hypovolemia. Crystalloid fluids such as LRS or 0.9% NaCl are appropriate for this. This will also help to decrease potassium values. If hyperkalemia is life threatening, sodium bicarbonate can be used. Dextrose supplementation can also help to decrease potassium values as well as helping to combat hypoglycemia if symptomatic. Glucocorticoid administration is generally needed, dexamethasone is ideally suited for this. Initial dosage is 1 to 4 mg/kg i.v. Mineralocorticoid supplementation can take the form of tablets (fludrocortisone acetate at 0.02 mg/kg daily) or injections (DOCP 1-3 mg/kg every 3 to 4 weeks).

• Monitoring: Sodium and potassium values should be monitored regularly. Once stable on medications every 3 to 6 months is sufficient.

• Complications: Supportive care and administration of glucocorticoids and mineralocorticoids will often successfully treat the pet presented in an Addisonian crisis. Very rarely have complications been noted with fluid therapy. As a result of the development of chronic hyponatremia, the brain compensates by decreasing intracellular osmolality (changes in inorganic ions and amino acids, so called idiogenic osmoles). The amino acids can only translocate slowly so it is possible for a significant diffusion gradient to develop. With correction of hyponatremia the cells dehydrate. This has been documented infrequently and occurs 3 to 4 days after treatment is initiated. Myelinolysis occurs with depression, weakness, ataxia spastic quadraparesis. The recommendation is to correct sodium <12 mEq/L/day.
Overview
Hyperthyroidism is a common disease of cats over 6 years of age. Feline hyperthyroidism is caused by adenomatous hyperplasia or adenoma of the thyroid gland; carcinomas are rare. The disease is bilateral in 70% of cases. The cause remains unknown. Clinical signs can vary depending upon which stage the disease is identified. Weight loss is present in about 90% of cases with polyphagia in about 50% of cases. Cats can become hyperactivity, anxious, and are prone to this especially when being examined by a veterinarian. Most cases have a thyroid slip.

Other signs:
- Polyuria and polydipsia
- Heart murmur or gallop rhythm
- Tachycardia
- Systemic arterial hypertension is common.
- Vomiting
- Diarrhea or increased fecal volume
- Panting or dyspnea
- Generalized weakness can occur because of impaired muscle function.
- Cervical ventroflexion is occasionally present and may be due to myopathy, hypokalemia, or thiamine deficiency.

In rare cases apathetic hyperthyroidism can occur with decreased appetite, weight loss and severe lethargy.

Routine laboratory tests
- Elevated liver enzyme (ALT, AST, alkaline phosphatase) activity is commonly present.
- BUN and creatinine are elevated in some cats, this is generally related to concurrent renal disease and not the hyperthyroid condition. The presence of this increases risk of treatment for the hyperthyroid state as more severe renal dysfunction may be unmasked.

Diagnostic imaging
Thoracic radiographs:
- Indicated when dyspnea, tachypnea, or muffled heart and lung sounds are present.

Ancillary testing
Electrocardiography is indicated when an arrhythmia is suspected.
Arterial blood pressure should be measured in all cases.

Specific tests for diagnosis
Hyperthyroidism is usually readily diagnosed by documenting elevated serum total T4 concentration. Normal serum T4 can be seen in hyperthyroid cats with mild hyperthyroidism, hyperthyroidism with a concurrent nonthyroidal illness, or a disease other than hyperthyroidism. If the total T4 is normal
and hyperthyroidism is still suspected, serum T4 should be measured again in 1-4 weeks. Alternatively free T4 with a dialysis procedure can be run. This is a more sensitive test (98% vs. 90% with just T4). Falsely elevated fT4 can be seen in cats with nonthyroidal illness.

Thyrotropin releasing hormone (TRH) stimulation test
As effective as the T3 suppression test in diagnosing hyperthyroidism in difficult cases.
Protocol:
Obtain blood sample for serum T4 concentration before and 4 hours after IV administration of 0.1 μg/kg TRH.
Interpretation:
Normal response is an increase in T4 on the 4 hour sample > 60% of the basal concentration. Hyperthyroidism is diagnosed when the serum T4 concentration is < 50% of baseline.

T3 suppression test
Protocol:
Obtain blood sample prior to initiating test for measurement of serum T4.
Administer T3 at 25 μg PO q 8 hours for 7 doses.
Obtain blood sample 2-4 hours after the final dose of T3 for measurement of serum T4 and T3 concentrations.
Interpretation:
Normal response is a decrease in T4 concentration to < 20 nmol/L. Hyperthyroidism is diagnosed when the serum T4 is > 20 nmol/L post-T3 administration. Serum T3 should be elevated on the post-treatment sample unless the T3 was not administered properly.

Treatment recommendations

Antithyroid drug treatment
Methimazole or carbimazole are the drugs of choice for medical management of hyperthyroidism. Methimazole inhibits synthesis of thyroid hormones. Methimazole should always be used prior to a more permanent treatment in order to assess the effects that resolution of hyperthyroidism has on renal function. Methimazole should be administered initially at 5 mg/day in a single dose or divided twice per day. Compounded methimazole has been shown to be variable in dosage and in some cases would probably not be absorbed.

Complications do occur with these drugs, some of which respond to dosage decreases or gradual reintroduction, others necessitate stopping the medications permanently. GI side effects (vomiting, diarrhea, anorexia) are the most common signs seen. Blood dyscrasias (thrombocytopenia, agranulocytosis, hemolytic anemia) occur rarely but are life-threatening. Other severe side effects include pruritus and hepatopathies. All the severe side effects necessitate stopping the medication permanently.

Monitoring treatment is vital given the chances of complications from these drugs. A CBC should be evaluated every 2 weeks for the first 3 months of treatment to monitor for neutropenia, anemia, and thrombocytopenia. Renal function should also checked every 2 weeks until serum T4 concentration has decreased into the normal range for at least 2 weeks.
When methimazole is poorly tolerated, the most effective alternative with the fewest side effects is ipodate or ipanoic acid, unfortunately these are generally not available. These oral contrast agents reduce T3 without changing T4. Administer 50 mg twice per day. These only work with mild hyperthyroidism and can stop working at a later date.

**Radioactive iodine**
Treatment of choice if available and affordable. Ideally only abnormal tissue will be destroyed, normal tissue should be spared. Equally effective if abnormal tissue is not located in the thyroid gland. Can also be used with adenocarcinomas (higher dose required). Cats do have to be relatively stable clinically to be treated and need to eat in clinic if treatment is to occur. Most cats are euthyroid within 1-2 months of treatment.

**Percutaneous ethanol injection:**
Has a learning curve, so needs experienced operator. Only one thyroid gland should be treated at a given time even if both thyroid glands are enlarged; the contralateral gland should be treated at a later date. Results with treatment of cats with unilateral involvement has been good, while hyperthyroidism has uniformly reoccurred in those with bilateral disease. Laryngeal paralysis is a common complication, which may be permanent or transient; bilateral laryngeal paralysis may be fatal. Horner’s can also occur.

**Surgical thyroidectomy:**
An effective, permanent treatment of hyperthyroidism with bilateral thyroidectomy being recommended given that 70% of cases have bilateral disease. Surgical removal can be staged if needed. Hypoparathyroidism can occur. Because of this calcium concentration should be monitored daily for 3 days after the procedure. Levothyroxine supplementation (0.1 mg QD) is recommended for 2 months following bilateral thyroidectomy. Laryngeal paralysis and Horner’s syndrome occur rarely due to intraoperative trauma.
**Gastric ulceration**

Prevention is always preferable to treatment. The same applies for gastric ulcers/erosions. In most cases this is of course not possible. One exception is ulceration associated with NSAID administration. The prostaglandin analogue misoprostol (3 to 5 µg/kg q 8h) has been shown to prevent ulcers in humans treated with NSAIDs. Proton pump inhibitors also show promise with this, whereas H2 blockers are not considered efficacious.

A variety of medications are available for treating erosions/ulcers, the majority work by suppressing stomach acid secretion. Both H2 receptor antagonists and proton pump inhibitors have been used in dogs and cats. It is not uncommon to see gastric acid suppressing agents being combined with sucralfate for the treatment of erosions/ulcers. This does not appear justified since in humans the combination is no more effective than using a single agent.

The H2 blockers bind to receptors on the acid producing parietal cells. This renders the cells less likely to respond to histamine, gastrin or acetylcholine. Commonly used H2 blockers used include cimetidine, famotidine and ranitidine. Ranitidine also has prokinetic effects in the GI tract that make it an antiemetic as well. Studies in dogs suggest that these agents do not suppress stomach acid production adequately. Proton pump inhibitors are more effective at significantly decreasing gastric acid secretion since the binding of the drug to the parietal cell is irreversible and inhibits stomach acid secretion. Omeprazole (0.2 to 0.7 mg/kg daily) has been used in dogs and is highly effective.

Sucralfate is a mucosal protectant that is often used with GI erosions/ulceration. It binds to defects in the mucosa, protecting the damaged area. Production of prostaglandins is also increased which results in increased mucous and bicarbonate production. Many additional effects have been documented as well.

**Promotility agents**

A variety of purported prokinetic medications are available, though evidence that they work in a clinical patient are limited. Metoclopramide (0.2-0.4 mg/kg q 8h) can be used and has been shown to be a prokinetic in healthy dogs. It has central antiemetic effects as well as speeding gastric emptying. A more potent effect with regard to gastric emptying can be achieved with cisapride (0.1 to 1.0 mg/kg q 8-12h). Erythromycin at low dosages will also promote gastric emptying by stimulating motilin receptors in the GI tract (0.5 to 1.0 mg/kg q 8h). Oral ranitidine and nizatidine also have prokinetic effects because they inhibit acetylcholinesterase activity thereby increasing parasympathetic tone.

**Antiemetics**

Antiemetics are commonly used in veterinary practice. By reducing nausea they improve the condition of the patient. They also reduce loss of fluids and electrolytes caused by persistent vomiting. Antiemetics can act at various receptors. Some are specific to individual receptors whereas others may influence multiple receptors. In patients response to a specific antiemetic can be
variable so that various agents or combinations may be needed to achieve the desired therapeutic benefit.

Metoclopramide is a commonly used antiemetic. It can be given via intermittent subcutaneous injections (0.2 –0.4 mg/kg q 6 h, SQ, IM) or via a constant rate intravenous infusion (1-2 mg/kg/day). The latter appears to be more efficacious. This medication predominantly affects the D2 dopaminergic receptors in the CRTZ and gut. It also affects the 5-HT3 serotonergic receptors in the CRTZ.

Phenothiazines such as chlorpromazine (0.2 – 0.4 mg/kg q 8h SQ) or prochlorperazine (0.5 mg/kg q 8h SQ or IM) are broad spectrum antiemetics with activity at the α2-adrenergic, D2-dopaminergic, histaminergic, and cholinergic receptors. These medications are a good choice in those patients that fail to respond to metoclopramide, it is possible to use both agents concurrently. They can cause hypotension and this should be monitored for. Sedation is also usually quite pronounced.

A limited number of medications are specific to the 5-HT3 serotonergic receptors. Ondansetron (0.5 – 1.0 mg/kg q 12 to 24h PO) can be helpful in some cases of vomiting associated with stimulation of the CRTZ.

H1-histaminergic receptor antagonists include diphenhydramine (2 – 4 mg/kg q 8h PO) and dimenhydrinate (4 – 8 mg/kg q 8h PO). These can be used for the treatment of motion sickness or vestibular disease.

Erythromycin at low dosages (0.5 – 1.0 mg/kg q 8h) can also be an antiemetic by stimulation of the motilin receptors that increase GI motility and promote gastric emptying.

Maropitant is a veterinary specific antiemetic that is widely used. It is a NK 1 receptor antagonist. It is approved for use in dogs older than 8 weeks of age and for 5 consecutive days. Efficacy is good, probably as good as most other antiemetics.

References:
Available upon request from the author.
Pancreatitis in Cats
Dr. Anthony P. Carr, DACVIM

Introduction
Pancreatitis is a common problem in cats and as with most things, cats are not small dogs. Clinical signs and outcomes tend to be different between the two species. As with dogs, pancreatitis needs to be on the differential list for most cats with signs of gastroenteritis. Diagnosis is not always straightforward, though this generally does not have a major impact on therapy as therapy for gastroenteritis and pancreatitis are very similar in cats.

Signalment and risk factors for pancreatitis
Most cats with pancreatitis are middle age or older. There is an association between GI disease, hepatic disease and pancreatic disease that is termed "triaditis" by some authors. This is based on a research paper that found an association with these diseases, however it was very specific forms of disease. Making a global association between these various diseases is not supported by scientific evidence.

Clinical Signs
Clinical signs are variable, though generally less severe than in dogs. Anorexia is common as is lethargy. Pancreatitis is a common cause of hepatic lipidosis, so that the signs of HL may predominate. Pain on abdominal palpation is also much rarer. With chronicity, weight loss can occur. In cats necrotizing pancreatitis can also have a very rapidly fatal course, though clinical signs also tend to not be very specific.

Diagnosis
There is no test that says a patient does or does not have pancreatitis. This is especially true since many other diseases can have pancreatic involvement without pancreatitis being the major issue (i.e. foreign body).

Imaging: Radiographs can show non-specific changes. Ultrasound is better, but sensitivity and specificity can be quite variable (usually severe cases can be diagnosed, but with mild cases common degenerative changes to the pancreas can mimic pancreatitis).
Laboratory Testing: The results of routine blood and urine analysis are not diagnostic, though at times they can help to assess severity (especially the CBC) and if concurrent issues such as diabetes are present. There may also be indications of cholestasis present. In cats hypocalcemia can be a clue to pancreatitis.

Amylase and Lipase: Sensitivity and specificity are very poor in cats and as such these analytes are of no value in the diagnosis of pancreatitis.

TLI: The TLI assay is not sufficiently useful to rely upon to rule pancreatitis in or out.
fPLI/fPL: This test can be useful in diagnosing pancreatic disease, however the test is not specific nor necessarily sensitive (depending on cut off around 60 to 80% for sensitivity and specificity). It is also important that a positive test not be interpreted in a way that stops the clinician from looking for
other reasons for the clinical signs present (i.e. GI foreign body) as this could result in major issues not being diagnosed.

**Treatment**
There is no specific therapy for pancreatitis, the therapy that is needed is extremely good supportive care. Fluid therapy is a major part of therapy for pancreatitis. By providing fluids, acidosis and hypoperfusion are limited which can contribute to progression of disease. Buffered crystalloids are generally preferred over other crystalloids. A fluid therapy plan should of course address any dehydration present to minimize the impact of hypoperfusion, especially to organs sensitive to this such as the kidney and GI tract. In those patients where peripheral edema becomes an issue or where blood pressure cannot be maintained with crystalloids, colloids such as Pentaspan or Hetastarch can be used.

Anti-emetic therapy is important in those cases where ongoing vomiting or nausea are major issues, though this is rarer in cats.

Analgesia is an important part of treating pancreatitis in cats that show signs of pain. Pain severity will be variable and as such analgesic protocols should be adjusted. Buprenorphine is a good choice in many cases.

The use of prophylactic antibiotics in humans and dogs with pancreatitis is controversial. In cats there is little indication for their use unless an infection is diagnosed (i.e. aspiration pneumonia). Nutrition is vital in cats with pancreatitis as they can not infrequently develop hepatic lipidosis if anorexic. Appetite stimulants rarely are efficacious enough to consider them as a viable option. Feeding tubes are generally the preferred way to provide nutrition. Given that cats can be quite unstable initially, placement of a naso-esophageal feeding tube is often the first step to meeting the nutritional needs of cats with pancreatitis. This allows feeding of a liquid diet. CRI administration is better than bolus administration as it seems that tolerance to feeding is better (less nausea and vomiting). In many cases this is adequate to allow the cats to stabilize and start to eat on their own. If signs are persistent, placement of an esophageal or gastrotomy tube can be considered for longer term therapy, especially if the patient is to be discharged from the hospital.

**References:**
Available on request from the author.
CKD and Stomach Ulcers
Increased stomach acid leads to stomach ulcers
Gastrin controls stomach acid
Proven
Gastrin is excreted by kidney so gastrin concentration goes up
Proven
Therefore ipso facto CKD leads to hyperacidity, stomach ulcers and we need to reduce acid production
Ulcers seem appropriate given GI clinical signs with CKD not uncommonly
Vomiting
Inappetance
Upper GI bleeding
Dogs study from 1979 looked at 4 dogs
Ulcers not found
Mineralization and edema found, atrophy of gastric glands (so how is acid produced??) as well as a vasculopathy
Peters et al, JVIM 2005
28 dogs that went to necropsy with a diagnosis of CKD at Cornell
7 control dogs without CKD or GI problems
9 known to be chronic, others less clear if chronic or not
Vasculopathy and mineralization were inversely related
In dogs renal disease commonly has GI pathology however not ulcers, mainly it is a vasculopathy
Even though vasculopathy, necrosis with ulceration uncommon (1 dog)
Gland atrophy common so unsure if stomach acid can be produced
Conclusion
In dogs signs of uremic gastropathy can occur, however ulcers don’t very often
Routine use of stomach acid suppressants is not indicated, use only if signs of upper GI bleeding or esophagitis

What about in cats?
Case series of 8 cats with ulcers, none had CKD (Liptak, JFMS, 2002)
2008 Ciancio et al, JAVMA
70 cats with renal failure from melamine contaminated food
14 died or were euthanized
3 had gastric mineralization
Involved 37 CKD cats, 12 controls
Looked at necropsy information
Cats were grouped based on severity of azotemia: mild (Scr: 1.6–2.8 mg/dL), moderate (Scr: 2.9–5.0 mg/dL), and severe (Scr: >5.0 mg/dL)
9 mild, 9 moderate, 18 severely azotemic
The higher that calcium phosphorus product the more likely mineralization was present
Edema and inflammation were not present which is different from humans and dogs
GI signs seen do not seem to be related directly to GI pathology
In this study 84% had a history of inappetance and 45% of vomiting
Conclusion in cats
CKD is commonly associated with GI signs
These signs are not linked to ulcers in almost all cases, therefore the routine use of anti-ulcer medications is not indicated
Treatment for GI signs should address symptoms present, i.e. vomiting, nausea, inappetance which may be central in origin
Medication that is used to stimulate appetite and suppress vomiting, especially in cats
Initial published study by Quimby in Vet Q 2013
Tetracyclic antidepressant (5HT3 antagonist)
Anti-emetic, appetite stimulant, anti-nausea
Initial study was a double masked placebo controlled cross-over study
11 cats with CKD and poor appetites or weight loss
Either 1.88 mg of mirtazapine (1/8th of a 15 mg tablet compounded by CSU pharmacy) or placebo every 48 hours for 3 weeks, washed out for 4 days, then switched to other treatment

Mirtazapine Toxicity

Abstract presented at 2014 ACVIM Forum, Ferguson, McLean, Quimby
Retrospective analysis of ASPCA calls on mirtazapine toxicity from 2006-2011
A total of 104 cases were identified
Most common dosage 1/8th or ¼ of a 15 mg tablet
After exclusions a total of 78 cases studied
Most common side effects were
Vocalization (58%)
Agitation (33%)
Vomiting (28%)
Others included tremors, ataxia, restlessness, hypersalivation, tachypnea, tachycardia
Side effects occurred in 25 cats given 3.75 mg, only one that was given 1.88 mg.
Accidental toxicity more likely if owners given whole tablets (owners didn’t split pills or not appropriately)

Quimby study
Monitored weight, BCS, activity level, behavioral changes, QOL scores, and lab work
During the trial 91% of cats on mirtazapine gained weight, 82% lost weight while on placebo (up to a pound gain, up to ½ lb loss)
BCS increased in 45%
No adverse effects
Appetite scores improved in 91%
Activity score increased in 55%
Definite positive effect on quality of life which means less likely to euthanize
Those cats whose BCS did not increase already had a good BCS
One cat did have an increase in ALT (700+), normalized after stopping medication, owner elected to continue on drug, ALT went up but cat was fine
CKD and Maropitant
ACVIM Forum 2014, Quimby et al.

41 cats enrolled in placebo controlled blinded study
Stable IRIS Stage 2 or 3 cats with inappetance and/or vomiting
Cats got either placebo or 4 mg of Cerenia daily for 2 weeks (0.6-2.9 mg/kg daily)
Monitored biochemical profiles, vomiting logs, appetite, activity, body weight, BCS
33 cats completed trial, 21 received drug, 12 placebo
Cerenia effective at controlling vomiting
In comparison to mirtazapine appetite, activity and body weight did not improve

Gastric Ulcer Therapy
Ulcers do occur as does esophagitis and then treatment is indicated
Among the most commonly not needed drugs used in veterinary medicine
If you do need to use them should be rational (vs. irrational) approach
Goal is generally pH > 3, better >4; in humans for GI ulcers should be >3 75% of the time, for GERDs >4 67% of the time
Proton pump inhibitors are most potent anti-ulcer medications
Thought to be delay in onset of effect (30% efficacy in 24 hours, maximal efficacy in 4 days)
Study by Tolbert et al, JVIM 2015
Looked at famotidine plus pantoprazole vs. pantoprazole alone in dogs
Famotidine given for rapid effect
Concerns that famotidine might decrease PPI efficacy since acidic environment is needed for PPI to become protanated and trapped in parietal cells
Not an issue in humans, in fact combo was better
Gave either famotidine and pantoprazole or just pantoprazole (1 mg/kg i.v.) q 12 hours for 3 days, washout 10 days, then crossover
Continuous pH monitoring via a Bravo pH capsule in 12 healthy dogs (recording every 6 seconds)
Combination had pH greater than 3 74% ± 19 of the time monitored, pantoprazole alone was 79% ± 17
Combination had pH > 4, 64% ± 23, pantoprazole alone was 68% ± 17
Vomiting and diarrhea occurred on occasion in both groups

Probiotics
What makes it a probiotic
Gets through GI tract
Colonizes GI tract
Has a health impact
Many probiotics do not meet these criteria
Rossi et al, ACIVM Forum 2015
10 cats with history of constipation, refractory to medical treatment, 3 diagnosed as megacolon, 7 as chronic constipation
No antibiotics for 1 month
Probiotics are promising in humans with constipation
Cats were given 200 billion bacteria orally for 90 days
Clinical, endoscopic and histopathologic exam at T=0 and after treatment
Histologically fewer interstitial cells of Cajal found vs. normal cats
After treatment considerable improvement in the Feline Chronic Enteropathy Activity Index (what’s that??)
Combination of gastrointestinal signs, endoscopic abnormalities, serum total protein, serum alanine transaminase/alkaline phosphatase activity, and serum phosphorous concentration

SF68 (Enterococcus faecium, Fortiflora) and Amoxi-Clav in Cats
ACVIM Abstract 2015 Forum
Camille Torres-Henderson et al.
With amoxicillin-clavanulate it is not uncommon to see vomiting, diarrhea and inappetance in cats
Double blind, placebo controlled trial
34 cats enrolled (research cats)
Initial monitoring period to exclude stress diarrhea cats
All got amoxi-clav BID for 7 days
Each day appetite, attitude, hydration, vomiting, and consistency of feces was monitored
Cats got either Fortiflora or the palability enhancer in the product
No vomiting during acclimation
With antibiotics 7 of the Fortiflora and 6 of the placebo cats vomited at least once
No difference between groups
Soft pile stools in 11 of the Fortiflora and 12 placebo cats
In the Fortiflora cats 61% had non-formed or puddle feces, whereas 86% of placebo had that
In the Fortiflora cats 61% had non-formed or puddle feces, whereas 86% of placebo had that
Only placebo cats had worst scores possible
68% of placebo stools were soft piles or worse vs. 44% on probiotic
Statistically significant difference
Overview
Can’t I just lance that? Learn to differentiate umbilical conditions in calves, including abscesses, urachal abnormalities, and hernias. In addition to diagnosis, we will cover medical therapy as well as surgical options and techniques for these conditions.

Objectives
• Review of umbilical anatomy
• Diagnosis
• Review of common conditions affecting bovine umbilicus
• Review of surgical techniques

Key Terms
• Omphalitis
  o Inflammation of the umbilical structures
• Omphalophlebitis
  o Inflammation of the umbilical vein
  o Omphalooarteritis
  o Inflammation of the umbilical vein
• Omphalourachitis
  o Inflammation of the urachus
• Patent urachus
  o Urachus fails to regress and continues to communicate with bladder. Animal able to pass urine through umbilical stalk.
• Richter’s (parietal) hernia
  o One wall of a luminal organ (most often abomasum) in hernia. Often nonreducible and may be mistaken for umbilical abscess.
• Urachal cyst
  o Urachus fails to regress and continues to communicate with the bladder. Animal does not pass urine through the umbilical stalk.
• Urachal remnant
  o Urachus fails to regress but no longer communicates with bladder

Common Clinical Conditions
• uncomplicated umbilical hernias
• umbilical hernias with subcutaneous infection/abscesses
• umbilical hernias with umbilical remnant infection
• umbilical abscesses/chronic omphalitis
• urachal cysts/ruptures

Anatomy
• Urachus
- Just one
- Connection between the bladder and allantoic sac
- Regresses completely

- 2 umbilical arteries
  - Branches of internal iliac artery
  - Regress to form round ligament of the bladder

- 2 Umbilical Veins
  - Combine into one structure upon entering the abdomen
  - Regress to form round ligament of liver (falciform)

**Key Clinical Diagnostic Points**

- **History**
- **Physical Exam**
  - Deep Palpation
    - This is easily performed in young calves particularly when they are in lateral recumbency. The umbilical mass should be palpated and evaluated for pain, signs of previous drainage, and reducibility. This is difficult in larger calves.

- **Ultrasound**
  - The scan starts at the naval and goes cranially toward the liver (right paracostal region) to evaluate the umbilical vein. Following evaluation of the umbilical vein, move back to the umbilical and follow the urachus and umbilical arteries caudally to the bladder. It may be difficult to visualize these structures as they leave the umbilicus. It is important to visualize the bladder as umbilical remnants may increase in size near the cranial pole of the bladder. A 3.5 mHz curvilinear probe is recommended, however a rectal probe may be appropriate in small calves.

### Mean diameter (mm) of umbilical structures in clinically normal calves over time from birth

<table>
<thead>
<tr>
<th>Location</th>
<th>24 hours</th>
<th>1 wk</th>
<th>2 wk</th>
<th>3 wk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vein within stalk</td>
<td>9.61 ± 4.41</td>
<td>2.61 ± 1.78</td>
<td>1.0 ± 1.52</td>
<td>n/a</td>
</tr>
<tr>
<td>Vein near stalk</td>
<td>17.67 ± 6.04</td>
<td>10.33 ± 5.05</td>
<td>7.89 ± 4.70</td>
<td>5.33 ± 4.56</td>
</tr>
<tr>
<td>Vein at midpoint</td>
<td>11.22 ± 3.60</td>
<td>7.56 ± 2.24</td>
<td>4.44 ± 3.40</td>
<td>1.22 ± 2.44</td>
</tr>
<tr>
<td>Vein near liver</td>
<td>10.44 ± 4.50</td>
<td>6.11 ± 3.86</td>
<td>2.78 ± 4.24</td>
<td>1.33 ± 2.83</td>
</tr>
<tr>
<td>Arteries</td>
<td>10.33 ± 1.80</td>
<td>8.94 ± 2.11</td>
<td>8.39 ± 1.92</td>
<td>6.82 ± 1.03</td>
</tr>
</tbody>
</table>

From Watson, et al.¹
Initial Treatment
What is diagnosis?
• Surgical fixation is recommended for hernias, urachal problems, or infection of internal umbilical structures.
• Infection of External Umbilicus
  o Drain/lavage external abscess
    ▪ This is only recommended for external abscesses without internal umbilical structure involvement. Ultrasound followed by aspiration of mass contents can aid in confirmation. This will result in fistula if Richter’s hernia is present, or enterotomy/evisceration if the diagnosis is not accurate.
  o +/- Hydrotherapy

Surgery
• Anesthesia
  o For field procedures, sedation with a high volume caudal epidural or local infiltration of lidocaine is recommended. Nasotracheal intubation is possible in these cases to provide protection for the airway.
    ▪ Sedation +/- high volume caudal epidural
      • Sedative combinations
        o Xylazine/butorphanol/ketamine
          ▪ Intramuscular: 0.05-0.2mg/kg xylazine, 0.025-0.05mg/kg butorphanol, 0.2-2mg/kg ketamine
        • High volume caudal epidural (2% lidocaine)
          o 0.15-0.4 cc/kg administered as caudal epidural (cattle < 300kg). Adding xylazine at 0.05mg/kg will extend the duration of the epidural. If this is done, intramuscular doses of xylazine must be reduced accordingly.
  o Complicated hernias, umbilical infections etc...
    ▪ General anesthesia recommended

• Pre-Op
  o Restrict feed (adults, weaned calves)
    ▪ Hay: 36-48 hours
    ▪ Grain: 24 hours
    ▪ Water: 12-24 hours
    ▪ Exception: Baby calves can be kept on milk up to time of surgery.
  o Antimicrobial
  o Anti-inflammatory

• Approach
  o The general approach is the same for hernias and umbilical remnants. Masses that are draining should be sutured closed prior to incision and final surgical preparation. In bulls or steers, the prepuce should also be sutured closed with a purse string that will be removed at the end of surgery. In heifers, an elliptical incision is made around the umbilical mass, taking care to leave enough skin for closure with as little tension as possible. The subcutaneous tissue is dissected around the umbilical mass, taking care not to penetrate the hernia sac or abscess. The white external sheath of the
rectus abdominis muscle will be exposed. A small body wall incision (large enough for one finger to be inserted in to the abdomen) is made just lateral to the mass, on the left side. This allows the surgeon to palpate for umbilical remnants and release any fibrinous adhesions that may be present. This body wall incision is then continued as an elliptical incision around the umbilical mass or hernia ring, taking care to avoid incising any infected structures that may be present. The incision may need to be extended to removed affected umbilical structures. Most commonly, the urachus is affected requiring caudal extension of the incision and potentially resection of the apex of the bladder. If this is necessary, the bladder is closed with 2-0 poliglecaprone 25 (Monocryl) in a double inverting pattern. Doyens should be used to prevent urine spillage from the bladder. Any holes in the omentum can be repaired with 2-0 poliglecaprone 25 (Monocryl). The rectus sheath is closed with polyglactin 910 (Vicryl) or polydioxanone (PDS) in size #0-2 depending on the size of the animal. If there is tension preventing easy closure, one to two near-far-near tension sutures are placed at the midpoint of the body wall incision. The remainder of the incision is closed using a cruciate or simple continuous pattern. The subcutaneous tissue is closed in a simple continuous pattern using absorbable, monofilament suture such as poliglecaprone 25 (Monocryl), taking care to eliminate dead space to prevent seroma formation. The skin is closed using 0.4-0.6mm polymerized caprolactam (Vetafil) in a Ford interlocking pattern. Sutures can be removed 10-14 days post-operatively.

Bulls/steers?
- Bull and steer calves present a logistical challenge due to the presence of the preputial orifice. Elliptical skin incisions can be used only in masses less than 3cm in diameter. The elliptic skin incision is only useful when the umbilical mass is less than 3 cm in diameter. In the majority of cases, the incision will need to be extended caudally, lateral to the prepuce. This allows the prepuce to be reflected to one side to avoid inadvertent penetration. This approach allows access to the caudal abdomen, including the bladder. Closure is performed as in the female, however there may be more dead space due to reflection of the prepuce.

Post-Op
- Antibiotics
  - Antimicrobial therapy should be based on initial diagnosis combined with surgical findings. For simple hernia repairs, a single dose of peri-operative antimicrobials is appropriate.
- Anti-inflammatory drugs
- Abdominal bandage
- Feed reduction
  - Animals are gradually re-introduced to forage over 3-5 days, to prevent excessive rumen fill and pressure on the incision.

Complications
- Swelling
- Seroma
- Abscess
- Re-herniation
Selected References:
The Bovine Abdomen: I’m In...Now What?
Amanda Hartnack, DVM, MS

Overview
A detailed review of bovine anatomy in both adult and juvenile animals with common indications for abdominal surgery covered. Will include a review of surgical techniques that can be performed in the field in both the standing and recumbent animal, and indications for referral.

Objectives
• Review of approaches to the abdomen in both juveniles and adults, including what structures can be visualized and palpated with each approach.
• Review of indications for abdominal surgery and what to expect intra-operatively.

Common Indications for Abdominal Surgery
• Cesarean section
• LDA/RDA/RVA
• Bladder/urachal remnant rupture
• Small intestinal obstruction
  o Intussusception
  o Volvulus
  o Intraluminal obstruction
  o HBS (hemorrhagic bowel syndrome)
• Cecal dilatation/volvulus/retroflexion
• Hardware disease
• Cryptorchidectomy
• Colic of unknown origin

Key Clinical Diagnostic Points
• History
• Physical Exam
  o Rectal Palpation
• Ultrasonographic examination
• CBC/Chemistry
• Abdominocentesis

Pre-Operative Treatment
What is diagnosis?
• Antimicrobial Therapy
• Anti-inflammatory therapy
• Fluid Therapy
  — Dependent on systemic status of animal

Surgical Approach
• Standing: In most, abdominal surgery can be performed standing in adult bovids with the use of local or regional anesthesia.
Right Side

- A right sided approach is appropriate for most clinical conditions and the most useful approach for an exploratory laparotomy in the standing animal.
- The following structures can be exteriorized from a flank incision
  - Abomasal pylorus and cranial duodenum
  - Descending duodenum
  - Most of the jejunum
  - Ileum
  - Cecum
  - Most of the spiral colon
  - Portions of the proximal and distal loop of ascending colon
- Most other structures can be palpated but not exteriorized, with the exception of the ascending duodenum, transverse colon, and portions of the distal loop of the ascending colon.
- A systematic approach to the abdomen should be taken. In normal animals, the first thing that should be seen when entering the abdomen from a right sided approach is the descending duodenum running horizontally and its associated omentum. When beginning the abdominal exploration, the caudal abdomen should be explored first, including the bladder, reproductive organs, and left kidney. Next, move to the cranial portion of the abdomen. The reticulum, diaphragm, and omasum can be palpated. The liver and gall bladder can be examined next, noting any irregularities including rounding of the liver edges and enlargement of the gall bladder. The gall bladder is variably enlarged in anorectic animals. The abomasum should be present along the right body wall, with the pylorus located at the level of the costochondral junction of the 9th and 10th ribs. The right kidney can be located under the last two ribs, dorsal to the descending duodenum. To examine the remainder of the intestinal tract, the omental sling must be pulled forward to access the supraomental recess. At this point, the cecum, spiral colon, ileum, distal flange of jejunum, and most of the rest of the small intestine can be examined. Following examination of the right side of the animal, the left side can be examined by going behind the omental sling and over the rumen.

Left Side

- A left sided approach is generally reserved for cesarean sections, surgery of the rumen, or abomasopexy. This approach does not allow a thorough exploration of the abdomen.

  - Recumbent
    - Ventral midline
      - This approach is most useful for cesarean section, particularly in the case of an emphysematous fetus.
  - Paramedian
    - A cranial right paramedian approach can be utilized for access to the reticulum, or correction of abomasal displacement or volvulus.
A caudal paramedian approach can be utilized to access the bladder or intra-abdominal testicles in cryptorchid animals.

- **Right Paracostal**
  - This approach can be utilized for access to the abomasum in adult cattle and calves, and for exploration of the abdomen in calves.

- **Flank/ Low flank**
  - This approach is useful for accessing the uterus in cattle that are recumbent or that are put into lateral or semi-sternal recumbency for surgery.

**Post-Operative Care**

- Antibiotics
  - What happened in surgery?
- Anti-inflammatory drugs
- Abdominal bandage?

**Complications**

- Peritonitis
- Incisional Infection/Dehiscence/Herniation
- Adhesion formation

**Selected References:**

Overview
Oxytetracycline didn’t resolve the lameness? We will take a systematic approach to diagnosing lameness in cattle, from the foot to the hip. We will also cover the most common causes of lameness and their treatments.

Objectives:
• Diagnostic techniques for lameness in cattle.
• A review of common causes of lameness and their clinical features.

While lameness issues are common in cattle, they can be difficult to diagnose and treat depending on the type of animal, the management situation, and the diagnostic and treatment modalities available to the practitioner. Often, lameness issues are attributed to the foot (and commonly to foot rot) and the animal is treated empirically with antibiotics and anti-inflammatory drugs. Veterinary care is often sought following one or more failed courses of treatment. While the foot is the origin of most lameness problems, the ability to differentiate between foot and upper limb problems may allow you to provide more appropriate recommendations to the producer, and potentially avoid violative residues if treatment is not determined to be economically viable.

Lameness Evaluation
• Evaluation at a distance
  o It is important to evaluate the animal from a distance if possible at the prior to beginning other parts of the exam. The posture of the animal should be evaluated, as well as the animal’s ability to bear weight while standing. The animal can also be evaluated at a distance for any obvious wounds, swelling, or abnormal
• Locomotion scoring
  o There are a number of scoring systems used to evaluate lameness in cattle, with the majority of systems designed for scoring lameness of dairy cattle. These scales all have some variability in their definitions of degrees of lameness, and score lameness on a three to five point scale. The ProAction initiative in Canada has established 2 scales for dairy cattle (locomotion and stall lameness scoring: https://www.dairyfarmers.ca/proaction/resources/animal-care). In finished cattle the North American Meat Institute established the NAMI Mobility Scoring System used in the packing industry, rating locomotion on a 4-point scale.
• Physical Examination
  o The importance of the physical examination in the lameness evaluation cannot be overemphasized. When possible, the limb should be If the cause of the lameness is unclear, the examination should begin at the foot and move up the limb. To examine the foot, the animal must be restrained with the foot tied in a standing position or placed on a tilt table intended for foot work.
Radiographic Imaging
  - Digital and wireless radiographic systems are making it easier for practitioners to utilize this modality in the field.

Ultrasound
  - Most useful for diagnosis of joint pathology, including septic arthritis and osteochondrosis.

Arthrocentesis

Specific Conditions

 Disorders of the Foot
  - Many foot problems can be diagnosed with a thorough hoof exam (including use of hoof testers). Common conditions affecting the foot include:
    - White line disease
    - Toe Ulcers
    - Pedal osteitis
    - Sole (Rusterholz) ulcer
    - Digital Dermatitis
    - Septic arthritis of the distal interphalangeal joint may occur secondary to other conditions, including foot rot. Clinical signs include severe lameness, and a characteristic unilateral swelling above the coronary band over the affected claw. Recommended treatment for septic arthritis of the distal interphalangeal joint in adult cattle is facilitated ankylosis of the joint.

 Joint Disorders
  - Septic Arthritis/Physitis
    - Septic arthritis is most often associated with trauma in adult cattle and hematogenous spread of bacteria in calves.
    - Treatment generally requires lavage +/- arthrotomy, local or regional antimicrobial administration, and systemic antimicrobial administration. Treatment is often cost-prohibitive in adults. Prognosis depends on duration of condition and joint affected.
  - Stifle, tarsus, and carpus are most often affected. May occur secondary to chronic clinical osteochondrosis.
  - Osteochondrosis dessicans
    - Mild to moderate lameness often with moderate to severe joint distension.
  - Coxofemoral Luxation
    - Dorsal and ventral luxations have different clinical presentations, and animals experiencing ventral luxations are unable to rise. Diagnosis requires manipulation of the rear limb. This requires heavy sedation in fractious animals. Palpation of the greater trochanter in a dorsal luxation will reveal the
greater trochanter in line with the tuber coxae and tuber ischia. Failure to palpate the greater trochanter suggests ventral luxation.

- **Tendon/Ligament Disorders**
  - CCL Rupture
    - Stifle swollen/distended and painful to palpation. Drawer test possible in some cattle.
    - Most commonly, animals are either culled or treated with NSAID and stall rest. Prognosis for return to function is poor. In valuable animals, surgical options do exist.
  - Peroneus Tertius Rupture
    - Occurs after hyper-extension of the tarsus while the stifle remains flexed. There is damage if the stifle can be flexed while the hock extended or vice versa.
    - Treatment of peroneus tertius rupture is simply stall rest and prognosis for return to function is good.
  - Gastrocnemius Rupture
    - With rupture of the gastrocnemius muscle, the animal is unable to bear weight on its rear limb, and its hock will be on the ground if it attempts to do so. Treatment of gastrocnemius rupture is a full limb cast. If this is not possible due to weight, temperament, or economics, humane euthanasia is recommended.
  - Upward Fixation of the Patella
    - The patella becomes locked on the medial trochlear ridge of the femur, resulting in the affected leg being held in extension behind the animal. This is generally an intermittent occurrence.
    - Treatment for upward fixation of the patella is desmotomy of the medial patellar tendon. This can be performed in the standing animal and carries a good prognosis.
  - Spastic paresis
    - These animals show a characteristic, intermittent caudal movement of the hindlimb if the gastrocnemius is affected. Quadriceps and mixed forms of spastic paresis have also been reported.
    - Either a gastrocnemius tenectomy or tibial neurectomy is recommended for salvage, with the tenectomy being the easier procedure to perform in the field. As this condition is often bilateral, the opposite limb may show more severe signs following surgical correction.

- **Nerve Disorders**
  - Radial Nerve Paralysis
- Seen when an animal has been in lateral recumbency on a hard surface. The elbow will be dropped and the carpus will remain partially flexed.
- Treatment involves a full limb splint to aid the animal in standing. Animals may recover in days to weeks.

  - Peroneal nerve paralysis
    - Animals affected often have a history of recently calving. These cows will knuckle on their hind limbs, and it is often bilateral. This condition often resolves within days.
    - Hydroflotation

  - Femoral nerve paralysis/patellar luxation
    - Seen most often in calves as a result of dystocia, particularly if the calf became hip locked during the delivery. Severe muscle atrophy can be observed within one week, and the calf will have a characteristic crouched stance, as well as difficulty rising. Patellar luxation in cattle almost always occurs secondary to femoral nerve paralysis, though it can occur on its own as a result of trauma in rare instances.

- **Bony Disorders**
  - Sequestrum
    - These are generally associated with chronically draining tracts on the limb and may occur secondary to traumatic injury. Surgical removal is required, and difficulty is dependent on duration and size.

  - Long Bone Fracture
    - With most long bone fractures, the animals are non-weight bearing lame on the affected limb. They may have an obvious angulation of the affected limb, particularly with distal limb or tibia fractures. Femur fractures can be difficult to diagnose due to the large amount of swelling that occurs over the femur following injury.

**Selected References:**


Introduction

- Reproduction is the essential driver of beef cattle operations' production and profitability.
- In the beef cow, estrus, ovulation, fertilization, implantation, pregnancy maintenance and successful birth of offspring are all complex physiologic processes.
  - Infectious disease agents can affect any of these processes, causing early or late reproductive failure.
    - Implicating infectious agents in early reproductive failure is often difficult, since reproductive failure is often not noticed until well after the infection.
    - Infectious disease agents causing late reproductive losses in beef cattle can be illustrated by abortion diagnoses at veterinary diagnostic labs (for example, Table 1):

<table>
<thead>
<tr>
<th>Table 1. Abortion Diagnoses, SDSU Diagnostic Lab, 2006-2016</th>
<th>% of total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Idiopathic (no diagnosis)</td>
<td>1540</td>
</tr>
<tr>
<td>Non-Specific Placentitis</td>
<td>815</td>
</tr>
<tr>
<td>T. pyogenes</td>
<td>170</td>
</tr>
<tr>
<td>Fungal - Mycotic</td>
<td>90</td>
</tr>
<tr>
<td>IBR Virus</td>
<td>79</td>
</tr>
<tr>
<td>Non-specific Bacteria</td>
<td>66</td>
</tr>
<tr>
<td>Neospora</td>
<td>44</td>
</tr>
<tr>
<td>Other</td>
<td>110</td>
</tr>
<tr>
<td></td>
<td><strong>2914</strong></td>
</tr>
</tbody>
</table>

Best Practices: Laboratory Diagnosis of Abortions

- Diagnostic success can be improved by promptly submitting the proper samples.
- While the following recommendations are likely sufficient for most laboratories, veterinarians should always confirm these with the lab they use.
- When possible, the entire fetus and placenta – chilled but not frozen – is the most desirable specimen.
  - The placenta is of particular importance and should be included whenever possible.
    - Significant histopathologic changes and agent identification often stem from examining the placenta.
- When field necropsies of aborted fetuses are performed,
  - Observe:
    - The carcass for trauma
    - Lungs for lesions and aeration
    - Liver and other organs for focal necrosis
  - Obtain a sterile sample of 3-5 ml of thoracic fluid should before going any further.
  - Samples to submit are listed in Table 2:
Table 2. Samples to send for abortion diagnosis

<table>
<thead>
<tr>
<th>Fresh and fixed:</th>
<th>Heart</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Lung (place fresh sample in its own whirl-pak)</td>
</tr>
<tr>
<td></td>
<td>Liver</td>
</tr>
<tr>
<td></td>
<td>Kidney</td>
</tr>
<tr>
<td></td>
<td>Spleen</td>
</tr>
<tr>
<td></td>
<td>Placenta (place fresh sample in its own whirl-pak)</td>
</tr>
<tr>
<td></td>
<td>Brain (place fresh sample in its own whirl-pak)</td>
</tr>
<tr>
<td>Fixed only:</td>
<td>Skeletal muscle such as tongue or diaphragm</td>
</tr>
<tr>
<td>In sterile disposable syringe or in sterile tube (use caution to transport)</td>
<td>Fetal stomach content (1-3 mL)</td>
</tr>
<tr>
<td></td>
<td>Fetal thoracic fluid or heart blood (3-5 mL)</td>
</tr>
</tbody>
</table>

- Include a complete, concise history to aid the diagnostician in choosing diagnostic tests. Information to include:
  - Number of animals in the herd, with recent purchases or movements
  - Number of abortions and previous diagnoses, if any
  - Age and breed of dams
  - Gestational age of abortions
  - Pertinent treatment or vaccinations

Serologic Diagnosis of Infectious Reproductive Failure
- Reproductive failure in beef herds often occurs without overt abortions, making serologic evaluation of the animals’ immune response to infectious disease a possible diagnostic tool.
- Possible strategies include:

  1. **Single serologic samples from an affected animal**
     - High single antibody titers can stem from prior vaccination as well as infectious disease exposure, making interpretation of results extremely challenging.

  2. **Paired serology**
     - Uses the premise that cattle recently exposed to an infectious disease agent will exhibit an antibody response to that germ that rises and falls according to the length of time post-infection.
     - May be considered for diseases commonly vaccinated against (e.g., BVDV, IBRV)
     - Serum is collected from a dam at the time of abortion (acute sample) and again 3 to 4 weeks later (convalescent sample).
       - Both acute and convalescent samples should be sent to the same laboratory for simultaneous testing.
     - A 4- to 8-fold difference between acute and convalescent titers indicates an active immune response to the disease agent.
• Variations in animals’ immune responses can confound interpretation.
• Additionally, the rise of an antibody titer in the dam may significantly predate the actual abortion, adding to interpretation difficulty.

3. **Case-control serology**
   • Blood is collected from affected (open) and non-affected (pregnant) animals and results compared between groups.
   • Interpretation may be clouded due to variations in exposures and individual animal immune responses.
     o Infections causing pregnancy loss might not necessarily stimulate a commensurate immune response
     o Likewise, non-affected animals may mount a robust antibody response sufficient to prevent pregnancy loss.

4. **Whole herd serologic profiling**
   • Serologic results in the face of a reproductive disease outbreak could be compared with profiled results obtained earlier from the herd.
   • Attention must be paid to herd events that have occurred between the sampling dates, such as vaccinations, and timing of sampling relative to the herd’s production cycle.

**Considerations for Specific Reproductive Diseases**

1. **Bovine Viral Diarrhea Virus (BVDV)**
   • Widespread vaccination for BVDV makes serology interpretation difficult.
   • Diagnostic workup of fetal tissues is ideal for implicating reproductive failure.
   • Persistently infected (PI) calves are the main reservoir for BVDV in beef herds.
     o Using ear notches to identify PI calves may demonstrate that the virus was present in the herd during breeding and early gestation.
     o Negative herd results do not necessarily mean BVDV was not present in the herd, due to potential losses of PI animals through death or culling; however, it does ensure no animals going out for the subsequent breeding season are persistently infected.

2. **Infectious Bovine Rhinotracheitis Virus (IBRV)**
   • Widespread vaccination for IBRV makes serology interpretation difficult.
   • Diagnostic workup of fetal tissues is ideal for implicating reproductive failure.
   • Interestingly, IBRV strains identified in fetal losses are increasingly found to be the same as IBRV strains commonly used in modified live vaccines.

3. **Neospora caninum**
   • Serology results are not confounded by vaccine titers.
   • Case-control serology approach.
   • Single titers are of limited use:
     o Titers may persist in a cow or heifer long after their exposure.
     o Positive serology in older cows may not correlate with increased risk of pregnancy loss.
   • Testing replacement heifers is recommended, because they have an increased risk of:
     o Reproductive failure.
- Persistently infected calves.
- Potential recontamination of feed if canines ingest placentas and fetal tissues.

4. Leptospirosis
   - Serology confounded by recent vaccination.
   - Paired serology may be useful
   - May be implicated by single titers, if very high.
   - L. interrogans serovar hardjo-bovis is host-adapted and evades the animal’s immune response.
     - PCR examination of urine may indicate its presence in a herd.
   - PCR examination of aborted fetuses.
     - Many PCR tests do not differentiate among strains of leptospirosis.

5. Trichomoniasis
   - No serologic tests.
   - Abortions due to “trich” are relatively rare – infertility and irregular returns to estrus are more common.
   - Presence in a herd usually relies on identifying the organism in the bulls.
     - Preputial scrapings for PCR testing.
   - Cows with pyometra are also diagnostic candidates.
     - PCR testing on a pus sample from the uterus.

6. Opportunistic Bacterial and Fungal Pathogens
   - Relatively commonly identified in individual cases of reproductive failure.
   - No validated serologic tests exist.
   - Microbiological analysis of placenta and fetal tissues is used.

Suggested Readings:
Reproductive Vaccines in Beef Cattle: Current Concepts
Russ Daly, DVM, MS, DACVPM (presenter), George Perry, PhD, South Dakota State University

- Infectious reproductive diseases can significantly damage reproductive efficiency and profitability for the beef herd.
- Examples of such pathogens include: Bovine Viral Diarrhea Virus (BVDV), Infectious Bovine Rhinotracheitis Virus (IBRV), Leptospira, Tritrichomonas foetus, Campylobacter fetus subsp. venerealis ("vibrio"), and Neospora caninum.
- These agents not only can cause abortion, they can also affect ovulation, fertilization, embryonic survival, perinatal survival, and disease persistence in offspring.
- Preparing and controlling these diseases include efforts directed at:
  - Biosecurity (keeping sources of new diseases out of the herd)
  - Environmental control (reducing stress and disease-favorable environmental conditions)
  - Vaccination (for some disease agents).
  - Commonly deployed vaccines include IBRV and BVDV in addition to other pathogens
    - Modified live (MLV) versions of these vaccines:
      o Stimulate immunity by actively infecting host cells.
      o Are considered more cross-reactive and more likely to stimulate cell-mediated immunity.
      o Exhibit longer duration of effect.
      o Provide flexibility in administration, due to boosters needed less commonly.
      o Also have the potential to revert to virulence and inflict damage they are designed to prevent.
    - Inactivated versions of these vaccines:
      o Are safe to use in a wide variety of circumstances.
      o Are considered less broad in their immune stimulation.
      o Exhibit shorter durations of effect.
- Effects of vaccination on reproductive parameters have been studied to a lesser extent than the effects of the pathogens themselves. This presentation will review pertinent research on the effects of the different versions of reproductive vaccines on reproductive parameters in beef cattle.

Effect of Modified Live Virus IBRV and BVDV vaccines on Reproduction in Naïve Cattle: Studies
- Live IBRV inoculated into previously unexposed (seronegative) heifers the day following estrus (Van der Maaten & Miller, 1985); and MLV IBRV vaccine given intravenously to 8 seronegative heifers (Van der Maaten et al., 1985):
  o Ovarian necrosis occurred in 7 of 8 heifers exposed to IBRV.
  o Mild to marked necrosis and inflammation in the corpora lutea and ovaries in 8 heifers given MLV IBRV intravenously.

- 3 different MLV vaccines were given to 18 seronegative heifers on day 4 after the 2nd dose of a 2-dose prostaglandin synchronization protocol (Smith et al., 1990):
  o Necrotic and inflammatory ovarian changes
  o IBRV was also isolated from 2 of 4 unvaccinated controls, apparently from vaccine virus excreted by vaccinated pen mates
• Modified live IBRV vaccines given to 8 seronegative heifers 14 days following breeding (Miller et al., 1989):
  o Infertility, embryonic death, and return to estrus in 4 of the 8 heifers.
  o Progesterone levels in bred animals demonstrated a disruption in luteal activity.

• 19 seronegative heifers synchronized and split into MLV vaccine and control groups (Chiang et al., 1990):
  o All 9 heifers not receiving the vaccine calved after a 35-day breeding period
  o Only 7/10 vaccinated heifers calved.

• 10 seronegative females vaccinated with MLV vaccine containing BVDV (Grooms et al., 1998):
  o Cytopathic BVDV isolated from ovaries up to 12 days post-vaccination
  o BVDV demonstrated with immunohistochemistry in ovaries 10 and 30 days post-vaccination

• 59 seronegative beef heifers vaccinated with MLV or inactivated vaccine IBRV & BVDV at synchronization program onset (Perry, et al., 2013):
  o Heifers given MLV vaccine had lower pregnancy rates and more abnormal estrous cycles.
    ▪ In heifers with abnormal estrous cycles, those given MLV were less likely to conceive at the return estrus

• Taken together, these studies provide ample evidence that vaccination of naïve heifers with modified-live IBRV vaccines will result in significant negative effects on pregnancy.

Effect of Modified Live IBRV and BVDV vaccines on Reproduction in Previously Vaccinated Cattle
While understanding the effects of MLV on naïve heifers is instructive, most beef heifers will have been previously vaccinated for IBRV and BVDV at least once prior to breeding. Therefore, studies examining the effects of MLV vaccines in previously-vaccinated cattle should be considered:
• In 10 previously-vaccinated heifers, MLV IBRV vaccination at the time of estrus did not result in recovery of IBRV from the ovaries (Spire et al., 1996).
• Yearling crossbred heifers (n = 295) of unknown prior vaccination status were vaccinated with MLV vaccine either 30 or 9 days prior to breeding (Stormshak et al., 1997):
  o No differences in pregnancy status or cyclicity between the two timing groups.
• 2 different MLV vaccines were given either 30 or 10 days prior to breeding (Rosenberg, 2004):
  o No reproductive differences among the groups except for more embryonic death in the 10-day group compared to the 30-day group.
• 799 previously-vaccinated heifers were given MLV vaccine either 40 or 3 days prior to breeding (Bolton et al., 2007):
  o No differences in conception rates.
• While the projects noted above used well-vaccinated animals, comparisons were only made regarding vaccine timing relative to estrus.
  o Non-vaccinated control groups were not included, so the effect of MLV vaccination by itself was not determined.

In recent years, studies including these control groups have been carried out:
1. 60 seronegative beef heifers were vaccinated with two doses of either a MLV vaccine or an inactivated vibrio-lepto vaccine, either 10 or 30 days pre-breeding (Walz et al., 2015):
• Heifers vaccinated with MLV vaccine had a 78% (31/40) calving rate.
• Heifers in the inactivated vaccine group had a 95% (19/20) calving rate.
• Numbers were too small for significant differences.

2. In 686 primiparous dairy cows, previously vaccinated with 4 MLV vaccine doses when younger, half were given MLV vaccine and half inactivated vaccine, 45 days pre-breeding (Walz et al., 2015):
   • No differences in conception rates between vaccine groups.
   • Less-than-ideal response to vaccines were noted overall.

3. A large, 2-year multi herd study, using 1304 well-vaccinated beef cows was designed with the power to detect smaller differences in reproductive parameters (Perry et al., 2016):
   • Vaccine groups included MLV, inactivated, and saline controls.
   • Animals were vaccinated 30, or 30 and 60 days prior to breeding, as per labels.
   • Conception rates to AI were higher, but not statistically significant, in the saline group compared to the MLV group, and were higher in the inactivated group compared to the saline group. Rates tended to differ between MLV and inactivated groups (P = 0.055).
   • At 56 d after AI, MLV animals had lower pregnancy rates compared to both inactivated and saline groups.
   • Breeding season pregnancy success was numerically higher in the saline group compared to MLV, and numerically higher in the inactivated group compared to saline. It was statistically significantly higher in the inactivated group compared to MLV.

4. A similar large-scale field study (10 herds with 1565 females) compared reproductive performance of well-vaccinated cows between pre-breeding vaccines with MLV or a chemically altered/inactivated vaccine (Perry, et al., 2018, submitted):
   • Conception rates to AI were significantly greater in the CA/IV vaccine group compared to the MLV vaccine group.
   • Animals vaccinated 38 to 89 d prebreeding had significantly higher conception rates than those vaccinated 27-30 d or 30-37 d prebreeding.
   • At all three vaccination intervals, conception rates to the CA/IV vaccine were higher than those to the MLV.
   • There were no differences between vaccine groups on whole breeding season pregnancy rates.

If inactivated vaccines result in fewer adverse reproductive effects than MLV vaccines, the question of efficacy against infectious disease agents remains. MLV vaccines are generally considered to provide a higher level of protection against viral pathogens than are inactivated vaccines.

• A recent study (Walz et al., 2017) examined heifers vaccinated with MLV prior to their first breeding, then split into MLV or CA/IV vaccine groups during subsequent pregnancies, then challenged with IBRV and BVDV:
  o Cattle vaccinated with the CA/IV vaccine before challenge during their second pregnancy had similar levels of abortions compared to the MLV group.

Conclusions
• Vaccines against infectious reproductive disease agents are valuable tools for protection against the potentially devastating effects of these germs.
• However, MLV versions of these vaccines can cause negative effects when given to naïve animals.
• Further evidence supports the notion that prior vaccination with MLV vaccines will mitigate negative effects when MLV vaccines are given prior to subsequent breedings, even closer to breeding than labels indicate.
• However, evidence is also emerging that MLV vaccines, even when given at labeled pre-breeding intervals, may negatively affect reproductive parameters compared to cattle vaccinated with inactivated vaccines.

When using reproductive vaccines in beef cattle herds, it’s necessary to consider the balance between needed protection and any potential adverse effects. It should be expected that no two beef herds have the same risk levels for infectious reproductive diseases. Therefore, veterinarians are in a unique and essential position to work with herds on an individual basis to form a vaccine program that works for each situation.
Introduction
Pre-weaning respiratory disease in beef herds has presented itself as a puzzle for veterinarian and beef producers alike. Yet it is an important phenomenon, as evidenced by the following points:

- Estimated to affect 20% of beef cattle operations in any given year.
- The #1 cause of death in beef calves from 3 weeks of age to weaning.
- Calves treated for respiratory disease are 17 pounds lighter at weaning compared to counterparts.
- Estimated to cost producers $4-6 per beef cow per year, representing a $144 million annual cost to the industry.

Its inconsistent incidence between herds and different years makes pre-weaning BRD a difficult disease issue to sort out. While factors that contribute to post-weaning BRD and dairy calf BRD have been characterized, the same is not true for pre-weaning BRD in beef calves.

Some of the aspects of pre-weaning BRD that will be discussed in this presentation include:

1. Herd level risk factors
2. Calf level risk factors
3. Epidemiology
4. Vaccination

Herd Level Risk Factors
Because the occurrence of pre-weaning BRD varies greatly from herd to herd and from year to year, it may be possible to compare herd-level differences between affected and non-affected herds, and between high-incidence years and low-incidence years. Several studies have looked at herd level risk factors for BRD. Their results are summarized here:

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While there are few consistencies across studies, some of the risk factors identified can be grouped:

1. Risk factors related to the introduction of new pathogens:
   a. Importing weaned steers
   b. Visits by outsiders
   c. Importing new pre-weaned calves
2. Risk factors related to increasing the opportunities for contacts between infected and susceptible calves:
   a. Longer calving seasons
   b. Larger herds
   c. Antibiotics in feed for preweaned calves
   d. Treating cows for BRD
   e. Calving season January-April
   f. Creep feeding
   g. Heat synchronization
   h. Intensive grazing

**Epidemiology**
Illness patterns in affected herds may help us better understand pre-weaning BRD. Two larger data sets that detail calf age at diagnosis show somewhat similar patterns. A biphasic distribution emerges:

1. Calves less than 60 days of age:
   a. Failure of passive transfer may be significant in this age group.
2. Calves 3-5 months of age:
   a. After increased contacts between susceptible and infected calves
   b. Declining maternal antibodies

![Graph showing incidence of BRD by age](image)

**Calf Level Risk Factors**
Fewer studies have examined individual calf risk factors in affected herds, comparing aspects of calves sick with pneumonia with those not affected. The factors that come out in those studies, however, seem to make sense:

1. Male calves affected more than female calves
2. Calves born to dams ≤ 2 yrs old
3. Inadequate passive transfer
4. Calves born during dystocia
5. Twin calves
6. Calves born to dams without high IBRV, BCV, or BVD titers
Veterinarians and cattle producers would also do well to consider whether some of the known risk factors for post-weaning BRD may apply to pre-weaning BRD, too:

1. Corticosteroids/Stress
2. Viral infections: IBR, BRSV, BVDV, PI-3, Coronavirus
3. High numbers of bacteria in environment
4. Cold air
5. Dehydration
6. Dust
7. Poor colostrum
8. Vitamin deficiency
9. Lack of prior exposure
10. Acidosis

In addition, those factors known to influence dairy calf BRD could be considered as well:

1. Navel infections
2. Poor nutrient intake
3. Feeding waste milk
4. Poor colostrum
5. Group housing w older calves
6. Poor ventilation in barns
7. Corticosteroids/Stress - weaning

**Diagnostics**

Could diagnostic work performed on pre-weaning beef calf submissions help us understand pathogenesis?

Postmortem diagnostic cases at the SDSU veterinary diagnostic lab from 2016-17 revealed:

1. Largely the same bacterial pathogens as one would expect with post-weaning BRD were present, including Mannheimia hemolytica, Pasteurella multocida, Histophilus somni, and Mycoplasma bovis.
   a. A general trend toward the involvement of M. hemolytica in younger cases.
   b. A general trend toward H. somni involvement in older calves.
2. Bovine coronavirus was identified in approximately 13% of post-mortem cases; the vast majority of those cases involved other pathogens as well.
3. No other viruses were involved in these cases.

Because mortality is not always a feature of pre-weaning beef calf BRD, antemortem nasal swabs are frequently submitted by veterinarians investigating those cases.

1. Pasteurella multocida and Mannheimia hemolytica were frequently isolated from nasal swabs.
2. Bovine coronavirus is frequently isolated from these cases, but not other viruses.

**Vaccination/Immunity**
Some of the same herd-level studies mentioned previously attempted to find factors related to vaccination as associated with pre-weaning beef calf pneumonia. None of those studies found significant differences in vaccination programs between affected and non-affected herds. Knowing that the bulk of pre-weaning beef calf BRD occurs in the 3-5 month of age range, it seems logical that stimulating active immunity prior to that point would be a worthy goal. Traditionally, stimulating immunity in this age of a calf is not easy, and would have to address immunity against all major BRD pathogens.

Many pieces of the pre-weaning BRD puzzle have yet to fall into place. However, the picture continues to become clearer as herd- and calf-level studies, and epidemiologic and microbiological studies are reported. It will take cooperation among veterinarians, cattle producers, animal health companies, and diagnosticians to better understand this phenomenon.
Introduction
One of the biggest challenges facing a veterinarian is their ability to diagnose and treat a veterinary patient for pain. Every species has a different subset of behavioral nuances associated with the presence of pain. Identification of pain is particularly difficult in prey species. It appears this is further complicated by the fact that different types of pain, i.e. acute vs chronic, somatic vs visceral, and physiologic vs pathological have differing behavioral cues. This makes the formulation of behavioral pain scales difficult to achieve. Once the period of physiologic pain has ceased, the presence of pain is no longer necessary for the well-being of the patient. Physiologic pain is protective against tissue injury and is transmitted along the Aδ-fibers. The duration of physiologic pain is measured in seconds to minutes. The notion of allowing an animal to remain in a state of pain to prevent further injury is erroneous. The use of analgesics will rarely abolish pain/nociception to the point where full function will be restored. There are obvious moral and ethical issues to be considered. The benefits of analgesia far outweigh the negative aspects. Earlier return to normal function, improved wound healing and reductions in metastatic rates of cancers are just a few of the benefits[1]. This article will investigate the different treatment options available: and how they affect the different stages of nociception, namely transduction, transmission, modulation and perception.

Transduction
A noxious stimulus (mechanical, thermal or chemical) is converted into an action potential via the nerve endings of Aδ- (physiologic; fast conduction) and C-fibers (pathologic, slow conduction) [2, 3]. This conversion is called transduction. Cytokines, kinins, leukotrienes, prostaglandins, H+, K+ and histamine (algogens) are released from damage tissue, which increase the sensitivity of these local neurons to noxious (hypersensitivity) and innocuous (alldynia) stimuli. These inflammatory mediators have the ability to up-regulate the sensitivity of Aβ-fibers (touch) and ‘sleeping’ fibers (High stimulus mechanonociceptors), thereby greatly increasing the number and area of neuronal input in response to the noxious stimulus. These are some of the mechanisms involved with peripheral sensitization. Peripheral sensitization is the increase in area over the original area of injury in which neuronal input is processed as a noxious stimulus. Peripheral nociceptors are inhibited by local anesthetics at the fast voltage-gated sodium channels. NSAIDs and corticosteroids will reduce the production of the pro-inflammatory mediators, minimizing the development of peripheral sensitization. De novo mu-opioid receptors will be expressed in inflamed tissue within three to four days of an injury. Opioids administered at the peripheral site of injury will activate these mu-opioid receptors causing the hyperpolarization of the cell membrane, decreasing the chances of an action potential being propagated.

Transmission
Transmission is the propagation of the action potential along these Aδ- and C-fibers to the dorsal root horn and synapses in the I, II, and V Rexed laminae of the spinal cord with the second order neuron of the nociceptive pathway. The second-order neurons then project to the brain via the neospinothalmic tract (Aδ-fibers), and paleospinothalamic tract (C-fibers). Transmission is inhibited
by the use of local anesthetics and α2-adrenergic agonists. Xylazine does have direct local anesthetic properties. Perineuronal infiltration of xylazine however, does cause cytotoxicity and sloughing of the tissues.

**Modulation**
The synapse of the first and second order neurons in the dorsal root horn is under the influence of the endogenous descending analgesic system (neurotransmitters: enkephalins, serotonin, norepinephrine) as well as Aβ-fibers and excitatory interneurons. Opioids, α2-adrenergic agonists, and norepinephrine potentiate this endogenous descending analgesic mechanism of reducing noxious neural input. Stimulation the Aβ-fibers (massage) close to the area of injury will inhibit the noxious neural input at the level of the dorsal root horn. The excitatory interneurons on the other hand, have the ability to enhance the noxious input as well as amplifying these effects to other ascending neural pathways. This mechanism is part of the phenomenon known as dorsal horn wind-up. This is also known as central sensitization. At the sub-cortical level, there is modulation of the noxious sensory input by higher cortical centers and the analgesic system. The noxious sensory input is therefore the summation of these interactions at the level of the dorsal horn, indicating the importance of analgesic therapies aimed at the spinal cord rather than the brain. Inhalant anesthetics, local anesthetics, opioids, α2-adrenergic agonists, tricyclic antidepressants, cholinesterase inhibitors, N-methyl D-asparate (NMDA) antagonists and anticonvulsants can all affect this interaction at the level of the dorsal horn.

**Perception**
Perception is the end-result of the transduction, transmission, modulation and sub-cortical integration of the noxious sensory input. Perception is the higher cortical center processing of this sensory input and the conscious, emotional response. It is possible to have a physiological (stress) response to a noxious stimulus without having a perception of it. Unconsciousness induced by anesthesia is not enough to block a noxious stimulus and response; some form of analgesia needs to be administered at the same time. Anesthetics, opioids, α2-adrenergic agonists, tricyclic antidepressants, benzodiazepines, and phenothiazines have the potential to alter the perception of a noxious stimulus.

**Equine Pain Control Plans**

**Preemptive analgesia**
The administration of analgesics prior to the initiation of a noxious stimulus is known as preemptive analgesia [2]. This preemptive administration of analgesia will reduce the chances of peripheral and central sensitization from occurring and result in a reduction in analgesics administered. Preemptive analgesia does not prevent pain but will often lead to a pain free state earlier in the treatment process. Local anesthetic techniques, opioids, α2-adrenergic agonists, and NSAIDs administered prior to a noxious stimulus will contribute to preemptive analgesia.

**Balanced analgesia**
The use of multiple analgesic agents that will affect preferably more than one level of the nociceptive pathway is more likely to be success in preventing the stress response. Multiple agents targeting more than one receptor type and anatomical location will prevent peripheral and central sensitization, tachyphylaxis, stress response, improve wound healing, reduce infection rates and improve mobility of the patient. No one single analgesic agent can achieve all of these observations,
with the exception of neuraxial analgesia/anesthesia using local anesthetics. This is virtually impossible to perform safely in the equine patient. Balanced analgesia may lead to a reduction in the amount of parenterally-administered drugs, resulting in fewer side-effects. The expected pharmacodynamic effects and pharmacokinetic properties of each drug used must be known to minimize not only adverse effects but also to enable the administrator to know when the analgesic therapy is not achieving its goal.

Some New & Old Therapies – Special Considerations

Fentanyl
Fentanyl patches are highly convenient however they do have some shortcomings. The fentanyl patch have been proven to be analgesic with a limited amount of adverse effects [4, 5]. The analgesic response to the fentanyl patches is highly variable. This is due to the high individual variability of transdermal drug absorption. Even within the same experimental lab the time to what is considered analgesic levels (1ng/mL) varied considerably[4, 5] and in research labs individuals failed to attain this level [6]. This variability is partly due to the location of patch application[7] and site preparation. The prescription and safe disposal of fentanyl patches does impose other complications to their use in veterinary practice. Regular visitations, cautious client selection and the use of an exchange patch program will help obviate some of these problems. Intra-operative fentanyl infusions will reduce the minimum alveolar concentrations of isoflurane[8]. Fentanyl has the advantage of a short half-life and tissue residency time, both of which will increase the predictability of its use. More research is needed to fully elucidate all the pharmacokinetic and pharmacodynamic properties of fentanyl in the equine patient.

Constant Rate Infusions
Lidocaine is one old drug that is enjoying a new renaissance. The systemic administration of lidocaine is useful for the treatment of mild to moderate pain. The exact mechanism for this action is unknown. Lidocaine is used intra- and post-operatively for analgesia. The anti-inflammatory and prokinetic properties of lidocaine are proving to be beneficial, in particular for the colic patient. Ketamine is another old drug experiencing a revival. Ketamine is an antagonist on the N-Methyl D-Asparate receptor (NMDA). Agonism of the NMDA receptor is one mechanism of central sensitization and the formation of a chronic pain state. Ketamine is best utilized as an adjunct to opioids and or α2-adrenergic agonists rather than as a sole agent. NMDA antagonist will reduce the chances of opioid tolerance developing. Common indications for inclusion of a NMDA antagonist are severe and chronic pain states. It is not uncommon to incorporate an opioid with lidocaine and ketamine (‘MiLK’) as a constant rate infusion. When administered together, the differing half-lives, active metabolites and side effects have to be appreciated and the dosage adjusted accordingly. Gabapentin another NMDA antagonist (and anticonvulsant) has been used in horses as an adjunct to treat moderate to severe pain states, without any adverse effects. Little work has been done on this drug however and is still in the infancy states of investigations. In Europe, tramadol has been used for some time in the medical field. It has received FDA approval in this country. Neuraxial tramadol has been reported in the horse without any adverse effects and analgesia comparable to morphine. Tramadol binds to mu-opioid receptors, as well been a serotonin and norepinephrine reuptake inhibitor. Further research needs to be performed to fully assess the potential of this drug.

Conclusions
A multimodal (multi-receptor) approach to analgesia will provide the best therapy for minimizing the development of peripheral and central sensitization, and chronic pain states. Adverse effects from any one particular drug will be minimized due to the synergic effects and subsequent reduction in dosages required. Return to normal function will be shortened. Where possible the use of local and regional anesthesia should be considered. Local anesthesia will not only provide analgesia, but obliterate the stress response. It is important to administer an analgesic therapeutic regime, assess the response to this therapy within an appropriate time frame for the drug administered, and adjust your dosing regime or combination of drugs; the AAA approach to analgesic therapy. Each individual will response in a different way and the analgesic therapy needs to be tailored to that individual.

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose (mg/kg IV)</th>
<th>Frequency</th>
<th>Special Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Opioids</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Morphine</td>
<td>0.05-0.1</td>
<td>q4-6h</td>
<td>ileus, urinary retention, histamine release, excitement.</td>
</tr>
<tr>
<td>Methadone</td>
<td>0.1-0.0.3 (IM)</td>
<td>0.05-0.1mg/kg/h</td>
<td></td>
</tr>
<tr>
<td>Fentanyl</td>
<td>0.005-0.02 (2x100μg/h)</td>
<td>6.8μg/kg/h</td>
<td>CRI-Anesthesia adjunct (MAC reducing)[8]</td>
</tr>
<tr>
<td>Meperidine</td>
<td>1-2mg/kg (IM)</td>
<td>q1-2h</td>
<td>Hypotension ~ histamine release. Spasmolytic</td>
</tr>
<tr>
<td>Buprenorphine</td>
<td>0.005-0.01 (12h) [9]</td>
<td>q6-8h</td>
<td>↑ locomotor activity</td>
</tr>
<tr>
<td>Butorphanol</td>
<td>0.02-0.04</td>
<td>q2-4h</td>
<td>ileus, urinary retention, excitement.</td>
</tr>
<tr>
<td></td>
<td>0.05-0.1 (IM)</td>
<td>13-24μg/kg/h</td>
<td></td>
</tr>
<tr>
<td><strong>α2-Adrenergic agonists</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Xylazine</td>
<td>0.5-1.0</td>
<td>1mg/kg/h</td>
<td>Bradycardia, ataxia, hypoventilation, ↓ Gut perfusion, CRI-Anesthetic adjunct</td>
</tr>
<tr>
<td>Medetomidine</td>
<td>0.005-0.02</td>
<td>3.5-5μg/kg/h</td>
<td></td>
</tr>
<tr>
<td>Detomidine</td>
<td>0.01-0.02</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Romifidine</td>
<td>0.04-0.08</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Anti-inflammatory Drugs</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Phenylbutazone</td>
<td>2-4</td>
<td>q12-24h</td>
<td>Gastrointestinal ulceration, Renal toxicity</td>
</tr>
<tr>
<td>Flunixin meglumine</td>
<td>0.2-1.1</td>
<td>q12-24h</td>
<td></td>
</tr>
<tr>
<td>Ketoprofen</td>
<td>1.1-2.2</td>
<td>q24h</td>
<td></td>
</tr>
<tr>
<td>Carprofen</td>
<td>0.5-1.1</td>
<td>q24h</td>
<td></td>
</tr>
<tr>
<td>Firocoxib</td>
<td>0.1-0.5 (PO)</td>
<td>q24h</td>
<td></td>
</tr>
<tr>
<td>Diclofenac 1%</td>
<td>5” strip topical cream</td>
<td>q12h</td>
<td></td>
</tr>
<tr>
<td><strong>Miscellaneous</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lidocaine 1%</td>
<td>1.3mg/kg IV (0.05mg/kg/min)</td>
<td>over 15min</td>
<td>Ataxia, agitation, somnolence, bradydysrhythmias. Prokinetic, immunomodulator</td>
</tr>
<tr>
<td>Ketamine</td>
<td>0.4-0.8mg/kg/h</td>
<td></td>
<td>Agitation, Mydriasis, hypersensitivity</td>
</tr>
<tr>
<td>Tramadol</td>
<td>1-2mg/kg</td>
<td>q4-6h</td>
<td>Opioid agonist, Reuptake</td>
</tr>
</tbody>
</table>
(IV,IM) inhibitor
Limited experience

[3, 10, 11]Disclaimer – The author has made every effort to show common clinical doses. It is up to the veterinarian to determine the best treatment and dose for each individual. Neither the publisher nor author assumes any liability for any injury and/or damage to persons or property arising from this publication.

References:

<table>
<thead>
<tr>
<th>Anesthetic</th>
<th>PKa</th>
<th>% RN at PH 7.4</th>
<th>Onset in minutes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mepivicaine</td>
<td>7.6</td>
<td>40</td>
<td>2 to 4</td>
</tr>
<tr>
<td>Etidocaine</td>
<td>7.7</td>
<td>33</td>
<td>2 to 4</td>
</tr>
<tr>
<td>Articaine</td>
<td>7.8</td>
<td>29</td>
<td>2 to 4</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>7.9</td>
<td>25</td>
<td>2 to 4</td>
</tr>
<tr>
<td>Prilocaine</td>
<td>7.9</td>
<td>25</td>
<td>2 to 4</td>
</tr>
<tr>
<td>Bupivicaine</td>
<td>8.1</td>
<td>18</td>
<td>5 to 8</td>
</tr>
<tr>
<td>Procaine</td>
<td>9.1</td>
<td>2</td>
<td>14 to 18</td>
</tr>
</tbody>
</table>
Sedation for Standing Equine Procedures
Patrick Burns BVSc, MANZCVS, DACVAA

Introduction
The use of sedation and regional anesthesia for standing restraint is becoming more popular as chemical agents for restraint improve, along with new surgical techniques. A brief list of the indications for standing restraint but not exclusive to, include dental surgery, diagnostic imaging, laceration repairs, obstetrical surgery, laparoscopic surgery, ocular surgery, and orthopaedic procedures. These protocols are becoming more frequent, especially in the aged equine population with co-morbidities. Standing restraint has two obvious advantages, obviating the risks associated with general anesthesia and the reduction in peri-operative time. Increased personnel risk and over-sedation are the main disadvantages to standing sedation. Standing restraint usually consists of the combination of sedatives, analgesics and regional anesthesia. As surgical techniques improve, there is a demand for a predictable level of sedation, for a longer time period. Constant rate infusions provide a more stable state of sedation compared to intermittent boluses thus enabling the operator to titrate the infusion to the desired level of sedation. An infusion also has the advantage of achieving the desired level of sedation with a lower total dose of sedation used.

Constant Rate Infusions
Alpha2-Adrenergic Agonists
This class of drug has become the mainstay for standing chemical restraint for the equine patient. There are subtle differences between these agents, which can be exploited to the operator’s advantage. An ideal sedative agent would cause a linear dose-dependent sedation, quick onset time, short duration of action, non-accumulating, with minimal cardiovascular and respiratory side effects. Unfortunately, there is no such agent available at this time. Each α2-adrenergic agonist has its own benefit. Xylazine has a short duration of action in the horse [1] which is ideal for a constant rate infusion (loading dose 0.6-1.0mg/kg IV, followed by 1mg/kg/h). The ataxia and head ptosis caused by xylazine tend to make it less ideal, especially for surgical procedures of the head.

There are reports in the literature describing detomidine as a constant rate infusion for standing restraint in the horse [2, 3]. Detomidine is the agent most studied in North American for the use of standing sedation. It is generally considered more predictable in producing sedation in horses which are excited or aggressive when compared to xylazine. One published receipe for an infusion of detomidine adds 24μg/kg of detomidine to a 250mL bag of normal saline. This infusion [2] starts with a bolus dose of 5-10μg/kg IV followed by an initial infusion of 8mL/min which is then halved every 15minutes. This takes into consideration the longer half-life of detomidine compared to (dex)medetomidine and xylazine.

Romifidine (40-80μg/kg IV) has been reported to cause the least amount of ataxia out of all the α2-adrenergic agonists [4], however the duration of romifidine appears to be the longest. The reduction in ataxia is an advantage particularly with aged or debilitated horses. It is difficult however to titrate an infusion of romifidine to effect.

A loading dose of 5-10μg/kg IV of medetomidine followed by 3.5μg/kg/h has been described as an adjunct to general anesthesia [5] and has been used in a clinical setting by the author. This infusion
rate may need to be adjusted over time. A reduction in dose may be required if co-administration with an opioid is instituted. The dose of dexmedetomidine would be slightly less than half the dose of medetomidine due to the antagonistic effects of levomedetomidine[6]. Dexmedetomidine may theoretically be the most suitable α2-adrenergic agonist for a constant rate infusion on the market at this moment [7]. Some advantages it possesses are relatively short duration of action, less ptosis of the head, milder degree of ataxia, and shorter duration of effect on the cardiovascular system [8]. The high affinity for the α2-adrenergic receptor decreases the risk of paradoxical excitement and disinhibition.

High risk patients for example, the aged, metabolic syndrome or those with cardiovascular disease you may decide to omit the bolus dose and just start the infusion at a slightly higher rate and monitor for the desired level of sedation. The infusion is then reduced to a lower rate. When using an α2-adrenergic agonist infusion there are some side effects which you may keep in mind when managing these patients. All α2-adrenergic agonists will cause a hyperglycemia and polyuria (of various mechanisms, not just an osmotic diuresis) which may cause dehydration after the procedure. This may necessitate instituting intravenous fluids to help reduce the risk of impaction, ileus or colic. See “When things go wrong” for further information.

Table 1 Recipes for alpha2-adrenergic agonist infusions

<table>
<thead>
<tr>
<th>Drug</th>
<th>Loading dose</th>
<th>Infusion Rate</th>
<th>Amount added to 500mL bag (10drops/mL)</th>
<th>Amount added to a 500mL bag (15drops/mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medetomidine</td>
<td>5μg/kg</td>
<td>3.5μg/kg/h</td>
<td>2000μg (4μg/mL)</td>
<td>3300μg (6.6μg/mL)</td>
</tr>
<tr>
<td>Detomidine</td>
<td>8μg/kg</td>
<td>40μg/kg/h</td>
<td>25000μg (50μg/mL)</td>
<td>37500μg (75μg/mL)</td>
</tr>
<tr>
<td>Butorphanol</td>
<td>20μg/kg</td>
<td>24μg/kg/h</td>
<td>15000μg (30μg/mL)</td>
<td>22500μg (45μg/mL)</td>
</tr>
<tr>
<td>Lidocaine</td>
<td>1.3g/kg</td>
<td>3mg/kg/h</td>
<td>1875mg (3.75mg/ml)</td>
<td>2800mg (5.6mg/mL)</td>
</tr>
</tbody>
</table>

Infusion rate 1drop/sec if using a 10drop/mL administration set to a 450kg horse. (Concentration of solution)

Adjunct agents

Butorphanol has been used commonly as an analgesic constant rate infusion for the horse. It may be added to α2-adrenergic agonist infusion to reduce the rate of infusion. Clinical research tends to suggest butorphanol may offer some advantages over the use of morphine [9]. A loading dose of 10-100μg/kg IV followed by 10-15μg/kg/h[10] in addition to an α2-adrenergic agonist infusion. Morphine or methadone can be used as alternatives to butorphanol; a loading dose of 100μg/kg IV slowly, followed by an infusion of 50-100μg/kg/h. Opioids are best administered after the bolus dose of the α2-adrenergic agonist to reduce the chances of excitement induced by the opioids.
Lidocaine is another agent that may also be added to an α2-adrenergic agonist infusion (Loading dose 1-1.3mg/kg IV over ten minutes; 50μg/kg/min). Agitation and pacing are some of the early indicators of adverse effects of lidocaine. If the infusion is not stopped this may progress to seizures. Careful dosing is required especially if lidocaine is also being used for local and regional anesthesia. Acepromazine (20-50μg/kg IV) administered prior to the α2-adrenergic agonist will help to blunt the sudden increase in blood pressure. This can be helpful especially in the geriatric equine patient.

**Antagonistic Agents**

It may be necessary in some cases to reverse the effects of α2-adrenergic agonists. The various α2-adrenergic agonists have differing receptor activities (α2: α1 selectivity: Yohimbine 40:1; Atipamezole 8526: 1) and affinities[11]. Ideally the receptor antagonist activity should match the receptor activity of the agonist. For example, atipamezole is a highly selective α2-adrenergic antagonist whereas tolazoline is a non-selective α2-adrenergic antagonist. It binds to α1-adrenergic receptors, imidazoline receptors, histaminergic receptors, cholinergic receptors as well as having direct vasodilatation effects. Yohimbine on the other hand, is considered a selective α2-adrenergic antagonist with some activity on dopaminergic and serotonergic receptors but no activity on imidazoline receptors [12]. The dose of the α2-adrenergic antagonists will vary depending upon the dose of α2-adrenergic agonist and the amount of metabolism that has already taken place. As a general rule with any antagonist, reversal should be done slowly to the desired pharmacodynamic effect. This can be done either by a slow intravenous infusion or by repeated intramuscular boluses. Refer to table 2 for a guide to dosing. The aim of the reversal is not to have the horse 'looking normal' again but rather to enable the horse to function. Full reversal of the cardiovascular effects induced by α2-adrenergic agonists is unlikely to be achieved by α2-adrenergic antagonists [13]. A relative overdose of an α2-adrenergic antagonist can lead to agitation, gut hypermotility, diarrhea, and colic. It is far better to be partially reversed rather than given too much reversal agent.

**Table 2 Pharmacological properties of alpha2-adrenergic antagonists [11-14]**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Species</th>
<th>Dose</th>
<th>Elimination half-life (min)</th>
<th>Adverse effects/overdose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Yohimbine</td>
<td>Horse</td>
<td>0.075–0.15 mg/kg IV</td>
<td>52.8 ± 27.8</td>
<td>Hypotension and excitement with rapid IV administration, Seizures at high doses, Reversal of analgesia (all α2-adrenergic antagonists) which may induce a stress response,</td>
</tr>
<tr>
<td></td>
<td>Cattle</td>
<td>0.125–0.2 mg/kg IV</td>
<td>46.7 ± 24.4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sheep</td>
<td>0.125–0.2 mg/kg IV</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Tolazoline</td>
<td>Horse</td>
<td>1-4 mg/kg IV slowly</td>
<td>60</td>
<td>Gastrointestinal disturbances, agitation, muscle fasciculations, tachycardia, mild hypertension, ventricular arrhythmias and death</td>
</tr>
<tr>
<td></td>
<td>Cattle</td>
<td>2-4mg/kg IV slowly (calves 1 mg/kg IV slowly)</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Llamas</td>
<td>2 mg/kg IM</td>
<td>70</td>
<td>Overdoses of tolazoline at five times the recommended dose</td>
</tr>
</tbody>
</table>
have been associated with fatalities in horses

<table>
<thead>
<tr>
<th>Atipamezole</th>
<th>Horse</th>
<th>0.06-0.15mg/kg IV slow</th>
<th>Doses above 0.16-0.2mg/kg used to treat an immediate overdose can cause agitation. Rapid IV administration to animals with vasoconstriction may result in cardiovascular collapse due to the rapid vasodilatation with persistent bradycardia. Agitation and possible aggression (all α2-adrenergic antagonists)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dairy cows§</td>
<td>0.2 mg/kg IV</td>
<td>35.2 ± 17.9</td>
<td>Agitation and possible aggression (all α2-adrenergic antagonists)</td>
</tr>
<tr>
<td>Calves§</td>
<td>0.2 mg/kg IV</td>
<td>52.1 ± 7.0</td>
<td>Agitation and possible aggression (all α2-adrenergic antagonists)</td>
</tr>
<tr>
<td>Sheep§</td>
<td>0.2 mg/kg IV</td>
<td>34.2 ± 11.9</td>
<td>Agitation and possible aggression (all α2-adrenergic antagonists)</td>
</tr>
</tbody>
</table>

§Following medetomidine 0.04 mg/kg IV

WHEN THINGS GO WRONG!

Respiratory Distress
There are two causes of respiratory distress following the administration of α2-adrenergic agonists and their clinical signs will vary slightly depending upon the etiology of the cause. Rapid identification of the problem is necessary to avoid any morbidity or even death associated with the use of the α2-adrenergic agonists.

Acute lung injury
The use of α2-adrenergic agonists in small ruminants may lead to activation of pulmonary macrophages and the formation of acute lung injury [12]. These macrophages cause a change in the pulmonary vascular endothelium increasing permeability resulting in pulmonary edema. These changes are not reversible with α2-adrenergic agonists. It has been observed by the author and other colleagues, horses with a pre-existing fever sedated with α2-adrenergic agonists may develop clinical signs of tachypnea/dyspnea (parenchymal pulmonary changes). On auscultation there are inspiratory crackles and breath sounds. This has been reported in cats occurring up to three days post administration (see product insert of Dextromitor). Treatment includes intra-nasal oxygen (2 x 15L/min), α2-adrenergic antagonists, furosemide and flunixin meglumine. Severe reactions are a rare occurrence in horses.

Pharyneal collapse
Pharyngeal relaxation is a common side effect of α2-adrenergic agonists however many horses do not require any intervention. The pharyngeal mucosal relaxation may be so severe as to collapse the airway during the inspiratory phase. This is more likely to occur with aged and obese horses. Occasionally a horse may require either a nasotracheal intubation to elevate the obstruction. The use of α2-adrenergic antagonists may be beneficial in these cases.

Paradoxical Aggression/ Excitement
A hypothetical reason for paradoxical aggression/excitement may be the effects of α2-adrenergic agonists on α1-adrenergic receptors, especially xylazine since it has a low α2:α1 selectivity. Alpha1-adrenergic activity is responsible for the level of arousal in the central nervous system. The author has not seen paradoxical behavior in horses sedated with α2-adrenergic agonists other than with xylazine. Another potential reason for this effect of xylazine may be associated with its lack of interaction with the imidazoline receptors however these receptors have not been reported to be associated with sedation or anesthesia. The addition of acepromazine, butorphanol or the cautious use of another α2-adrenergic agonist such as detomidine or dexmedetomidine may help to alleviate this problem. If possible wait for the animal to calm down before sedation will also help reduce the chances of paradoxical aggression/excitement. You may elect to cancel the procedure and start again another day using a difference α2-adrenergic agonist preceded by acepromazine and butorphanol.

**Overdose (relative and absolute)**

The primary treatment for an overdose of a α2-adrenergic agonist is the use of a α2-adrenergic antagonist. Case reports in the literature describe using extremely high doses of α2-adrenergic antagonists to antagonize the effects of α2-adrenergic agonists. A pony given an overdose of detomidine 0.2mg/kg which was treated with a total dose of atipamezole 1100mcg/kg [15]. Remember the amount of α2-adrenergic antagonists that has to be administered must match the tissue concentration of the α2-adrenergic agonists. A cautious approach to the use of a α2-adrenergic antagonist is often the best. You need to slowly increase the dose until the desired effect. Intravenous fluids may also be required to counteract the diuretic effects and the predisposition for ileus and impaction. The head will also need to be elevated to prevent nasal congestion. Thermoregulation is also impaired in these individuals so attention to body temperature is important to prevent hyperthermia or hypothermia. Be aware however, the peripheral vasoconstriction will cause the periphery to become cool to touch while the core temperature may increase. Relatively small overdoses may be treated symptomatically.

**Table 3 Clinical signs of an overdose of alpha2-adrenergic agonists**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Magnitude of overdose</th>
<th>Clinical signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Xylazine</td>
<td></td>
<td>Cardiac arrhythmias, hypotension, respiratory depression which may require mechanical ventilation</td>
</tr>
<tr>
<td>Detomidine</td>
<td>5X (0.2mg/kg)</td>
<td>Tolerated by horses. Severe respiratory and cardiovascular changes that may lead to death.</td>
</tr>
<tr>
<td></td>
<td>10-40X (0.4-1.6mg/kg)</td>
<td></td>
</tr>
<tr>
<td>Romifidine</td>
<td>5X (600mcg/kg)</td>
<td>Bradydysrhythmias, apneustic respiration, respiratory stridor, sweating, and polyuria.</td>
</tr>
</tbody>
</table>

**Intra-Arterial Injection**

Intra-arterial injection is an uncommon complication following the insertion of a intravenous catheter. It is most commonly performed by inexperienced personnel. Clinical signs of an intra-
arterial injection usually occur within one jugular-brain circulation time. Extensor rigidity followed by generalized uncontrolled tonic-clonic type convulsions is typically observed following accidental intra-arterial injections of sedatives. Treatment is symptomatic. Diazepam or midazolam (0.05-0.1mg/kg IV) followed by thiopental if needed. Guaiifenesin would be an alternative/adjunct to the benzodiazepines. In these situations, it is prudent to postpone the procedure.

**Human Exposure**

There a numerous case reports in the medical literature describing accidental and intentional exposure to α2-adrenergic agonists [16-20] including one description of an ocular exposure [20]. A man lavaged his eyes accidentally with 800mg of xylazine and started to exhibit clinical signs of hypotension and bradycardia two hours post exposure despite copious ocular lavage with saline [20]. Most of the reports describe the exposure to xylazine from small doses of 200mg to over 1.5g[18]. There has been one report of deliberate exposure (intramuscular) to detomidine 50mg (+ butorphanol 100mg) in a man. Remarkably the person was awake, responsive and breathing spontaneously six hours post exposure. The human clinical signs mimic those observed in horse, bradycardia, profound somnolence, coma, hyperglycemia, miosis, hypertension or hypotension (long-term) and apnea. Most of the patients were treated supportively with mechanical ventilation and intravenous fluids. Interestingly some people accidentally injected with small amounts of xylazine still required respiratory support. Currently in North American there a no registered drugs for the treatment of α2-adrenergic agonist intoxication despite clinical trials evaluating the use of atipamezole as a reversal agent for dexmedetomidine [21, 22]. It is important to seek immediate medical attention if an exposure to any veterinary product has occurred.

**References:**

Loco-regional Anaesthetic Techniques for the Equine Head

Patrick Burns BVSc, MANZCVS, DACVAA, MRCVS

Ophthalmic Blocks
To properly exam the eye of a horse, sedation is usually required. This may take the form of an intravenous bolus or a constant rate infusion (see Standing Sedation). It is important to realize that not all the local blocks described here provide analgesia. The auriculopalpebral nerve block provides
akinesis to the eyelids. It is a motor block only. The following is a brief discussion of the techniques used to cause complete anesthesia of all the structures of the eye[1-3].

Sensory Nerve Blocks

**Corneal anesthesia**
Topical anesthetics such as tetracaine and proparacaine are used to examine the cornea of the eye as well as for corneal scrapings, swabs of the eye and other minor surgical procedures. It can also be helpful when performing an eye enucleation. Local anesthetics are cytotoxic so therefore should not be used for long-term analgesia. The onset of analgesia in humans is within one minute and duration of action is approximately 15-30 minutes. The metal shaft of a 25G needle may be broken off to help facilitate the instillation of the local anesthetic into the eye.

**Supraorbital nerve anesthesia**
The supraorbital nerve is the terminal ending of the opthalmic nerve (CN V). It is the sensory supply to the superior eyelid. It is used mainly for lacerations to the superior eyelid, placement of a subpalpebral lavage tube and enucleations. The supraorbital foramen can be located using Tóth’s law which states, when the thumb and third finger are placed on the medial and lateral canthi, the index finger will automatically be placed over the opening of the foramen. A 22-25G needle is inserted over the opening of the foramen and 2mL of local anesthetic may be injected. The needle can either be withdrawn and the operator wait for a few minutes before processing to insert the needle directly into the foramen or attempt to insert the needle into the foramen immediately. This technique may also block some of the small fibers coming from the auriculopalpebral nerve, thus resulting in motor blockade of the medial aspect of the superior eyelid[3]. The lacrimal nerve (ophthalmic nerve; CN V) a sensory nerve which supplies the lateral aspect of the superior eyelid, will also need to be desensitized. A line block (2-3mL) along the supraorbital rim extending from the lateral third portion of the eyelid to the lateral canthus will desensitize the lacrimal nerve. An alternative to the above technique is to make a line block along the entire superior orbital rim.

**Retrobulbar anesthesia**
The techniques described here will anesthetize the optic (CN II), oculomotor (CN III), trochlear (CN IV), abducens (CN VI) and parts of the trigeminal (CN V) nerve.
Four-point block: This is the most commonly used technique to anesthetize the ocular muscles of the eye. Either a 22 or 20G 2.5 inch spinal needle (with a slight curve) is inserted through the orbital septum at the 12:00, 3:00, 5:00 (or 7:00) and 9:00 positions. Avoiding the 6:00 position will minimize the chances of damaging the optic nerve. At each site deposit 5-10mL of local anesthetic. Ocular conus block: This is another technique used to anesthetize the ocular muscles of the eye. Palpate the lowest point to the supraorbital fossa. Insert a 22 or 20G 2.5 inch spinal needle which is directed 5° off vertical in a lateral to medial, and rostral to caudal directions. Observing the eye during the insertion, the eye will move dorsal as the tip of the needle engages the ocular conus. As the conus is penetrated by the needle the eye will rotate back to a neutral position again. Local anesthetic (10mL) may now be injected. No resistance should be detected.

**Anesthesia of The Inferior Eyelid**
There are two nerves which innervate the inferior eyelid, the infratrochlear and zygomaticofacial nerves both of which are branches of the opthalmic nerve (CN V). The infratrochlear is located
within a notch along the superior orbital rim near the medial canthus. It provides the sensory innervations to the medial canthus, nictitans, and lacrimal glands. The zygomaticofacial is located on the lateral and inferior aspect of the orbital rim. The zygomaticofacial supplies approximately 75% of the sensation of the inferior eyelid. Local anesthesia 3-5mL may be deposited at each site. An alternative is to make a line block along the inferior orbital rim which extends dorsally at the medial canthus.

**Motor Nerve Block**
The auriculopalpebral nerve is one of the terminations of the facial nerve (CN VII). The auriculopalpebral nerve crosses the medial aspect of the zygomatic arch near the base of the ear, and then follows the arch along its dorsomedial aspect. It supplies the motor innervations to the orbicularis oculi muscle. This block is used to produce akinesis of the eyelids and to facilitate an eye examination. A 22G needle may be inserted along the dorsomedial aspect at the most dorsal aspect of the zygomatic arch. A small amount of local anesthetic (3-5mL) may be deposited in a fan-like manner at this site under the fascial plane. Do not forget to use some artificial tears or ointment for a few hours post-administration because of the inability to blink and to prevent dessication of the cornea.

**ADJUNCT THERAPIES**
The onset of action of lidocaine can be shortened by the addition of bicarbonate. For every 9.5mL of lidocaine 2% add 0.5mL of sodium bicarbonate 8.4%. By alkalizing the solution, the lidocaine will penetrate the neuronal tissue quicker, thus the onset time will be shorter. Alkalization of the solution will also reduce the pain on injection of lidocaine. If using bupivacaine, the amount of sodium bicarbonate required is only one-tenth the volume. The addition of epinephrine 5µg/mL (0.1mg epinephrine diluted to 20mL of saline) to the solution will approximately double the duration of a blockade by lidocaine by reducing the amount of vascular absorption of the local anesthetic from the infiltration area. This is not true for bupivacaine. The addition of the epinephrine also has the advantage of causing a transient tachycardia if an accidentally intravascular injection has taken place. Monitoring the heart rate during the performance of the locoregional anesthesia can help detect this phenomenon.

**Dental Blocks**
Anaesthesia of the upper lip, nose and incisors
The domain blocked by the desensitisation of the infraorbital nerve as it exits the infra-orbital canal consists of the upper lip, nostril, the roof of the nasal cavity and the skin rostral to the infra-orbital canal. This anatomical location can be found by drawing an imagery line between the nasomaxillary notch and the rostral end of the crest of the face. Pushing the levator labii superioris muscle dorsally at the mid-point of imaginary this line, the infra-orbital foramen can be detected. It will be dorsal to the line by approximately 2.5cm. A small needle can be used to infiltrate this region with 3 to 5 mL of local anaesthetic. Note this will not desensitize any of the teeth since all the nerves supplying the teeth branch off the infra-orbital nerve from within the canal. In order for any teeth to be desensitized, the local anaesthetic must be deposited within the infra-orbital canal. By inserting a short needle into the canal with the bevel facing axially towards the nerve will help to minimise the risk of nerve injury. This will encourage the needle to move more laterally within the canal.
Occluding the foramen during the injection will facilitate the passage of the local anaesthetic along the canal, thus desensitizing the teeth.

*Anaesthesia of the lower lip, gingiva, incisors and premolars*
A similar approach is used to desensitise the soft tissue structures of the rostral mandible and incisors depending upon where the local anaesthetic is infiltrated. The mental foramen is located within the interdental space and on the lateral aspect of the mandible by pressing down on the tendon of the depressor labii inferioris dorsally. It is possible to have multiple foramina with an individual. Depositing a few mL of local anaesthetic at the entrance to the mental foramen will desensitise the soft tissues only. In order to desensitise the incisors, the local anaesthetic must be deposited within the canal. To facilitate this, a 30 degree bend is made at the shaft of the needle and hub. Caution must be used when inserting the needle into the canal. I often inject a small amount of local anaesthetic at the entrance before processing to insert the needle into the canal.

*Anaesthesia of the maxillary teeth*
The maxillary nerve block will desensitise the entire ipsilateral maxillary dental arcade, paranasal sinuses, and parts of the nasal cavity. The maxillary nerve enters the infra-orbital canal via the pterygopalatine fossa, which is located on the caudal aspect of the maxillary bone. The point of insertion of the needle is ventral to the zygomatic arch and is located approximately in line with the lateral third of the eye. The needle is inserted perpendicular to the skin to a depth of 5 to 6.5 cm. Make sure you aspirate first before you injected the local anaesthetic (10 to 15mL) as the maxillary vessels are nearby. To increase your chances of success, the local anaesthetic can be deposited along this vertical line in a fan-like manner.

*Anaesthesia of the mandibular teeth*
The success of the mandibular nerve block is the most difficult to achieve consistently. The landmarks vary and change over time with the age of the horse due to the wear of the teeth. The mandibular foramen is located on the medial aspect of the mandible and is approximately in line with the occlusal surface. Draw an imaginary line from the lateral canthus of the eye, which is perpendicular to the line along the occlusal surface. The foramen is located ventral to this intersection. The needle is inserted up against the medial aspect of the mandible to a depth of 10 to 15 cm directing it towards the lateral canthus of the eye. If the needle is inserted too far medially, it is possible to desensitise the ipsilateral same of the tongue.

References:
Introduction
The diagnosis and treatment of sinus disease in the field is complicated by the complex anatomy of the structures, difficulty in access and their large size. This session will emphasize examination and diagnostic interpretation in the field setting and available field treatment options for select pathologies.

Anatomy
The extensive paranasal sinus system of the horse consists of 6 pairs of compartments: the dorsal, middle and ventral nasal chonchal sinuses, the sphenopalentine sinus, the frontal sinuses and the maxillary sinus divided into rostral and caudal compartments (Figure 1). While disease can occur in any compartment, that of the frontal and maxillary compartments are most common, and most amendable to diagnostics and treatment in the field. However, because of communication between different compartments, extension of disease into the smaller, less accessible sinuses can occur and can be a cause of disease refractory to treatment or recurrence.

The maxillary sinus opens into the middle nasal meatus via the nasomaxillary opening and this is the main drainage point for the extensive sinus system. Because of direct communication to the nasal cavity and its close association with the alveoli of the cheek teeth the maxillary sinus is more predisposed to disease that the other compartments.

Physical Exam Findings Associated with Sinus Disease
Physical exam findings can raise a high suspicion of sinus disease without the use of more advanced diagnostics such as radiography or endoscopy. Most sinus diseases cause a unilateral nasal discharge as the nasomaxillary opening draining the sinuses is rostral to the nasal septum. If bilateral discharge is present, the guttural pouches, pharynx or lower respiratory system should be investigated. Another common feature of sinus disease is distortion of the facial bones due to pressure from underlying pathology. Close inspection for subtle (or not so subtle) changes in facial asymmetry can indicate disease. By the same token, masses, fluid and general inflammation can also compress the nasal passage reducing airflow through the affected side. Placing hands over each nostril as the horse breaths is an easy way to determine airflow asymmetry. Abnormal respiratory noise by impingement of the chonchae into the nasal passage or septum deviation can also be present. Exophthalmos and epiphora can also be noted on the affected side if there is compression of the orbit or nasolacrimal duct that traverses the maxillary sinus.

Percussion can be used to detect space-occupying material within the sinuses. To percuss the sinuses, the fingers of one hand are sharply tapped against the overlying bone from one side to the other. A ‘duller’ or ‘lower’ tone can be appreciated versus the normal side when the space is filled with fluid or soft tissue. An oral exam should also be performed to detect dental abnormalities that may be associated with secondary sinusitis and/or neoplasia.

**Adjunctive Diagnostics**
If possible, obtaining sinus/dental radiographs in the field is valuable. Digital systems have vastly improved field interpretation by allowing manipulation of the images post acquisition making perfect technique less essential. Lateral, oblique and DV views are possible in the field and standing sedation is recommended for adequate head positioning. Halters should either be removed or replaced with a rope variety. Lateral and oblique views with the affected side displaced proximally are most valuable for examination of the roots of the cheek teeth in communication with the maxillary sinus and evaluation for fluid lines. Suggested technique is 80 kVp and 1.4 mAs with standard field units for lateral and oblique views with the beam centered 5cm below the medial canthus of the eye. The dorsoventral view is valuable for comparing soft tissue densities within the sinuses from side to side, as well as, determination of septal deviation. The beam is centered right between the eyes on midline with a suggested technique of 80 kVp and 1.6 mAs. Common errors in acquiring images include a tendency to get too close to the animal when shooting, which results in a ‘fuzzy’ image and makes anatomic landmarks difficult to recognize, and not labeling the images correctly. Getting as much anatomy on the plate as possible and using proper labels at the time of acquisition is helpful for making interpretations. A nice review of equine skull radiography in the field can be found here: Equine vet. Educ (2013) 25 (12) 643-652 doi:10.111/eve.12086

Endoscopy via the nasal passages is limited in ability to detect abnormalities of the sinuses. Occlusions of the nasal passage, blood or pus draining from the nasomaxillary opening, masses protruding into the nasal passages and abnormalities of the ethmoturbinates can be indications of sinus disease. Direct endoscopy via sinus trephination (later session) can be of value and can be performed in the field. Direct examination of communicating tooth roots and masses can occur in this fashion. Biopsy instruments can often be passed through the same trephine hole should sampling be desirable.

**Diagnostic Features & Treatment of Paranasal Sinus Disease**
Sinusitis
Sinusitis can primary in nature or secondary, usually to tooth root infection. Distinguishing between the 2 can be challenging but is necessary for resolution.

Clinical Features: Unilateral nasal discharge is the most common sign of both types of sinusitis although with secondary the discharge tends to be more fetid. Facial distortion is not common with primary unless in very chronic stages but can be present with apical root infections. Sinus tracts from the tooth root to the skin can be present in cases of secondary sinusitis. Epiphora may be present. Oral exam may reveal tooth abnormalities with secondary sinusitis.

Radiographic Findings: Primary – soft tissue density +/- presence of fluid lines within the maxillary and possibly frontal sinus. Secondary – as per primary with the addition abnormalities associated with 1 or more tooth roots. Blunting, lysis, distortion of the root are common findings and distinct soft tissue masses above a root may be indicative of an inflammatory granuloma

Endoscopic Findings – limited to drainage from the nasomaxillary opening
Treatment: Primary sinusitis diagnosed early can usually be addressed in the field via repeated sinus lavage with saline via sinus trephination (later session). The trephine is placed through the frontal sinus to allow gravity flow of saline throughout the entire paranasal sinus system. Lavage is repeated daily until the flush retrieved through the nostril is clear (anywhere from 2-6 days). Systemic antibiotics are based on culture & sensitivity of sample obtained at the time of trephine. Chronic sinusitis may form inspissated pus that is difficult to clear with lavage only. Debridement through a trephine hole is recommended before serial lavage. Some of these cases may be refractory treatment and may require more aggressive surgical debridement through a large flap. The inciting cause of secondary sinusitis must be addressed before sinus infection can be resolved. Typically this involves removal/repulsion of the tooth in question.

Ethmoid Hematoma
Clinical Features: Intermittent, unilateral epistaxis, reduced airflow through the affected side, airway noise at exercise, percussion is usually unremarkable, facial asymmetry not a typical finding.

Radiographic Findings: Smooth walled, well circumscribed soft tissue opacity, typically in the area of the ethmoid turbinates. On DV view the mass can be seen in the nasal passage but usually does not deviate the septum.

Endoscopic Findings: The lesion can often be seen extending into the nasal passage at the level of the ethmoid turbinates but absence of this finding does not rule out a lesion contained within the sinus. Occasionally hematomas in locations other than ethmoid region are found on direct sinus endoscopy.

Treatment: Field treatment is often limited to intralesional formalin injection under endoscopic guidance. Briefly, through an injection catheter placed through the endoscope approximately 50 ml of 10% neutral buffered formalin (4% formaldehyde) is injected into the protruding nasal component of the hematoma or through a trephine hole into the hematoma's sinus component. Injections are repeated every 3-4 weeks until lesion is too small to treat. Those refractory to treatment may need...
to be removed through a surgical flap. Recurrence is common and repeat endoscopy should occur every 6 months to treat recurring lesions while they are small.

**Paranasal Sinus Cyst**

Clinical Features: Facial asymmetry is a common finding, as well as, unilateral nasal discharge, reduced airflow and dullness on percussion. Oral exam is unremarkable.

Radiographic Findings: Often multiloculated soft tissue densities and fluid lines – more commonly in the maxillary sinuses are noted. On DV view there is often considerable nasal septal deviation due to soft tissue opacity protruding into the nasal passage. Tooth roots may appear flattened or distorted and can mimic tooth root abscess.

Endoscopic Findings: Nonspecific. Can be difficult to pass the scope. In very large cysts, protrusion through the nasomaxillary opening is possible.

Treatment: Not amendable to treatment in the field. Draining the cyst through a trephine hole will only temporarily resolve the problem. Radical surgical resection of the cyst and involved mucosal lining is required.

**Neoplasia**

Clinical Features: Squamous cell carcinoma is the most common sinonasal tumour of the horse. Clinical features include unilateral (often fetid) nasal discharge and facial distortion/swelling. Epistaxis, exophthalmos, epiphora and enlarged regional lymph nodes may be present.

Radiographic Findings: Soft tissue opacity within sinus often with septal deviation. Sinus tumours are often invasion so bone should be carefully evaluated for disruption, cortical bone reaction and lysis.

Endoscopic Findings: Masses often protrude into the sinus cavity and can be visualized on endoscopy. Neoplasia can be difficult to distinguish between paranasal sinus cyst in early cases. Biopsy (preferably though a trephine directly in the sinus) is diagnostic. Typically when you biopsy a paranasal sinus cyst copious amount of clear yellow cyst fluid flow through your trephine site. This does not occur with a neoplastic mass.

Treatment: Sinonasal tumours are aggressive and respond poorly to surgical treatment due to their extensive nature and invasiveness by the time of diagnosis.
Anatomy
The guttural pouch (GP) is a cornucopia of potential life-threatening issues for the horse, largely because its unique anatomy provides intimate contact with the nervous, respiratory and cardiovascular systems. The GPs are paired extensions of the eustachian tubes that connect the pharynx to the middle ear. Each pouch is divided into a medial and lateral compartment by the stylohyoid bone and communicates with the pharynx via the pharyngeal orifice. The medial compartment contains the internal carotid artery, cervical sympathetic trunk, and vagus, glossopharyngeal, accessory & hypoglossal nerves (see diagram). The lateral compartment contains the external carotid & maxillary arteries and facial arteries. The mandibular lymph nodes, salivary glands, esophagus, and pharynx all border the limits of the GP and can also be affected by disease.

Examination
Clinical signs associated with disease of the GP are similar to those of other upper respiratory conditions, namely, nasal discharge and dyspnea. In addition, dysphagia and abnormal head position may be observed. When hemorrhage from the nostrils is observed, GP mycosis should always be ruled out.

The GP can be examined by palpation, endoscopy and radiography. Palpation just caudal to the ramus of the mandible and ventral to the base of the ear may provide a clue that the guttural pouch may be involved in the upper respiratory signs. Distention of the pouch(es) from empyema or tympany can be palpated externally.

http://cal.vet.upenn.edu/projects/eqairway/nrmlgutt2.htm
Lateral radiographic projections of the pouches can demonstrate fluid lines, fractures of bony structures, exostoses of the stylohyoid bone, and space occupying masses. Be sure to center the beam caudal to the ramus of the mandible to ensure the entire GP is imaged.

GP endoscopy provides the most information regarding GP disease. Signs of hemorrhage or purulent material draining from a guttural pouch opening on endoscopy examination of the upper airway indicate that the pouch itself should be investigated. However, no upper airway endoscopic examination is complete without including both GPs. Entrance into the pouch with the endoscope can be tricky and requires a guide wire to assist the scope through the pharyngeal orifice. The guide wire is passed down the biopsy channel of the scope and inserted into the pouch. The endoscope is then passed over the wire with a 90 degree clockwise rotation of the wrist to enter the pouch. A Chambers mare catheter passed into the pouch from the opposite nostril can also open the orifice enough to allow passage of the scope. Once in the pouch it is examined with particular attention paid to the roof for signs of fungal plaques and floor for signs of lymph node enlargement or empyema.

**Guttural Pouch Tympany**

GP tympany is a condition seen in young foals and up to 1 year of age and appears more commonly in fillies and the Arabian breed. Essentially it is an accumulation of air in 1 or both pouches as a result of a ‘one way valve’ due to a functional defect of the pharyngeal orifice. In most cases, there is no gross anatomic abnormality of the GP opening. The affected pouch is distended forms a non-painful, elastic swelling in the throat latch area. Severe distention is usually well tolerated by the animal but can cause dyspnea, dysphagia and inhalational pneumonia. Secondary infection is not uncommon.

Tympany is diagnosed based largely on clinical signs and signalment. Radiographs will show large amounts of air in the guttural pouch but it can be difficult to distinguish between unilateral or bilateral involvement.

Temporary alleviation can be achieved by Foley catheter placement in the affected pouch. A recent case series showed ability to permanently resolve the condition by placing indwelling Foley catheters for 3 weeks. Catheters are placed under sedation and endoscopic guidance and then sutured to the nostril. Maintenance can be difficult, especially in nursing foals. In most instances, surgical treatment is necessary. Laser fenestration of the median septum between the pouches offers a fairly non-invasive surgical procedure that can be performed on a standing, sedated patient. This allows the trapped air to escape out the ‘normal’ opening of the unaffected pouch. If both pouches are affected, creating a salpingopharyngeal fistula with the laser through the pharynx into the GP is usually successful. If transendoscopic laser surgery is unavailable, conventional surgical approaches to the GP are used to create a median septum fenestration (later session).

**Empyema**

Empyema is defined as the presence of purulent material and/or chondroids within 1 or both GPs. Chondroids consist of concretions of inspissated purulent material that form in approximately 20%
of empyema cases. Empyema can affect any age of horse but usually occurs in younger horses. Streptococcus zooepidemicus and Strep equi from an upper respiratory tract infection are the most common cause of the empyema but retropharyngeal lymph node abscessation & rupture, stylohyoid bone fracture or pharyngeal perforation by a nasogastric tube are also reported causes.

Clinical signs include intermittent nasal discharge, throatlatch area swelling, dyspnea and difficulty swallowing. Diagnosis is made on palpation and endoscopic examination. Purulent material can usually be seen draining from the affected pouch opening and, in severe cases, the pharyngeal collapse may be present. Direct endoscopy of the pouch may demonstrate purulent material, presence of chondroids or evidence of a ruptured retropharyngeal lymph node through the floor of the pouch. Fluid aspirates should be taken for culture to rule out Strep equi as the source.

Treatment is based on daily lavage of the affected GP and systemic antibiotic therapy. Lavage can be accomplished by placing indwelling nasal catheters or by daily repetition of endoscopic lavage with 1-2 L of saline. While it may be tempting to add disinfectant or antibiotic to the lavage solution, this provides little or no benefit, and increases risk of chemical injury to the cranial nerves that traverse the pouch. Lavage is continued until the pouch is clean and systemic antibiotics are continued for 2-3 weeks. If chondroids or severe dyspnea is present surgical therapy may be necessary. In cases of chondroids, the guttural pouch is approached through a conventional open approach to remove the chondroids. Lavage is then carried out as above. If severe dyspnea is present, temporary tracheotomy may be necessary before proceeding with the lavage therapy (later session).

**Mycosis**
Mycosis is the most potentially life threatening condition of the GPs; not because of the fungus itself, rather where the fungus likes to grow within the pouch. There is no apparent age, breed or geographic predisposition for the disease and the cause is unknown. Fungal plaques consist of diphtheric membranes of various sizes composed of fungal hyphae, necrotic tissue & cell debris and secondary bacterial infection. Lesions are usually present on the roof of the guttural pouch in either the medial or lateral compartment, directly on top of the internal carotid or maxillary arteries. In rare occasions, lesions are localized away from the major vessels overlying the nerves in the medial compartment. Eventually the lesions erode through the wall of a major vessels leading to acute, severe, bilateral epistaxis. The 1st episode of hemorrhage is fatal in approximately 50% of cases.

Outward signs of disease are usually not apparent to the owner until the 1st episode of epistaxis. However, more subtle signs associated with inflammation of the cranial nerves of the GP such as Horner’s syndrome, dysphagia, cough, aspiration pneumonia or laryngeal hemiplegia may be present. In other cases, mild intermittent episodes of epistaxis may occur before the major bleed. Endoscopy is essential for diagnosis. However, during an acute hemorrhage episode, it will be impossible to see anything other than active hemorrhage from the GP opening. In fact, attempting to get into the pouch may be contraindicated as a forming clot may be disturbed. It is necessary to wait several days before the clot is organized enough to actually observe the plaque, and its location, in the affected pouch. Location is important in determining what vessels need to be addressed in treatment.
Horses presented in active severe hemorrhage should be stabilized and supported by IV fluids and blood transfusion (if available). Some surgeons advocate temporary common carotid artery ligation via cut down in the jugular area to help stop the hemorrhage. However, due to collateral circulation of the Circle of Willis, there is potential actually to increase blood flow via retrograde flow to the internal carotid artery.

After stabilization of the horse, definitive treatment should ensue and requires surgical referral. In the case of mycosis without vessels involvement, repetitive topical treatment of fungal plaques with endoscopic guided application of anti-fungals can be successful. However, surgical occlusion of affected vessels via a number of different methods is the treatment of choice. This prevents the risk of fatal hemorrhage through the affected vessel and it is widely accepted that occlusion of the affected vessels also hastens spontaneous resolution of the mycotic lesion.

If the horse survives the major hemorrhage episode, and occlusion procedures are performed, prognosis is generally fair to good. Concurrent dysphagia or other neurologic problems associated with mycosis may take several months to recover function after resolution of the lesion.

**Temporohyoid Osteopathy**

Temporohyoid osteopathy (THO) is a progressive disease of the middle ear and the components of the temporohyoid apparatus. The cause is thought to be hemotogenous spread of infection of the inner/middle ear to the bones of the temporohyoid apparatus causing them to thicken and lead to fusion of the TH joint. In older horses, degenerative changes to the joint may underlie development of the disease (although usually milder).

Early clinical signs can include head shaking, reluctance to take bit or position head or other non-specific behavioral changes. An acute onset of asymmetric ataxia, head tilt and spontaneous nystagmus with the slow component to the affected side can occur. Signs may be exacerbated by blind-folding. Signs of facial nerve dysfunction may also be present (check for corneal ulcers).

Radiographs of the skull may depict proliferation and osteitis of the affected bones. A DV view with both TH joints on the film can be helpful. However, endoscopy of the GP in most cases is a more sensitive method for detecting stylohyoid bone and TH joint involvement – leading to diagnosis of THO. Both sides should be examined as the disease can be bilateral with the more severely affected sign showing the clinical signs.

Medical treatment includes broad spectrum anti-biotics and anti-inflammatories. Neurologic signs may persist, especially if treatment is delayed. Referral for surgical treatment may be required. Surgery is aimed at disrupting the temporohyoid apparatus to decrease painful movement of the joint and inflammation (ceratohyoidectomy). In one study, surgical management allowed for substantial improvement at 1 year in 89% of horses.
**Introduction**
Many surgical procedures for treatment of upper airway conditions in horses are amendable to performance in the field. While some procedures are moderately challenging and require general anesthesia, many are amendable to standing sedation and local anesthesia and are not difficult to perform with a thorough understanding of anatomy. Less than ideal aseptic conditions are acceptable as, by nature, the upper airway is contaminated and is impossible to sterilize allowing field conditions to be a viable operating environment. Clipping and prepping incision sites as per surgical protocol is warranted and sterile gloves should be worn. Caps, masks, gowns, and drapes are optional.

**Tracheostomy**
Level of difficulty (1-5): 1 – unless the horse is actively trying to die – then 5
Standing Procedure: Yes
Special Instruments: No

Tracheostomy may be performed with the horse standing or under general anesthesia. In emergency situations, the horse may be extremely panicked when unable to breathe and it is important to keep self-preservation in mind. Therefore, it is best to perform this procedure if there is any anticipation of upper airway obstruction BEFORE the upper airway actually becomes obstructed. Tracheostomy is performed at the junction of the upper and middle thirds of the neck. The trachea is located very superficially at this level. Aftercare requires twice daily cleaning of the site and tube to prevent accumulation of tracheal secretions. Once removed the site heals by 2nd intention over the next several weeks.

**Technique:**
1. Understanding sedation palpate the trachea at the junction of the upper and middle thirds of the neck and inject 5-10 ml of local anesthetic in a line block on midline. (If the horse is actively trying to die, forgo this part)
2. Make a 10 cm incision with a #10 scalpel blade on the ventral midline overlying the trachea through the skin, subcutaneous tissues and the cutaneous colli muscle.
3. The paired sternothyrohyoid muscle bellies are bluntly divided along the ventral midline for a distance of 8 cm and are held in retraction to expose the trachea.
4. Palpate the tracheal rings and the annular ligament between them. The annular ligament between 2 adjacent cartilage rings is incised parallel to the orientation of the rings and is lengthened to allow placement of the tracheal cannula. The incision should NOT exceed one third the circumference of the trachea.
5. A finger can be inserted into the lumen of the trachea to help guide the tracheal cannula into position. PRACTICE TIP: - in a pinch, if you don’t have a tracheal cannula, the handle off a 4L bleach or mineral oil container makes an excellent tracheal cannula. So does a segment of garden hose.
Sinus Trephination
Level of difficulty: 1
Standing Procedure: Yes
Special Instruments: Jacob’s Chuck and ¼ - 5/8 inch Steinmann Pin

Sinus trephination is useful for diagnostic and therapeutic access to the paranasal sinus cavities. Trephination can be performed to collect culture or histology samples, explore the sinus endoscopically or provide access for lavage. The frontal, caudal and rostral maxillary sinus trephination sites are depicted in the diagram (end of notes). In horses under 3 years of age, the alveoli of the cheek teeth may impede access to the maxillary sinuses but as the horse ages, the space within these sinuses becomes larger.

Technique:
1. The trephination site for the frontal sinus (Figure below ‘A’) is at the midway point of a line drawn between the midline of the face and the highest point of the orbit. After placing a 1ml SQ bleb of local anesthetic make a small stab incision down to the bone with a #15 scalpel blade.
2. Assemble your Jacob’s with 1 inch of Steinmann pin exposed at the working end of the chuck.
3. Use the Steinmann Pin and Jacob’s chuck to trephine a hole through the facial bone into the frontal sinus. Free up and down movement of the working end of the pin within the sinus indicates full penetration.
4. For the caudal maxillary sinus (Figure below ‘B’) the trephination site is 1 inch below the medial canthus of the eye at the midway point of an imaginary line drawn from the medial canthus to the infraorbital foramen (this represents the path of the nasolacrimal duct and needs to be avoided) and the facial crest. Repeat steps 1-3 to penetrate the caudal maxillary sinus.
5. The trephination site of the rostral maxillary sinus (C) is approximately 2 inches rostral to the site of the trephination site for the caudal maxillary sinus, again at the midpoint of the path of the nasolacrimal duct and the facial crest. Repeat steps 1-3 to penetrate the rostral maxillary sinus.
6. If recurring lavage is the reason for trephination a regular extension set with the luer lock cut off can be placed into the hole and finger-trapped in place for fluid delivery.
7. Upon completion of lavage procedures or specimen collection the trephine site is left open and heals within several days.

Surgical Approach to the Guttural Pouch
Level of difficulty: 3 (but it’s a bit scary with all those big vessels close by)
Standing Procedure: Yes, depending on approach
Special Instruments: No but endoscopic guidance is preferable

Multiple approaches can be used to open the guttural pouch for removal of pus, chondroids, foreign bodies, mycotic plaques and to establish drainage but the Modified Whitehouse approach is the most amendable to standing field surgery (Figure below). Due to the vast density of major vessels and nerves in the pouch, extreme care and knowledge of anatomy is critical to prevent serious surgical complications. In all approaches, entrance through the pouch mucosa is done with a blunt
instrument and the incision is enlarged by blunt finger dissection to avoid sharp transection of important structures.

In the Modified Whitehouse approach, the skin incision is placed just ventral to the linguofacial vein. The major advantage of placing the skin incision here rather than on ventral midline (Whitehouse approach) is that dissection is placed through natural tissue planes instead of between muscle layers. Entrance to the pouch is the same as with the Whitehouse. Advantages of both Whitehouse approaches are direct access to the roof of the pouch due more ventral entrance to the pouch itself, excellent ventral drainage and, if necessary, it allows access to the medial septum so both pouches can be operated simultaneously. Although both approaches involve deep dissection, they do not appear to have a higher complication rate than the other approaches.

Technique:
1. The horse is heavily sedated and the head is placed on a stand to extend the head and neck and increase the size of the operating area.
2. A line block with 10-20 ml of local anesthetic is placed just ventral and parallel to the linguofacial vein.
3. A 12 cm incision is made at this site and the underlying omohyoideus muscle fascial attachment to the linguofacial vein is sharply separated with scissors.
4. Blunt dissection with fingers is then used to separate the fascia lateral to the larynx in a rostral-dorsal direction towards the ventral aspect of the pouch.
5. A Chambers mare’s catheter (bent 30 degrees) is passed through the ipsilateral nostril into the medial aspect of the pouch and directed towards the floor under endoscopic guidance by an assistant. The tip of the catheter can then be palpated through the surgical incision to help guide entrance into the pouch.
6. The wall of the ventral aspect of the pouch is then perforated by elevating it with Allis tissue forceps placed ½ cm apart and bluntly inserting Mosquito forceps through the wall. The incision is enlarged by opening the jaws of the instrument. Sharp incision or dissection should be avoided.
7. Alternatively, the blunt end of the Chamber’s catheter can be forced through the floor of the medial aspect of the GP under palpation guidance through the surgical incision and the hole can be widening with fingers.
8. Chondroids or inspissated pus evacuation or median septum fenestration can then occur.
9. The incision is left open for lavage and drainage

**Laryngotomy and Ventriculectomy**
Level of difficulty: 4
Standing procedure: Not recommended
Special instruments: Roaring burr

Laryngotomy refers to the approach used to enter the lumen of the larynx to perform such procedures as ventriculectomy, ventriculocordectomy, arytenoidectomy or soft palate surgery. Ventriculectomy refers to removal of the mucosal lining of the laryngeal ventricle located caudal to the vocal fold. It is usually performed to eliminate upper respiratory noise associated with left idiopathic laryngeal hemiplegia but its DOES NOT produce abduction of the arytenoid cartilage. It is not recommended as a sole procedure for racing horses affected with ILH. However it reduces soft
tissue collapse during exercise and be can be quite successful when performed on show and draft horses. It can be performed on a standing sedated horse but the blood drips in your eyes making visualization challenging. I recommend placing the horse in dorsal recumbency on triple drip injectable anesthesia. The laryngotomy incision is cleaned twice daily after surgery and heals by 2nd intention. Horses can return to exercise in 6-8 weeks.

**Technique:**

1. The horse is placed in dorsal recumbency on triple drip anesthesia and the landmarks of the larynx are palpated. A triangular depression can be felt between the cricoid cartilages and the paired thyroid cartilages.
2. A 10 cm incision is made though the skin centered at the level of the vertical rami of the mandible.
3. Once through the skin the paired sternothyrohyoideus muscles are exposed. A faint white fibrous band depicts the natural separation between the 2 muscles bellies. The muscles are separated (with scissors) until the ventral aspect of the larynx is exposed. Retraction of the muscles bellies with Weitlaner retractors makes exposure of the larynx easier.
4. The characteristic ‘V’ formed by the thyroid cartilages and the cricothyroid membrane caudal to the thyroid cartilages is palpated. Using your #10 scalpel blade, a sharp stab incision on midline is made through the cricothyroid membrane to gain exposure to the lumen of the larynx. Extend this incision cranially and caudally to maximize exposure but try not to cut the bordering cartilages. Place your Weitlaner retractor into the cricothyroid ligament incision can help maximize exposure of the luminal structures.
5. With an index finger, locate the laryngeal ventricles and then pass the roaring burr into the site. Once fully inserted, slowly rotate the burr to engage the ventricular mucosa while slowly drawing the burr back out of the ventricle.
6. Once enough mucosa is everted, clamp it with a Kelly hemostat and transect it proximal to the clamp with scissors.
7. Repeat the procedure on the other ventricle.
8. The surgical site is left open for drainage and heals by second intention.
Sinus trephination sites for the horse

Figure 45-14. Surgical approaches to the guttural pouch. A, Hyovertebrotomay. B, Viborg’s triangle. C, Modified Whitehouse. D, Whitehouse. 1, lateral compartment of the guttural pouch, which is partly separated from the medial compartment: 2) by the stylohyoid bone (3); 4, vertical ramus of the mandible; 5, wing of the atlas. (Redrawn from Freeman DE: Diagnosis and treatment of diseases of the guttural pouch: Part II. Comp Cont Educ Pract Vet 1980;2:525.)

Surgical approaches to the guttural pouch

1 Both figures Courtesy of Dr. David Freeman
Animal Welfare: Large Animal

Animal Welfare in the Context of Social Sustainability
Marina A. G. von Keyserlingk* and Daniel M. Weary

Introduction
Questions concerning the sustainability of food-animal producing industries have become the focus of intense public debate by social critics, animal advocates, and scientists. Specific concerns about the welfare of dairy cattle is nothing new; producers and veterinarians have always been concerned about the condition of animals in their care and have tried to ensure that they are healthy and well nourished (von Keyserlingk et al., 2009). In the tradition of good animal husbandry, good welfare can be seen largely as maintaining high levels of production and the absence of illness or injury. However, recent interest in farm animal welfare stems more from concerns about pain or distress that the animals might experience, and concerns that animals are kept under “unnatural” conditions, with limited space and often a limited ability to engage in social interactions and other natural behaviours. For instance the results of a recent survey indicated that providing assurances that dairy cattle are well treated, developing methods to incorporate pasture access and assurance of healthy products without relying on antibiotics or hormones, are all aspects deemed to be important by citizens when asked what they views on the ideal characteristics of a sustainable dairy farm (Cardoso et al., in press).

In addition to the tremendous increase in scientific research on the welfare of cattle, some new work has begun to investigate stakeholder views on dairy farming and practices common in the dairy industry (see review by Weary et al., 2015). An objective of the current paper is to summarize some of our recent work on stakeholder views. We focus on four common management practices (tail docking, pain mitigation for disbudding/dehorning, access to pasture and cow calf separation) and describe how research in the natural sciences and social sciences can be integrated to identify more sustainable practices.

Farm Animal welfare
For the purposes of this paper we have adopted the three part definition of animal welfare proposed by Fraser et al. (1997): 1) animals should exhibit good physical health and biological functioning, 2) animals should have the ability to live reasonably natural lives including the ability to perform natural behaviours that are important to them, and 3) animals should experience minimal negative psychological states and the presence of at least some positive psychological states. These different types of concerns can and do overlap. For instance, a lactating dairy cow unable to seek shade on a hot day (natural living) will likely feel uncomfortably hot (affective state) and may show signs of hyperthermia, and ultimately reduced milk production (poor biological functioning) (von Keyserlingk et al., 2009).

These three key concepts of animal welfare have been included in official definitions such as the World Organization for Animal Health (OIE) which defines an animal as being in good welfare if it is “healthy, comfortable, well nourished, safe, able to express innate behavior, and it is not suffering from unpleasant states such as pain, fear, and distress” (OIE, 2013).
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Definitions of sustainability frequently include three pillars, economic, environment and social, which should be weighted equally (see von Keyserlingk et al., 2013). Traditionally academics working in agriculture (for example Steinfeld et al., 2006; Foley et al., 2011), and farmers and others working in food animal production systems, have placed greater emphasis on the economic pillar. More recently sustainability discussions on animal agriculture have focused on the environmental concerns resulting in this aspect receiving much attention. For example, debates frequently discuss the role that food-animal production plays in competition for natural resources i.e. water, land, and energy, and how to mitigate any negative effects of food animal agriculture on the environment (Thornton, 2010). The fact that the social pillar has received the least amount of attention may be a consequence of it having an aspect of human values (Thompson, 1997), and because it is difficult to quantify using traditional natural science based metrics. Furthermore, values are influenced by cultural norms within societies (Boogaard et al., 2011). Despite these difficulties there is a growing recognition that the social pillar is an important component of sustainability (von Keyserlingk et al., 2013). This may be particularly true for production that takes place in intensive housing systems that are the subject of increased societal criticism (Thornton, 2010).

Animal welfare is an important social concern and, as such, needs to be integrated into the concept of sustainable agriculture, rather than made to ‘compete’ with environmental goals (Hötzel, 2014) and economic goals (von Keyserlingk and Hötzel, 2015). To achieve this we argue that those not directly involved in farming must be accepted as credible stakeholders in the discussions on the way farm animals are cared for.

Stakeholder engagement on contentious practices in dairy industry
Our perspective is that rather than focusing efforts on one-way efforts to ‘educate’ the public, we should instead develop methods of facilitating constructive, informed engagement among the stakeholders. We suggest that this approach will likely to be more effective in identifying shared concerns and potential solutions likely to find general appeal.

At The University of British Columbia (UBC) we have been using web-based platforms to provide opportunities for people within the dairy industry to discuss dairy management practices with each other and with members of the public interested in these issues. For example, UBC’s Cow Views site provided the opportunity for people to state their views, and also vote on the views of others. The idea was to get people discussing uncomfortable issues in dairy farming. Our aim was to use these discussions to provide farmers and the industry a better basis for making informed decisions about management on farms and policy for the industry.

For each issue, participants were given a brief background of the perceived advantages and disadvantages associated with each practice (see tail docking below for example). They were then asked to vote on whether or not the practice should continue or not. We recruited participants into multiple virtual ‘town hall’ meetings, such that participants could see each other’s responses, but participants in one meeting could not see the reasons discussed in other meetings. In this way each meeting provides an independent test of how this type of discussion unfolds. Also, an especially persuasive reason can only influence the votes within a single town hall meeting.
Our intention was not to collect a random or representative sample of any specific population, but rather to include a diverse range of participants to increase our chances of achieving saturation in views. The forum was made available on the Internet so anyone with Internet access could participate. To encourage participation of people in the North American dairy industry, we published brief articles in producer magazines (Progressive Dairyman and Ontario Farmer) that invited readers to participate. For the broader public samples we recruited online via Mechanical Turk (MTurk, www.mturk.com). Several studies have assessed this tool and concluded that this approach results in high-quality and reliable data (e.g. Buhrmester et al., 2011; Saunders et al., 2013; Rouse, 2015) that is more representative than many other samples (Mason and Suri, 2012; Rouse, 2015).

To provide context, for each of the specific issues we have summarized below we also state the current position in Canada’s Code of Practice, and where relevant have described policy in other parts of the world.

**Should we continue docking the tails of dairy cattle?**
The responses to this question are fully described in Weary et al. (2011).

Briefly, 178 participants were provided the following context:

“Tail docking dairy cattle first became common in New Zealand where workers thought this could reduce their risk of diseases like leptospirosis that can be carried by cows. Some milkers also preferred working with docked cows because the shortened tail was less likely to hit them in the parlor. Some people also felt that docking improved cow cleanliness, and cleaner cows should be exposed to fewer pathogens and have improved udder health.

There may also be disadvantages associated with docking. For some, at least, there is a ‘yuk’ factor of seeing cows without their tails. Docking might also cause pain, and prevents cows from using their natural fly-swatter. For these reasons several European countries including Norway, Sweden, the Netherlands, the United Kingdom, and Switzerland have prohibited tail docking of dairy cattle.

More recently, Canada’s new Code of Practice for the Care and Handling of Dairy Cattle states that dairy cattle “must not be tail docked”.
In the United States, about 40% of dairy cows have docked tails.”

Participants were then asked, “Should we continue docking the tails of dairy cattle?”

Approximately 79% of participants were opposed to docking (i.e. responded “No” to the question). Responses varied with participant demographics (e.g. females were more likely than males to oppose docking), but in every demographic sub-group (e.g. by gender, age, country of origin and dairy production experience) the majority of respondents were opposed to tail docking. Common reasons for opposition to docking included the lack of scientific evidence that docking improves cleanliness or udder health, that docking is painful for cows, that docking is unnatural and that tails are important for controlling flies. Some respondents in favour of docking cited cow cleanliness as an issue, despite the scientific evidence showing no positive effect of docking on cow cleanliness or udder health. Additional reasons included protecting producer safety.
These results illustrate the range of reasons that are cited for supporting and opposing tail docking. This approach can be used to better target outreach efforts (e.g. improving farmer education on the lack of positive effects of docking on cleanliness and udder health while addressing concerns about producer safety).

Given the extent of public opposition to this practice it is not surprising that in some countries tail docking has been banned, including Norway, Sweden, the Netherlands, the United Kingdom and Switzerland. This has also likely motivated corporations to take a stand on this issue as part of their corporate social responsibility practices. For example, Nestle, the world’s largest food company, has announced their objection to tail docking.

In Canada, dairy producers have taken a clear position on this issue. Our Code of Practice for the Care and Handling of Dairy Cattle has a requirement that cows “must not be tail docked unless medically necessary.” This is also the position of the Canadian Veterinarian Association and the American Association of Bovine Practitioners. Most recently the National Federation of Milk Producers in the US announced that members of their assurance program will be prohibited from tail docking their cows effective January 1, 2017.

**Should we provide pain relief for disbudding and dehorning dairy calves?**
The responses to this question are fully described in Robbins et al. (2015).

For this issue participants were provided the following context:

“The developing horns of dairy calves are typically removed to reduce the risk of injuries to farm workers or other cattle that can be caused by horned cattle. Horns of calves three months of age or older are normally removed surgically (“dehorning”) by scooping, shearing or sawing. Horn buds of younger calves are typically removed (“disbudding”) using a caustic paste or a hot iron.

There is considerable scientific evidence that all of these procedures cause pain. The immediate pain can be reduced using a local anesthetic to provide a nerve block – this procedure has been used safely for decades and costs just pennies a shot. Pain can persist 24 hours or more; this longer lasting pain can be reduced using non-steroidal anti-inflammatory drugs (like the ibuprofen you take for a headache). Providing calves a sedative before the procedure can reduce handling stress and make the procedure easier to carry out.

In many countries some pain relief is required. For example, Canada’s new Code of Practice for the Care and Handling of Dairy Cattle requires that pain control be used. Approximately 18% of dairy farms in the United States report using pain relieving drugs for disbudding or dehorning dairy calves.”

Participants then answered the question “Should we provide pain relief for disbudding and dehorning dairy calves?”

Participant composition was as follows: dairy producer or other farm worker (10%); veterinarian or other professional working with the dairy industry (7%); student, teacher or researcher (16%); animal advocate (9%) and no involvement with the dairy industry (57%).
Of 354 participants, 90% thought pain relief should be provided when disbudding and dehorning. This support was consistent across all demographic categories suggesting the industry practice of disbudding and dehorning without pain control is not consistent with normative beliefs. The most common themes in participants’ comments were: pain intensity and duration, concerns about drug use, cost, ease and practicality and availability of alternatives.

These results show a clear disconnect between current practice (with many farmers failing to provide pain control) and the attitudes of participants (including dairy producers) in these virtual town hall meetings. Causing pain to animals under our care, especially when this pain can easily be prevented, no longer seems acceptable. Our challenge is to find ways of getting pain control techniques applied widely on dairy farms.

In Canada, dairy producers have also taken a clear position on this issue. The Code of Practice for the Care and Handling of Dairy Cattle requires that “Pain control must be used when dehorning or disbudding.” In many countries (i.e. Sweden, Denmark, Netherlands, New Zealand and Australia) pain control for disbudding and dehorning is a legal requirement (ALCASDE, 2009; NAWAC, 2005; PIMC, 2004).

**Should dairy cows be provided access to pasture?**
The responses to this question are fully described in Schuppli et al. (2014).

For this issue participants were provided the following context:

“On many dairy farms cows are always kept indoors. Some dairy farmers believe that well-designed indoor housing provides a more comfortable and more suitable environment for the cows. In addition, some farmers keep cows indoors to more easily provide and control diets formulated to sustain high milk production.

Others consider pasture access to be important. For example, some believe that grazing is more environmentally sustainable, that pasture provides a healthier and more comfortable environment for cows, and that grazing is a natural behaviour important for cows.

Participants then answered the question “Should dairy cows be provided access to pasture?”

A total of 414 people participated. Providing access to more natural living conditions, including pasture, was viewed as important for the large majority of participants, including those affiliated with the dairy industry. This finding is at odds with current practice on the majority of farms in the United States where less than 5% of lactating dairy cows have routine access to pasture (see USDA 2007). To our knowledge there is no research indicating about how many lactating cows in Canada have routine access to pasture.

Participant comments showed that the perceived value of pasture access for dairy cattle went beyond the benefits of eating grass; participants cited as benefits exposure to fresh air, ability to move freely, ability to live in social groups, improved health, and healthier milk products. To
accommodate the challenges of allowing pasture access on farms, some participants argued in favor of hybrid systems that provide a mixture of indoor confinement housing and grazing.

Despite the public indicating that access to pasture is important (see also Cardoso et al. 2016), the Code of Practice is largely silent on this issue, recommending only “for bedded-pack or composted-pack barns, provide access to pasture or an exercise or an exercise yard to decrease labor and bedding requirements.” In contrast, Sweden requires that cows be given pasture access during summer months (Ministry for Rural Affairs -Government Offices of Sweden, 2009).

**Should dairy calves be separated from the cow within the first few hours after birth?**

The responses to this question are fully described in Ventura et al. (2013).

For this issue 195 participants were provided the following context:

“Dairy farmers often remove the calf from within the first few hours of birth. This is done in response to several concerns including the following: the calf may become infected from pathogens carried by the cow or her environment; the calf may become injured by the cow or the barn equipment; the calf will not be able to nurse from the cow and receive adequate colostrum (first milk produced by the cow after birth) and milk; the calf will drink too much milk which increases the farmer’s cost of feeding and increases the risk of diarrhea; allowing the cow and calf to bond will result in greater separation distress when separation does occur; farms are often not well designed for cow-calf pairs, so keeping cows and calves together can be considered an extra chore. Others consider that some form of cow-calf contact is an important element of natural behavior, and believe that this contact is beneficial to the cow and calf. On these farms the cow and calf are kept together for days or even weeks after birth.”

Participants then answered the question “Should dairy calves be separated from the cow within the first few hours after birth?”

Opponents of early separation contended that it is emotionally stressful for the calf and cow, it compromises calf and cow health, it is unnatural, and the industry can and should accommodate cow-calf pairs. In contrast, supporters of early separation reasoned that emotional distress is minimized by separating before bonds develop, that it promotes calf and cow health, and that the industry is limited in its ability to accommodate cow-calf pairs. Opponents of separating calves from their cows in the first few hours after birth often based their views on the emotional experiences of cows and calves. They compared the bond of a cow and her calf to the bond between mother and offspring in other species.

A major theme raised by proponents was that separation was inevitable, and that early separation was easier on the cow and calf than separation at a later age. There is considerable scientific evidence in support of this claim. Separating calves at an older age results in a much stronger response (high rates of vocalization and other activities) in comparison with calves separated soon after birth (Flower et al., 2003). Some respondents also believed that early separation minimized disease transmission from the cow. We are aware of little evidence to support this link.

The Canadian Dairy Code of Practice (NFACC 2009) states the following:
“Generally, dairy calves are separated from their mothers shortly after birth. There are benefits to both calf and dam by allowing the pair to bond. Allowing the calf to spend a longer period of time with the dam may result in lowered morbidity and mortality in the calf; however, separation stress to both the cow and calf will be higher the longer they are together. Cow health is generally improved by allowing the calf to suckle (related to oxytocin effects on the post partum uterus)”.

Based on this summary of information the Code provides the following recommended best practice – “reduce separation distress by either removing the calf shortly after birth or by using a two-step weaning process.”

The fact that cows and calves are routinely separated at birth is an issue that the public is largely unaware of (Ventura et al., submitted), perhaps explaining why this issue has received little attention within non-dairy audiences. However, we speculate that external stakeholders will become increasingly unwilling to accept this practice.

Conclusions
The examples illustrated in this paper show how social science methodologies can document the shared and divergent values of different stakeholders, the associated beliefs regarding the available evidence, and the barriers in implementing changes. In some cases we documented shared values amongst the majority of stakeholders (e.g. that dehorning causes pain), but we also found important disconnects between current dairy production methods and widely held public values. Understanding the attitudes of people affiliated and unaffiliated with the dairy industry allows for the identification of contentious topics as well as areas of agreement; this is important in efforts to better harmonize industry practices with societal expectations.

We have also identified where the Code of Practice on the Care and Handling of Dairy Cattle aligns with stakeholder expectations and where gaps exist. We encourage the dairy industry to work to overcome these gaps.

Acknowledgements
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23. CEAH, Fort Collins, CO.
Public Concerns About Animal Welfare: How Should the Industry Respond?

Marina A. G. von Keyserlingk* and Daniel M. Weary

Introduction

Questions concerning the sustainability of food-animal producing industries have become the focus of intense public debate by social critics, animal advocates, and scientists. Specific concerns about the welfare of dairy cattle is nothing new; producers and veterinarians have always been concerned about the condition of animals in their care and have tried to ensure that they are healthy and well nourished (von Keyserlingk et al., 2009). In the tradition of good animal husbandry, good welfare can be seen largely as maintaining high levels of production and the absence of illness or injury. However, recent interest in farm animal welfare stems more from concerns about pain or distress that the animals might experience, and concerns that animals are kept under “unnatural” conditions, with limited space and often a limited ability to engage in social interactions and other natural behaviours. For instance the results of a recent survey indicated that providing assurances that dairy cattle are well treated, developing methods to incorporate pasture access and assurance of healthy products without relying on antibiotics or hormones, are all aspects deemed to be important by citizens when asked what they views on the ideal characteristics of a sustainable dairy farm (Cardoso et al., in press).

In addition to the tremendous increase in scientific research on the welfare of cattle, some new work has begun to investigate stakeholder views on dairy farming and practices common in the dairy industry (see review by Weary et al., 2015). An objective of the current paper is to summarize some of our recent work on stakeholder views. We focus on four common management practices (tail docking, pain mitigation for disbudding/dehorning, access to pasture and cow-calf separation) and describe how research in the natural sciences and social sciences can be integrated to identify more sustainable practices.

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> More recently, Canada’s new Code of Practice for the Care and Handling of Dairy Cattle states that dairy cattle “must not be tail docked”.

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The responses to this question are fully described in Robbins et al. (2015).

For this issue participants were provided the following context:

“The developing horns of dairy calves are typically removed to reduce the risk of injuries to farm workers or other cattle that can be caused by horned cattle. Horns of calves three months of age or older are normally removed surgically (“dehorning”) by scooping, shearing or sawing. Horn buds of younger calves are typically removed (“disbudding”) using a caustic paste or a hot iron.

There is considerable scientific evidence that all of these procedures cause pain. The immediate pain can be reduced using a local anesthetic to provide a nerve block – this procedure has been used safely for decades and costs just pennies a shot. Pain can persist 24 hours or more; this longer lasting pain can be reduced using non-steroidal anti-inflammatory drugs (like the ibuprofen you take for a headache). Providing calves a sedative before the procedure can reduce handling stress and make the procedure easier to carry out.

In many countries some pain relief is required. For example, Canada’s new Code of Practice for the Care and Handling of Dairy Cattle requires that pain control be used. Approximately 18% of dairy farms in the United States report using pain relieving drugs for disbudding or dehorning dairy calves.”

Participants then answered the question “Should we provide pain relief for disbudding and dehorning dairy calves?”

Participant composition was as follows: dairy producer or other farm worker (10%); veterinarian or other professional working with the dairy industry (7%); student, teacher or researcher (16%); animal advocate (9%) and no involvement with the dairy industry (57%).
Of 354 participants, 90% thought pain relief should be provided when disbudding and dehorning. This support was consistent across all demographic categories suggesting the industry practice of disbudding and dehorning without pain control is not consistent with normative beliefs. The most common themes in participants’ comments were: pain intensity and duration, concerns about drug use, cost, ease and practicality and availability of alternatives.

These results show a clear disconnect between current practice (with many farmers failing to provide pain control) and the attitudes of participants (including dairy producers) in these virtual town hall meetings. Causing pain to animals under our care, especially when this pain can easily be prevented, no longer seems acceptable. Our challenge is to find ways of getting pain control techniques applied widely on dairy farms.

In Canada, dairy producers have also taken a clear position on this issue. The Code of Practice for the Care and Handling of Dairy Cattle requires that “Pain control must be used when dehorning or disbudding.” In many countries (i.e. Sweden, Denmark, Netherlands, New Zealand and Australia) pain control for disbudding and dehorning is a legal requirement (ALCASDE, 2009; NAWAC, 2005; PIMC, 2004).

**Should dairy cows be provided access to pasture?**
The responses to this question are fully described in Schuppli et al. (2014).

For this issue participants were provided the following context:

“On many dairy farms cows are always kept indoors. Some dairy farmers believe that well-designed indoor housing provides a more comfortable and more suitable environment for the cows. In addition, some farmers keep cows indoors to more easily provide and control diets formulated to sustain high milk production.

Others consider pasture access to be important. For example, some believe that grazing is more environmentally sustainable, that pasture provides a healthier and more comfortable environment for cows, and that grazing is a natural behaviour important for cows.

Participants then answered the question “Should dairy cows be provided access to pasture?”

A total of 414 people participated. Providing access to more natural living conditions, including pasture, was viewed as important for the large majority of participants, including those affiliated with the dairy industry. This finding is at odds with current practice on the majority of farms in the United States where less than 5% of lactating dairy cows have routine access to pasture (see USDA 2007). To our knowledge there is no research indicating about how many lactating cows in Canada have routine access to pasture.

Participant comments showed that the perceived value of pasture access for dairy cattle went beyond the benefits of eating grass; participants cited as benefits exposure to fresh air, ability to move freely, ability to live in social groups, improved health, and healthier milk products. To accommodate the challenges of allowing pasture access on farms, some participants argued in favor of hybrid systems that provide a mixture of indoor confinement housing and grazing.
Despite the public indicating that access to pasture is important (see also Cardoso et al. 2016), the Code of Practice is largely silent on this issue, recommending only “for bedded-pack or composted-pack barns, provide access to pasture or an exercise or an exercise yard to decrease labor and bedding requirements.” In contrast, Sweden requires that cows be given pasture access during summer months (Ministry for Rural Affairs -Government Offices of Sweden, 2009).

Should dairy calves be separated from the cow within the first few hours after birth?
The responses to this question are fully described in Ventura et al. (2013).

For this issue 195 participants were provided the following context:

“Dairy farmers often remove the calf from within the first few hours of birth. This is done in response to several concerns including the following: the calf may become infected from pathogens carried by the cow or her environment; the calf may become injured by the cow or the barn equipment; the calf will not be able to nurse from the cow and receive adequatecolostrum (first milk produced by the cow after birth) and milk; the calf will drink too much milk which increases the farmer’s cost of feeding and increases the risk of diarrhea; allowing the cow and calf to bond will result in greater separation distress when separation does occur; farms are often not well designed for cow-calf pairs, so keeping cows and calves together can be considered an extra chore. Others consider that some form of cow-calf contact is an important element of natural behavior, and believe that this contact is beneficial to the cow and calf. On these farms the cow and calf are kept together for days or even weeks after birth.”

Participants then answered the question “Should dairy calves be separated from the cow within the first few hours after birth?”

Opponents of early separation contended that it is emotionally stressful for the calf and cow, it compromises calf and cow health, it is unnatural, and the industry can and should accommodate cow-calf pairs. In contrast, supporters of early separation reasoned that emotional distress is minimized by separating before bonds develop, that it promotes calf and cow health, and that the industry is limited in its ability to accommodate cow-calf pairs. Opponents of separating calves from their cows in the first few hours after birth often based their views on the emotional experiences of cows and calves. They compared the bond of a cow and her calf to the bond between mother and offspring in other species.

A major theme raised by proponents was that separation was inevitable, and that early separation was easier on the cow and calf than separation at a later age. There is considerable scientific evidence in support of this claim. Separating calves at an older age results in a much stronger response (high rates of vocalization and other activities) in comparison with calves separated soon after birth (Flower et al., 2003). Some respondents also believed that early separation minimized disease transmission from the cow. We are aware of little evidence to support this link.

The Canadian Dairy Code of Practice (NFACC 2009) states the following:

“Generally, dairy calves are separated from their mothers shortly after birth. There are benefits to
both calf and dam by allowing the pair to bond. Allowing the calf to spend a longer period of time with the dam may result in lowered morbidity and mortality in the calf; however, separation stress to both the cow and calf will be higher the longer they are together. Cow health is generally improved by allowing the calf to suckle (related to oxytocin effects on the post partum uterus)’.

Based on this summary of information the Code provides the following recommended best practice – “reduce separation distress by either removing the calf shortly after birth or by using a two-step weaning process.”

The fact that cows and calves are routinely separated at birth is an issue that the public is largely unaware of (Ventura et al., submitted), perhaps explaining why this issue has received little attention within non-dairy audiences. However, we speculate that external stakeholders will become increasingly unwilling to accept this practice.

Conclusions
The examples illustrated in this paper show how social science methodologies can document the shared and divergent values of different stakeholders, the associated beliefs regarding the available evidence, and the barriers in implementing changes. In some cases we documented shared values amongst the majority of stakeholders (e.g. that dehorning causes pain), but we also found important disconnects between current dairy production methods and widely held public values. Understanding the attitudes of people affiliated and unaffiliated with the dairy industry allows for the identification of contentious topics as well as areas of agreement; this is important in efforts to better harmonize industry practices with societal expectations.

We have also identified where the Code of Practice on the Care and Handling of Dairy Cattle aligns with stakeholder expectations and where gaps exist. We encourage the dairy industry to work to overcome these gaps.

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Introduction
Calf care is possibly the most challenging job on the dairy farm, in part because milk-fed calves are the animals most likely to become ill. New methods of calf rearing are becoming available that can benefit both producers and their calves, providing the potential for widespread improvements in calf care over the next decade. We predict that in the coming years producers will begin feeding dairy calves more milk than they are now commonly fed, increasingly using labour-saving milk delivery systems that facilitate more natural milk drinking behaviour. These improved feeding systems will ease the move towards group housing of calves before weaning, saving producers time and money. However, changes in feeding and housing systems pose new challenges for producers and their calves that require much innovation and research. In this presentation we will describe how new milk feeding methods promote rapid growth and more natural calf behaviour. New feeding systems facilitate keeping calves in groups, but group housing can result in increased competition and increased risk of disease transmission. Therefore, we will also discuss the challenges involved in using new feeding methods, and how to reduce these problems.

Calf Feeding
Methods of feeding calves in modern dairying differ markedly from those found in nature (von Keyserlingk and Weary, 2007), but knowing more about the natural behavior of cow-calf pairs can help us develop better ways of feeding calves (von Keyserlingk et al., 2009). On many dairy farms, calves are separated from their mothers within 24h of birth and then fed milk by bucket or bottle until 4 to 12 wks of age. Separating cow and calf early is thought to allow for better supervision of colostrum, milk and solid food intake and help prevent transmission of disease. Early separation also reduced the distress response of both the cow and calf. For example, Flower and Weary (2001) examined some of the effects of the age of separation on cow and calf behaviour and found that cows and calves that were separated (14 days versus 1 day) had higher levels of activity and vocalized more often. However, the calves separated at 14 days gained 16.5 kg over this period, versus just 4.5 kg for those separated early, and the calves maintained this weight advantage even after separation from the dam. The higher growth of calves kept with the cow may have been due, at least in part, to higher milk intakes – the spread between the cow-fed and people-fed calves shows the opportunity we have for improved gains with improved feeding management of dairy calves.

In conventional management schemes, calves are normally provided milk at 10% of their body weight (~ 4 kg / day), are vulnerable to disease, often fail to gain adequate weight and can sometimes experience high levels of mortality. We have tested the effects of feeding calves ad libitum by teat (Appleby et al., 2001; Jasper and Weary, 2002). In each experiment we compared weight gain, milk intake, starter intake and number of days with diarrhoea for calves fed milk conventionally (i.e. twice daily by bucket at 10% of body weight per day) versus ad libitum from a teat. In our first experiment, we found that weight gains during the first 2 weeks after birth were less than 0.4 kg/d for the conventionally fed calves versus 0.85 kg/d for the teat-fed ones; during the next 2 weeks gains were 0.58 and 0.79 kg/d respectively (Appleby et al., 2001). In a second experiment we again found that the teat-fed calves gained weight more quickly (0.78 versus 0.48...
kg/d from birth to weaning at 37 days of age) (Jasper and Weary, 2002). We also found that calves maintained their advantage in body weight after weaning. In both experiments the differences in weight gain were likely due to teat-fed calves drinking approximately twice as much milk as the calves fed conventionally. For example, the ad libitum fed calves consumed on average 8.8 litres of milk per day, compared to 4.9 litres per day for the conventionally fed calves (Jasper and Weary, 2002). Calves limit fed according to conventional practices also show behaviours indicative of chronic hunger (de Paula Vieira et al. 2008).

It is commonly thought that feeding less milk will encourage solid feed intake. Indeed, we have found that over the first 5 weeks of life, feeding calves less milk does increase starter consumption (0.17 versus 0.09 kg per day) but this practice also severely limits weight gains (Jasper and Weary, 2002; de Paula Vieira et al. 2008). Moreover, we have found that the ad libitum milk-fed calves quickly caught up to the conventionally fed calves in their intake of starter after weaning; both groups consumed on average 1.9 kg per day during the two weeks after weaning.

Improving access to milk raises practical problems, such as maintaining milk quality throughout the day, especially during warm weather. An alternate approach to continuous access is to provide unlimited availability of milk but only for a few hours each day. Previous research has found that calves provided unlimited access to milk spend just 45 minutes per day drinking milk, and that the largest meals occur just after the delivery of fresh milk (Appleby et al., 2001). In another study, we tested the effects of limited access to milk (4 h/d) versus continuous (24 h/d) access on milk intake, weight gain and behaviour of dairy calves (von Keyserlingk et al., 2004). Calves consumed as much milk in the 4 h/d treatment as they did in the 24 h/d treatment. An added advantage of the 4 h/d treatment, for some facilities at least, is that the same equipment can also be used to supply water to calves.

Much research and on-farm innovation is required to maximize the benefits of these new calf-feeding methods. In particular, little is known about how best to wean rapidly growing calves fed high milk rations. Current recommendations for weaning age and method are specific to slow growing calves fed conventionally, but new work is showing that slowly reducing milk intakes in the days before weaning can be helpful (Khan et al., 2007). In one study with calves fed up to 12 L/d (Sweeney et al., 2010), we compared calves weaned abruptly with calves weaned gradually over 4, 10, or 22 d. Calves weaned over 22 d ate the most starter, but also had the lowest weight gains before weaning. The abruptly weaned calves ate the least amount of calf starter but had the best weight gains before weaning. After weaning, calves on the 22 and 10 d treatments ate more starter and had better weight gains than calves on the more abrupt treatments. These findings suggest that weaning over 10 d is optimal. This type of gradual weaning is easily accomplished using automated calf feeders.

**Group housing**

For the past decades, common wisdom among North American dairy experts was that calves should be housed individually, in separate pens or hutches (e.g. Quigley, 1997). This practice was considered to maximize performance and minimize the risk of disease. Individual housing also helps avoid behavioural problems such as competition and cross-sucking.
The new calf-feeding methods described above work well for individually housed calves, but also facilitate group housing. Group housing provides more space for calves and allows for social interactions. Research and practical experience show that group rearing of calves can result in considerable benefits through reduced labour requirements for cleaning pens and feeding. One study on a commercial farm in New York State showed that calves kept in groups required one third of the labour that went into caring for the individually housed and fed calves (de Passillé et al., 2004). Calves are social animals that need exercise and keeping dairy calves in groups may provide a number of advantages to both producers and their calves. Successful adoption of group housing will mean avoiding problems such as increased disease and competition. Recent research provides some insights into how these risks can be minimized.

We evaluated the behaviour and growth rates of calves housed in pairs versus individually (Chua et al., 2002); calves gained weight steadily regardless of treatments. Interestingly, during the week of weaning (approximately 5 weeks of age), pair-housed calves continued to gain weight normally but the individually housed calves experienced a slight growth check. There were no differences between groups in the amounts of milk, starter or hay consumed, or in the incidence of scouring or other diseases. Aggressive behaviour and cross-sucking were almost never observed (less than 0.2% of time).

In a more recent study, de Paula Vieira et al. (2010) found that calves housed in pairs vocalized less during weaning than did individually housed calves. The results of this study also illustrated some longer-term costs to housing calves individually. When all calves were eventually introduced to a group pen after weaning calves that had previously been single housed took on average 50 h to begin feeding, in comparison to just 9 h for the pair-reared calves. These results suggest that individual housing may result in at least temporary deficits in cognitive or social tasks.

Successful group rearing requires appropriate management, including feeding method and group size. Large epidemiological surveys of U.S. and Swedish dairy farms found increased mortality and disease on farms keeping calves in large groups (more than 7 or 8) (Losinger and Heinricks, 1997; Svenson et al., 2000). Thus, small groups are likely a better alternative than large ones.

Calf immunity and the design and management of the housing systems, such as its cleanliness and ventilation, likely affect disease susceptibility more than group housing per se. Our work shows that housing young dairy calves in small groups is viable in terms of calf health, performance and behaviour. New research is now required on management strategies that will help prevent disease. For now, we encourage producers to consider keeping a closed herd (i.e. no new animals entering the herd), keeping groups small and physically separated from one another (e.g. in super hutches), and managing group pens in an all-in-all-out basis.

Calves in groups sometimes compete with pen mates. In one experiment using a simple teat-feeding system, we found that group-housed calves can displace one another from the milk teat many times each day if there are not enough teats (von Keyserlingk et al., 2004). However, giving each calf access to its own teat greatly reduced these displacements. This improved access to teats resulted in longer feeding times and increased milk intakes. However our most recent research (reviewed by Costa et al., 2016) provides a number of lines of evidence that calves reared in social isolation from
birth have increased food neophobia, impaired cognition and reduced performance, particularly during the milking feeding and weaning phases (Costa et al., 2016).

Other research has focused on how computerized feeding stations can be managed to reduce competition between calves. Increasing the daily milk allowance for calves from 5 to 8 liters per day reduced by half the number of times calves visited the feeder, reducing occupancy time and displacements from the feeder, and improving the efficient use of this equipment (Jensen and Holm, 2003; de Paula Vieira et al. 2008). Our research shows that young calves can be introduced into a group with little disruption when they are trained to feed from the computerized feeding station prior to the introduction (O’Driscoll et al., 2006). Although the calves visited the feeder less frequently on the day of mixing, they were able to compensate by increasing both the duration and amount consumed per meal, and established their pre-mixing feeding pattern after just one day.

**Conclusion**

Current research on dairy calves is paving the way for new methods of managing and housing these animals that will facilitate calf care and improve living conditions for these young animals. Calf care is arguably the most difficult job on the dairy farm. For the good calf manager, the research that we will describe provides opportunities to further improve calf care and reduce labour. However, like any new method, these are best adopted first by the best and most innovative managers. New methods require new skills and a careful eye to ensure that these are implemented in the best ways possible.

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**References:**

Veterinary medicine is a key component of the positive aspects of the human-animal bond. Veterinarians help preserve the symbiotic relationships between people and animals that can improve physiologic and behavioral health across species lines. In recent years, there has been additional interest in what is called the “dark side” of the human-animal bond, i.e., the adverse impacts of animal cruelty, abuse and neglect upon human health and well-being as well as upon the animals. The nexus where animal abuse intersects interpersonal violence is called “The Link.”

Veterinarians’ prevention of animal abuse may be seen as a One Health intervention.

The Link manifests in several stereotypical scenarios, including:

- Harm or threats to companion animals, and to a lesser degree livestock, as coercive control of intimate partners in domestic violence scenarios.
- Harm or threats to animals to keep sexually-abused children silent or compliant.
- Youths’ perpetration or witnessing of animal abuse, including animal fighting, which may lead to emotional distress and desensitization to violence and be a precursor to antisocial behaviors in adolescence and adulthood.
- Animal sexual assault or bestiality, which is receiving increased attention as a widespread aberrant and criminal behavior linked to child pornography.
- Animal hoarding, now widely seen as a mental health and public health issue rather than an eccentricity.

With its twin heritages in agriculture and medicine, veterinary medicine has a long-established history in human health. The Link has extended this role to include a proactive response to suspected animal cruelty, abuse and neglect. Mandated or permitted reporting of suspected abuse – with immunity from civil and criminal liability – is now codified by statute in 35 U.S. states, 8 Canadian provinces, and by CVMA, AVMA, AAHA, RCVS, and NZVMA codes of practice. This species-spanning, One Health response to prevent animal, human and environmental health problems has the potential to reduce animal suffering, to stop such acts from escalating into interpersonal violence, to set the highest standards for animal welfare, and to position the practitioner as being as responsive to animal abuse as physicians have long been vis-à-vis child abuse.

Increased public and professional awareness of the impact of animal abuse on human well-being has led to more aggressive law enforcement and prosecution of animal cruelty cases. AVMA data suggest that 99% of Americans perceive animals as close companions or family members. Consequently, animal abuse is now considered to be one form of family violence which often co-occurs with, or predicts, interpersonal violence and polyvictimization.

There are few reliable data describing the incidence of animal cruelty, as these crimes heretofore have not been documented in national or state crime incidence statistics (Lockwood, 2008). A new FBI procedure will change this dynamic. Veterinarians will increasingly be called upon to serve as animals’ advocates, to articulate the nature of animal abuse cases, and to serve as expert witnesses.
Animal Abuse and Domestic Violence
Abuse of companion and farm animals is a component of the Duluth Model of coercive control utilized by perpetrators of intimate partner violence to dominate and induce fear and subservience in their victims. Fear for animals’ welfare is a significant barrier to battered women and their children leaving abusive situations.

Roguski (2012) described pets as “pawns” in complex and multifaceted animal cruelty in households marked by domestic violence. Animal abuse:

- Creates a culture of normalized violence and psychological and emotional abuse.
- Is conducted purposefully by batterers who believe that police will see animal cruelty as not warranting investigation.
- Occasionally includes forced use of pets as sexual objects.
- Emanates from the perpetrator’s jealousy of his partner’s affections directed elsewhere.
- May also target animals belonging to friends and family members who abet her escape.

Orchestrated harm to animals creates a level of intimidation that secures families’ obedience and is a deterrent to victims’ asserting any independence. Emotional abuse involving animals is one of the first indications of escalated and broadened physical violence towards family members. 32.7% of survey participants with children reported that one or more of their children had witnessed threats to injure or kill an animal. An additional 24.5% had witnessed actual killing or injury (Roguski, 2012). Comparable findings have been reported in the U.S., Canada, Australia, the Bahamas, and Ireland. Women residing in domestic violence refuges have reported that their partners were 11 times more likely to hurt or kill pets than were a comparison group of non-abused women. Batterers who also abuse animals were described as more dangerous and as using more forms of violence than batterers who do not harm animals (Simmons & Lehmann, 2007). A history of pet abuse is one of the 4 most significant risk factors for becoming a batterer (Walton-Moss et al., 2005). 41% of intimate partner violence offenders were reported to have committed an act of animal abuse (Febres et al., 2014).

Animal Abuse and Child Maltreatment
The child protection movement originated in animal welfare, with Societies for the Prevention of Cruelty to Animals handling child abuse cases from the 1870s through the 1960s. Many humane organizations maintained dual responsibilities in animal and child protection. Animal abuse is listed as a criterion for conduct disorder and is believed to be one of the earliest manifestations of this condition, emerging at about the age of 6.5 years. The perpetration or witnessing of animal abuse may desensitize youths against violence and contribute to an intergenerational cycle of violent behaviors (DeGue & DiLillo, 2009; Ascione, 1993). Many in the Link field believe that perpetration or witnessing of animal abuse should be considered an Adverse Childhood Experience which can harm children’s developing brain architecture and lead to long-term hyper-responsiveness to perceived threats and to negative physical and mental health outcomes.

Animal Abuse and Elder Abuse
Elders with declining mental capabilities, financial and transportation limitations, and social isolation are at increased risk for neglecting their animals. Adult protective services case workers report that
animal neglect is frequently a marker for co-occurring self-neglect and a variety of mental health disorders. Animal hoarders are statistically over-represented by older women. They may live in unhealthy and squalid environments surrounded by dozens or hundreds of living and dead animals in a self-fulfilling cycle of social isolation. Four distinct types of animal hoarders have been identified (Patronek et al., 2006):

- Mentally ill.
- Overwhelmed caregivers.
- Rescuers whose identity is tied to their animals.
- Exploiters falsely claiming to be legitimate animal welfare charities

**Animal Abuse and Dog Bites**

Public health research links histories of animal abuse with increased risk for morbidity from dog bites. Patronek et al. (2013) reported that 21.1% of 256 canine attacks resulting in human fatalities involved dogs that had been abused. DeViney, Dickey & Lockwood (1983) reported that families under investigation for child abuse experienced 11 times more dog bites than did non-abusing households.

**Community and Professional Responses**

Growing recognition of animal abuse’s linkages with human well-being has resulted in numerous legislative and programmatic developments. 32 U.S. states plus the District of Columbia and Puerto Rico now specifically allow courts to include animals in domestic violence protection-from-abuse orders. Over 500 domestic violence shelters have foster care programs for companion animals with animal shelters and colleges of veterinary medicine; over 100 in the U.S., Canada, Australia, the Netherlands and New Zealand have built on-site kennels to enable domestic violence survivors to bring their pets.

**Conclusion**

Multiple theories explain why adults and juveniles commit acts of animal abuse. There is no single unifying theory explaining numerous complex, multivariate etiologies of cruelty phenomena. Animal abuse may be: causal or co-relational with interpersonal violence; a construct of a graduation hypothesis or part of a pattern of general deviance; a context of dysfunctional home environments and abusive experiences; or symptomatic of other confounding factors. It is widely recognized anecdotally and through empirical research, however, that animal abuse left untreated frequently indicates or predicts other antisocial behaviors that threaten animals and human members of families and communities.

Veterinary recognition and reporting of suspected animal abuse are the first steps to resolve unhealthy situations and make communities safer for all. In so doing, veterinarians can work within a One Health approach that unites human and veterinary medicine in common concern for the vulnerable, victimized and at-risk. Part 2 of this track will help practitioners identify general clinical and diagnostic indicators which usually suggest Non-Accidental Injury and other forms of animal abuse and neglect.
References and Recommended Reading:


Session 2: How Do I Know if it’s Abuse? Clinical and Diagnostic Indicators of Potential Animal Abuse

Phil Arkow, Coordinator, The National Link Coalition

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. Most practitioners will be presented with cruelty cases. While cruelty cases are not seen regularly, they are invariably problematic and difficult to resolve.

Even such seemingly simple definitions as “animal,” or the even more subjective “cruelty,” vary across jurisdictional, cultural, community, personal, and professional boundaries and can shift from time to time and situation to situation.

A key point is that practitioners do not have to “know” whether a suspicious situation is abuse, cruelty or neglect in order to trigger a report to law enforcement or humane authorities. An investigator, prosecutor, and court will make that determination. The veterinarian’s roles are:

• To serve as the animal’s first line of defense.
• To report suspicions to the appropriate agency.
• To document findings objectively and be prepared to present them in court if necessary.

Animal cruelty, abuse and neglect generally imply socially unacceptable actions or omissions that inflict unnecessary pain or distress. What constitutes “unnecessary” varies widely and the prevailing standard can be determined only by the courts.

The following terms generally describe socially unacceptable conduct:

• Animal cruelty: the most prevalent term in statutes. It implies intentional infliction of pain from which the offender derives enjoyment or amusement.
• Animal abuse: a more neutral term, modeled after child protection, describing willful or negligent maltreatment regardless of the perpetrator’s intent, motivation or mental state.
• Animal neglect: an act of omission signifying a lack of care; the most common form of maltreatment.
• Animal hoarding: neglect on a significant scale involving large numbers of animals often kept in deteriorating conditions below minimal standards of nutrition, sanitation, and veterinary care.
• Animal physical abuse: a wide range of injurious acts requiring active engagement such as beating, kicking, suffocating, throwing, shaking, poisoning, burning, etc. The clinical presentation includes injuries to the skeleton, soft tissue or organs sustained as a result of beating or repeated trauma. Non-Accidental Injury (NAI), a term borrowed from child protection, is a synonym for physical abuse.
• Animal sexual abuse: abusive acts or sexual conduct with an animal involving the rectum, anus or genitalia. The term is preferred over the more archaic bestiality (in which penetration must occur) and zoophilia (a strong erotic preference for animals).
A new typology is being developed as a result of the FBI’s inclusion, beginning in 2016, of four types of animal abuse within its National Incident-Based Reporting System. This standardized checklist of crimes is utilized by 18,000 law enforcement agencies nationwide.

- Simple neglect or gross neglect (animal hoarding)
- Intentional abuse or torture
- Organized animal abuse (animal fighting)
- Animal sexual abuse

It should be noted that unlike child maltreatment, emotional abuse does not constitute a crime in animal welfare.

Clinical conditions that should raise the Index of Suspicion and inclusion of abuse in the differential diagnosis include:

Presenting Clinical Factors
- Unexplained or repetitive injuries to an animal, which may show up on examination, ultrasound or x-ray
- History of unexplained or repetitive injuries to multiple animals
- Evidence of rib injuries, either current or from previous trauma
- Low weight or low body condition scores
- Unexplained poisoning, burns, bruising, stab wounds
- Fractures: Tong (2015) reported the following 5 features should raise the Index of Suspicion and support a diagnosis of NAI, especially among young male dominant breeds such as Staffordshire bull terriers:
  - Presence of multiple fractures
  - Fractures occurring on more than one region of the body
  - Transverse fractures
  - Fractures presenting at a later stage of healing
  - Multiple fractures at different stages of healing
- Gunshot wounds
- Ingrown collar
- Scars, wounds and traumas consistent with dog- or cock-fighting competitions (Intarapanich et al., 2017)
- Obvious severe neglect, such as heavy ectoparasite infestation, dental disease, severely matted fur, failure to treat adverse medical conditions, dehydration, emaciation, or overgrown claws, horns or hooves
- Signs of disease, pain, distress or injuries needing treatment, such as blood from orifices, vocalization, vomiting, lameness, shivering, or diarrhea
- Sexual abuse, such as unexplained trauma to the anus or genitalia
- Animal displays fear of its owner or of people in general
- Animal displays an unexplained change in behavior

Client Profile Factors
- Client is new to the practice or visits several clinics to avoid raising suspicion
- Discrepancies in names, addresses and ownership of animals
- Prior history with client has raised concerns
- History of high turnover of animals, especially with repetitive histories of behavioral problems
- Family is known to be under economic, marital, substance abuse, family violence, or other pressures
- Client’s knowledge, skills and attitude compromise proper animal husbandry

**Client Behavior Factors**
- History as presented is inconsistent with the nature of the injuries
- Family members present changing or discrepant histories
- Client lacks concern and is indifferent to animal’s injuries
- Client repeatedly fails to follow-up on treatment of serious medical conditions
- Unexplained delay in seeking medical attention
- Weak emotional attachment to animal
- Client is argumentative or aggressive; family members appear intimidated, apprehensive or deferential
- Client expresses not feeling safe at home
- Client blames someone else or unknown causes for the trauma
- Client’s methods of disciplining or housebreaking animals raise concerns
- Children’s responses to questions about their pets raise concerns
- Munchausen Syndrome by Proxy involving the animal

**Environmental Factors**
- Hoarding excessive number of animals under substandard care
- Animals’ living environment is unsuitable
- Availability, nutritional composition and quality of feed is insufficient for animals’ metabolic needs
- High incidence of viral, bacterial and fungal infections, heavy intestinal or heartworm burdens, or dermatitis or other skin conditions indicative of flea infestations or sarcoptic and demodectic mange
- Animal has been abandoned by owners who have departed

**Animal Fighting Factors**
Telltale injuries in dogfighting include: scarring on faces or legs; missing or amputated ears or tails; and characteristic bite wounds on the head, neck and legs. Suspicious environmental signs include: excessive numbers of fighting breeds segregated from each other with logging chains or caging; makeshift fighting pens; breaking sticks used to separate the jaws; treadmills and other conditioning equipment; and the presence of self-administered steroids, antibiotics and iron supplements (Lockwood, 2012).
Similarly, fighting birds with injuries and torn feathers, segregated housing, makeshift pens, and razor-sharp gaffs for slashing are cause for concern.

**Conclusion**
The practitioner is reminded that a single incident should not automatically cause animal abuse to be added to the differential diagnosis. Rather, a variable combination of factors, a pattern of actions and behaviors, or injuries which are not clearly or adequately explained should lead to a raised Index
of Suspicion. The person presenting the animal may not be the perpetrator or may be under some coercion from the person who caused the injury.

Part 3 of this track will address practice management concerns to help alleviate concerns for the safety, exposure to liability, economic, and patient/client confidentiality issues involved in responding to suspected animal abuse.

References and Recommended Reading:

Practitioners and their staffs experience confounding dilemmas when presented with suspected animal cruelty, abuse or neglect. They need to balance economic, safety, confidentiality, legal, liability, and management concerns with ethical principles, personal beliefs and professional standards while responding compassionately and effectively to the needs of patients, clients and society. Additional pressure is exerted when egregious cases generate extensive news media coverage. With increasing public, legislative and prosecutorial interest in animal welfare, failure to respond appropriately places the profession at risk of adverse criticism and litigation. A cruelty case can bring into question a fundamental dilemma of veterinary medicine: is the practitioner’s primary responsibility to the patient or the client?

These dilemmas are not unlike those faced by physicians in human healthcare vis-à-vis the response to interpersonal violence (Arkow & Munro, 2008). Analogues from those experiences can assist practitioners in developing protocols to cover themselves and members of the veterinary team.

Resolving veterinarians’ concerns regarding these contentious issues has involved a 6-stage process of: raising professional awareness and a sense of responsibility; professional and peer support; legislative support; development of diagnostic, clinical and forensic indicators; promulgation of practice management guidelines; and publication of a directory of agencies to which allegations of abuse are to be reported.

While veterinarians remain divided on whether reporting suspected abuse should be mandated or voluntary, statutes requiring or permitting practitioners to report with immunity from civil and criminal liability, combined with professional codes of practice encouraging such response, eliminate contentious moral dilemmas and make the decision easier to explain to clients. Once a determination has been made that a situation is sufficiently suspicious, the issue becomes how to implement an appropriate reporting process to comply with the duty. Veterinarians are again reminded that they are medical experts, not legal ones, and the determination as to whether a situation is actionable and constitutes illegal criminality will be determined by the criminal justice system.

**Peer, Professional and Statutory Support**

Veterinarians’ reporting responsibilities are now embedded in professional codes of conduct. The Canadian Veterinary Medical Association declares that veterinarians have a “moral obligation” to report suspected animal maltreatment; in return, “society has an obligation to support those veterinarians who report in good faith using their professional judgment.” CVMA encourages veterinary schools to train students in recognizing and reporting animal abuse. It urges veterinary associations to lobby their provincial governments to make this reporting mandatory with immunity from civil and criminal liability. CVMA likewise recognizes animal abuse as an important social issue affecting families and communities, and believes that veterinarians may help break the cycle of family violence and create safe, humane communities. Veterinarians in 8 Canadian provinces and territories are required or permitted to report suspected animal cruelty. The Government of
Saskatchewan (2016) recently published an animal welfare handbook addressing these issues, noting that practitioners in the province have an ethical and professional obligation to report as a way to protect animal welfare and alleviate animal suffering. AAHA in 2015 expanded its earlier policy and now supports the reporting of suspicions of animal abuse to appropriate authorities. AAHA encourages the adoption of laws mandating veterinary professionals to report with immunity from legal liability when reporting in good faith. This policy is based, in part, upon studies that have shown a link between animal abuse and other forms of violence, including child, spousal, and elder abuse. Reporting suspicions of animal abuse is important as it will trigger an investigation that may ultimately protect both animals and humans. It upholds the veterinary oath to prevent animal suffering and promote public health.

AVMA’s Policy on Animal Abuse and Neglect in 2012 recognized that veterinarians may observe cases of animal abuse or neglect as defined by laws and that it is their responsibility to report cases promptly to appropriate authorities to protect the health and welfare of animals and people, regardless of whether reporting is mandated by law. This peer support from AAHA and AVMA has helped fuel legislative initiatives whereby 35 states have enacted laws mandating or permitting veterinarians to report suspected animal maltreatment with immunity from civil and criminal liability. (California and Colorado require veterinarians to report suspected child abuse, and Illinois mandates reporting suspected abuse of elders and vulnerable adults.)

New Zealand’s Code of Professional Conduct lists animal welfare as the first of seven fundamental principles, calling it “a special responsibility” and “an over-riding professional duty.” Veterinarians must act immediately to remedy situations where they have cause to suspect unreasonable or unnecessary pain or distress or possible breaches of animal welfare legislation. If the animal’s caregiver is a client, the veterinarian should discuss the situation and develop an action plan to relieve the concerns. The matter must be reported to an animal welfare inspector: if issues cannot be discussed with the caregiver; if the action plan’s improvements are not achieved; or if the case involves severe cruelty or neglect. Valid and justifiable reasons allow disclosure of personal information. Acknowledging research linking animal abuse with human violence, an explanatory note within the Code encourages veterinarians to consider whether people within the home might also be at risk: if so, practitioners should use their best judgment to determine whether police or Child, Youth & Family authorities should be informed.

The New Zealand Veterinary Association (2011) and Veterinary Council of New Zealand (2013) have published guidances to assist large- and small-animal practitioners in communicating their concerns with clients. NZVA in 2015 took the additional step of endorsing a national campaign against domestic violence, describing veterinary medicine as a “three-dimensional” profession with a unique voice that transcends animal, human and environmental health. A similar inter-species public health approach has been suggested in the U.S. as well (Allison et al., 2017).

Great Britain’s Royal College of Veterinary Surgeons’ Code of Professional Conduct (2016) encourages practitioners to include Non-Accidental Injury (NAI) in the differential diagnosis. If the examination of the animal leads to a suspicion of abuse, the veterinarian should first attempt to discuss these concerns with the client. When this would be inappropriate or the client’s reaction increases rather than allays concerns, the veterinarian should contact relevant authorities. Real and immediate risk to animals and the public interest justify breaching client confidentiality restrictions.
The Code similarly extends the veterinary response to report suspected child abuse and domestic violence.

**Primary Considerations When Faced with a Possible Case of Cruelty**

1. Safety first. Never compromise the safety of humans or animals. If risk of harm, call 911.
2. Treat the animal. Collect and document the evidence — but do not compromise the timely treatment of the animal.
3. Recognize that animal cruelty is a legal, not a medical, determination. As with medical practitioners who deal with child, elder or domestic abuse, the situation is not always black and white — and yet, to protect the vulnerable, health professionals must be prepared to act.

**Preparing the Practice to Respond**

First, be open to a differential diagnosis of Non-Accidental Injury (NAI). Identify in advance whether reporting is mandated or permitted in the province and which law enforcement or humane agencies are charged with investigating allegations of cruelty. Have an internal decision-making process framed in advance so junior staff members understand the chain of command as to who may make a report.

A risk-assessment form, client questionnaire and decision tree can be helpful in assessing risks to the patient, client, staff, and other animals or human members of the household. These can be administered when welcoming a new client to the practice or whenever an abusive etiology is suspected. These materials are available in a Guidance published by AVMA and the National Link Coalition (Arkow, Boyden & Patterson-Kane, 2011).

**Engaging the Client**

Good interpersonal communication skills are critical when discussing animal welfare concerns with clients who may be abusive, indifferent, or lacking in acceptable animal husbandry skills. Think about how you will approach the client. Clearly state that your concern is for the welfare of the animals: ask the client if s/he is also concerned. Deliver your opinions clearly and respectfully. Use language that diffuses the situation: do not escalate by being judgmental or assigning blame. Separate the person from the problem. Be supportive and encourage the client to find solutions. Actively listen. Acknowledge when clients have tried to do something positive (even if it hasn’t worked). Provide clients an opportunity to have a support person present. Use objective standards and relevant laws as reference points against which to measure the situation. Emphasize that welfare of their animals is their responsibility and they must take action. Be aware of your own triggers and responses to high emotion.

**Five Steps of Response**

1. Prepare the practice. Be familiar with provincial laws and where to report. Develop a protocol and train staff.
2. Patient history. Create a client profile and patient history.
3. Medical examination. This may include examinations, tests, radiographs, photographs, pathology, serology, and other procedures as necessary. The animal may have to be held for additional observation. Diagnose, treat and monitor the animal.
4. Evidence collection, documentation and preservation. This may involve ante- and post-mortem cases.
5. Report writing. Comprehensive, contemporaneous and accurate reports of findings must be entered into the patient’s record and be suitable for court testimony in the event of a subpoena. If necessary, another veterinarian can conduct a second, documented examination, which may support or contradict your findings.

Case Management
1. Assess the situation. Include the animal’s food supply, environmental conditions, the client’s animal husbandry expertise, and prior experience with the client.
2. Planning. Write an unbiased assessment noting your professional concerns. Identify what the client has and has not done, what could and should be done, support that may be needed, and available resources. Agree with the client on an action plan and timeline for further follow-ups.
3. Inform the client of your responsibility to report suspected abuse. Provide the client with referrals to agencies that might assist.
4. Review. Return to the case periodically to ensure actions have been taken and the condition is resolving.

Strategies to Reduce Liability Exposure
Practitioners should maintain good insurance coverage and work with their provincial association’s legal department to resolve concerns. Hold-harmless agreements with animal shelters can reduce exposure. Careful and comprehensive documentation of observations, findings, communications, and test results can further protect the practitioner. The confidentiality of patient records, and whether they may be released to the client and/or outside agencies with or without a court order, varies widely. The practitioner is advised to check with legal counsel for the most current information. When in doubt, seek clarification from your veterinary association or legal counsel. Whether the decision is made to report, to advise a client or to monitor a case further, staff members should be fully briefed on the situation and understand the implications. Remember that it is not up to the veterinarian to establish any offense, but rather to report the situation to those who have the legal power to investigate and who will make a determination whether filing of criminal charges is warranted.

References and Recommended Reading:
During the post-operative period, there is a critical transition from unconsciousness and a protected airway to semi-consciousness and a partially protected airway. It is important to closely monitor the patient during this phase of anaesthesia. The level of pain experienced by the patient is also changing during this phase due to changes in sedation and stimulus. This article is a brief discussion the difficulty in assessing pain during this the recovery phase. It further discusses some of the issues related to the identification and treatment options available to the general practitioner.

During the recovery phase, your patients will challenge you as to how you interpret their clinical signs as they pertain to their level of pain. It is important to have a logical approach to this problem. Remember the acronym “AAAA”.

Assimilate the situation of the case.
Administer an analgesic drug to the patient
Assess the response of the patient after an appropriate period of time.
Adjust the following dose of analgesic depending upon the previous response.

Assimilate: Assimilating the situation will incorporating all the information you have at hand including the signalment (age, breed, sex), history of the events leading up to this point, co-existing diseases, physical examination, and medications administered. Assessing the level of pain an animal is experiencing is extremely difficult. Many of these pain scales are based upon subjective measures that cannot be applied to all the clinical scenarios for example assessing an animal following a soft tissue procedure whilst using a scale that was developed for orthopaedic procedures. There are limitations when using objective measures of pain as well such as von Frey devices or force plate analysis.

Administer: According to the WHO, the main reason why human beings experience surgical pain is still due to the lack of administration of drugs. The ideal drug will match the needs of the patient with respect to their level of pain. Either treating an animal with too little or too much analgesia can lead to an adverse event.

Assess: Immediately following the administration of a drug, you should be continually assessing the animal for a reduction in the level of pain. The exact timing of this will be based upon the pharmacokinetics of the drug, i.e. the time to peak effect and duration of action. If you have doubts as to the level of pain an animal is experiencing you can always choice an ultra short-acting drug like fentanyl and then assess them within minutes of the administration. A drug like buprenorphine will take up to 15 minutes for its full effect.
Adjust: This is the step that is most often missed when an analgesic has been administered, the simple act of administering a second or third dose of an analgesic to the patient. Again the patient will need to be assessed for their level of pain at each time point. The introduction of a pain scale will assist in speeding up this process.

Before considering an animal is in pain or discomfort, it is important to rule out the cause of the behavioural changes may be due to agitation. With respect to the observed clinical signs, there is a continuum from mild changes associated with agitation to more obvious changes associated with pain. There will be a gradual increase in sympathetic tone as you move from clinical scenario to the next. Generally, it is hardest to delineate mild pain from agitation and emergent delirium as compared to other levels of pain. Causes of agitation include a distended bladder or colon, tight bandages, internal bleeding and drug-induced psychosis. Simple measures can be introduced to your workflow to help minimise the chances of agitation. Ensuring the bladder is empty and checking bandages are not too tight will help with this. Another approach is to use local and regional anaesthetic techniques thereby reducing the need for sedating opioids during the recovery. The animals can thus reanimate with a ‘clear’ head.

**Emergent Delirium vs Pain**

Differentiating between emergent delirium and pain is perhaps one of the most difficult challenges facing an anaesthetist. The behaviours of both conditions are more of a continuum of clinical signs ranging from very mild changes to obvious signs of pain. Most patients wake up with some form of emergent delirium, however most are mildly effected not warranting any treatment but a soothing voice. Some patients may be misdiagnosed as having emergent delirium or pain when their only cause of agitation is a full bladder or colon, tight bandages, internal bleeding or drug-induced psychosis (atropine, scopolamine, ketamine).

<table>
<thead>
<tr>
<th>Table 1 Comparison of emergent delirium and pain</th>
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<tbody>
<tr>
<td>Emergent delirium</td>
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<tr>
<td>-------------------</td>
</tr>
<tr>
<td><strong>Sympathetic tone</strong></td>
</tr>
<tr>
<td>• Normal rhythm (sinus arrhythmia)</td>
</tr>
<tr>
<td>• Normo- to hypertension</td>
</tr>
<tr>
<td>• Eupnoea to mild tachypnoea</td>
</tr>
<tr>
<td>• Miosis to mid-range</td>
</tr>
<tr>
<td>• Cat - mydriasis</td>
</tr>
<tr>
<td><strong>Vocalisation</strong></td>
</tr>
<tr>
<td>• Short duration</td>
</tr>
<tr>
<td>• Low intensity cry</td>
</tr>
<tr>
<td><strong>Diarrhoea</strong></td>
</tr>
<tr>
<td>• Secondary effect of opioids</td>
</tr>
<tr>
<td><strong>Blank expression</strong></td>
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To differentiate between emergent delirium and pain, you first must rule out any other causes that may cause agitation such as those described above. Mild delirium may only need gentle reassurance and the least amount of restraint possible. The use of excessive restraint can cause the escalation of
the delirium. Acepromazine 0.01 – 0.03mg/kg IV will help reduce the anxiety. Naloxone 1-5mcg/kg IV slowly or flumazenil 0.5-3mcg/kg IV slowly to effect will reverse the drug-induced dysphoria associated with opioid or benzodiazepine (respectively) administration. The patient needs to be re-evaluated after each dose of reversal agent for any worsening of pain. If there is a problem associated with dysphoria, there will be signs of improvement with 3-5 minutes of administration. It may also be possible the clinical signs of pain may become more obvious at this time. You need to be flexible and consider your initial assessment of dysphoria was incorrect. Dexmedetomidine 1-3mcg/kg IV to effect may be necessary if the signs of delirium are severe. Remember, heavy sedation will slow the rate of recovery and increase the nursing needs of the patients. Heavy sedation will prevent quicker return to eating, increase risk of aspiration pneumonia and may mask other problems.

When considering treating an animal for pain, you first must consider the history (duration of pain before surgery, type of surgery, type of pain), physical examination and your evaluation of the level of pain. There are a number of pain scales that have been described in detail in the literature (visual pain scales, facial pain scales, Melbourne Pain Scale). If you think it hurts, it probably does! A general approach to the treatment of pain is firstly to administer a drug, assess the response of the drug after an appropriate period of time, and then adjust your next dosing regime based upon this initial response. For example, you only need to wait 2-3 minutes after administering fentanyl to assess the initial response unlike buprenorphine where you may have to wait up to 15 minutes for a good response.

If there has been any tissue trauma or an inflammatory state, you should always ask yourself is there any reason why I cannot use a non-steroidal anti-inflammatory drug. Table 6 contains a guide as to the various analgesics therapies that may be used during the post anaesthetic period. It is important not to think of this as a set recipe but a starting point. Remember to administer, assess and adjust your therapy to each individual. The aim is to have a patient pain-free, functioning normally and without excessive sedation. This is not always possible.

Table 2 Guide for an analgesic plan during the post anaesthetic period

<table>
<thead>
<tr>
<th>Locoregional anaesthesia</th>
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<tbody>
<tr>
<td>• Blocks nociceptive processing</td>
</tr>
<tr>
<td>• Blocks the stress response</td>
</tr>
<tr>
<td>• MAC-sparing effect</td>
</tr>
<tr>
<td>• Shortens recovery</td>
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</tbody>
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<table>
<thead>
<tr>
<th>Mild to moderate pain</th>
</tr>
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<tbody>
<tr>
<td>• Buprenorphine 0.005-0.02mg/kg</td>
</tr>
<tr>
<td>• Butorphanol 0.05-0.2mg/kg</td>
</tr>
<tr>
<td>• Hydromorphone 0.05mg/kg</td>
</tr>
<tr>
<td>• Lidocaine 1mg/kg IV bolus; 50mcg/kg/min</td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Moderate pain</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Hydromorphone 0.05-0.15mg/kg</td>
</tr>
<tr>
<td>• Morphine 0.05-0.2mg/kg/hr</td>
</tr>
<tr>
<td>• Fentanyl 2-10mcg/kg/hr</td>
</tr>
</tbody>
</table>

| Severe pain (Opioid + any combination of drugs) |
- Fentanyl >10-45mcg/kg/hr (mu agonist)
- Ketamine 0.25-1.0mg/kg/hr (NMDA antagonist)
- Dexmedetomidine 0.25-1mcg/kg/hr (alpha2-adrenergic agonist)
- Lidocaine 1mg/kg IV bolus; 50mcg/kg/min (Sodium channel antagonist)

Delayed Emergence

Occasionally a patient will have a delay in recovery. This is usually due to residual anaesthetic, analgesic or sedative effects in combination with hypothermia. It is important to rule out other causes that may delay emergence. Metabolic disturbances such as hypoxaemia, hypercapnia, hypoglycaemia, hyponatraemia, hypercalcaemia and hypermagnesaemia can all delay recovery. Pathology of the central nervous system is an important differential to rule out. A review of the history, physical examination followed by a blood gas, glucose and electrolytes will aid in identifying the underlying cause. The treatment plan may include correction of glucose and electrolyte disorders, the use of a reversal agent such as naloxone, flumazenil or alpha2-adrenergic antagonists or potentially correction of a raised intra-cranial pressure.
This presentation will focus on the techniques that are easy to do and that do not require specialized equipment. It is difficult to cover all the local and regional techniques especially when new techniques are being developed currently.

**Declaws (onchectomy)**

Regional anesthetic techniques are easy to perform to provide intra-operative analgesia for the surgical removal of claws in the front legs of cats. An additional benefit of using regional anesthetic techniques during the surgery is the prevention of central sensitization and “dorsal horn wind-up”.

In general this will have a positive effect on the comfort level once the local anesthetic block has worn off and thus may reduce the amount of analgesics agents required. Firstly it is important for you to calculate the maximum safe dose of local anesthetic that can be administrated to the cat. Cats are more sensitive to the adverse effects of local anesthetics in general. The commonly reported maximal doses for lidocaine and bupivacaine are 2mg/kg and 1mg/kg respectively.

There are three sites, which must be desensitized with local anesthetics to induce total anesthesia of the digits of the front leg paw. Traversing the dorsomedial aspect of the carpus are the superficial branches of the radial nerve. To desensitize the dorsum of the paw (with the exception of the fifth digit) a line block is performed over the bony protuberances of the dorsomedial aspect of the distal radial epiphysis. There are two sites are located on the palmar aspect of the paw. The median nerve is located on the medial and proximal aspect to the accessory carpal pad. A depot of local anesthetic agent may be infiltrated subcutaneously in this area. This will anesthetize most of the palmar aspect of the paw. There is an additional branch of the ulnar nerve, which is located on the axial aspect of the accessory carpal pad. This branch supplies the innervation to the fifth and part of the abaxial aspect of the fourth digits. The third depot of local anesthetic can be infiltrated on the proximal, lateral aspect of the accessory carpal pad (ulnar). The superficial and deep peroneal nerves are located on the distal, dorsal aspect of the tarsus. The tibial nerve is located on the distal, plantar aspect of the tarsus. A line block on the dorsal and plantar aspect of the tarsus will block all the digits of the foot.

**Dental Blocks**

Dental procedures can be very painful especially when performing endodontic procedures and extractions. Certain conditions in cats for example, feline odontoclastic resorptive lesions (FORL) and immune-mediated stomatitis are particularly painful. There are a number of techniques described below which can be used to prevent the discomfort associated with dental procedures. One fascinating observation is the complexity of the anesthesia used during a dental procedure. The majority of these cases are old and often have co-morbidities requiring a heightened level of surveillance. The most extreme of which is the insertion of a temporary pacemaker. This is one situation where you should not think, “it is just a dental procedure”.

**Mental Block:** The mental foramen can be palpate on the lateral aspect of the rostral mandible at approximately the level of the first premolar tooth. The mental foramen is a small depression is easily palpated by flipping the lower lip downward to reveal the gingivia. You have two options depending upon the site with which you wish to block. If you only need to desensitize the most
rostral portion of the hemi-mandible (lip and incisors) then you can simply deposit the local anesthetic at the entrance to the mental foramen. To desensitize the canine, you need to infiltrate the local anesthetic into the mental foramen. To insert the needle, hold the needle parallel to the mandible and insert carefully. You may do this with the lip pushed out of the way or simply inject through the skin. Remember the artery, vein and nerve are all within this foramen. Avoid excessive probing with the needle or you risk vascular and or nerve injury. The needle should pass easily into the correct position and there should be no resistance to the injection. If resistance does develop, stop injecting and move to the mandibular foramen. If you are having difficulty then simply change to a mandibular nerve block. Also use the smallest needle possible for example a 25 or 27 G needle. The volume of the injectate will vary (0.25 – 1mL) depending upon the size of the animal (cat to a large dog).

**Alveolar Mandibular Block:** This is probably the most difficult dental nerve block to perform consistently well. This is because in small patients it can be difficult to access via the mouth and the location of the tip of the needle during the injection is unknown. There are two basic approaches to the mandibular block via the mouth and externally. With the oral approach, the vertical aspect of the ramus of the mandible is identified which is just caudal to the last molar. Externally the corner of the mandible is palpated. An imaginary line is draw between these two points. The needle is inserted along this line on the medial aspect of the mandible. The needle also needs to be as close to the bone as possible. The foramen usually sits approximately halfway between these two points. A line block can be made along this imaginary line. For the external approach you need to identify the slight depress in the mandible just ventral to the ramus of the mandible. The correct location for the insertion is at the most profound depth of the depression. The needle is inserted on the medial aspect of the mandible and directed towards the lateral canthus of the eye. The insertion depth of the needle is approximately halfway of the height of the mandible, i.e. from the bottom of the mandible to the bottom of the gingival line. To increase your chances of success you may infiltrate the local anesthetic in a fan-like manner.

**Infraorbital block:** Similar to the mental block it is possible for you to desensitize only the structures rostral to the infraorbital foramen or to desensitize the ipsilateral structures of the dental arcade. The infraorbital foramen is located by palpating inferiorly to the eye at the level of the second or third premolar. When you fold the upper lip up and out of the way, you will see a tense band of gingiva extending dorsally from the second or third premolar. The infraorbital foramen is often just caudal to this. The needle held parallel to the long axis of the nose is inserted into the infraorbital foramen carefully. You may insert the needle with the upper lid folded dorsally or insert the needle with the lip down. If you experience any difficulty with the location of the block or injection, just simply perform a maxillary block instead. The advantage with the mental and infraorbital nerve blocks is you are injecting the local anesthetic around the nerve. You know where the location of the nerve is exactly. This is not the case with the other nerve blocks.

**Maxillary block:** Again there are the oral and external approaches to the maxillary block. Desensitizing the maxillary nerve will block the entire dental arcade and rostral soft tissue structures. Use a mouth-gag to help facilitate the block when performing the oral approach. Identify the last molar tooth and palpate the most caudal aspect of the hard palate just medial to the tooth. Bend the needle to approximately a 45-degree angle. Insert the needle to a depth of approximately ¼ to ½ inch depth at a location just medial to the last molar and caudal to the hard
palate. Infiltrate the local anesthetic in a fan-like manner. There is a small branch of the maxillary nerve, which branches before it enters the hard palate in a more medial aspect. This is important to anesthetize if you are performing surgery on the hard palate e.g. a cleft palate repair. Location of the insertion point for the external approach starts with palpating along the dental arch of the maxilla and identifying the last molar tooth. Follow the caudal border of the maxilla dorsally to where it meets the zygomatic arch. The needle is inserted perpendicular to the skin along this line, just dorsal to the last molar. The depth of the insertion will vary depending upon the size of the animal. By opening the mouth and retracting the lip caudally, you can use the needle cap as a depth gauge by measuring from the location you would you for the oral approach to the level of the skin. Infiltrate the local anesthetic in a fan-like manner in the transverse plane. To redirect the needle it is better to pull the needle almost all the way out before changing the direction of the needle insertion. This will minimize the risks of soft tissue damage.

Retrobulbar Block
This block requires respect from the operator since there is a real chance of death of the patient if this block is performed incorrectly. There are several techniques that have been described such as the Peterson Block, Four-point technique, and Lateral approaches. The approach I will describe I believe is one of the simplest, safest and most reliable compared to the other techniques, some of which risk the injection of local anaesthetics near the optic foramen. This technique is a modification of the technique shown to me by the ophthalmology service of the North Carolina State University. It is a dorsolateral approach. The orbital ligament is palpated and the needle is place caudal to this structure on the dorsolateral aspect of the orbit. A small depression is often present just caudal to this ligament. Depressing your finger into this depression helps to identify this structure. The 1.5” spinal needle is angled towards the center of the eye but in a plane that is caudal to the eye. In fact the tip of the needle needs to be angled slightly rostral during the insertion of the needle. The majority of the globe is in front of the orbital ligament so the risks of damage to the eye are small. In order to penetrate the globe the angle of insertion needs to be very acute with respect to the surface of the skull. As the needle hits the conus ocularis, the eye will rotate in a dorsolateral direction towards the insertion point. Once the needle pierces the conus ocularis the eye will return to a neutral direction again. The location of the tip of the needle is now known. Aspirate for the presence of blood and then inject 0.5 to 2 mL of local anaesthetic depending upon the size of the patient. Lidocaine is safer anaesthetic to use if you have a concern about an intravascular injection. As the needle is withdrawn the eye will rotate in the opposition direction. The entire globe and the extra-ocular muscles will be desensitized by this technique. There will be no effect on the eyelids.

Brachial Plexus Blocks
The classic description of a brachial block will be presented here only. There are new techniques being developed and evaluated for efficacy that are technical demanding and requires the use of an ultrasound machine. The classical brachial block is indicated for any procedures, which require the desensitization of the fore leg from the level of the distal humerus on down. The skin just medial to the shoulder joint is clipped and prepped. Pushing the shoulder joint temporarily caudally will aid with the palpation of the first rib. You will palpate blood vessels, which run cranial to the first rib at approximately midway along the rib. It will feel like a noodle run over the front aspect of the rib. These vessels approximate the level of the brachial plexus. The brachial plexus is located medial to the shoulder joint at the level of the caudal aspect of the joint when in a neutral plane. This can be used as a depth gauge as to the extent with which you insert the needle. The length of the needle
required will vary from a 1.5 to 3.25 inch spinal needle. The needle is inserted parallel with the long axis of the body at the level where the blood vessel run across the first rib. You need to move the needle slightly dorsal to help avoid hitting the vasculature. The needle also needs to be angled slightly in a medial to lateral direction to avoid puncturing the thoracic cavity. A depression is created medial to the shoulder joint when the leg is pulled laterally. At the deepest point of the depression is an approximation of the insertion point. This is at the level of the shoulder joint. Identify the insertion point and supinate the shoulder to open up the space medial to the shoulder. Insert the needle to the pre-determined depth and perform a line block as you pull the needle out.

You must aspirate before injecting each time you move the needle because of the high risk of intravascular injection or intra-pleural injection. To increase the chance of success the local anesthetic can be diluted with saline. This will however decrease the duration of the block. A nerve stimulator can also be used to help identify the exact location of each of the nerves. A smaller, more concentrated volume of local anesthetic can be used. A mixture of lidocaine and bupivacaine may be mixed in the same syringe to help increase the volume. If using bupivacaine, remember it will cause seizing and cardiovascular collapse if administered intravascularly.
**Epidural**
The following are some definitions commonly used.

- **Epidural** ~ Injection of a drug into the epidural space i.e. the space between the dura mater and the wall of the vertebral canal.
- **Spinal** ~ injections into the subarachnoid space i.e. the space between the arachnoid mater and pia mater.
- **Caudal** ~ injection into the epidural at the level of the sacro-coccygeal vertebrae.
- **Neuraxial anesthesia** ~ is the complete blockade of both sensory and motor fibers of the spinal cord.
- **Neuraxial analgesia** ~ is the sensory blockade only of the spinal cord. It is possible to use a low concentration of local anesthetics to achieve this, however it is usually performed using an opioid only.

An epidural is indicated when there is surgery of the tail, hind legs or abdomen. It is possible to use neuraxial analgesia (opioids only) with a large volume with some degree of success for procedures being performed on the thoracic cavity. This however has not been critically evaluated.

<table>
<thead>
<tr>
<th>Absolute</th>
<th>Relative</th>
</tr>
</thead>
<tbody>
<tr>
<td>Allergies to medications used</td>
<td>Hemodynamically unstable</td>
</tr>
<tr>
<td>Coagulopathies – therapeutic and</td>
<td>Anatomical defects e.g. spina bifida</td>
</tr>
<tr>
<td>pathological</td>
<td></td>
</tr>
<tr>
<td>Systemic and local infections</td>
<td>Sacral fractures</td>
</tr>
<tr>
<td></td>
<td>Neurological deficits</td>
</tr>
<tr>
<td></td>
<td>Raised intra-cranial pressure</td>
</tr>
</tbody>
</table>

The epidural sac terminates at a different level depending upon the species and size of the animal. In medium to large dogs, the epidural sac terminates at the level of L5-L7. In cats and small dogs it may terminate at the level of the 1st sacral vertebra, therefore the risks of a spinal injection in cats and small dogs is quite high. If cerebrospinal fluid is aspirated the dose of the epidural is generally halved or some anesthetists perform to use an opioid only. If you inject the normal epidural dose into the subarachnoid space the resulting concentration will be a lot higher than originally intended. The dura mater acts as a barrier to the passage of therapeutic agents thus reducing the concentration of the drugs in the subarachnoid space. The other fact to bear in mind is the CSF is circulating. With a spinal injection the mass of the drug determines the cranial spread of the block, whereas with an epidural the volume of the injection determines the cranial spread. Using the same dose injected into the subarachnoid space would result in a higher cranial spread and potentially cardiovascular and respiratory collapse. These are the reasons why the amount of medication needs to be reduced if you aspirate CSF during your epidural attempt.

Position the animal in either sternal or ventral recumbency. Sternal recumbency does allow for the operator to use the hanging drop method which is preferred by novices however there are
disadvantages to sternal recumbency. If the animal has a fracture of the pelvis or hind leg it will increase the discomfort for the animal during the procedure. If the animal has a good body condition score, the structures are harder to feel for the operator. In lateral recumbency the anatomical structures are easier to palpate and is more comfortable for the animal. The area over the lumbosacral region is clipped and prepped as for a surgical procedure. Wear surgical gloves during the procedure. Draw an imaginary line between the tuber sacrale. The dorsal spinous process of L6 is closest to this line. Palpating the mid-line, the spinous process of L7 is deeper and more rounded as compared to L6. The cranial aspect of the sacrum slopes caudally and the L7-S1 space is wider as compared to L6-L7. A 1.5 to 3.25 inch spinal needle may be required for the epidural puncture. Insert the needle perpendicular to the skin at a point mid-way between L7-S1. It is important to stay on the mid-line. The operator often will feel a “popping” or stepping sensation as the needle is inserted through the ligamentum flavum (see table 2). You can then perform a number of methods to confirm the correct placement of the spinal needle. In young animals it is possible to insert the needle into the ventral aspect of the vertebral canal. If you experience resistance pull back slightly on the needle, and try to confirm correct placement again. The dose of morphine commonly used is 0.1 to 0.2mg/kg diluted with saline or a local anesthetic to a total volume of 1mL/3.5kg (lean weight).

Table 2 Methods used for the detection of the epidural space.

<table>
<thead>
<tr>
<th>Confirmation of a correct placement of an epidural injection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sensation of the needle as it inserted through the ligamentum flavum. Once in the correct place the needle will be held tightly by the ligamentum flavum.</td>
</tr>
<tr>
<td>Hanging drop method – Saline is placed in the hub of the needle and when the tip of the needle is in the correct place the saline is sucked into the epidural space (normally sub-atmospheric)</td>
</tr>
<tr>
<td>Loss of resistance – a small amount of air or saline is attached to the epidural needle once the needle has been inserted through the skin. The plunger is repeated tested for the loss of resistance to the injection once the needle passes into the epidural space.</td>
</tr>
<tr>
<td>Bubble compression test – This is similar to the loss of resistance test. Some air is put into a saline filled syringe. Once the needle is suspected of being in the epidural space the syringe is attached to the needle and a test injection is made. There should be very little compression of the air bubble within the syringe if the needle is correctly placed.</td>
</tr>
<tr>
<td>Nerve twitch – If the needle is touching the nerves you may observe a tail twitch. Pull back on the needle and redirect the needle to avoid injecting directly into the nerves</td>
</tr>
<tr>
<td>Nerve stimulator – If using an insulated epidural needle it is possible to use a nerve stimulator to detect when the needle is close to the cauda equine.</td>
</tr>
<tr>
<td>Test dose – injection of a small volume of lidocaine and epinephrine (2 to 5mcg)</td>
</tr>
<tr>
<td>• Lidocaine – Anal relaxation</td>
</tr>
<tr>
<td>• Epinephrine – transient tachycardiac response if administered intravascularly</td>
</tr>
<tr>
<td>Radiography – usually for the detection of an epidural catheter, if required.</td>
</tr>
</tbody>
</table>

Table 3 The major clinical signs observed as the epidural injectate moves cranially along the spinal cord.

The cranial migration of an epidural and the order of the loss of sensibility and function
Relaxation of the tail and anus
Paresis / Paralysis of the pelvic limbs
Abdominal wall relaxation
Orthostatic hypotension (mild tachycardia)
Sympathetic blockade (bradycardia and hypotension)
Increased respiratory effort – blockade of respiratory muscles
Schiff-Sherrington-like symptoms
Horner syndrome (unilateral or bilateral)
Loss of diaphragmatic respiration

**Neurologic Effects**
The neurological effects resulting from the use of local anesthetics are dependent upon the concentration of the drug present in the epidural space. The sensitivity of the different types of nerves differs with the sympathetic fibers being the most sensitive and the motor fibers the less sensitive in a clinical setting. Motor fibers are more resistant to local anesthetics and are the first neurologic function to return following an epidural. It is therefore possible to inject a certain concentration of local anesthetic, which causes a sensory blockade but not a motor blockade. This is the basis of a differential blockade. Table 1 documents the major effects observed as the epidural injectate moves cranially along the spinal cord.

**Cardiovascular Effects**
The effects of an epidural on the cardiovascular system are complex and are dependent upon the blood volume, level of blockade, Bainbridge reflex, baroreceptor reflex and the Bezold-Jarisch reflex. This interaction is too complex to discuss here in full, so the following is a simplified version. Local anesthetic agents block the sympathetic fibers, which control the vascular tone, will result in vasodilation. This will decrease the systemic vascular resistance and the mean arterial pressure. In a non-anesthetized patient, the portion of the body cranial to the level of the sympathetic blockade will vasoconstrict to compensate for the fall in vascular resistance. The level of anesthesia will influence this compensatory response to the point where it may inhibit the response especially if using isoflurane or sevoflurane. Both of which will cause vasodilation and suppress the baroreceptor reflex. The net effect will be a fall in mean arterial pressure and a modified tachycardic response. Another scenario may result when the level of epidural blockade is too far cranial and has blocked the sympathetic fibers (T1 – T4, cardioaccelerator fibers) that stimulate the heart directly. The net result will be vasodilation, bradycardia and a reduction in inotropy. The blood pressure in this instance will fall precipitously. In rare situations, it is possible for the patient to suffer a cardiac arrest.

**Respiratory Effects**
An epidural normally has little effect on the respiratory function of a veterinary patient. There may be some paralysis to the caudal respiratory muscles, however this does not normally impair respiratory function. The use of an epidural (high volume) should be used with caution if there is a severe anatomical anomaly of the thoracic cage. Respiratory arrest is usually associated with hypotension and a reduction of the perfusion of the respiratory center rather than a blockade of the respiratory muscles. The phrenic nerve normally exits the spinal cord at C4 to C5 therefore you would need to inject a lot of volume to desensitize the spinal cord at this level. You would also
observe cardiovascular collapse with a blockade at C4. There may be modest reductions in the vital and total lung capacities, however there are no changes in airway resistance, functional residual capacity, PaO2 or the alveolar – arterial oxygen gradient.

Gastrointestinal Effects
Local anesthetic agents will block the splanchnic fibers and will result mainly in venodilation. There are subtle species differences based upon the basal vagal tone. In general the vagal tone will be increased to the gut. The muscular tone of the gut will increase along with active peristalsis. There is usually a quicker return to normal gut function following an epidural. Gut flow is better during an epidural rather than systemically administered analgesia. Hepatic blood flow may reduce in parallel to the reduction in the mean arterial blood pressure.

Urogenital Effects
Neuraxial blockade has no effect on renal autoregulation, however the autonomic control of the urinary bladder is affected. Urinary retention is one of the common problems associated with epidural. By simply using a urinary catheter or emptying the bladder at the end of the procedure you can minimize this problem. Large breed male dogs are the most commonly affected by this and are usually associated with a hind leg orthopedic procedure.

Metabolic and Endocrine Effects
The stress response is blunted or blocked by neuraxial analgesia or anesthesia depending upon the concentration of the local anesthetic agented used. It is particularly important in severely debilitated patients. Blocking the T11 sympathetic fibers will blunt the adrenal pathways i.e. catecholamine release however vasopressin is usually increased in response to the loss of neurogenic vasomotor control. For the best results to inhibit the stress response, it is better to administer neuraxial analgesia or anesthesia before the surgical intervention.

Conclusions
These effects may be complicated if the injection is administered intravascularly or into the subarachnoid space which will result in the medications spreading more cranially than expected. There are also the adverse systemic effects of these agents if a relative or absolute overdose is administered. Surgical position may also potentiate the hypotensive effects of an epidural such as the positioning for perineal surgery. It is important to have a good knowledge of these effects when considering using an epidural. In general, the benefits of an epidural usually outweigh the risks associated with an epidural. How to treat the hypotension following an epidural is the subject for another discussion.
This presentation will not include respiratory monitoring since it will be covered in another presentation discussing capnography.

**Introduction**

The true meaning of life from the point of view of any anesthetist is to ensure that the oxygen delivery (DO2) is always more than the oxygen consumption of the tissues (VO2). Oxygen delivery is equal to the product of the cardiac output (CO) and the arterial oxygen concentration (CaO2) \[\text{DO2} = \text{CO} \times \text{CaO2}\]. There are many cardiovascular monitors available which can assess the CO and the CaO2 indirectly. CO is estimated by measuring the arterial blood pressure since mean arterial blood pressure is equal to the product of the CO and the systemic vascular resistance (SVR) \[\text{MAP} = \text{CO} \times \text{SVR}\]. We also know that the CO is the product of the heart rate and stroke volume \[\text{CO} = \text{HR} \times \text{SV}\]. By combining these two equations it will result in the following relationship \[\text{MAP} = \text{HR} \times \text{SV} \times \text{SVR}\].

Stroke volume is dependent upon three cardiovascular parameters, i.e. preload (blood volume), inotropy (contractility) and afterload. It is possible to measure each one of these parameters either directly or indirectly to assess each of the different aspects of the cardiovascular system.

When using the MAP as an indicator of the CO, we are assuming the SVR is normal. Any agent, which greatly affects the SVR, will obviously nullify this assumption. There is no single monitor, which evaluates every aspect of the cardiovascular system. The following discussion will describe the various characteristics of each device. Within the scope of private practice, this article will not discuss cardiac output monitors because they are not commonly used at this point in time.

**ELECTRICAL ACTIVITY**

Electrocardiography – Heart rate, chronotropy

Cardiac function can be categorized into two major parts, i.e. the electrical and mechanical activity of the heart. The electrocardiograph is the modality of choice to monitor the electrical activity of the heart. It is possible to monitor the heart rate and rhythm, as well as electrolyte disorders indirectly by the changes in heart rate and the appearance of the ECG complex for example hyperkalemia. Myocardial ischemia may induce changes to the ST segment of the ECG complex however this is always difficult to interpret because of the large variable between individuals. Changes in the size of each wave are also difficult to interpret with a patient under general anesthesia because it is relatively rare to have a patient in the reference position for an ECG (i.e. right lateral recumbency for small animals). General speaking you only interpret changes in the intervals of an ECG, e.g. PR, QRS, and QT intervals. An ECG is not an indicator of mechanical activity. The classic example of this is pulseless electrical activity (or electromechanical dissociation).

**MECHANICAL ACTIVITY**
Esophageal Stethoscope
An esophageal stethoscope is a simple device to measure the heart rate and rhythm especially when attached to a speaker. The surgery team will have the comfort of hearing the soothing sounds of the regular heart beat rather than simply attaching the stethoscope to ear-pieces. The intensity of the cardiac sounds is a subjective measure of inotropy and is often the first sign to indicate when a person has forgotten to open the pop-off valve.

Doppler
A Doppler device detects the flow of blood in an artery using a piezoelectric crystal, which emits an sound wave when electrically stimulated. A transceiver detects the change in frequency of the wavelength of the sound wave induced by the movement of the red blood cells. A pressure cuff is placed proximal to the probe to act as a resistor to the flow of blood. The width of the cuff is a function of resistance therefore you need to use a standardized width e.g. 40% of the diameter of limb where the cuff is place (~ 30% for cats). In theory, this device is an estimate of the systolic blood pressure at normal blood pressures. Its performance during hyper- and hypotension is less predictable. At low blood pressures it tends to be a better indicator of the mean arterial blood pressure. Compared to oscillometry it tends to be more repeatable however the absolute value is less accurate. A Doppler is better at indicating the overall trends of the arterial blood pressure over time rather than giving an accurate reading at one point of time. This tends to be true for oscillometry also. It is generally accepted that a Doppler device is better for small animals less than 8-10kg and arrhythmias.

Oscillometry
An oscillometry device detects the arterial turbulence with the pressure cuff rather than a specific sensor like the Doppler device. The same sized cuff is used as that of a Doppler. The cuff is inflated above the systolic arterial pressure and then slowly deflated. Small oscillations are transmitted to the cuff and the return signal is analysed to detect any changes in this signal. The arterial pulsations will disturb the returning signal. The pressure in the cuff at which this disturbance is detected is considered the systolic arterial pressure. The point of maximal disturbance of the oscillations is the mean arterial pressure and the diastolic is calculated from first measures. This is a general approach, however the exact signal extraction is different with each device. Similar to the Doppler, oscillometry functions better during normotension and its performance deteriorates during hypotension and bradydysrhythmias. The systolic arterial pressure during hypertension is often under-estimated because of the narrow peak of the curve and the relatively slower signal-sampling rate of the oscillometry.

For both the Doppler and oscillometry devices the cuff must be at the same level as the heart. If the cuff is above the heart, the readings will under-estimate the true arterial blood pressure (1 mmHg = 1.36 cmH2O) and the reverse is true if the cuff is below the heart.

Direct arterial blood pressure monitoring
With a little bit of practice it is relatively easy to place an arterial catheter in a medium to large dog. Once you have mastered this, you can try to do the same thing in a small dog or cat. The dorsal pedal artery is the easiest site to use. Other possible sites include the coccygeal, lingual, carpal arteries and depending upon the type of dog, the auricular artery. It is possible to use a simple manometer attached to some tubing and a three-way stop-cock. This is cheap and easy to do
however due to the high dampening coefficient of this type of system you will only allow you to measure the mean arterial blood pressure. The needle of the manometer will oscillate around the value of the mean arterial pressure.

By using a pressure transducer, it is possible to measure the systolic, mean and diastolic pressures as well as the heart rate. You have the added benefit of being able to evaluate indirectly the preload (blood volume) (diastolic arterial pressure and Delta pressure), degree of inotropy (slope of the up-sweep, pulse pressure) and vascular tone (pulse pressure). A low DAP is indicative of a low preload or profound vasodilation. Bradycardia may also cause a low DAP due to the long run-off time during the diastolic time. One definition of inotropy is $\Delta$Pressure/$\Delta$Time, which is the slope of the curve, the more vertical the slope, the greater the degree of inotropy. SAP is another indicator of inotropy, vascular impedance and compliance. A large variable in arterial blood pressure during the respiratory cycle is the best indicator hypovolemia. Hypovolemia will cause all three arterial pressures to vary more than 10% of the expiratory values during the inspiratory phase. This is because of the positive pressure during mechanical ventilation reducing venous return. You make a comparison of these values between the peak of expiratory and inspiratory. You may use the SAP alone or the pulse pressure for this comparison. Both of these are called the Delta pressure and have a better predictive value than Central Venous Pressure. CVP is traditionally used as an indicator of preload (blood volume), however it may be influenced by right-sided heart disease, pleural space disease, and the position on the table. CVP measurements also require a long catheter with the tip of the catheter resting in either the cranial or caudal vena cava. Jugular filling and emptying times are crude indicators of blood volume and venous return.

<table>
<thead>
<tr>
<th>Characteristic of the arterial curve</th>
<th>Indicator of ...</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diastolic pressure</td>
<td>Preload, vascular resistance, coronary perfusion</td>
</tr>
<tr>
<td>Mean arterial pressure</td>
<td>Perfusion pressure of organs</td>
</tr>
<tr>
<td>Systolic arterial pressure</td>
<td>Inotropy, afterload, vascular impedance and complancce</td>
</tr>
<tr>
<td>Pulse pressure</td>
<td>Inotropy, vascular impedance</td>
</tr>
<tr>
<td>$\Delta$Pressure/$\Delta$Time</td>
<td>Inotropy</td>
</tr>
<tr>
<td>Delta pressure</td>
<td>Preload</td>
</tr>
</tbody>
</table>

**PERIPHERAL PERFUSION**

So far we have concentrated the discussion directly on the heart function alone, however we need to consider the perfusion of the peripheral tissues. All the devices have been evaluating the global function of the cardiovascular system. It is possible to have a good arterial blood pressure whilst having a poor peripheral perfusion. A good example of this is the use of a vasoconstrictor such as an alpha1- or alpha2-adrenergic agonist. In this instance, blood may simply by-pass the capillary bed resulting in tissue hypoperfusion and hypoxia. There are many parameters, which may be used to assess this efficiency of the peripheral perfusion many of which are easy to perform.

**Clinical Signs**

For normal tissue functioning, many organs require a critical perfusion pressure. A MAP of more than 60mmHg is required to have a normal level of consciousness and urine output. Peripheral skin
temperature is another simple indicator of perfusion. The greater the difference between the core and peripheral temperatures, the lower the perfusion pressure of the skin and is a sign associated with hypovolemic shock. The presence of a pulse (SAP – DAP) is another indirect indicator of the MAP. The presence of a pulse in the dorsal pedal artery generally indicates a MAP of approximately 80mmHg. A femoral pulse indicates a MAP of approximately 60mmHg. The femoral pulse will disappear below 60mmHg. These statements are obvious generalizations with a large margin of error.

**Pulse Oximetry**
CaO2 is based mainly upon the arterial oxygen saturation (SaO2) and hemoglobin concentration ([Hb]) and the PaO2 in plasma. The portion of oxygen dissolved in the plasma is extremely small and is not required to estimate the CaO2. The major portion of the oxygen carrying capacity of the blood is based upon the SaO2 and [Hb]. In most clinical situations the SaO2 can be estimated by using a pulse oximetry (SpO2) assuming there is not a dyshemoglobinemia such as carbon mono-oxide poisoning or methemoglobinemia. The [Hb] can be estimated using the hematocrit or packed cell volume, assuming there is normochromasia, if not you need to measure the hemoglobin concentration directly.

The SpO2 probe may be place on a peripheral tissue (tongue, ear, vulva, penis, or toe) to measure the efficiency of peripheral oxygen delivery.

**Conclusion**
MAP may be used as a surrogate for the CO assuming the SVR is within normal limits. CO is one of the depending factors of DO2. CaO2 is the other and can be estimated by measuring the hematocrit and SpO2 in most instances. Therefore, it is possible to ensure a normal DO2 by measuring the MAP, SpO2 and hemocrit ([Hb]). By measuring as many parameters as possible of the cardiovascular system, you may limit the possibility of an adverse event during general anesthesia.
The Physics of Capnography
Capnography is the measurement of carbon dioxide via infrared spectroscopy during the respiratory cycle. The amount of infrared light absorbed is proportional to the concentration of CO2. The respiratory gases are sampled using two methods either sidestream or mainstream sampling. Sidestream is the most common method. Respiratory gases are continually aspirated from the end of the endotracheal tube and analysed by infrared spectroscopy. The gases are then either scavenged or returned to the circuit. Mainstream sampling consists of placing the infrared spectroscope directly onto the end of the endotracheal tube. This is more advantageous for long-term ventilated patients. Capnography displays this information as either the partial pressure of CO2 (mmHg) in the gas or as a fraction (%) in a graphical format. Capnometry on the other hand displays only the end-tidal CO2 (ETCO2) value at one instance in time. Capnometry does not have any of the additional benefits of capnography and improved patient security.

The Benefits of Capnography
Medical research has shown the combination of capnography and pulse oximetry monitoring detected 93% of problems associated with general anaesthesia before these problems became an issue. The main reason to monitor capnography is to assess ventilation indirectly. The only way to truly assess ventilation is to use blood gas analysis. Once you have the PaCO2 and simultaneous ETCO2 measurements you can then use the ETCO2 as an indicator of ventilation. Taking a blood gas analysis is not routinely performed. In patients with a high risk of a ventilation-perfusion mismatch, right-to-left shunt or if there is a suspicion of hypoventilation is a blood gas analysis performed. In healthy patients it is usually unnecessary to perform a blood gas analysis. There are other benefits to monitoring capnography.

The normal arterial – ETCO2 gradient is 2-5mmHg. If this gradient is higher than this, it normally suggests a problem (as previously described above). A high gradient is suggests dead space ventilation (High V/Q mismatch). As the alveolar dead space increases the gradient also increases. There is no CO2 exchange in the alveolar that are not being perfused. The problem is with the pulmonary blood flow. This gas then mixes with and dilutes the CO2 from the other perfused alveoli. This results in a reduction in the ETCO2 and retention of CO2 in the blood. With a low V/Q ratio (Shunt perfusion), the problem is with the lungs. The alveoli are collapsed. The resulting hypoxaemia is resistant to oxygen therapy. The hypoxaemia stimulates ventilation so the arterial – ETCO2 is usually only slightly elevated (4-10mmHg).

Capnography also indirectly measures the metabolic rate and cardiac output of the patient. As both these parameters decrease so will ETCO2. The inverse of this statement is also true. Subtle changes in the capnograph can indicate some forms of respiratory disease. Another advantage of capnography is its ability to monitor the security of the breathing circuit. Capnography can assess the integrity of the patient airway, integrity of the breathing circuit, verification of proper endotracheal tube placement, the function of unidirectional valves, or if the soda lime is exhausted.
Components of the Capnograph

The graphical display of the capnography consists of time along the x-axis and either the percentage (%) or partial pressure of CO2 (mmHg) along the y-axis. If you image a column of gas is inspired into the lungs, then the last portion of gas to enter the mouth and upper airways has not come into contact with the alveoli, therefore there will be no CO2 in this last portion of gas. Upon expiration, during the initial phase (A-B), the gas will be coming from the upper airways and mouth exit the endotracheal tube again there will be no CO2 and hence the initial portion of the curve will be zero. The next portion of gas will come from the lower conducting airways (B-C). There will be some CO2 mixed in the gases from the lower airways from the previous breath and hence the reason why the graph sudden increases. The graph reaches a plateau (C-D) once the gases are originating from the alveoli. This plateau should be smooth and have a slight slope upward. The peak of this gentle slope is the ETCO2 and the last phase of expiration. This is immediately followed by inspiration (D-E). The percentage of CO2 in the inspired gases must be zero, if not there is a major problem with the breathing circuit. The graph should fall almost vertically back to zero. Failure to return to zero indicates rebreathing. Rebreathing occurs when the patient inspires gas from the previous breath which contains CO2. Rebreathing in this instance may be due to a large equipment dead space, inadequate fresh gas flow with a non-rebreathing circuit, malfunctioning unidirectional valves, exhausted soda lime or a faulty capnograph. The normal capnograph is approximately rectangular in shape (see figure 1 below). It is important that all the components of the capnograph are normal. The graph must start and return to zero.

<table>
<thead>
<tr>
<th>Changes in End-Tidal Carbon Dioxide (ETCO2)</th>
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<tbody>
<tr>
<td><strong>Increased ETCO2</strong></td>
</tr>
<tr>
<td>Decreased alveolar ventilation</td>
</tr>
<tr>
<td>• Reduced respiratory rate</td>
</tr>
<tr>
<td>• Reduced tidal volume</td>
</tr>
<tr>
<td>• Increased equipment dead space</td>
</tr>
<tr>
<td>• Mucus plugging, Pneumonia</td>
</tr>
<tr>
<td>• Endobronchial intubation</td>
</tr>
<tr>
<td>Increased CO2 production</td>
</tr>
<tr>
<td>• Fever</td>
</tr>
<tr>
<td>• Hypercatabolic state</td>
</tr>
<tr>
<td>• Shivering</td>
</tr>
<tr>
<td>• Bicarbonate administration</td>
</tr>
<tr>
<td>• Tourniquet release</td>
</tr>
<tr>
<td>Increased inspired PCO2</td>
</tr>
<tr>
<td>• Rebreathing</td>
</tr>
<tr>
<td>• CO2 absorber exhausted</td>
</tr>
<tr>
<td>• External source of CO2</td>
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Sampling error
- Inadequate tidal volume
- Water blocking sampling line
Normal Capnograph

Abnormal Capnographs

Oesophageal intubation: Small flat curves without a plateau, which gradually diminish in size. It is not uncommon for the stomach to contain a small amount of CO2. A normal capnograph is the gold standard for the verification of correct placement of an endotracheal tube.

Obstruction in the airway: As the size of a partial obstruction increases, airway resistance increases and air velocities fall. There is a slow upsweep to the phase C-B and the plateau. The inspiratory phase will eventually become sloped as well when the airway resistance is high. Possible causes – kinked endotracheal tube, mucus in the tube, obstruction in the expiratory limb of the breathing circuit or bronchospasm.

Curare cleft (reversal of muscle relaxants): There is a cleft in the plateau indicating voluntary breathing during the breath generated by the ventilator. The depth of the cleft is inversely proportional to the drug activity.

Cardiogenic oscillations: Rhythmical oscillation of the last portion of the plateau and the inspiratory phase. The oscillations are synchronized with the heart rate. This is a benign effect that is associated with slow respiratory and cardiac rates.

Inadequate seal around the ET tube: Phase B-C appears normal with a sudden cessation to the plateau and a slow rate of decline to the phase D-E.

Hypoventilation – increase in ETCO2: There is a gradual increase in the peak of the plateau or ETCO2. This may be due to a reduction in respiratory rate, decrease in tidal volume, increase in metabolic rate or a rise in body temperature.

Hyperventilation – decrease in ETCO2: A gradual slow reduction in the ETCO2. This may be due to an increase in respiratory rate, tidal volume of a decrease in metabolic rate or body temperature.

Rebreathing: The baseline rises above zero. This is an important pattern to recognize. The potential causes of rebreathing include a clogged expiratory filter, a malfunctioning expiratory valve, inadequate inspiratory flow, insufficient expiratory time, exhausted soda lime or resistance to the expired gas flow.

Sudden decrease in ETCO2: A drop in pulse oximetry and an increase in arterial – ETCO2 gradient is indicative of a pulmonary embolism – thrombus, fat, gas embolism.
1. Identify the following components:
   a. The supply of oxygen
   b. The flowmeter
   c. The vaporizer
   d. The common gas outlet. Why is this important?
   e. The rapid oxygen valve. When should you use this?
   f. The unidirectional inspiratory valve
   g. The inspiratory segment of the circle circuit
   h. The expiratory segment of the circle circuit
   i. The unidirectional expiratory valve
   j. The reservoir bag
   k. The exhaust valve
   l. The circuit manometer
   m. The exhaust system.
   n. Define the dead space of the breathing circle.

2. Perform a leak test for a circle circuit and non-recycling circuit e.g. Bain circuit.

   Is it safe to use an anaesthetic machine that has passed a leak test?

3. Why does an anesthesia machine need a reservoir bag?
   a. Switch on the flow of oxygen
   b. Breathe through the filter circuit using a mask
   c. Look at the gauge of the circuit
   d. What is the circuit pressure during the breathing cycle?
   e. Remove the reservoir bag and block the connection of the breathing bag with your hand;
   f. Breathe through the filter circuit again
   g. What is the circuit pressure during the breathing cycle?
   h. What does this mean?

**Breathing with a Reservoir Bag**

The pressure of the circuit should approximate zero during the respiratory cycle. The reason for this is because of the reservoir bag. The fresh gas flow fills the respiratory circuit only and not the patient. The patient breaths in the gases (oxygen and the volatile anaesthetic) from the breathing circuit, i.e. the reservoir bag during inspiratory phase therefore the pressure in the circuit remains close to zero. During the expiratory phase the patient breaths out into the circuit thus increasing the volume of gas in the circuit by the tidal volume. The reservoir bag will increase in size during the expiratory phase thus allowing the volume of the circuit to expand, thanks to the high compliance of the reservoir bag. The reservoir bag allows the volume of the respiratory circuit to shrink and expand by the same volume as the tidal volume.
Breathing without Reservoir Balloon

When you remove the reservoir bag from the respiratory circuit, the compliance of the respiratory circuit will decrease, i.e. for a given change in volume of the respiratory circuit there will be a greater change in pressure as measured on the manometer. During the inspiratory phase the pressure of the respiratory circuit will become more negative as the volume of the respiratory circuit is decreased by a volume equal to the tidal volume. This will also increase the work of breathing for the patient. During the expiratory phase, the pressure of the respiratory circuit will increase by the volume of the tidal volume but not by the same magnitude as during the inspiratory phase. This is because the spring-loaded escape valve will open when the respiratory circuit is pressurised thus allowing some of the expired gases to escape the respiratory circuit.

The reservoir bag allows the pressure within the respiratory circuit to remain close to zero. By doing this, the resistance of the respiratory circuit and the work of respiration are both decreased.

4. What will happen if the reservoir bag is too small?
   a. Replace the reservoir bag with a small bag
   b. Breathe deeply through the filter circuit
   c. Once the tank bag collapses, look at the gauge of the circuit
   d. Describe how you feel?

By replacing the appropriate-sized reservoir bag with a smaller reservoir bag where its volume is smaller than the tidal volume. During the inspiratory phase, the reservoir can collapse completely before the inspiratory phase has been completed. This will result in the airway pressure of the respiratory system becoming negative. The work of breathing will increase because of the need to generate an even more negative pleural pressure. During the expiratory phase the reservoir bag may become distended depending on the degree of disparity between the size of the patient and the size of the respiratory bag. The respiratory circuit will become pressurized by a smaller degree in the respiratory circuit during the inspiratory phase. This is because of some expiratory gas escapes through the escape valve.

5. What will happen if there is a hole in the inspiratory segment of a Bain circuit or coaxial circuit (e.g. F-circuit)?
   a. Look at the capnograph: what is the effect?
   b. What is the effect of increasing the flow of fresh gas?

What is the dead space of the respiratory circuit when there is a hole in the inner tube of a coaxial breathing system?

What will be the effect if there is a hole in the outer tube of a coaxial breathing system?

What will happen to the SpO2% as the FiCO2 increases over time?

The baseline of the capnogram will become elevated, i.e. the inspired CO2 will become positive. Normally the inspired CO2 must equal zero. Increasing the fresh gas flow depending upon the location of the hole may have little effect on the rebreathing of CO2. To correct this situation, we
must replace the respiratory circuit. A hole in the outer tube will pollute the work environment and may allow entrainment of air room into the inspired gases thereby reducing the fraction inspired concentration of the anaesthetic gas.

The SpO2% will stay normal despite the fact the FiCO2 is elevated. As long as the patient is able to increase their respiratory minute ventilation to compensate for this, there will be little change to the PaCO2 and SpO2% levels. If the anaesthetic depth is such that it inhibits the stimulus to increase the respiratory minute volume, then the PaCO2 will increase causing a reduction in pH. The SpO2 will most likely remain normal because of the enrichment of the fraction inspired oxygen will compensate for the relative small increase in CO2.

6. **What will happen if the unidirectional valve is defective?**
   a. Remove the dome from the unidirectional expiratory valve;
   b. Remove the expiratory valve disc
   c. Replace the dome of the one-way expiratory valve;
   d. Breathe through the circle circuit
   e. Look at the capnograph
   f. What is the effect?
   g. What is the effect of increasing the flow of fresh gas?

In this situation, we are simulating a situation when a unidirectional valve is open all the time. This occurs when the anaesthetic machine is on a tilt. The other common scenario occurs when the humidity in the circuit is high. The high humidity causes the disc to stick on the dome of the unidirectional valve. When the disc is removed, the gas flow in the respiratory circuit is bidirectional. This implies that the patient is now rebreathing the exhaled gas of the previous breathe. Increasing the fresh gas flow has little effect on correcting rebreathing. We must correct the underlying problem.

### Differential Diagnosis of rebreathing of CO2

<table>
<thead>
<tr>
<th>Circle circuit (recycles exhale gas)</th>
<th>Non-recycling system</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased dead space of the breathing circuit</td>
<td>Increased dead space of the breathing circuit</td>
</tr>
<tr>
<td>A hole in the inspiratory segment of the coaxial circuit</td>
<td>A hole in the inspiratory segment of the Bain circuit</td>
</tr>
<tr>
<td>A defective one-way valve</td>
<td>A defective exhaust valve in the Lack breathing system</td>
</tr>
<tr>
<td>Exhausted soda lime</td>
<td>-</td>
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<tr>
<td>Defective capnograph</td>
<td>Defective capnograph</td>
</tr>
<tr>
<td></td>
<td>Insufficient fresh gas flow</td>
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<tr>
<td></td>
<td>Lack of a respiratory pause</td>
</tr>
</tbody>
</table>

7. **What will happen if the exhaust valve is left closed?**
   a. DO NOT do this test if you have a medical history of respiratory illness, for example, asthma
b. Breathe through the filter circuit normally
c. Now close the exhaust valve
d. Look at the pressure gauge of the filter circuit
e. Continue to breathe until you are uncomfortable or the pressure is 10 to 15 cm H2O between each breath
f. How do you feel?

This test is to demonstrate how dangerous anaesthetic machine could be for the patients. When the escape valve is closed the pressure in the respiratory circuit quickly increases. This is particularly so with the Bain circuit, where the time required for the increase to be lethal is only a few minutes. The increase in pressure in the respiratory tract is transmitted to the thoracic cavity of the patient. The increase in intrathoracic pressure will decrease the diameter of the cranial and caudal vena cavae and hence reduce the flow of blood into the thoracic cavity, i.e. reduce the venous return. The patient will die because of acute cardiogenic shock and not due to barotrauma
Companion Animal: Dermatology
Canine Atopic Dermatitis on the Horizon
Andrea Lam, DVM, DACVD

Paper not available in time for online posting.

Soap and Suds: More Than Adjunct Therapy
Andrea Lam, DVM, DACVD

Paper not available in time for online posting.

Otitis: Diagnosis and Treatment of Ear Canal Disease
Andrea Lam, DVM, DACVD

Paper not available in time for online posting.
Clinical Approach to Canine Alopecia
Manon Paradis, DMV, MScV, Dipl. ACVD

Introduction
Causes of alopecia are numerous in dogs and include infections (e.g., dermatophytes, demodicosis, bacterial folliculitis, leishmaniosis), self-inflicted hair loss (from hypersensitivities or parasitism), immune-mediated diseases (sebaceous adenitis, dermatomyositis, alopecia areata, etc.) endocrinopathies, follicular dysplasias, etc.

Hair growth is influenced, among other things, by gonadal, adrenal, thyroidal, pituitary and pineal hormones. Excesses, deficiencies and hormonal imbalances have been incriminated in a myriad of clinical syndromes in dogs. In some endocrinopathies (e.g., hypothyroidism, hyperadrenocorticism, hyperoestrogenism and pituitary dwarfism) the hormonal implication is well understood and these disorders are relatively well characterized clinically. However, other alopecic disorders may resemble endocrinopathies clinically (e.g., canine recurrent flank alopecia, alopecia-X, colour dilution alopecia and other follicular dysplasias) and, in many instances, the final diagnosis can be more difficult to establish.

The aim of this presentation is to provide the clinician with a methodical clinical approach to canine alopecia, especially the non-pruritic, non-inflammatory, symmetrical hair loss.

Clinical Approach
A complete history should be taken and a general physical examination conducted to detect any abnormality present in other organs. A history of polyuria-polydipsia, the presence of a pendulous abdomen or abnormal genitalias (testicular asymmetry or cryptorchidism, vulvar enlargement) may greatly influence further tests to be carried out. The history and the dermatological examination should allow the clinician to rule in/out the presence of pruritus. If present, it should be investigated first. If pruritus is absent or minimal, one should determine whether the pattern of hair loss is focal or symmetric and diffuse. In addition, one should look for presence of inflammation and/or any primary skin lesions such as papules and pustules. Skin scrapings, skin cytology and/or dermatophyte cultures are often indicated if such skin lesions are present. If pruritus, inflammation or any other primary lesions are absent, the next most pertinent diagnostic procedure to perform will be influenced by age of onset, breed and sexual status.

1. Signalment and history
Consideration of the dog’s age at the time of onset of alopecia, the rate of progression of the alopecia, its spontaneous resolution or its progression, and the presence of a cyclical pattern will help in compiling a list of differential diagnoses.

Age and time of onset. The onset of alopecia should always be related to the dog’s age and any physiological and/or pathological event, management change or treatment. Alopecia sometimes occurs a few weeks after physiological events, such as pregnancy and lactation, or pathological events, such as severe systemic disease, shock or surgery (e.g., telogen defluxion). Failure of hair regrowth after clipping is suggestive of hypothyroidism, hyperadrenocorticism and alopecia-X. In Nordic breeds it is a common finding as in these breeds hair follicle cycle is longer than in other breeds.
Many disorders have an age at onset that is quite predictable. Congenital alopecia is present at birth; canine pattern alopecia often develops between 6 and 12 months of age, demodicosis usually occurs before 1 year of age. Clinical signs due to hypothyroidism typically develop between 3 to 6 years of age, and spontaneous hyperadrenocorticism occurs generally in middle-aged to old dogs.

**Breed.** Some breeds are predisposed to alopecic conditions such as alopecia-X (e.g., Pomeranian, Keeshond, Malamute, miniature poodle), canine pattern alopecia (e.g., Dachshund, Chihuahua) and canine recurrent flank alopecia (e.g., boxer, airedale).

**Coat color.** Coat color may provide useful diagnostic information in color-linked alopecia such as black hair follicular dysplasia, color dilution alopecia, and follicular lipidosis.

**Sexual status.** Hyperestrogenism due to Sertoli’s-cell tumors and ovarian cysts or tumors may lead to alopecia. Prolonged estrus can be seen in bitches with hyperestrogenism whereas anestrus has been reported in bitches with hyperadrenocorticism and hypothyroidism.

**Spontaneous remission.** Spontaneous remission usually occurs in canine recurrent flank alopecia, anagen and telogen defluxions, and post-clipping alopecia not secondary to endocrinopathies. Alopecia areata may also resolve spontaneously and is often associated with regrowth of white hair (leucotrichia). Spontaneous remission is also seen in localized demodicosis and dermatophytosis; however, clinical inflammation and scaling is usually observed.

**Progression.** Slow progression of alopecia is more indicative of a systemic problem (e.g., endocrinopathies). Seasonality is more indicative of canine recurrent flank alopecia and flea or pollen allergy.

**Signs of internal disease.** Owners of dogs with hyperadrenocorticism often report polyuria, polydipsia and polyphagia. In canine hypothyroidism, the owner may describe signs that reflect the slowing of metabolism, such as lethargy and weight gain.

**Previous treatments.** Endogenous and exogenous corticosteroids are notorious in causing Cushing’s syndrome. One should not underestimate the effect of corticosteroids on hair growth (even when only applied topically such as in eyes), and on alkaline phosphatase and thyroid hormone levels. Focal alopecia may develop at site of injection, especially rabies vaccines.

2. **Physical examination**

   Pendulous abdomen and hepatomegaly is frequently seen in hyperadrenocorticism. Enlarged lymph nodes can be seen in leishmaniasis. Abnormal genitalias (e.g., gynecomastia, testicular asymmetry or cryptorchidism) can be observed in hyperestrogenism.

3. **Dermatologic examination**

   Alopecia may be a feature of a myriad of skin diseases; therefore, thorough clinical examination of hair coat and skin is important. Presence of primary or secondary skin lesions (e.g., papules,
pustules, scaling, crusts), follicular casts (e.g., in demodicosis and sebaceous adenitis), skin thickness, aspect of hair shafts (broken or not) are some of the findings that can be very helpful to orient toward more specific diagnoses.

Erythema, papules, pustules, lichenification, self-trauma (recognized by broken hairs and excoriations) are all suggestive of an inflammatory process and pruritus. Thinning of the skin with prominent subcutaneous vessels and calcinosis cutis are pathognomonic of hyperadrenocorticism, whereas hypothyroidism is often accompanied by thickened and hyperpigmented skin without inflammation, unless a secondary bacterial infection is present. In canine recurrent flank alopecia, the alopecic areas are typically well demarcated and hyperpigmented.

*Coat and skin color change.* Coat color change (e.g., brown discoloration), especially noticeable in white dogs, is suggestive of licking. A regrowth of white hair, where the hair coat was formerly pigmented, is suggestive of alopecia areata.

Intense skin hyperpigmentation of alopecic areas is most commonly seen in dogs with recurrent flank alopecia and alopecia X. However, hyperpigmentation is also often observed in response to chronic inflammation.

*Pattern of alopecia.* The pattern of the hair loss (e.g., focal, multifocal, moth-eaten, asymmetric, symmetric and diffuse) should be documented. Infectious alopecias generally develop a more asymmetric, multifocal, sometimes moth-eaten pattern, whereas endocrine alopecias and other hair cycle abnormalities are more symmetric in pattern. Allergies, however, may also present with bilaterally symmetric alopecia but the history will reveal that the dog exhibits pruritus toward the affected areas.

Canine pattern alopecia (CPA), as the appellation implies, follows a predetermined distribution and usually start around 6 months of age. In CPA ventral type, the alopecia develops along the ventral neck, chest and abdomen, the caudomedial aspect of thighs, perineum, and the base of the convex aspect of pinnae. In CPA pinnal type, the alopecia involves the entire convex aspect of the pinnae.

4. **Further investigation**

If the results of the above evaluation have failed to produce a definitive diagnosis, further tests are necessary. These should be selected according to the index of suspicion. Microscopic examination of skin scrapings, hair plucks (trichoscopy), Wood’s lamp examination (+/− fungal culture) and cytological evaluation of impression smears or swabs may all be required if primary or secondary lesions such as papules, pustules, erythema, scaling, crusting, follicular casts are seen.

Hematology, biochemistry and urine analysis may be useful to evaluate the general health status of adult dogs with an alopecic condition or if a systemic disease, which may lead to alopecia, is suspected. Hormonal tests (e.g., thyroid hormone profile, ACTH stimulation test, low dose dexamethasone suppression test) should be carried out if the clinical signs and results of blood or urinalysis suggest an endocrinopathy. In contrast, skin biopsies may be the initial and unique diagnostic procedure performed if immune-mediated alopecias such as sebaceous adenitis is strongly suspected.
ALOPECIA, CANINE

Alopecic breeds
- Chinese crested dog
- Mexican hairless dog
- Peruvian hairless dog
- X-linked ectodermal dysplasia

Congenital hypotrichosis/alpecia

Congenital?
Yes → Acquired
No → Self-inflicted?

Yes → Symmetric/diffuse alopecia
No → Immune-mediated (microscopic inflammation)

Infections
- Staphylococci
- Demodex
- Dermatophytes
- Malassezia
- Leishmania

Endocrinopathies
- Hypothyroidism
- Hyperadrenocorticism
- Hyperestrogenism
- Pituitary dwarfism

Miscellaneous
- Telogen defluxion
- Anagen defluxion
- Pattern alopecia
- Pinna type
- Ventral type
- Neoplastic alopecia (e.g. epitheliotropic lymphoma)

Follicular dysplasias

Coat colour-linked?
Yes → Follicular dysplasias
No → Colour dilution alopecia

- Black hair follicular dysplasia
- Breed associated follicular dysplasias
- Red or black Doberman f.d.
- Weimaraner’s f.d.
- Rottweiler follicular lipidosis

- Alopecia-X
- Recurrent flank alopecia
- Breed associated follicular dysplasias
  - Portuguese Water dog
  - Irish Water spaniel
  - Chesapeake Bay retriever
  - Curly coated retriever
  - Pont Audemer spaniel

Adapted from Manon Paradis, Clinical Veterinary Advisor 3rd ed. 2014

References:


Infectious Causes of Canine Alopecia
Manon Paradis, DMV, MScV, Dipl. ACVD

Introduction
Skin infection (e.g., bacterial folliculitis, Malassezia dermatitis, dermatophytes, demodicosis) are frequent causes of focal or multifocal alopecia. The hair loss usually results from inflammation and direct damage to the hair follicles. In addition, in pruritic disorders, self-inflicted alopecia often aggravates the hair loss caused by the infection itself. The aim of this presentation is to overview the clinical features and diagnosis of the infectious causes of alopecia.

Bacterial Infection
Bacterial pyoderma is common in dogs and most cases are caused by coagulase-positive Staphylococcus pseudintermedius, a normal component of canine skin flora.

Classification. Pyoderma can be subdivided in (1) surface pyoderma (infection restricted to the surface layer of the epidermis) which includes intertrigo (skin fold pyoderma), acute moist dermatitis (pyotraumatic dermatitis, hot spots), and bacterial overgrowth syndrome (BOGS); (2) superficial pyoderma (infection involving the epidermis and the infundibular portion of the hair follicle) which includes impetigo, superficial bacterial folliculitis (SBF), exfoliative superficial pyoderma (superficial spreading pyoderma), and mucocutaneous pyoderma, and; (3) deep pyoderma (bacterial infection extending beyond the hair follicle to involve the dermis and subcutis, which may lead to cellulitis) which include acne, pyotraumatic furunculosis, nasal folliculitis and furunculosis, interdigital furunculosis/ pododermatitis, infected acral lick dermatitis, callus pyoderma, postgrooming furunculosis, and German shepherd pyoderma.

Underlying etiologies. Some type of pyoderma, particularly SBF and BOGS are often secondary to underlying etiologies such as allergic skin diseases (atopic dermatitis, cutaneous adverse food hypersensitivity, flea bite hypersensitivity), endocrinopathies (e.g., hypothyroidism, hyperadrenocorticism), parasitic skin disease (e.g., Sarcoptes, Demodex spp.), immune-mediated diseases, cornification disorders (e.g., sebaceous adenitis) and follicular dysplasia (e.g., colour dilution alopecia). Deep pyoderma may be associated with demodicosis and other underlying immuno-incompetence. Any therapeutic plan for controlling pyoderma without considering underlying predisposing factors is destined to fail.

Clinical features. In SBF, one can observe papules, pustules, epidermal collarettes, and patchy alopecia producing a “moth-eaten” appearance of the haircoat over the trunk. Resolving lesions may show central hyperpigmentation (“bull’s-eye” lesion). In exfoliative superficial pyoderma, large epidermal collarettes with an erythematous edge is seen over the trunk. The associated exudate may form crusts.

Level of pruritus can be quite variable in superficial pyoderma, varying from absent to severe. In addition to the alopecia resulting from the inflammatory process, a variable level of alopecia may result from self-trauma due to pruritus.
Diagnosis. The dermatological examination alone may provide a strong suspicion of pyoderma. Epidermal collarettes are extremely useful secondary skin lesions to look for (clip some hair if needed); they are strongly suggestive of a superficial pyoderma. Presence of a hemorrhagic discharge, resulting from a breach in the basal membrane, is evocative of a deeper bacterial infection (furunculosis).

Skin cytology is very useful to confirm the infection (e.g., degenerated neutrophils, free and phagocytized cocci). Bacterial culture and sensitivity may be required, particularly if there has been a failure to respond to rational antibiotic therapy or if bacilli are noted on skin cytological examination.

Differential diagnosis. Dermatophytosis and demodicosis have a similar clinical presentation. Pemphigus foliaceus and epitheliotrophic lymphoma may also present as pyodermas that fail to respond to appropriate antibiotic therapy. In these instances, skin biopsy is indicated to confirm the diagnosis.

Treatment. Treatment usually consists of topical antiseptic treatment (e.g., chlorexidine) more commonly under the form of shampoo (usually sufficient for most cases of surface pyoderma and many cases of superficial pyoderma). Oral antibiotic (for a minimum of 3 weeks and for 1-2 weeks beyond clinical cure) is normally required for more severe cases of SBF and in most cases of deep pyoderma.

Fungal Infection
Yeast (Malassezia) dermatitis and dermatophytosis, two superficial fungal skin infections, are relatively common cause of alopecia in dogs.

Yeast (malassezia) dermatitis
Yeast dermatitis is a common pruritic dermatitis caused by the overgrowth of Malassezia spp. yeast on skin surface.

Etiology. Most infections are caused by the lipophilic unicellular organism Malassezia pachydermatis, which is part of the normal skin microflora. The yeast can become opportunistic invaders when changes occur in the cutaneous microclimate (e.g., lipid composition, relative humidity) or defense mechanisms (e.g., immunosuppression, epidermal barrier dysfunction). Once colonization takes place, yeasts release proteases and lipases that alter cutaneous homeostasis, allowing for continued yeast overgrowth. In addition, in some atopic dogs with cytologic demonstration of yeasts, Malassezia may elicit a type-I cutaneous hypersensitivity reaction.

Specific disorders that predispose to cutaneous Malassezia overgrowth include allergic skin disease (e.g., atopic dermatitis, adverse cutaneous food reaction, flea bite hypersensitivity, contact allergy), endocrinopathies (e.g., iatrogenic or spontaneous hyperadrenocorticism, hypothyroidism, hyperthyroidism, diabetes mellitus), cornification disorders, nutritional deficiencies and metabolic diseases (e.g., superficial necrolytic dermatitis, zinc-responsive dermatosis).
Clinical features. Moderate to intense pruritus is the most common complaint but rancid offensive odor, oily coat, alopecia, erythroderma, lichenification, scaling are frequently observed. Skin lesions reflect existing pruritus and are not specific to Malassezia dermatitis.

Localized yeast dermatitis involves the face (perioral, muzzle, ears), ventral aspect of the trunk (neck, axillae, inguinal area), ventral tail and perianal area, skin folds, or paws (interdigital web and nail fold).

Generalized yeast dermatitis involves several regions as described above. Lesional skin may be erythematous, scaly, greasy or dry, alopecic, saliva-stained, and excoriated. Hyperpigmented lichenification is seen in more chronic lesions. A brown waxy discharge may be observed in the claw folds with extension onto the claws proper.

Diagnosis. Skin cytology is the most reliable diagnostic procedure and should be performed on every dog with compatible historical and physical findings. Skin scrapings are also indicated to exclude ectoparasites.

Differential diagnosis. The differential diagnosis includes most pruritic skin diseases encountered in dogs, particularly BOGS and superficial pyoderma, allergic skin disease and sarcoptic mange.

Treatment. Treatment consists of keratomodulating and antiyeast topical treatments, and systemic antifungal treatment such as ketoconazole (5-10 mg/kg PO q24h with food). It is important to look for and treat the underlying predisposing diseases.

Dermatophytosis
Dermatophytosis is a contagious superficial fungal skin disease of hair, skin or claws.

Etiology. In order of prevalence, causative fungal species include Microsporum canis, Microsporum gypseum, and the Trichophyton group. Dermatophytes are not part of the normal fungal flora. Dermatophytosis is a self-curing disease in healthy animals and most infections will resolve without treatment in 1 to 3 months. Recovery is typically associated with development of a strong cell mediated immunity.

Clinical features. Lesions are characterized by focal to multifocal areas of alopecia, scaling, crusting, with or without erythema, and variable pruritus.

Diagnosis. Diagnosis is based on a combination of compatible clinical signs and confirmation of infection of the lesions. Active infections are documented by Wood’s lamp, microscopic examination of infected hairs and fungal culture. M. canis is the only veterinary pathogen that fluoresces (in > 90% of the cases) when exposed to UV light. Positive fluorescence is apple green and found only on the hair shaft (never nails or crusts). Wood’s lamp positive hairs can be plucked and mounted in mineral oil for direct microscopic examination or used for fungal culture. All suspect colonies must be microscopically confirmed, using methylene blue or lactophenol cotton blue stain.

Differential diagnosis. Superficial pyoderma and demodicosis have a similar clinical presentation and are more common in dogs; therefore, these disorders should be suspected first.
**Treatment.** Treatment consists of antifungal topical treatment such as enilconazole or lime sulfur dips, and systemic antifungal treatment such as ketoconazole or itraconazole.

**Parasitic Infestations**
Various ectoparasitic infestations can cause alopecia by their direct damage to the hair follicle (demodicosis) or from self trauma due to the intense pruritus (sarcoptic mange and flea bite hypersensitivity).

**Demodicosis**
Demodicosis is a common alopecic disease in dogs.

**Etiology.** Skin lesions develop secondary to overgrowth of Demodex canis mites in the hair follicles.

**Clinical features.** Canine demodicosis is classified as localized or generalized demodicosis. Localized demodicosis is most common in young dogs between 3 to 8 months of age. Lesions, consisting of one or more localized areas of alopecia, erythema, and scaling, are typically found on the face and forelimbs.

Canine generalized demodicosis may be classified as juvenile onset (affecting dogs from 3 to 18 months of age) or adult onset (affecting middle-aged to older dogs, often immunocompromised animals with underlying hyperadrenocorticism, hypothyroidism, diabetes mellitus, immunosuppressive drug therapy, or neoplasia). Clinical signs are variable and may start with localized lesions that spread. Secondary deep bacterial infection (furunculosis) is very common.

**Diagnosis.** Diagnosis is confirmed with deep skin scrapings or hair plucks (trichoscopy) revealing numerous adult D. canis mites and its immature forms (nymph, larvae and ova). General health of the dog should be evaluated, particularly in the adult-onset form, and any underlying diseases or concurrent infections should be treated.

**Other parasitic infestations**
Flea bite allergy dermatitis and sarcoptic mange (canine scabies) can result in significant amount of alopecia. However, pruritus, is usually the initial and most important clinical signs.

In sarcoptic mange, the mites often affect the ear pinnae margins and elbows. Female mites burrow through the epidermis and lay their eggs in the resulting tunnel. Many dogs develop a hypersensitivity reaction to mite antigens. Clinical features include an intensely pruritic, nonseasonal dermatitis with papules, excoriations, thick yellowish crusts, and alopecia. Lesions may rapidly generalize, but the dorsum is usually spared.

In flea bite hypersensitivity, pruritus and lesions (papules, crusts, excoriations, erythema, and alopecia) typically develop over the dorsal lumbosacral region, tail head, and caudomedial thighs.
References:

Immune-Mediated Causes of Canine Alopecia
Manon Paradis, DMV, MScV, Dipl. ACVD

Introduction
There are several immune-mediated dermatopathies causing alopecia in dogs. The aim of this presentation is to provide the clinician with an overview of selected immune-mediated skin disorders causing alopecia. These include sebaceous adenitis, dermatomyositis, post-rabies vaccine alopecia, adult onset generalized ischemic dermatopathy, alopecia areata (pelade) and isthmic lymphocytic mural folliculitis (pseudopelade).

SEBACEOUS ADENITIS
Sebaceous adenitis is an uncommon idiopathic skin disease in the dog.

Etiopathogenesis. The exact etiopathogenesis of this disease remains unknown. A genetically inherited cell-mediated immune reaction directed against a component of the sebaceous glands is suspected. The common feature of the disease is an inflammatory infiltrate affecting the sebaceous glands resulting in their destruction. Alopecia occurs by hair shafts breaking easily, most likely due to a decreased amount of sebum produced by the sebaceous glands.

Clinical features. Sebaceous adenitis is most commonly seen in standard poodles and Akitas but has been diagnosed in various other breeds including Vizsla, German shepherd dog, Hovawart, Lhasa apso, Bernese mountain dog, and mongrels. Young adult to middle-aged dogs are most commonly affected. No sex predilection has been reported.

There is a marked variability of clinical presentation depending on individual breeds and severity. Follicular casts (white scales adherent to hair shafts) is a common feature. They most likely result from the lack of sebum in the hair follicle infundibulum, where epidermis-like keratinization and desquamation occur.

In long-coated dogs (e.g., standard poodles, Akitas) the first sign of disease is follicular casts protruding from the hair follicle. When hairs are plucked, the follicular keratinaceous debris cast the root of the hair shafts. In standard poodles, the disease starts most often on the dorsal muzzle and temporal region, spreading to the dorsal neck and thorax, whereas in Akitas the alopecia is typically more extensive. Broken hair shafts, dull and brittle hair coat, excessive scaling, change in hair colour, and musty odour may be seen. Pruritus is variable but may be marked, especially if secondary bacterial or yeast infection is present. In the Akita, where the disease can be more severe, fever, anorexia, and lethargy have been reported.

In short-coated dogs such as Vizsla, clinical presentation consist of coalescing patches of scaly alopecia with adherent scales developing more commonly on the face, head, and trunk.

Diagnosis. Presence of follicular casts and alopecia in a susceptible breed is highly suggestive of the disease. Skin biopsy and histopathological examination is necessary to confirm the diagnosis.

In the early phase, dermatohistopathological changes are characterized by discrete perifollicular inflammation at the isthmus level of hair follicles. Later, nodular, granulomatous to
pyogranulomatous inflammatory reaction around the sebaceous glands is seen. In addition, orthokeratotic hyperkeratosis and follicular plugging is observed in long-coated breeds. These hyperkeratotic changes tend to be milder in short-coated breeds. In advanced stages of the disease, the sebaceous glands are completely destroyed and the inflammatory reaction may disappear. Telogenization of hair follicles or follicular atrophy may occur. Suppurative folliculitis or furunculosis can be found when secondary staphylococcal infection is present.

Skin scrapings and hair plucks (trichoscopy) are useful for ruling out ectoparasites. Casts of keratosebaceous material adherent to hair shafts can be seen on trichoscopic examination, which is suggestive of sebaceous adenitis, particularly in absence of Demodex mites. Cytologic examination to assess for presence of secondary bacterial or Malassezia infection, and Wood’s lamp and dermatophyte culture to rule out dermatophytosis may also be required.

Differential diagnosis. Secondary sebaceous gland destruction with similar clinical hyperkeratosis can also occur with demodicosis, leishmaniasis, and severe granulomatous and histiocytic folliculitis. Differential diagnosis also includes dermatophytosis, bacterial folliculitis and various cornification disorders such as ichthyosis.

Treatment. Topical treatment with oil soaks, humectants (propylene glycol 50-75%) and shampoos are often effective but quite laborious. Oral cyclosporine (5 mg/kg q24h) has been shown to improve the clinical signs and to reduce inflammation greatly, as well as achieving regeneration of sebaceous glands. It is, however, an expensive treatment option. There is evidence of a synergistic benefit on both scaling and alopecia if topical therapy is combined with oral cyclosporine. Isotretinoin has been reported to be effective in Vizsla. However, it is difficult to comprehend why considering it is known to reduce sebaceous gland size and decrease sebum secretion.

DERMATOMYOSITIS
Canine dermatomyositis is a hereditary, idiopathic inflammatory skin and muscle disease well-characterized in Collies and Shetland Sheepdogs.

Etiopathogenesis. Etiopathogenesis of dermatomyositis is still unknown, although an autosomal dominant mode of inheritance with variable expression has been proposed. In Shetland Sheepdogs, inheritance of dermatomyositis has been linked to canine chromosome 35.

An immune-mediated or autoimmune basis is possible but it is unclear if immune reaction is the cause of the disease or is in response to pre-existing muscle or skin damage. Lesions could be induced by drugs, vaccines, infection (especially viral), toxins, internal disease, but causal relationship is unproven. Mechanical trauma and sunlight (UV), and reproductive stress (estrus, parturition, and lactation) may worsen the lesions.

Vascular lesions and local ischemia appear to play a central role in disease process, explaining the distribution of lesions which occur at pressure points and areas of low sustained circulation (ear and tail tips).
**Clinical features.** A familial basis as been reported in collies, Shetland sheepdogs, Beauceron shepherds; however, the disease has been diagnosed in many other breeds including Australian cattle dog, Welsh corgis, and chow chow.

Clinical signs are usually first noticed in dogs less than 6 months, and as early as 7 weeks of age. They wax and wane and vary from minor skin lesions (patchy alopecia, rarely vesicles) to severe ulceration of the skin, with a generalized debilitating myositis affecting the head and distal limbs.

Skin lesions are generally characterized by alopecia, erosions and crusting around the eyes, on the bridge of the nose, pinnae, bony prominences (elbows, hocks, digits), and the tail tip. Vesicles and ulceration may be seen. Some dogs also present with onychodystrophy. Pruritus is normally absent unless complicated by another condition such as pyoderma.

Many dogs show some degree of skeletal muscle involvement that can vary from subtle atrophy of temporal and masseter muscle to generalized muscular atrophy with megaesophagus and lameness.

**Diagnosis.** Young dogs presenting with alopecia and crusting on face, pinnae, and tail tip along with muscle wasting in a predisposed breed (e.g., collie, Shetland sheepdog) is highly suggestive of dermatomyositis.

Initial data base may include skin cytological examination to rule out pyoderma and/or Malassezia overgrowth, skin scrapings to rule out demodicosis, and Wood’s lamp and fungal culture to rule out dermatophytosis. Serum biochemistry profile may show elevated creatine kinase. CBC and urinalysis results are usually unremarkable.

Histopathological evaluation of skin biopsy is characterised by hydropic degeneration in the basal cell layer and cell-poor lymphohistiocytic interface dermatitis. Follicular atrophy may be noted in chronic lesions. Vasculitis is occasionally seen.

**Differential diagnosis.** Differential diagnosis includes demodicosis, dermatophytosis, bacterial folliculitis, Malassezia dermatitis, discoid lupus erythematosus, and vasculitis, and adult-onset generalized ischemic vasculopathy.

**Treatment.** The lesions wax and wane and response to therapy is difficult to evaluate. For acute flares, prednisone 1 mg/kg q24h, weaning down to an alternate-day regimen based on a favorable response is recommended. Prednisone can be use in conjunction with pentoxifylline (25 mg/kg q12h) for severe acute flares. Focal lesions can be treated with topical 0.1% tacrolimus.

Chronic management includes pentoxifylline (25 mg/kg q12h) alone, Vitamin E 200-800 IU/12h, oral cyclosporine, topical tacrolimus, and/or oral tetracycline and niacinamide (250 mg q8h PO for dogs < 10 kg or 500 mg q8h PO for dogs > 10 kg of each drug given for a minimum of 3 months, weaning down based on a favourable response). Flares can be minimized by avoidance of sunlight. Affected dogs should be neutered.

**POST-RABIES VACCINE ALOPECIA**
Localized or generalized ischemic vasculopathy may result from rabies vaccine administration.
Etiopathogenesis. The causal pathomechanism remains unknown, but it is likely that the formation of rabies antigen-antibody complexes that become lodged in vessel walls (a type III hypersensitivity response) is involved. Rabies viral antigen has been demonstrated in vessels and in the epithelium of hair follicles.

Clinical features. Rabies vaccine-associated ischemic dermatopathy is usually seen in adult dogs. It is typically seen in small breeds such as miniature poodle, bichon Frisé, Shih Tzu, Lhasa Apso, Maltese, Yorkshire terrier, and Chihuahua.

The onset of clinical signs is typically 2 to 3 months after vaccine administration, but occasionally takes longer to develop. It usually consists in a focal alopecic lesion at the site of vaccine-administration, but occasionally can be widespread (see adult-onset generalized ischemic dermatopathy). The local form typically occurs over the shoulders, back or the posterolateral thighs and is characterized by a firm circular patch of erythema and alopecia. Old lesions often have a shiny appearance with mild scaling.

Diagnosis. Histopathological changes are characterized by nodular perivascular accumulations of lymphocytes, plasma cells, and histiocytes in the deep dermis and panniculus. Macrophages occasionally contain cytoplasmic basophilic material that is believed to be phagocytized vaccine product. The dermal changes also include moderate to severe follicular atrophy, hyalinization of collagen, mild interface dermatitis and mural folliculitis. These changes are often accompanied by cell-poor vasculitis (often quite subtle) of small blood vessels in the panniculus and the deep dermis.

Treatment. This localized form of ischemic dermatopathy does not necessarily require therapy. If inflammation is prominent, short course of glucocorticoids may be used. Pentoxifylline (25 mg/kg q12h) in combination with vitamin E (200 to 800 IU q12h) also can be used if needed.

ADULT-ONSET GENERALIZED ISCHEMIC DERMATOPATHY (rabies vaccine-induced or idiopathic)

Clinical features. Post-rabies vaccination alopecia associated with concurrent multifocal ischemic dermatopathy can rarely occur. Clinically, it emulates canine dermatomyositis, suggesting that the disease is caused by local ischemia of skin regions that are prone to impaired circulation. The main difference is the older age of onset. A similar clinical manifestation has been reported in Jack Russel terriers with no obvious history of previous vaccination.

In rabies vaccine-induced generalized ischemic dermatopathy, multifocal skin lesions develop within a few months after the appearance of the initial skin lesion at the injection site. However, some dogs never develop a lesion at the injection site. Lesions consist in alopecia, crusting, hyperpigmentation, erosions, and ulcers on the pinnal margins, periocular areas, skin overlying boney prominences, tip of the tail, and paw pads. Lingual erosions and ulcers can also bee seen.

Diagnosis. The dermatohistological changes observed are indistinguishable from those observed in canine dermatomyositis, suggesting a common aetiopathogenesis of immunological damage to the vessels, resulting in ischemic damage to susceptible tissue. In addition, an atrophic, ischemic myopathy paralleling the onset of skin disease can also be seen.
**Treatment.** The generalized form of ischemic dermatopathy requires systemic therapy. As for dermatomyositis, pentoxifylline (25 mg/kg q12h) or prednisone (1 mg/kg q24h) can be used. Dapsone (1 mg/kg q8h) has also been recommended. Some patients may need a more aggressive immunosuppressive therapy with higher doses of prednisone, cyclophosphamide or azathioprine.

**ALOPECIA AREATA (Pelade)**

Alopecia areata is rare in dogs, in contrast to human where it is a relatively common disease.

**Etiopathogenesis.** It is believed that the hair bulb melanocyte is a target cell population. Deposition of both immunoglobulin (particularly IgG) and complement around hair follicles, and the presence of circulating IgM and IgG to hair follicle-specific antigens have been documented. However, the pathogenic potential of anti-hair follicle autoantibodies in canine alopecia areata remains unclear.

**Clinical features.** Age of onset is highly variable, ranging from one to 11 years. Dachshunds appear predisposed. Lesions usually consist of spontaneously arising and well-demarcated alopecic patches developing first on the head (muzzle, chin, forehead, peri-ocular, ears) and occasionally on the legs. Facial lesions usually exhibit a bilateral symmetry. In some cases, alopecia can progress to a more generalized distribution. In multicoloured-coated dogs, alopecia usually occurs first in dark brown or black areas (preferentially targeting of pigmented hair).

Spontaneous and complete hair regrowth occurred in many dogs. Such regrowth is commonly of white hair, a feature also seen in humans and rodents with alopecia areata. In humans, in the more extensive forms of the disease (alopecia totalis and alopecia universalis), spontaneous remission is rare.

**Diagnosis.** Histopathological examination of skin biopsies are required to confirm the diagnosis. The histological hallmark is the so-called “swarm of bees” consisting of a mild to marked mononuclear cell infiltrate, predominantly composed T-lymphocytes, focusing in (bulbitis) and around (peribulbitis) the anagen hair bulb.

**Differential diagnosis.** Differential diagnosis includes demodicosis, dermatophytosis, bacterial folliculitis, isthmic mural lymphocytic folliculitis (pseudopelade), ischemic alopecias such as dermatomyositis and rabies vaccine-associated alopecia, and vitiligo.

**Treatment.** Oral cyclosporine administration appears to be effective in dogs with alopecia areata.

**ISTHMIC LYMPHOCYTIC MURAL FOLLICULITIS (Pseudopelade)**

Isthmic lymphocytic mural folliculitis is a very rare immune-mediated disease in dogs.

**Etiopathogenesis.** Isthmic lymphocytic mural folliculitis in dogs bears some clinicalpathological resemblance to pseudopelade of Brocq in humans which is characterized by a slowly progressive cicatrical alopecia and to the clinical similarity (pseudo) to alopecia areata (pelade) hence the term pseudopelade. However, since “pseudopelade” in humans represents the end stage of several diseases and thus is an ill-defined term, it has been suggested to use the descriptive term of “isthmic lymphocytic mural folliculitis”.


Of note, canine isthmic mural folliculitis can also be observed in conjunction with demodicosis, dermatophytosis and sebaceous adenitis. As for alopecia areata, it is unknown whether the occurrence of autoantibodies to keratinocyte proteins is a primary or a secondary event in the pathogenesis of this disease.

Recently, a non-infectious, mural, mucinotic, isthmus folliculitis alopecia has been reported in Norwegian puffin dogs (lundehunds). It is characterized by a multifocal or serpiginous alopecia, follicular plugging, dry skin, slight scaling and pruritus. A lymphoplasmacytic, mural, isthmus folliculitis/perifolliculitis with follicular and perifollicular mucin was observed on histopathological examination of skin biopsies.

Clinical features. Clinically, the disease is characterized by gradually progressing non-pruritic, non-inflammatory focal or multifocal well-demarcated patches of alopecia. Scales and hyperpigmentation may be present.

Diagnosis. The major histological finding is a mild to marked predominantly lymphocytic infiltration targeting the mid hair follicle (isthmus) sections. A perifollicular mononuclear infiltrate is also present around the isthmus. In late stage lesions severe follicular atrophy and variable atrophy of sebaceous glands, and sparse inflammation is seen. In lundehunds, follicular and perifollicular mucin is also observed.

Differential diagnosis. Differential diagnosis includes demodicosis, dermatophytosis, bacterial folliculitis, alopecia areata, ischemic alopecias such as dermatomyositis and rabies vaccine-associated alopecia.

Treatment. Due to the small number of cases that have been described in the scientific literature, there is no established therapy for isthmic mural folliculitis in dogs. However, cyclosporine was shown to be effective for this disease. In the mucinotic, isthmus folliculitis of the lundehunds, oral prednisolone reduced pruritus but was not effective in resolving clinical lesions. However, all dogs treated with cyclosporine went into remission.

References:


Companion Animals: Dental Instrument Care

The Important “Bits”
Tara Evans

Knowledge and skill of the veterinary professional are instrumental to the successful outcome of any dental procedure. A third and often overlooked contributing factor to a successful procedure is the instrumentation and equipment used. Not only is the appropriate selection and correct use of dental instrumentation and equipment important, the care and maintenance they receive greatly impacts their effectiveness. Dental handpieces (both high & low speed) and dental machines that are properly cared for will prevent unnecessary, premature equipment failure and potential patient contamination. While dental equipment maintenance can be intimidating to those unfamiliar with it, it is in fact quite simple. This lecture will address all aspects of handpiece and dental machine maintenance, including turbine care and replacement, compressor care, filter replacement, cleaning of dental lines, etc.

Keep Your Motor Running
Tara Evans

Although the statistics presented by various authors vary somewhat, all agree that by age two, most dogs and cats are affected by some form of periodontal disease. As client awareness of dental disease has increased, so has the demand for professional dental care. As a result, dental extractions may be the most common surgical procedure performed in veterinary medicine today, making high speed dental drill engines one of the most regularly used tools in the veterinary profession. Despite this, high speed handpieces remain one of the least understood tools in the veterinarians’ arsenal. High speed handpieces are like cars. Any car in good working order will get you from point A to point B, but there is a great deal of variation between cars in terms of efficiency, features, benefits, etc. This lecture will address differences, features, benefits and advantages of various types of high speed handpieces, as well as where each may be best employed. Understanding these differences will enable the user to select the handpiece best suited to his or her needs and budget. The most commonly used friction grip burs will also be discussed, including recommended use and which handpiece(s) they are most appropriate for use with. Utilizing the right bur in the right high speed handpiece can make your engine rev that much more efficiently!

Dental Instrument Forensics 101
Tara Evans

Dental instruments and equipment need to be utilized correctly and cared for properly in order to maximize their effectiveness and longevity. While this sentiment is generally shared by veterinary dental authors and lecturers, many veterinary professionals are uncertain as to what constitutes correct use and proper maintenance, or when it’s time to retire an instrument. Often the result is
unnecessary damage or wear to their instrumentation, and/or utilizing instrumentation long past its useful life expectancy, to the detriment of the user and the patient. To enable attendees to better troubleshoot, understand and avoid problems that may arise with their own instrumentation, during this lecture, we will look at a variety of dental instrumentation that has been damaged, or is in otherwise substandard condition, and will discuss the reasons for their condition. Instrumentation we will cover includes luxators and elevators, root tip picks, periosteal elevators, scalers and curettes, turbines and high-speed handpieces, dental burs, ultrasonic scaler tips, etc. We will identify common causes of damage and premature end of usefulness, whether it be the result of improper use, a care and maintenance issue, or another cause. The typical lifespan of common instrumentation will also be addressed. Pictures and video will be used throughout this presentation to emphasize proper use and care of various instrumentation to help attendees prevent or mitigate issues in future.
When faced with a patient with a systemic disease...do not forget to look at the eyes! Ocular lesions could be the first clinical sign of a systemic illness. Ocular examination findings can either help clarify a diagnosis and or influence prognosis. This is especially true with infectious and metabolic diseases, as well as neoplasia. Systemic diseases can also negatively influence the eyes and result in ocular diseases that require therapy. In this session, we will explore the ocular lesions associated with systemic diseases. Diagnosis and therapy of these associated ocular diseases will be emphasized during this session.

The main message of this lecture is the following: if you diagnose any of the systemic diseases listed below, have a look at the eyes! Keep in mind that ocular lesions may the first symptom of a systemic disease!

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<th>Etiology</th>
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<th>Ocular tests</th>
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<tr>
<td>Corneal ulcers (SCCEDS)</td>
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<td>Cushing’s disease</td>
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<td>Corneal stromal hemorrhages</td>
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<td>Corneal stromal hemorrhages, Punctate retinal hemorrhages</td>
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<td>Uveodermatological syndrome</td>
<td>Uveitis, Depigmentation of iris, Depigmentation of choroid</td>
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<td>Lymphoma, Third eyelid thickening, Conjunctival thickening, Uveitis</td>
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<td></td>
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<td>STT, Fluorescein stain, Tonometry, Conj. biopsy</td>
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Many of the ocular lesions associated with these aforementioned systemic diseases repeat themselves, such as KCS, corneal ulcers, uveitis and secondary glaucoma. With increasing severity of disease, additional clinical signs appear or existing clinical signs intensify in presentation. With regard to therapy of these ocular lesions, severity of the clinical signs will help determine the appropriate therapy.

Table 2: Description of ocular lesions associated with severity of ocular disease

<table>
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<th>Ocular lesions</th>
<th>Mild presentation</th>
<th>Moderate presentation</th>
<th>Severe presentation</th>
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<td>KCS</td>
<td>Mucoid discharge</td>
<td>Blepharospasm</td>
<td>Blepharospasm</td>
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<td></td>
<td>Mild conjunctivitis</td>
<td>Blepharospasm</td>
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<td></td>
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<td>Mucopurulent discharge</td>
<td>Mucopurulent</td>
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<td></td>
<td></td>
<td>Moderate conjunctivitis</td>
<td>discharge</td>
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<td></td>
<td>Dull cornea</td>
<td>Moderate conjunctivitis</td>
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<td></td>
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<td>Corneal blood vessels</td>
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<tr>
<td>Corneal ulcers</td>
<td>Superficial (simple)</td>
<td>Superficial (SCCEDS)1</td>
<td>Deep (complex)</td>
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<td></td>
<td>Serous discharge</td>
<td>Serous discharge</td>
<td>Mucopurulent</td>
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<tr>
<td></td>
<td>Mild conjunctivitis</td>
<td>Moderate conjunctivitis</td>
<td>discharge</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Corneal blood vessels</td>
<td>Severe conjunctivitis</td>
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<td>Uveitis</td>
<td>Mild conjunctivitis</td>
<td>Moderate episcleritis</td>
<td>Severe episcleritis</td>
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<td></td>
<td>Mild episcleritis</td>
<td>Limbal corneal edema</td>
<td>Diffuse corneal</td>
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<td></td>
<td>Low IOP2</td>
<td>Mild aqueous flare</td>
<td>edema</td>
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<td></td>
<td></td>
<td>Miosis</td>
<td>Aqueous flare</td>
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<td></td>
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<td>Low IOP2</td>
<td>Fibrin in anterior</td>
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<td></td>
<td></td>
<td></td>
<td>chamber</td>
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<tr>
<td>Secondary</td>
<td>Mild episcleritis</td>
<td>Moderate episcleritis</td>
<td>Diffuse corneal</td>
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<tr>
<td>glaucoma</td>
<td>Mydriasis</td>
<td>Limbal corneal edema</td>
<td>edema</td>
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<td></td>
<td>? PLR4 ?</td>
<td>Mild diffuse corneal</td>
<td>Mydriasis</td>
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<tr>
<td></td>
<td>IOP 30-35 mmHg</td>
<td>edema</td>
<td>Fixed pupil</td>
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<tr>
<td></td>
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<td>Miosis</td>
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</table>

1KCS: keratoconjunctivitis sicca; 2SCCEDS: spontaneous chronic corneal epithelial defect syndrome; 3IMHA: immune-mediated hemolytic anemia; 4ITP: immune-mediated thrombocytopenia
Fixed pupil
IOP: 35-45 mmHg

1SCCEDS: spontaneous chronic corneal epithelial defect syndrome; 2Low IOP: intraocular pressure is lower in affected eye; 3High IOP: intraocular pressure is higher in affected eye; 4?PLR?: pupillary light reflex may be weakly present

As mentioned, therapy will be impacted by severity of disease exhibited. As the severity intensifies, the number of medications needed to address the disease at hand also increases. Owner compliance is a must for successful outcomes; however, with severe disease, appropriate therapy and good owner compliance may not always result in resolution of clinical signs and may require removal of the ocular globe for pain management.

Table 3: Therapeutic considerations based on severity of clinical ocular disease

<table>
<thead>
<tr>
<th>Ocular lesions</th>
<th>Mild disease</th>
<th>Moderate disease</th>
<th>Severe disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>KCS</td>
<td>Artificial tears TID</td>
<td>Artificial tears QID</td>
<td>Artificial tears QID</td>
</tr>
<tr>
<td></td>
<td>Optimimmune BID</td>
<td>Bacteriostatic antibiotic1 Optimimmune BID</td>
<td>Bactericidal antibiotic2 QID</td>
</tr>
<tr>
<td>Corneal ulcers</td>
<td>Bactericidal antibiotic2 QID</td>
<td>Bactericidal antibiotic2 QID Topical Atropine BID 2 days</td>
<td>Bactericidal antibiotic3 q 4 hrs</td>
</tr>
<tr>
<td></td>
<td>Artificial tears QID</td>
<td>Artificial tears QID</td>
<td>Topical atropine BID 2 days Artifical tears QID</td>
</tr>
<tr>
<td>Uveitis: Diabetic</td>
<td>Topical NSAID4 BID</td>
<td>Topical NSAID4 TID</td>
<td>Topical NSAID4 QID</td>
</tr>
<tr>
<td></td>
<td>Artificial tears BID</td>
<td>Artificial tears TID</td>
<td>Artificial tears QID</td>
</tr>
<tr>
<td></td>
<td>Optimimmune BID</td>
<td>Optimimmune BID</td>
<td>Optimimmune BID</td>
</tr>
<tr>
<td>Uveitis: Non-diabetic</td>
<td>Topical NSAID4 BID or Topical steroid5 SID</td>
<td>Topical steroid5 TID</td>
<td>Topical steroids5 q 4-6 hours Oral prednisone 1mg/kg/day</td>
</tr>
<tr>
<td>Secondary glaucoma</td>
<td>Topical CAI6 TID</td>
<td>Topical CAI6 TID</td>
<td>Topical CAI6 QID</td>
</tr>
<tr>
<td></td>
<td>Topical steroid5 TID</td>
<td>Topical beta blocker7 BID</td>
<td>Topical beta blocker7 BID</td>
</tr>
<tr>
<td></td>
<td>Gabapentin 10mg/kg TID</td>
<td>Topical steroid5 QID</td>
<td>Topical steroid5 q 4-6 hours Oral prednisone 0.5mg/kg/day</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Gabapentin 10mg/kg TID</td>
<td>Gabapentin 10 mg/kg TID</td>
</tr>
</tbody>
</table>

1Bacteriostatic antibiotic: Isathal eye gel; 2Bactericidal antibiotic: tobramycin (dog and cat), BNP (dog); 3Bactericidal antibiotic: fluoroquinolone (ofloxacin, moxifloxacin); 4Topical NSAID: Voltaren, Nevanac. Ketorolac; 5Topical steroid: Maxidex, Prednisolone acetate 1%; 6Topical Carbonic Anhydrase Inhibitor: Azopt (dog), Trusopt (cat); 7Topical beta blocker: Timolol 0.5%

References:
Making Sure Not to Miss the Next Glaucoma Patient  
Chaorte L. Pinard DVM, MSc, DACVO

Introduction
Glaucoma is the true ocular emergency as it can cause permanent vision loss. This potentially blinding disease can have many presentations because there can be several underlying causes that trigger its occurrence. Signalment, ocular examination findings, as well as diagnostic tests can give the practitioner an ideal of therapy and prognosis. The choice of anti-glaucoma medications can be daunting but individualized patient therapy will be the best arsenal against this vision threatening disease. It is hoped that by the end of this session, the practitioner will be on the look-out for this devastating disease and have the arsenal of therapies ready to give their patients’ eyes a fighting chance for retention of vision!

Pathophysiology
The key to the diagnosis and treatment of glaucoma is the understanding the underlying mechanisms or causes of this disease. The main pathological event of glaucoma is the lack of outflow of aqueous humour leading to an increased amount within the eye and compression of ocular tissues. This compression will lead to ischemia and hypoxia of the retina as well as physical compression of the optic nerve head.

Table 1: Causes of glaucoma

<table>
<thead>
<tr>
<th>Type:</th>
<th>Primary</th>
<th>Secondary</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mechanism</td>
<td>Malformed iridocorneal angle</td>
<td>Malformed iridocorneal angle</td>
</tr>
<tr>
<td></td>
<td>Inflammatory debris/cells</td>
<td>Lens and vitreous blocking</td>
</tr>
<tr>
<td></td>
<td>angle</td>
<td>aqueous flow</td>
</tr>
<tr>
<td>Underlying cause</td>
<td>Embryological mistake with</td>
<td>Genetic Trauma Chronic</td>
</tr>
<tr>
<td></td>
<td>heritability</td>
<td>uveitis</td>
</tr>
<tr>
<td></td>
<td>Trauma Systemic disease</td>
<td>Genetic? Chronic uveitis?</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Retinal detachment</td>
</tr>
<tr>
<td></td>
<td>Ciliary body tumour</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Keratitis</td>
<td></td>
</tr>
<tr>
<td>Signalment</td>
<td>Any breed</td>
<td>Any breed</td>
</tr>
<tr>
<td></td>
<td>Any sex &lt; 6mos of age</td>
<td>Any breed</td>
</tr>
<tr>
<td></td>
<td>Adult to geriatric</td>
<td>Adult to geriatric</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Adult to geriatric</td>
</tr>
<tr>
<td>Ocular findings</td>
<td>Buphthalmos</td>
<td>Episcleral congestion</td>
</tr>
<tr>
<td></td>
<td>Episcleral congestion</td>
<td>Conjunctivitis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Episcleral congestion</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Episcleral congestion</td>
</tr>
<tr>
<td>Episcleral congestion</td>
<td>Corneal edema</td>
<td>Episcleritis Limbal corneal edema</td>
</tr>
<tr>
<td>-----------------------</td>
<td>--------------</td>
<td>----------------------------------</td>
</tr>
<tr>
<td>Corneal edema</td>
<td>Fixed dilated pupil</td>
<td>Mid-size pupil</td>
</tr>
<tr>
<td>Fixed dilated pupil</td>
<td>Lens in place</td>
<td>Aqueous flare</td>
</tr>
<tr>
<td>Lens in place</td>
<td>Optic nerve cupping</td>
<td>Fibrin clot</td>
</tr>
<tr>
<td>Optic nerve head</td>
<td>cupping</td>
<td>Hypopion or hyphema</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Diagnostic tests</th>
<th>Tonometry</th>
<th>Tonometry Ocular ultrasound</th>
<th>Tonometry Ocular ultrasound</th>
<th>Tonometry Ocular ultrasound</th>
<th>Tonometry Ocular ultrasound</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Tonometry</td>
<td>Ocular ultrasound</td>
<td>Tonometry Ocular ultrasound</td>
<td>Tonometry Ocular ultrasound</td>
<td>Tonometry Ocular ultrasound</td>
</tr>
<tr>
<td></td>
<td>Tonometry</td>
<td>Fluorescein</td>
<td>Tonometry Ocular ultrasound</td>
<td>Tonometry Ocular ultrasound</td>
<td>Tonometry Ocular ultrasound</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Medical Therapy</th>
<th>Triple cocktail 2</th>
<th>Triple cocktail 2</th>
<th>Prednisolone acetate 1% CAI3</th>
<th>Mannitol IV CAI3 Couching 4</th>
<th>Enucleation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Triple cocktail 2</td>
<td>Triple cocktail 2</td>
<td>Prednisolone acetate 1% CAI3</td>
<td>Mannitol IV CAI3 Couching 4</td>
<td>Enucleation</td>
</tr>
<tr>
<td></td>
<td>Prednisolone acetate 1%</td>
<td>CAI3</td>
<td>Beta blocker</td>
<td>Mannitol IV CAI3 Couching 4</td>
<td>Enucleation</td>
</tr>
<tr>
<td></td>
<td>CAI3</td>
<td>Beta blocker</td>
<td>Mannitol IV CAI3 Couching 4</td>
<td>Enucleation</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Prognosis</th>
<th>Grave</th>
<th>With time: grave</th>
<th>Guarded to grave</th>
<th>Guarded to grave</th>
<th>Grave</th>
<th>Grave</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Grave</td>
<td>With time: grave</td>
<td>Guarded to grave</td>
<td>Guarded to grave</td>
<td>Grave</td>
<td>Grave</td>
</tr>
</tbody>
</table>

1PIFM: pre-iridofibrovascular membrane; 2Triple cocktail: topical prostaglandin analogue, carbonic anhydrase inhibitor, and beta blocker; 3CAI: carbonic anhydrase inhibitor; 4Couching: pushing lens into posterior chamber

**Principles of Therapy**

Topical and systemic medications are used to treat glaucoma. Depending on the severity of the disease, one or more medications can be prescribed to lower the intraocular pressure (IOP) within normal range. The lowering of IOP is mainly done by manipulating the outflow or production of aqueous humour, or by reducing the intraocular fluid volume with osmotic diuretics. Topical medications carry fewer side effects than systemic drugs and therefore are preferred for most cases. Medical therapy can delay the onset of blindness significantly for patients; however, as glaucoma progresses, the beneficial effects of medication become less apparent and therapeutic failure is often inevitable.

**Prostaglandin Analogues**

Prostaglandin analogues are effective topical anti-glaucoma drugs due to their ability to decrease IOP by increasing the uveal-scleral outflow in dogs; this class of anti-glaucoma medications is not
effective in cats. Drugs in this class are best reserved for canine primary glaucoma. The effect of this medication can be seen within 30-60 minutes.

Conjunctival hyperemia is usually apparent following administration. An important side effect of this class of medications is a severe miosis lasting 12-18 hours; patients at risk for lens luxation should not be prescribed this medication. Another side effect to consider prior to prescription is that they cause a breakdown of the blood-aqueous barrier; consequently, canine patients with glaucoma secondary to severe uveitis should not be prescribed these medications. Several derivatives are available in human pharmacies.

Table 2: Prostaglandin analogues commercially available

<table>
<thead>
<tr>
<th>Drug</th>
<th>Frequency of Administration</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bimatoprost 0.003%</td>
<td>1 drop q 12-24 hours</td>
<td></td>
</tr>
<tr>
<td>*Latanoprost 0.005%</td>
<td>1 drop q12-24 hours</td>
<td>Requires refrigeration</td>
</tr>
<tr>
<td><em>Travaprost 0.004%</em></td>
<td>1 drop q 12-24 hours</td>
<td></td>
</tr>
<tr>
<td>Unoprostone 0.15%</td>
<td>1 drop q 12-24 hours</td>
<td></td>
</tr>
</tbody>
</table>

* Latanoprost and Travaprost are commonly prescribed for our canine patients and they are typically at a frequency of every 24 hours

**Carbonic Anhydrase Inhibitors**

Carbonic anhydrase inhibitors decrease IOP by lowering the production of aqueous humour within the eye. Time to effect is ~1-2 hours and this class of drugs remains an important adjunct therapy for primary and secondary glaucoma in both dogs and cats. Topical formulations moderately decrease IOP. No effects on pupil size or blood-aqueous barrier are seen with this class of medication. The main advantage of topical formulations is the far fewer side effects compared to systemic counterparts.

Systemic carbonic anhydrase inhibitors are thought by some to be more effective than topical drops; however, topical administration has been shown to be as effective as systemic formulations and no additive effects were seen when both formulations were given to glaucomatous dogs. The main disadvantages of systemic formulations are as follows: systemic acidosis, vomiting, diarrhea, general malaise, anorexia, panting, lameness due to paresthesia and ataxia (cats especially). Long-term supplementation with potassium is also needed due to the diuresis that induces hypokalemia.

Table 3: Carbonic anhydrase inhibitors commercially available

<table>
<thead>
<tr>
<th>Topical drug</th>
<th>Frequency of Administration</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Brinzolamide 1%</td>
<td>1 drop q 6-8 hours</td>
<td>Not useful in cats</td>
</tr>
<tr>
<td>Dorzolamide 2%</td>
<td>1 drop q 6-8 hours</td>
<td>Can be irritating</td>
</tr>
</tbody>
</table>

**Systemic drug**

<table>
<thead>
<tr>
<th>Drug</th>
<th>Frequency of Administration</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetazolamide</td>
<td>10-20 mg/kg q 8-12 hours</td>
<td>Many side effects</td>
</tr>
<tr>
<td>Methazolamide</td>
<td>2-5 mg/kg q 8-12 hours</td>
<td>Many side effects</td>
</tr>
</tbody>
</table>

**Beta Adrenergic Drugs**

Beta adrenergic blockers, or beta blockers, decrease IOP ~1-2 hours by lowering the production of aqueous humour in. The mode of action is different from carbonic anhydrase inhibitors and is
thought to include inhibition of cyclic AMP in the ciliary body. A very modest decrease in IOP has been reported in both dogs and cats with topical beta blockers. Positive additive effect is seen with combinations of beta blockers and carbonic anhydrase inhibitors.

Mild miosis can occur with these medications in both species. An important side effect with these medications is bradycardia; this author has had cardiac patients on topical beta blockers experience worsening of their cardiac disease. This class of medication should not be used in cats with asthma.

<table>
<thead>
<tr>
<th>Table 4: Beta blockers commercially available</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug</td>
</tr>
<tr>
<td>--------------------------------</td>
</tr>
<tr>
<td>Betaxolol 0.5%</td>
</tr>
<tr>
<td>Carteolol 2%</td>
</tr>
<tr>
<td>Levobetaxolol 0.5%</td>
</tr>
<tr>
<td>Levobunolol 0.5%</td>
</tr>
<tr>
<td>Metipranolol 0.3%</td>
</tr>
<tr>
<td>*Timolol 0.5%</td>
</tr>
<tr>
<td>Combination products</td>
</tr>
<tr>
<td>Cosopt</td>
</tr>
<tr>
<td>Duotrav</td>
</tr>
</tbody>
</table>

*Timolol 0.5% is commonly prescribed for our patients

**Parasympathomimetics**
Parasympathomimetics decrease IOP by opening the aqueous outflow channels due to contraction of the ciliary body. They also decrease IOP by causing a break in the blood aqueous barrier and therefore increasing vascular permeability. This class of medications has been effectively replaced in practice by prostaglandin analogues as the latter do not require frequent administrations and have better effects in patients with very high IOP (>40 mmHg). The only commercially available topical formulation of a parasympathomimetic is 1, 2 or 4% pilocarpine.

Conjunctival inflammation and miosis are side effect of these medications. These medications are contraindicated in patients with anterior lens luxation, subluxation, and uveitis.

**Diuretics**
Osmotic diuretics can be effective at lowering IOP by reducing the aqueous and vitreous volumes within the eye. The effect of diuretics can be seen as early as 10 minutes but is usually detectable within 20-30 minutes and can last 5-12 hours.

These medications are contraindicated in patients with congestive heart failure, systemic hypertension, renal insufficiency, and diabetes mellitus.

<table>
<thead>
<tr>
<th>Table 5: Osmotic diuretics commonly used in veterinary ophthalmology</th>
</tr>
</thead>
<tbody>
<tr>
<td>Drug</td>
</tr>
<tr>
<td>--------------------------------</td>
</tr>
</tbody>
</table>

Mannitol 1-2 g/kg IV over 20-30 minutes  Restrict water consumption for 2-3 hours; keep in fluid warmer to prevent crystallization of the solution
Glycerin 1-2 g/kg per os  Vomiting occurs if solution is not diluted

By knowing the type of glaucoma and the general status of your patient, some medications are indicated and some are contraindicated.

Table 6: Contraindicated anti-glaucoma medications

<table>
<thead>
<tr>
<th>Drug</th>
<th>Contraindication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mannitol i.v.</td>
<td>Diabetes, renal failure, cardiomyopathy, dehydration, uveitis</td>
</tr>
<tr>
<td>Topical Prostaglandins</td>
<td>Uveitis, lens luxation</td>
</tr>
<tr>
<td>Topical Beta blockers</td>
<td>Cardiac disease, feline asthma</td>
</tr>
<tr>
<td>Oral carbonic anhydrase inhibitors</td>
<td>Renal failure, cardiomyopathy, dehydration</td>
</tr>
</tbody>
</table>

Algorithm for primary glaucoma therapy:

What To Do About Red Eye…
Chantale Pinard, DVM, MSc, Dip. ACVO

This session will aim to answer the following question: What to do with a “red” eye?

Unfortunately, many eye diseases present with a “red” eye and they all have their specific diagnostic tests and therapies. The location of the redness is the first step towards an accurate diagnosis. In order to best achieve this goal, ocular tests help to narrow down the list of differentials. Once the diagnosis has been determined an appropriate treatment plan can be executed.

By identifying the type and location of the redness, differential diagnoses emerge.

**Conjunctival hyperemia**
- Superficial branching blood vessels
  - Conjunctivitis
  - KCS
  - Corneal ulcer
  - Blepharitis
  - Retrobulbar abscess cellulitis

**Subconjunctival hemorrhage**
- Trauma
- Proptosis
- Coagulopathy

**Episcleral hyperemia**
- Straight thick blood vessels
  - Uveitis
  - Glaucoma
  - Episcleritis KCS
  - Scleritis (rare)
  - Panophthalmitis (rare)

**Corneal vascularisation**
- Superficial vessels are branching
- Deep vessels are straight
  - Chronic corneal ulcer
  - Pannus
  - Eosinophilic keratitis
  - Uveitis (360 perilimbal)

**Intraocular hemorrhage**
- Hyphema or blood clot
  - Trauma
  - Uveitis
  - Coagulopathy
  - Systemic hypertension
  - Intraocular neoplasia

**Red raised nodule**
- Hemangioma
- Hemangiosarcoma

To help narrow down the list of differentials, a comprehensive ocular exam, including a neuro-ophthalmic exam, Schirmer tear test (STT), fluorescein stain uptake, and tonometry, is warranted for each patient with a red eye. Each ocular test can help to rule in or out a particular ocular disease. Bundling the STT, fluorescein stain and tonometry costs into one charge can help owners give consent to a comprehensive ocular exam. Marking down the values obtained onto an ocular exam sheet as well as drawing the ocular lesions seen can be instrumental in communicating with fellow associates and ensure accurate ongoing care.
Listed below are common ocular diseases that cause a “red” eye. Some of these diseases are primary to the eye while others are part of a systemic disease and may warrant additional diagnostic tests.

<table>
<thead>
<tr>
<th>Diseases</th>
<th>Common Causes</th>
<th>Associated common clinical signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blepharitis</td>
<td>Infectious</td>
<td>Mucopurulent discharg, blepharospasm</td>
</tr>
<tr>
<td></td>
<td>secondary to orbital disease</td>
<td></td>
</tr>
<tr>
<td>Coagulopathy</td>
<td>Rodenticide toxicity</td>
<td>Petechiation on other mucous membranes</td>
</tr>
<tr>
<td></td>
<td>immune-mediated</td>
<td></td>
</tr>
</tbody>
</table>
### Therapy of common ocular diseases that cause a “red eye”:

#### Blepharitis

**Canine:**

**Oral:** cephalaxin 22-30 mg/kg BID 3 weeks with prednisone 0.5-1mg/kg/day for 3 weeks; consider consultation with dermatologist
**Conjunctivitis**
Feline:
Topical: erythromycin TID, teargel TID; oral: lysine 500mg BID, Onsior 3-6 days
Canine:
Topical: fusidic acid BID if mucopurulent discharge, maxitrol TID if follicular hyperplasia, teargel TID; oral NSAID 5-7 days

**Corneal ulceration**
Simple:
Topical: tobramycin or BNP (dogs only) QID, teargel QID; oral NSAID 5 days
Indolent:
Topical alcaine, corneal debridement, grid keratotomy, corneal contact lens, e. collar;
Topical: tobramycin QID, atropine BID 2 days, teargel QID; oral NSAID
Complex:
Topical: bactericidal antibiotic q 2-4 hours, if melting serum q 2-4 hours, atropine BID 2 days; oral: NSAID; oral cephalosporin if risk of perforation; oral doxycycline if melting

**Eosinophilic Keratitis**
Feline:
Topical: tobramycin QID if corneal ulcer, cidovir 0.5% BID or ganciclovir QID 2 weeks; maxidex SID-BID following antiviral therapy, teargel TID; oral lysine 500mg BID

**Episcleritis**
Canine:
Topical prednisolone acetate 1% or maxidex TID-QID if diffuse; if nodular oral tetracycline 500mg and niacinamide 500mg each TID if >10kg

**Glaucoma**
Primary canine:
Topical: travatan SID-BID, azopt TID-QID and timolol BID; topical maxidex SID; oral pred 0.5-1mg/kg/day 5 days; oral amlodipine 5-7 days
Secondary canine:
Lens luxation – surgical extraction;
Uveitis- topical prednisolone acetate q 1-6 hours and oral prednisone 1mg/kg/day;
Neoplasia - enucleation
Feline:
Topical trusopt TID and timolol 0.5% BID (if not asthmatic); topical prednisolone acetate 1% q 4-8 hours

**Keratoconjunctivitis sicca**
Canine:
Topical: Optimmune BID, teargel QID, fusidic acid BID if no corneal ulceration or topical maxitrol BID; oral NSAID
Feline:
Topical: erythromycin TID, teargel QID; oral Onsior 3 days
Pannus (chronic superficial keratitis)
Canine:
Topical: prednisolone acetate 1% or maxidex q 4-12 hours, Optimmune BID, teargel QID if low STT

Uveitis
Trauma:
Topical NSAID TID if mild or diabetic; topical prednisolone acetate 1% or maxidex BID-QID if moderate and q 2-4 hours if severe uveitis
Infectious:
Treat underlying infection; topical NSAID TID if mild; prednisolone acetate 1% or maxidex BID-QID if moderate; oral prednisone 0.5mg/kg/day if severe
Immune mediated:
Oral prednisone 0.5-1.5 mg/kg/day; topical prednisolone acetate 1% q 2-12 hours; re-evaluate every 1-2 weeks
Lens induced:
Topical NSAID BID if diabetic; topical prednisolone acetate 1% or maxidex q 4-12 hours if non-diabetic or concerned about IOP; oral NSAID

Golden retriever:
Topical: NSAID BID if low IOP and presence of pigment on anterior lens capsule; topical prednisolone acetate 1% SID-QID if fibrin present

References:
Introduction

Parenteral antibiotics administered to treat infections diffuse into the intestine and influence the population and antimicrobial susceptibility of the intestinal bacteria. However, there is little is known about the concentration or persistence of these antimicrobials. Earlier studies have not measured drug concentrations in the gastrointestinal tract (GIT), and rarely assess microbial changes over time. Measurement of active, unbound drug concentrations at the site of action (GIT lumen) is critical to correlate pharmacokinetic-pharmacodynamic (PK-PD) indices with microbial changes.

Use of therapeutic antimicrobials is critical to cattle health, and many antimicrobials used in cattle are prescription drugs deemed “critically important” for human use. Epidemiological associations between systemic antimicrobial administration in cattle and resistance in enteric organisms, and studies reporting the association between antimicrobial use and subsequent presence of antimicrobial resistant bacteria within the feces are of limited value as they usually evaluate only a single bacterial species or isolate. Changes in the microbiome have been observed, but only at a single time point after treatment. To our knowledge, no study has correlated the intestinal pharmacokinetics (PK) of the antimicrobials, shedding of foodborne pathogens, and changes in the microbiome over time after treatment. This is primarily due to an inability to measure active drug concentrations in the GIT. The objective of these studies was to develop a method to measure these drug concentrations and correlate these findings with changes in fecal microbiota.

Sampling of the Gastrointestinal Tract: Fluid from the lumen of the gastrointestinal tract (GIT) is commonly collected in research projects ranging from nutrition to pharmacology in a wide variety of laboratory animals from rodents to cattle. A variety of approaches to collection of luminal GIT fluid have been used. These include euthanasia of multiple animals in order to collect GIT contents at multiple time points, requiring a significant number of experimental animals and yielding a relatively low number of animals per time point. Currently, this approach has only been used in pigs and has not been reported in cattle. Several cannulas and tubes have been used for similar purposes including rumen and duodenal cannulas, and gastrotomy tubes. Implanting these devices requires significant surgical skill and carries inherent risks of leakage, dislodgement, and peritonitis. Additionally, we are somewhat limited in the sections of the GIT into which these devices can be placed. They can also disrupt normal intestinal motility and flow of ingesta as they must be anchored to the abdominal wall.

Measurement of drug concentrations in the feces of cattle has been reported in environmental studies, but the use of these concentrations in PK-PD studies is problematic. These measurements include both protein-bound and unbound drug, and may not reflect the actual concentration of drug higher in the GIT. Drugs may be concentrated by extraction of water in the colon or degraded or transformed to inactive forms by intestinal bacteria.

Ultrafiltration probes have been used to collect interstitial fluid (ISF) in a number of species to measure many of these same substances. Typically, the probes are placed subcutaneously, though they can be placed in the muscle, bone, or pleural space. The ultrafiltration probe typically contains...
3 semi-permeable loops connected to a non-permeable tube extending to the exterior of the animal and attached to a Vacutainer® tube. The membrane in the loop consists of pores allowing water, electrolytes, and low molecular weight molecules (less than 30,000 Daltons) to pass while excluding the passage of protein, protein-bound drugs, and other large molecular weight compounds. The Vacutainer® provides the negative pressure for fluid collection through the small pores in the loop membrane.

**Surgical Method**

The ultrafiltration probes can be placed in the GIT using either general anesthesia or in a standing animal with regional anesthesia of the flank.9 The right paralumbar fossa is clipped and scrubbed for sterile surgery. A vertical 20 cm skin incision is made 6 cm below the transverse processes of the lumbar vertebrae and approximately equidistant from the last rib to the tuber coxae in the paralumbar fossa. Sharp dissection continues through the external and internal abdominal oblique muscles, transverse abdominal muscle, and peritoneum to enter the peritoneal cavity. The cecum is identified and exteriorized caudally to expose the ileocecal fold. The ileocecal fold is used to identify the ileum, the location of the first probe placement.

The site for probe insertion is on the antimesenteric border of the ileum, approximately 20cm orad to the ileocecal orifice. Intestinal contents are gently milked orad and aborad from the chosen site. A pursestring is pre-placed in the ileal wall using 3-0 polydioxanone (PDS II; Ethicon). A stab incision is made with a number 11 scalpel blade in the center of the pursestring. An introducer needle is placed through the stab incision into the lumen of the ileum. The collecting end of the ultrafiltration probe is inserted through the introducer needle aborally towards the cecum so the entire collecting apparatus is within the lumen. The introducer needle is fed off the probe, and the pursestring is tightened and tied to secure the probe in place and produce a leak-free seal. The remaining 3-0 PDS suture is used to fingertrap the free end of the probe for added security.

The cecum is then reflected cranially to locate and exteriorize a portion of the spiral colon. A probe is introduced into the lumen of the spiral colon in the same manner as the ileum in an aborad direction. The free ends of the ileal probe and spiral colon probe are identified and kept separate. The abdominal contents are replaced in the body cavity, while allowing the free ends of the probes to lightly settle in the abdominal cavity between the omental sling and abdominal wall. The free ends of the probes are exteriorized cranial to the dorsal third of the skin incision by using the introducer needle to create a tunnel from the abdominal cavity through the muscle layers. Mild slack is left in the tubing inside the abdominal cavity to allow normal intestinal motility, and avoiding intestinal entrapment. Fingertrap sutures using 2-0 nylon (Ethilon; Ethicon) are placed to secure the probe tubing to the skin.

**Surgical Outcomes**

Surgical implantation of ultrafiltration probes is easily accomplished from a right flank celiotomy using the cecum as the landmark for identification of the ileum and spiral colon. There were no complications postoperatively, and the steers tolerated the probes well. At 3-4 days post-surgery, there were no clinically significant findings at necropsy in any of the 12 steers. The suppurative inflammation found in the peritoneal fluid was consistent with that found after routine abdominal surgery in cattle. Using the probes, we were able to obtain an adequate number of samples for pharmacokinetic study.
We believe there are several key benefits to use of ultrafiltration probes to collect GIT fluid. The first is the ability to continuously sample the same animals over time without euthanasia, allowing for significant reduction in the number of experimental animals. Additionally, continuous sampling will reduce inter-animal variation in the data. As implantation is relatively quick with a short postoperative period, several animals can be instrumented immediately prior to a study; whereas animals with rumen and duodenal cannulas are typically surgically prepared well in advance and must be maintained between studies. For PK studies, the collection of a protein-free ultrafiltrate is ideal. This ensures that drug measurements only include the active, non-protein bound drug. Other fluid collection techniques will necessarily include both free and protein-bound drug and, therefore, will overestimate the amount of active drug present in the fluid. Because of the small size of the ultrafiltration probes, they could potentially be implanted into many different laboratory animals. The locations are not limited to those used in this study, as implantation would be feasible in any surgically accessible portion of the GIT. As there is no need to anchor the loop of bowel being sampled, multiple probes can be inserted in one animal with no impact on intestinal motility. The surgery is relatively atraumatic with rare complications, and we feel this may be a more humane alternative to other approaches of collecting intestinal fluid.

Potential drawbacks to this technique include the longevity of the probes in the GIT as most will cease collecting between two to three weeks after implantation. Further, this may vary from one location to another. In this study, there was no observed decrease in volume of fluid collected over 5 days, in spite of significant accumulation of ingesta on the membrane at necropsy. Further, the fragile membrane can easily be damaged by drier, firmer ingesta. When the probes were implanted in a canine colon, the membranes were rapidly damaged and the tubing clogged. The size selectivity of the ultrafiltration probes is a distinct advantage for PK studies, but this will hinder the use of these probes in other studies. Nothing larger than 30,000 Da can be collected which will exclude many proteins and all fiber and bacteria. Therefore, clear knowledge of the protein of interest is critical before using these probes for that particular purpose. Finally, it would be ideal to collect bacteria from the GIT over time to measure changes to the flora in response to drug administration or diet changes, but these organisms would be excluded from the fluid collected by this technique.

**Antibiotic Concentrations in GI Tract**

To compare two FDA-approved dosing regimens of enrofloxacin (Baytril 100, Bayer Animal Health), 12 steers were administered either 5 mg/kg once a day for 3 days or 12.5 mg/kg of enrofloxacin once after surgical implantation of the ultrafiltration probes. The concentrations of these fluoroquinolones in intestinal sampling sites exceed the plasma concentrations and ISF concentrations by a large fraction (Figure 1). Therefore, this suggests a transfer mechanism delivering these antimicrobials into the intestinal lumen against the concentration gradient. The likely mechanism(s) are direct intestinal transport via membrane transporters, and/or delivery via the bile.11

To compare two different FDA-approved formulations of ceftiofur, 12 steers were administered either a single dose of the slow-release formulation ceftiofur crystalline-free acid (CCFA; Excede®, Zoetis) at 6.6 mg/kg or 3 daily doses of the short-acting formulation ceftiofur hydrochloride (Excenel® RTU EZ, Zoetis) at 2.2 mg/kg. Because of the slow release of ceftiofur from CCFA, the maximum concentrations (Cmax) of ceftiofur and metabolites in the plasma, ISF, and GIT were...
significantly lower than from injection of ceftiofur hydrochloride (Figure 2). These low concentrations in the GIT persisted for a longer time as the half-life was 2-3 times greater in the ileum and colon for CCFA. The early peak of ceftiofur hydrochloride in the ileum that is most apparent on day 2 may be related to biliary excretion, and the rapid elimination is likely related to bacterial degradation.10

**In Vivo Assessment Microbiological Impact of GI Tract Antibiotic Concentrations**

Fecal samples in these studies were collected repeatedly from the 12 steers treated with two different dosing regimens of enrofloxacin (above data). The E. coli levels at 12, 24, and 36 hours were significantly different from time zero in the high dose study (Figure 3). By 72 hr post-treatment, the E. coli levels had returned to baseline. In the low dose study, E. coli CFU/g at 24, 36, 48, 60, and 72 hours were significantly different from time zero (Figure 4), and then returned to baseline by 96 hours post treatment. The high dose regimen caused a greater decrease in E. coli CFUs, while the repeated dosing caused a more prolonged decrease. The high dose regimen significantly increased the median MIC at only 24 hours post treatment, while the repeated low dose therapy increased the MIC at 12, 24, and 48 hours (Figure 4). Additionally, the median MIC of the E. coli isolates was significantly higher in the low dose group compared to the high dose group starting at 12 hours, and continuing through the end of the study.

CCFA had a significant and prolonged impact on the concentration of E. coli in the feces in spite of the low concentrations. Ceftiofur hydrochloride did not significantly reduce CFUs of E. coli, potentially due to the short duration in the GIT (Figure 5). CCFA significantly increased the median MIC of E. coli isolates starting at 36 hours and continuing through 7 days post-treatment. Ceftiofur hydrochloride also increased the median MIC from 36 hours to 4 days after treatment and the median MICs of these isolates were significantly higher than those of the CCFA group at each time point (Figure 6).

**Conclusions**

Continuous sampling of the GIT in cattle is both feasible and practical, allowing for accurate PK-PD modeling. This data correlates well with changes in the fecal microbiota, and suggests that single doses of enrofloxacin or CCFA cause a smaller and shorter increase in MIC of fecal E. coli.

**References:**

Figures

Figure 1: Combined enrofloxacin and ciprofloxacin concentrations in the plasma, ISF, and GIT of steers receiving either 5 mg/kg once a day for 3 days or 12.5 mg/kg once.

Figure 2: Combined concentrations of ceftiofur and its metabolites from steers treated with either 6.6 mg/kg of ceftiofur crystalline-free acid once or 2.2 mg/kg of ceftiofur hydrochloride once a day for 3 days.
Figure 3: Fecal *E. coli* concentrations in steers receiving either 5 mg/kg of enrofloxacin once a day for 3 days or 12.5 mg/kg once.

Figure 4: MIC of *E. coli* isolates from steers treated with either 5 mg/kg of enrofloxacin once a day for 3 days or 12.5 mg/kg once.

Figure 5: Fecal *E. coli* concentrations in steers receiving either 6.6 mg/kg of ceftiofur crystalline-free acid once or 2.2 mg/kg of ceftiofur hydrochloride once a day for 3 days.

Figure 6: MIC of *E. coli* isolates from steers treated with either CCFA once or ceftiofur hydrochloride once a day for 3 days.
Airway Concentrations of Antimicrobials
Derek Foster, DVM, PhD, DACVIM (Large Animal)

Introduction
Antimicrobial concentration at the site of infection is a critical piece of information to predict clinical efficacy. In respiratory disease of cattle, the initial site of infection is the airways of the lower respiratory tract as bacterial pathogens move from the upper airways with the assistance of superimposed viral infections and the stress of transport and comingling. Therefore sampling the bronchi and bronchioles has become the gold standard. Furthermore, once respiratory disease is established, bacterial pathogens may invade across the epithelium and interstitial fluid moves into the airways across the damaged epithelium. This suggests that measuring drug concentrations in the interstitial fluid of the lungs may also be useful in understanding the efficacy of antimicrobial therapies. As discussed in the previous presentation, the approach to sampling the airways and interstitial fluid to measure drug concentration can influence the results that are obtained. Nonetheless, multiple studies have been performed to attempt to determine the drug concentrations at these sites.

Antibiotic concentrations in interstitial fluid (ISF) can be predictive of the active concentration necessary for treating most infections. However, the respiratory tract presents another challenge: the diffusion of antibiotics across the blood-alveolar barrier (also referred to as the blood-bronchus barrier in some publications). The concentration of drug that penetrates the blood-alveolar barrier can be assessed by collecting the pulmonary epithelial lining fluid (PELF). The PELF may be an important site of infection in pneumonia, but there are limitations on the interpretation of these data. The importance of adequate antibiotic concentrations in PELF notwithstanding, we also recognize that lung infection can disrupt the alveolar wall and invade the interstitial space. Therefore, a healthy pulmonary epithelium may not represent the actual environment during clinical infection. In addition, during established pneumonia which may occur in BRD, the area of consolidation may not resemble PELF. In reality, both ISF and PELF concentrations may be important to evaluate: ISF drug concentrations may be predictive of respiratory concentrations during infection when disruption of the blood alveolar barrier occurs; but drug concentrations in PELF may be more predictive of drug concentrations in the airway secretions and may be helpful for infecting agents that colonize the airways. It is possible that drugs that attain adequate concentrations in PELF (adequate to meet PK-PD targets) are appropriate to prevent colonization in the airway. When the airway is inflamed and the blood-alveolar barrier disrupted, the infection can spread to the interstitium where the ISF fluid concentration is relevant. Currently prediction of these concentrations based on drug properties is difficult. Protein binding is believed to largely drive ISF concentrations, but the influence of protein binding or lipophilicity on PELF concentrations is unclear.

Indirect Measurements
Due to the invasive nature of sampling the airways, drug concentrations in the lungs has often been extrapolated from indirect measurements. Samples may be collected from the plasma, a transudate or exudate in a tissue cage or interstitial fluid. There are inherent advantages of these techniques as the sampling can be humanely repeated overtime in the same animals without requiring euthanasia. This reduces both the cost and animal use associated with the study, and reduces the inter-animal variability. Limitations of these techniques come from the observation that these fluids may not
accurately reflect drug concentrations in the airways or lung tissue. Unfortunately, the correlation of drug concentrations between these fluids and the airways can vary dramatically between drug classes, and even within drugs, making extrapolation difficult.

**Plasma Concentrations**

Historically pharmacokinetic modeling was based primarily on plasma concentrations due to the ease of sample collection. There is still value in assessing plasma concentrations to assess drug absorption, distribution, and elimination. Yet it is now clear that use of plasma concentrations to predict drug concentrations at the site of infection is problematic. This is particularly true of the macrolides. For this class of drugs, antimicrobial concentrations in plasma are low and rapidly eliminated while samples collected from the lung demonstrate significantly higher drug levels for much longer periods of time.1-3 Most other classes of antimicrobials, on the other hand, typically achieve higher concentrations in the plasma than any other measured areas as demonstrated by the combined enrofloxacin and ciprofloxacin concentrations in steers treated with enrofloxacin (Figure 1).

![Figure 1. Combined enrofloxacin and ciprofloxacin concentrations in plasma, interstitial fluid (ISF), and pulmonary epithelial lining fluid (PELF) from steers treated with enrofloxacin.](image)

**Tissue Chambers**

Tissue chambers are perforated, inert chambers that can implanted subcutaneously in a wide variety of species. Initially, acute inflammation leads to fibrin deposition around the cage. Over time, this resolves and a mild, chronic inflammatory process persists, but stabilizes by 40 days post implantation. By this time the chamber is encapsulated by fibrous tissue.4 Samples can be repeatedly collected by aspirating fluid with a needle and syringe from the chamber. The chambers can be injected with an inflammatory agent to create a sterile inflammatory process or inoculated with bacteria. A non-inoculated chamber in the same animal can be sampled to assess the differences due to inflammation or infection. Due to the large size of cattle and the ease of standing flank anesthesia, insertion of tissue cages in the flank of steers has been routinely used to evaluate the distribution of antimicrobials into these inflammatory transudates and exudates.
Advantages of this approach include the relative ease of placement and sampling. The cages can be as simple as plastic, practice golf balls. Inoculation of the cage with clinically relevant pathogens such as Mannheimia hemolytica allows for determination of pharmacokinetic and pharmacodynamic parameters by repeatedly culturing the fluid to assess drug concentrations and bacterial counts.

Disadvantages of this approach include the long delay from implantation to sampling, and the invasive nature of implanting the chambers. The inflammatory conditions associated with implantation are milder and more chronic than would be expected in most cases of respiratory disease, and the drug concentrations measured will include both bound and unbound drug. Both of these confound interpretation of the pharmacokinetic data derived from these studies. Most studies demonstrate that drug diffusion into the chamber and subsequent elimination lags behind what would be expected, and is driven by the ratio of the surface area to volume of the chamber. In studies in which both are measured, it does not appear that the tissue cage drug concentration reflects the airway concentration, while correlations between tissue cage concentrations and infected lung tissue are unclear.

**Ultrafiltration**

Measurement of active drug concentrations in the extracellular fluid may be the preferred method to correlate PK-PD indices to clinical efficacy with some classes of antibiotics as unbound antibiotic concentrations in the interstitial fluid at the target site are responsible for the antibacterial effect in extracellular infections. In order to sample interstitial fluid without implantation of a tissue chamber, ultrafiltration probes have been placed subcutaneously in cattle. The ultrafiltration probes contain three semi-permeable loops connected to a nonpermeable tube extending to the exterior of the animal and attached to a serum collection (red top) vacutainer tube. Samples are collected by changing the vacutainer tube. The vacutainer tube provides the negative pressure for fluid collection through the small pores in the loop membrane. The membrane in the probe consists of pores allowing water, electrolytes, and low molecular weight molecules (<30 000 Da) to pass and excludes the passage of protein, protein-bound drugs, cells, and other large molecular weight compounds. Studies in cattle have demonstrated that sampling of interstitial fluid from the subcutaneous space, pleural cavity, and intramuscularly yields similar drug concentrations.

Due to the small size of the pores in the ultrafiltration probes, protein bound drug is excluded from the sample allowing for direct measurement of the active drug concentration. Further, implantation and removal of the probe is simpler than a tissue chamber and is assumed to create less inflammation, allowing for sampling immediately after implantation. Studies comparing drug concentrations in interstitial fluid with pulmonary epithelial lining fluid demonstrate that they do not directly correspond and interstitial fluid can both over (Figure 1) and underestimate (Figure 2) airway concentrations. Yet it is assumed that these peripheral interstitial fluid drug concentrations would reflect the interstitial fluid concentrations in the lung and would be relevant in cases of respiratory disease, but this has not been proven.
Figure 2. Florfenicol concentrations in plasma, interstitial fluid (ISF), and pulmonary epithelial lining fluid (PELF) from steers.

Direct Measurements
In order to overcome the inaccuracies of extrapolation from the indirect measurement techniques mentioned above, various methods of directly sampling the lungs of cattle have been used. Due to the morbidity (and mortality in the case of lung homogenates) associated with these techniques, the samples obtained are typically obtained less frequently than plasma or interstitial fluid sampling.

Lung Homogenates
Originally, lung sampling was done by collecting a portion of the lung from calves euthanized at specific time points in the study. The tissue is then homogenized and the drug extracted and measured. The obvious immediate limitation to this technique is the large number of animals required to have adequate replicates at each time point for statistical relevance. The cost, animal welfare concerns, and inter-animal variability associated with this technique has made it largely fall out of favor. Furthermore, the data obtained is likely less clinically relevant than we would like to believe. Certainly any drug in the airway and interstitial fluid of the lung would be measured, but this is confounded by the blood contamination, intracellular drug concentrations and protein binding. These clinically irrelevant concentrations lead to overestimation of lipophilic drugs and underestimation of more hydrophilic drugs.

Bronchoalveolar Lavage
Originally developed as a diagnostic test for various respiratory diseases, bronchoalveolar lavage (BAL) has more recently been used to sample the airways in order to determine drug concentrations. To collect a sample, a catheter is passed through the nose, into the trachea, and lodged into place in a bronchi. There a balloon is inflated, and sterile saline is infused. For animals greater than 200kg, typically 120-240ml of saline is used. After infusion, the saline is rapidly aspirated in an attempt to recover as much as possible. Recovery volumes vary from 10-50% of the infused saline. The recovered saline can be directly analyzed or can be centrifuged to pellet the leukocytes suspended in the saline. Drug concentrations in the recovered fluid versus the supernatant and the cell pellet can differ greatly.2,10 In order to control for the dilution due to the lavage, a urea correction must be performed. As urea should be equivalent between the pulmonary epithelial lining fluid and the
plasma, urea concentrations in the recovered saline can be compared to plasma samples obtained at the same time and control for the dilution.

With this approach a relatively large area of the lung will be sampled as fluid is flooded into the airways distal to the bronchus where the catheter is wedged. The sample will also collect fluid from both larger bronchi and smaller airways as the fluid is lavaged in and aspirated. Disadvantages of this approach include the limited frequency of sampling due to the volume of saline infused. Additionally, the calculated drug concentrations can be extremely variable due to the urea correction method. Increasing the dwell time of the saline by as little as 60s can increase the measured urea concentration in the lavage fluid by 200%.

Swab of Pulmonary Epithelial Lining Fluid
An alternative to BAL is the collection of pulmonary epithelial lining fluid (PELF) via a guarded swab. The guarded swab is passed through either the nose or mouth, down the trachea to a bronchus. The swab is then passed into the bronchus, and PELF is absorbed in the swab. PELF is then extracted from the swab allowing for measurement of drug concentration. Advantages of the swab technique include direct measurement of drug concentrations without the need to correct for dilution and the ability to repeatedly sample the same animal without concern for the volume of fluid required for sampling. Collection of BAL and PELF swabs from the same animals have demonstrated that there is significantly less variability with this technique. Correlation of the drug concentrations between the two collection methods appear to depend on the drug of interest. With enrofloxacin, BAL concentrations exceed the PELF, plasma and ISF, while with tilmicosin, the BAL and PELF concentrations are relatively similar. These reasons for this are unclear at this time. The limitations of this technique include the small area of the lung sampled, which is not as deep as a BAL. The samples cannot be partitioned to assess drug concentrations in the resident leukocytes, and it would be expected that fewer leukocytes would be obtained with the swab compared to a BAL.

Ceftiofur
Ceftiofur protein binding ranges from 52% and 63%, representing an average fraction unbound (fu) of 0.42. Terminal plasma half-life (T½) for the slow-release formulation, as expected, was over 103 hours. The ISF concentrations were lower than anticipated based on the protein of ceftiofur but correspond closely with protein binding of the metabolite, with penetration to the ISF only 5% of the plasma concentration. This is in contrast to a study by Halstead in which they demonstrated much higher penetration into tissue cages after a ceftiofur sodium dose of either 2.2 or 4.4 mg/kg. The measured penetration in that study (calculated from a ratio of the AUC values) ranged from a mean of 47% to 79%, depending on the dose and frequency of administration. This difference can be explained by the method of collection. Tissue cages collect fluid containing protein and other transudate components. Drug concentrations in transudate represent both the bound and unbound form. Our study collected protein-free ultrafiltrate, which represents the biologically active form.

Penetration of ceftiofur and associated metabolites to the PELF was higher and represented 40% of the plasma concentration. This value is close to the degree of penetration reported in the study by Halstead who used a similar method of collecting PELF. They reported penetration of 30.5% and 42.2% at the high and low dose, respectively. These values (from both studies) agree with the fraction of drug unbound (fu) described earlier of 0.42. In a review only three comparisons
between drug concentration in PELF and plasma were available for β-lactam antibiotics. For each study, the ratios for these β-lactams were < 0.5.

PELF concentrations exceeded the MIC90 cited by the manufacturer for BRD pathogens (0.2 µg/mL) for 192 hours. Concentrations in PELF exceeded the MIC breakpoint listed by CLSI for susceptible BRD pathogens (≤ 2 µg/mL) for only a short time at approximately 48 hours after administration. Because of low penetration, concentrations in the ISF never reached a level above the MIC90 cited by the manufacturer for BRD pathogens (0.2 µg/mL) at any time point.

**Enrofloxacin**

Terminal T½ for enrofloxacin and its metabolite ciprofloxacin in plasma was 9.23 hr and 14.7 hrs, respectively. This was slightly longer than the half-life from a previous study in other calves using a higher dose.9 Protein binding was approximately 46% for enrofloxacin and 19% for ciprofloxacin, representing a fu of 0.54 and 0.81, respectively. The ISF concentration for enrofloxacin and ciprofloxacin was 52% and 78% of the plasma concentration, respectively, agreeing closely with the expected concentrations predicted from the fraction unbound (fu), as one expects the unbound plasma concentration in plasma to be in equilibrium with the interstitial fluid concentration. PELF concentrations were 24% and 40% of the plasma concentration for enrofloxacin and ciprofloxacin, respectively. The PELF concentrations (enrofloxacin + ciprofloxacin combined) exceeded the reported MIC90 of 0.06 µg/mL at 48 hours after administration. The PELF area-under-the-curve (AUC) concentrations were 5.72 µg hr/mL. Using a ratio of AUC/MIC > 100, this indicates that AUC in PELF is close to achieving the target for MIC values < 0.06 µg/mL. Using the same target AUC/MIC ratio of 100, the AUC for the ISF of 12.85 µg hr/mL exceeds the target for MIC values of 0.06 µg/mL by a factor of more than 2-fold.

The enrofloxacin and ciprofloxacin penetration from plasma to the tissue fluid (ISF) was similar to predictions and consistent with other studies. But it is unclear what factors caused lower concentrations in PELF. The concentrations of enrofloxacin and ciprofloxacin in PELF were 24% and 40%, respectively of the plasma concentration. However, this appears to be in agreement with a previous study in which enrofloxacin was administered to calves at a dose of 2.5 mg/kg.8 In that study the authors point out that collection of bronchial secretions in calves with their technique was difficult and there were not enough samples for pharmacokinetic analysis. However, estimating the bronchial fluid concentrations from their figures it appears that peak (Cmax) PELF concentrations were 24% and 45% of the enrofloxacin and ciprofloxacin concentrations, respectively. Their study did not report AUC for the bronchial fluids. In a follow-up study we examined enrofloxacin and ciprofloxacin concentrations in PELF after administration of 12.5 mg/kg to calves and the same technique used in this study. At the higher dose, we observed the drug concentration in the PELF to be 124% of the plasma concentration. Therefore, there are obviously differences among studies, the cause of which are undetermined, but it may be related to differences among groups of calves or because of the higher dose administered in our most recent study.

**Florfenicol**

Florfenicol protein binding was only 5% at the high concentration and was negligible at the low concentrations, representing a fu of essentially 1.0. The terminal T½ was approximately 28 hours, agreeing with label information. The ISF concentration was almost 98% of the plasma concentration, as expected from the low protein binding (high fu). The PELF concentrations
exceeded both plasma and ISF concentrations and were over 200% of the plasma concentrations. The PELF concentrations exceeded the breakpoint of ≤ 2 µg/mL, and the MIC90 for Mannheimia haemolytica (1.0 µg/mL) for BRD pathogens for the duration of the study.

**Tulathromycin**

Tulathromycin concentrations in PELF greatly exceeded the plasma drug concentrations throughout the collection period. PELF exposure was over 9 x higher than plasma. In other studies, the terminal half-life was (mean) 90 and 64 hours in beef calves and Holstein calves, respectively. In this study we calculated a half-life of 81 hours (CV 38%). The reported plasma area-under-the-curve from zero to infinity (AUC) in Villarino’s paper was listed as 18,700, 14,100, and 14,000 ng hr/mL, depending on the study. In our study we found 14,478.84 (CV 28.61%). Thus, we believe based on these comparisons that our study was consistent with others with respect to plasma concentrations.

The PELF AUC to plasma AUC ratio in the study by Cox et al.13 was 53 and in our study was 9.1. The PELF half-life for tulathromycin in Holstein calves was 330 hours13 and in our study it was 153 hours (CV 52.5%). The peak (Cmax) and AUC in PELF were reported as 3,730 ng/mL and 492 µg hr/mL13 and in our study was 867 ng/mL (CV 29.5%), and 87.6 µg hr/mL (CV 21.74%), respectively. We calculated the AUC from samples out to 288 hours and the Cox study calculated to 360 hours. But this is probably not enough to account for the large differences in PELF concentrations between the two studies.

The differences in PELF concentrations between our study and the Cox study13 can be attributed to several factors. PELF concentration measurements are inherently highly variable and affected by the method of collection. The Cox et al study, as well as others listed in the Villarino review1 collected samples for PELF measurement by bronchoalveolar lavage (BAL). This method flushes the entire airway, including alveolar space. The direct sampling method used in our study samples the bronchial fluid directly. The alveolar tissue is more richly perfused with blood and there is faster equilibrium between blood and the PELF. The bronchial mucosa receives less blood and is slower to equilibrate. This may partially explain the lower concentrations measured in our study. Further, a recent study directly comparing the two techniques demonstrated to increased variability with BAL sampling.14

The discrepancies in sampling techniques notwithstanding, these concentrations measured in this study can be viewed in relation to the reported MIC values for cattle pathogens. As reported by the drug sponsor (Zoetis Animal Health, Florham Park, NJ, USA) BRD pathogens have MIC90 values that range from 1 to 4 µg/mL, with Histophilus somni the highest at 4 µg/mL and Pasteurella multocida the lowest at 1 µg/mL. At no point, either in plasma or PELF as measured in our study, was the mean concentration above the lowest MIC90 (1.0 µg/mL). In fact, only 1 out of the 6 calves had PELF concentrations slightly above this value. This raises questions about the property of tulathromycin that produces clinical benefits in treated animals. Perhaps, as suggested in other reviews, the anti-inflammatory effects of macrolides are responsible for clinical effects.7 Tulathromycin was shown to have significant effects on neutrophils and inflammatory cytokines.8 Obviously, further study is needed to characterize the effects of tulathromycin and other macrolides in animals with pneumonia as there is significant evidence demonstrating their efficacy.

Impact of Drug Properties on ISF and PELF Concentrations
There are differences among drugs in the penetration of antimicrobials into the ISF and PELF that may affect the therapeutic use. It appears that some antimicrobials are best for control of respiratory disease based on high PELF concentrations while others may be more effective for treatment of pneumonia due to high ISF concentrations. In Table 1, we present the penetration values for ISF and PELF in relation to the drug’s protein binding (shown as fraction unbound) and lipophilicity (shown as LogD). For all drugs (or drug metabolite for ceftiofur) there is a relationship between protein binding and the penetration to the ISF; but, a similar relationship is not present for PELF. Likewise, lipophilicity (the higher the LogD value, the higher the lipophilicity) does not appear to influence the penetration into either ISF or PELF. Thus, the properties of protein binding and lipophilicity – often cited as determinants of drug penetration into the PELF – did not have an influence on the penetration of the antimicrobials administered to the calves of this study.

Antimicrobials from different classes have distinct differences in the penetration into both the ISF and PELF that do not always directly correlate with protein binding or lipophilicity. Drugs including florfenicol and ceftiofur with high PELF concentrations are expected to be effective in the control of respiratory disease while those with high ISF concentrations including enrofloxacin and florfenicol may be more effective in treatment of active respiratory infections.

Table 1. Effect of Lipophilicity and Protein Binding on Penetration into Interstitial Fluid (ISF) and Pulmonary Epithelial Lining Fluid (PELF): LogD = log of the partition coefficient at pH 7.4. A higher LogD value indicates a greater lipophilicity (Source: ChemSpider, www.chemspider.com). Fraction unbound is the mean from three replicates. * Data for ceftiofur metabolite was derived from incurred samples (after calves converted ceftiofur to main metabolite).

<table>
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<th>Drug</th>
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<th>PELF</th>
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</table>

References:
Rumen Distension and Dysmotility
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Introduction
Rumen distension and dysmotility are not uncommon presentations in both cattle and small ruminants. These clinical signs often are linked as dysmotility can lead to rumen distension and distension can lead to dysmotility. Identifying the underlying cause of the distension and dysmotility and determining if it is truly gastrointestinal of origin is critical to appropriate treatment. Generally, a thorough physical exam combined with some routine diagnostics can accurately identify the reason for rumen dysfunction, and guide appropriate treatment and prognosis.

Normal Rumen Contour and Motility
Examination of rumen shape, fill and motility should be a part of the physical exam on all ruminants. Assessment of abdominal shape and rumen fill provides crucial information on feed intake and potential causes of distension. Decreased rumen motility can be a sensitive indicator of disease, though not specific as many inflammatory processes and increased sympathetic tone will decrease normal rumen motility.1

Abdominal and rumen contour
Assessment of abdominal shape is preferably done early in a physical exam while observing a cow from a distance. While standing directly behind the cow, determine if the cow’s abdomen appears gaunt, normal or distended.2 Abdominal shape is not entirely dictated by rumen shape, but rumen size is the most common reason for abnormal distension.3 Abnormalities identified at this time can be useful in guiding a more thorough examination of the forestomach during the remainder of the physical exam. Nonetheless, practitioners must remember that other conditions including intestinal distension, peritoneal effusion, pathologic accumulation of uterine fluid, and rupture of the prepubic tendon can affect abdominal shape and must be considered.

In a normal cow or small ruminant, the abdomen should be slightly wider than the stifles bilaterally. Typically, it will be somewhat symmetrical, though slight differences from right to left are not uncommon. The most prominent distension on left in a normal cow is typically around the level of the stifle in the mid abdomen due to the fiber accumulation in the rumen. On the right, the normal shape is a slight enlargement below the stifle due to the small intestine.

The rumen should be palpated in the left paralumbar fossa and rectally. The normal rumen stratification can be identified on physical exam. There should be a gas cap in the caudodorsal rumen, a fiber mat throughout most of the rumen, and fluid ventrally. The gas cap, found dorsally, is softer and will immediately return to its previous shape when compressed. The doughy fiber mat is the most easily distinguished layer on palpation as one can press into the rumen wall and leave an indention when it is palpated rectally. On palpation through the flank, the fiber mat simply feels firm. The fluid layer is found in the ventral left flank. This area is softer than the fiber mat, but ballottement of this area is difficult due to the weight of the rumen contents.

Normal Rumen Motility
Rumen motility should similarly be evaluated as a part of the physical exam of all ruminants. Simultaneous auscultation and palpation in the left paralumbar fossa will allow the examiner to assess the frequency and strength of rumen contractions while also hearing any abnormal sounds associated with the contraction. The normal rate is 1-3 contractions per 2 minutes. Each contraction should be strong enough to lift the examiner’s hand on the paralumbar fossa. The sound should grow louder and then softer as the fiber mat turns inside the rumen and brushes along the rumen wall. There should not be any splashes or bubbling sounds associated with the contraction. 2 This assessment of rumen motility measures the contraction rate of the dorsal rumen sac, and does not differentiate primary versus secondary contraction as the dorsal sac will contract with both patterns. In most cases, simply determining the overall rumen contraction rate is adequate.

**Abnormal Abdominal and Rumen Shape**

Finding that the cow’s abdomen is narrower than her stifles suggests prolonged anorexia as completely emptying the rumen can take several days. While specific in identifying a significant and prolonged decrease in feed intake, a gaunt abdomen provides little guidance as to the underlying problem.2

If cow is found to have a distended abdomen, first characterize the location of the distension, the organ leading to abdominal distension, and determine if the distension is due to the accumulation of gas, fluid or feed material. The distension is most commonly found in the mid abdomen and dorsally on the left, ventrally on the right, dorsally on the left and ventrally on the right, or ventrally bilaterally. Other locations (i.e. just ventrally on the left or just dorsally on the right) are less common due to the abdominal anatomy of ruminants.3

Once the distension is localized, ballottement of the abdomen and rectal palpation can be used to determine the organ or organs leading the change in abdominal shape and whether the abnormal distension is due to gas, fluid or feed material. Based on the location and type of distension, the veterinarian can then develop a relatively short differential diagnosis list (Table 1).

**TABLE 1**

<table>
<thead>
<tr>
<th></th>
<th>Dorsal Left</th>
<th>Ventral Right</th>
<th>Dorsal Left and Ventral Right</th>
<th>Ventral Bilaterally</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Gas Distension</strong></td>
<td>Type 1 Vagal indigestion</td>
<td>Uncommon</td>
<td>Uncommon</td>
<td>Uncommon</td>
</tr>
<tr>
<td><strong>Fluid Distension</strong></td>
<td>Rumen acidosis</td>
<td>Type 3 or 4 Vagal indigestion, Small intestinal distension</td>
<td>Type 2 or 3 Vagal indigestion</td>
<td>Peritoneal effusion, hydrops conditions</td>
</tr>
<tr>
<td><strong>Feed material</strong></td>
<td>Rumen impaction</td>
<td>Abomasal impaction</td>
<td>Uncommon</td>
<td>Uncommon</td>
</tr>
</tbody>
</table>
Unilateral distension on the left side is almost always due to enlargement of the rumen. Palpation of the rumen at paralumbar fossa and rectally will allow practitioners to determine the reason for the distension. Excessive gas will accumulate dorsally, and will feel like a large balloon. This is consistent with a type 1 vagal indigestion (failure of eructation). Excessive fluid distension of the left side is consistent with a rumen acidosis and the subsequent fluid shifts that occur due to osmosis. Early type 2 vagal indigestion (failure of rumen outflow) cases may only have fluid distension on the left, but most commonly they are distended bilaterally. An enlarged, doughy rumen is consistent with a feed impaction due to poor quality feedstuffs or inactivity of the rumen microorganisms. Additional information on these disorders are provided later in this chapter and elsewhere in this volume.

Distension ventrally on the right side is most commonly either fluid or feed. If the distension is due to fluid, the most likely reasons are type 3 (failure of abomasal outflow) or 4 (failure of pyloric outflow) vagal indigestion or small intestinal distension. With type 3 or 4 vagal indigestion, the abomasum initially becomes distended and then ultimately, the rumen becomes distended as well. Therefore, most of the animals present with bilateral distension. Cattle with small intestinal distension, on the other hand, may not have rumen distension, as they often present with signs of abdominal pain due to the stretch of the intestinal wall earlier in the disease process. Feed distension on the lower right side is consistent with an abomasal impaction.

Bilateral distension most commonly occurs due to fluid accumulation in the rumen or rumen and abomasum. As fluid is trapped in the rumen, it initially distends on the left in the midflank. Overtime, the ventral sac of the rumen expands greatly towards the right such that there is now distension of both sides. If there is a type 3 vagal indigestion, distension of the abomasum will contribute to the ventral, right-sided distension, and eventually fluid will back up into the rumen and cause the left sided distension. In either case, the distension on the left is more diffuse and located in the middle to dorsal region of the flank (“apple” shaped), while the distension on the right is in the ventral flank (“pear” shaped). This combination leads to the description of these cows as “papple” shaped due to their asymmetric bilateral distension.

Bilateral ventral distension is generally due to fluid accumulation in the abdomen or uterus, and therefore, rarely GI in origin (Figure 3). Differentials for these animals include pathologic accumulations of fluid in the uterus due to placental or fetal abnormalities, peritoneal effusion, or uroabdomen. Appropriate history, rectal palpation, and abdominocentesis can be used to differentiate these, but this is beyond the scope of this chapter.

**Abnormalities of Rumen Motility**

Hypermotility of the rumen is a relatively uncommon finding, though in actuality it likely occurs quite frequently. In cases of early rumen distension, hypermotility may be noted as the moderate stretch receptors in the rumen wall are stimulated. The rumen continually senses this distension as a recent meal, and increases the rate of primary contractions in response to this distension. Therefore, in most cases of pathologic rumen distension, there is an early phase associated with rumen hypermotility.13 Due to the early nature of the disease course and mild distension, it is unusual for an owner to present an animal for examination at this stage, and the hypermotility is missed. As the distension increases, the severe stretch then stops rumen contractions, and it is at this stage at which animals are typically examined.
Hypomotility is a much more common finding in clinically ill ruminants. As mentioned above, systemic inflammation or increased sympathetic tone from a variety of causes will decrease rumen motility. Hence, most cases of rumen hypomotility are from causes outside the rumen. A thorough physical exam is necessary to rule out other causes of decreased rumen contractions. Hypomotility due to rumen diseases are most commonly associated with rumen distension or rumen acidosis. When the rumen is severely distended, rumen contraction rate will slow down and ultimately stop. Some disorders, traumatic reticuloperitonitis for example, may first disrupt normal motility, leading to rumen distension, which then further slows the contraction rate. Other disorders, such a physical obstruction of the omasal canal, lead to a primary rumen distension, and the distension ultimately slows and stops rumen contractions. This distinction is important prognostically, as cases with primary motility disorders are less likely to return to productivity after relieving the distension and underlying problem, while those with hypomotility due to distension are more likely to return to normal function after relieving the distension.

Disorders Associated with Rumen Distension and Dysmotility

Ruminants with both rumen distension and dysmotility typically are diagnosed with vagal indigestion, though rumen acidosis and rumen impactions should also be considered depending on the animal's abdominal shape, rumen fill, and dietary history. In spite of the name, clinical cases of vagal indigestion have been repeatedly shown to not involve the vagus nerve in most cases. Further, Hoflund's original description of the disease based on experimental transection of the vagus nerve does little to guide diagnostic and therapeutic decisions. The classification scheme of 4 types of vagal indigestion by Ferrante and Whitlock provide a more clinically useful approach to understanding these diseases and will be used here. No matter the underlying cause, the disease typically progresses from mild rumen distension leading to hypermotility, then progressive distension causes rumen hypomotility. At this point, the animal usually presents with severe rumen distension, decreased rumen contraction rate, and anorexia.

Type 1 vagal indigestion is associated with a failure of eructation. These animals present with gas distension of the dorsal left flank, and rumen hypomotility. This can occur due to a failure of secondary contractions, an inability to clear the cardia of fluid, failure of the cardia to open, or esophageal obstruction. A loss of secondary contractions appears to be relatively rare though this may play a role in the bloat that can be seen in some calves with chronic respiratory disease. It is hypothesized that the vagus nerve can become inflamed in the thorax secondary to the respiratory disease. Bloat that is seen with in laterally recumbent ruminants is due to fluid flooding the cardia in spite of normal rumen motility. Similarly, the froth that can be created from consumption of legumes is sensed as fluid at the cardia, and prevents eructation. Damage to the rumen epithelium in the area of the cardia from rumenitis can damage the receptors responsible for sensing the presence of gas at the cardia allowing it to open for eructation. Obstruction of the esophagus can occur from an intraluminal obstruction (swallowing an apple), an extraluminal mass (tracheobronchial lymphadenopathy in cases of respiratory disease), or a mass at the cardia (papilloma). Note that in all of these cases the distension arises from a failure to eructate, not from an increased rate of gas production. Even with significant gas production from fermentation, the normal ruminant can increase eructation adequately to eliminate the gas.

Animals with type 2 vagal indigestion present with bilateral distension of the abdomen due to fluid accumulation in the rumen. The abdomen is distended at the midflank and dorsally on the left and
ventrally on the right. On rectal exam, the classic finding of an “L” shaped rumen is felt due to the significant expansion of the ventral sac towards the right flank. The fluid accumulation arises from a failure of rumen outflow with continued food and water intake and saliva production. The obstruction of the omasal orifice can be either functional or mechanical. Functional failures are most commonly due to traumatic reticuloperitonitis leading to inflammation and adhesions around the reticulum. Without normal reticular contractions, primary contractions are disrupted, and fluid is not aspirated into the omasal canal.16 Other causes of peritonitis in the cranial abdomen including liver abscesses may present similarly.17 Mechanical obstructions can occur secondary to consumption of a foreign body including rope, hay netting, or placenta.18 Masses including fibropapillomas and other neoplasias can also obstruct outflow.19 In these cases, primary contractions are not disrupted initially, and they serve to maintain the foreign body lodged in the omasal orifice. Once the rumen becomes overly distended, then the rumen contractions stop.

Type 3 vagal indigestion presents similarly to type 2 in that the animal has the classic “papple” shape and fluid distension of the rumen. The difference is that the distension is due to a failure of abomasal motility and outflow. Reflux of abomasal fluid leads to the rumen distension, and the abomasum and rumen both contribute to the abdominal distension that is seen externally. The combination of abomasal and rumen distension leads to rumen hypomotility. Like type 2 vagal indigestion, type 3 also can be due to a functional or mechanical failure of abomasal motility. Functional causes include abomasal lymphosarcoma,20 traumatic reticuloperitonitis16 and abomasal damage after an abomasal volvulus.21 Roughly 15% of cattle with an abomasal volvulus will go on to develop abomasal motility disorders. This appears to be due to ischemic damage to the abomasal wall, peritonitis, and/or damage to the vagus nerve.21 Mechanical obstructions here are less common, though lymphosarcoma and feed or sand impactions can also physically disrupt pyloric outflow. Iatrogenic causes should be considered including inappropriately placed pyloropexy or incorrect placement of a toggle suture.

Type 4 vagal indigestion is a less well-defined syndrome of partial pyloric obstruction or generalized ileus. These animals have less abdominal distension compared to those with type 2 or 3 vagal indigestion. A common reason for this presentation is late term pregnancy, as the fetus may physically impede pyloric outflow or proximal intestinal motility.22 Other causes are related to severe systemic disease including hypocalcemia, peritonitis, septicemia, and enteritis leading to reduced intestinal motility.

Rumen acidosis is another cause of rumen distension and hypomotility. Due to the rapid production of volatile fatty acids from grain fermentation that exceeds the absorptive capacity of the rumen, water is pulled by osmosis into the rumen. This accumulation of fluid in the rumen causes a left-sided abdominal distension that may initially appear similar to a type 2 or 3 vagal indigestion. The abnormally low pH of the rumen fluid stops rumen contractions as the rumen attempts to slow fermentation. These animals with rumen acidosis will typically be more depressed and dehydrated than those with vagal indigestion, and examination of the rumen pH allows for easy differentiation of these diseases.

Animals with a rumen impaction will present with a firm, left-sided abdominal distension due to feed accumulation in the rumen. Rumen contraction rate will be variable depending on the degree of distension, and could range from increased to absent. The underlying pathogenesis of this disease
could be either a lack of appropriate rumen microbial populations or feeding a low quality, largely indigestible forage. The former can be seen in young animals who begin consuming large amounts of forage prior to developing a functional rumen or in an adult animal who has lost the normal rumen bacterial population after acidosis, anorexia, or antimicrobial administration. When fed indigestible forage, the rumen bacteria cannot adequately breakdown the plant material or the fermentation is excessively slow. This leads to an accumulation of fiber within the rumen as the animal continues to consume a large volume of feed material, yet cannot meet its nutritional needs. Hence, in chronic cases, animals will present with severe rumen distension but extremely poor body condition. The severe weight loss may be overlooked by owners due to the animal’s large abdomen.

**Diagnostic Approach to Animals with Rumen Distension and Dysmotility**

**History and Physical Exam**

When examining an animal with rumen distension and dysmotility, a complete physical exam will generally provide practitioners with a reasonably short list of differentials that can be further assessed with minimal diagnostic testing. Prior to examining the animal, it is useful to gather an appropriate nutritional and housing history. How much grain is fed? What is the quality of forage that is provided? Any exposure to legumes? Recent construction or building of fences? Evidence of trash or other potential foreign bodies in the pasture or animal’s enclosure? Has the animal had a recent abomasal volvulus, pyloropexy, or toggle procedure? Then the animal is observed prior to restraint to properly assess abdominal contour as described above.

Rumen contraction rate and strength should be assessed by auscultation of the left paralumbar fossa. Most of these animals will have few or no rumen contractions. If the animal does have some contractions, simultaneous auscultation the reticulum with palpation of the rumen will determine if the contractions are primary or secondary contractions. During the exam, particular attention should be paid to those potential diseases that can lead to vagal indigestion. A withers pinch should be performed. A lack of response could be due to any cause of cranial abdominal pain, though traumatic reticuloperitonitis is the classic disease associated with this finding. Other considerations include a ruptured liver abscess or a perforating abomasal ulcer. Practitioners may get some indication of the underlying problem if the cow responds more severely to sternal pressure on the right or left as traumatic reticuloperitonitis will typically cause more pain on the left, while other causes are more likely located on the right. On auscultation of the thorax, is there evidence of respiratory disease or muffling of the heart associated with traumatic reticulopericarditis? Is there any lymphadenopathy that might be suggestive of lymphosarcoma? On rectal exam, the rumen size and texture is assessed to determine if there is fluid distension of the ventral sac. Also, the pregnancy status of the animal is determined, internal lymph nodes are palpated, and the viscera are palpated for evidence of peritonitis and adhesions.

**Ancillary Diagnostic Testing**

**Rumen fluid analysis**

After completing the physical exam, passing a stomach tube is valuable diagnostically and therapeutically. In many cases of type 1 vagal indigestion, gas will be released when the tube is passed. With type 2 or 3 vagal indigestion, fluid may spontaneously reflux from the tube. If not, fluid should be siphoned off the rumen to reduce the distension and provide a sample for diagnostic evaluation. Upon collection of the fluid, the pH should be evaluated to rule out rumen acidosis. In
cases of vagal indigestion, the pH will be normal (5.5-7) or slightly alkaline due to anorexia. The reflux of abomasal fluid with type 3 vagal indigestion is not sufficient to reduce rumen pH out of the normal range. When collecting rumen fluid orally, it is critical to collect several hundred milliliters of fluid to minimize the impact of saliva contamination on the pH. Excessive saliva contamination in a small volume sample will artificially elevate the pH due to the buffering capacity of ruminant saliva. A drop of the fluid should be placed on a microscope slide and evaluated at low magnification to assess protozoal activity. There should be numerous protozoa of varying sizes rapidly moving across the field. This can be used as a proxy measure of general microbial activity as the protozoa appear to be more susceptible to changes in the rumen environment. In particular, the larger Holotrich protozoa appear to be especially sensitive to changes in the rumen environment. Acidosis or prolonged anorexia in vagal indigestion are the most common causes of decreased protozoal numbers. This assessment needs to be done relatively rapidly as these protozoa can be quite susceptible to changes in temperature and exposure to oxygen. Bacterial populations can be further investigated by Gram staining a sample of fluid, and measuring the methylene blue reduction time.

A sample of rumen fluid should also be strained for measurement of chloride content. In normal rumen fluid, the chloride content should be less than 30 mEq/L. Abomasal outflow obstructions (type 3 vagal indigestion) cause an increase in rumen chloride as the chloride secreted into the abomasum refluxes back into the rumen. It remains sequestered there due to the rumen epithelium’s relatively poor ability to absorb electrolytes. This finding is quite useful in differentiating type 2 and type 3 vagal indigestion as they often present similarly. It has been demonstrated that acetate in the rumen fluid can falsely elevate chloride measurement when assessed using routine potentiometric blood chemistry analysis. This interference is of less concern in animals with anorexia as the acetate levels will be lower. Further, a chloride level less than 30 mEq/L can be reliably interpreted as normal, while an elevated rumen chloride concentration could be due to abomasal reflux or increased acetate levels. Therefore it is critical to interpret rumen chloride concentrations in concert with blood chemistry analysis.

| TABLE 2 |
|-----------------|---------------------------------------------|
| **Rumen Fluid Analysis**                           |                                                                 |
| **Color**                                           | Yellow-green to olive green depending on diet               |
| **pH**                                              | 5.5-7                                                       |
| **Protozoal Activity**                              | Abundant protozoa of different sizes                      |
| **Methylene Blue Reduction**                         | Less than 5 min                                            |
| **Chloride**                                        | Less than 30 mEq/L                                         |

**Blood Chemistry Analysis**
Assessment of serum chloride and bicarbonate can be useful in distinguishing between type 2 and 3 vagal indigestion for similar reasons as rumen chloride. Reflux of the chloride and subsequent sequestration in the rumen leads to a severe hypochloremia as the chloride is normally reabsorbed in the duodenum. Similarly, the hydrogen ions secreted into the abomasum to acidify the contents...
are associated with bicarbonate moving into the bloodstream. Normally, the bicarbonate from the bloodstream is then taken by the duodenum to neutralize the abomasal pH when ingesta enters the proximal small intestine. When this flow is disrupted, a severe metabolic alkalosis occurs as the bicarbonate remains in the circulation. Hence, animals with a type 3 vagal indigestion will have a severe hypochloremic metabolic alkalosis. Those with other rumen motility disorders may have similar electrolyte and acid-base derangements, but not to the same degree. The hypochloremic, metabolic alkalosis in these cases is associated with reduced abomasal motility due to anorexia and systemic disease. Other findings on the blood chemistry analysis can also be instructive as an increased globulins would suggest a chronic inflammatory process such as traumatic reticuloperitonitis.

**Ultrasound of the Reticulum**

To definitively identify reticular contractions, it is helpful to use ultrasound to visualize the reticulum as auscultation can be difficult, and does not let one evaluate the strength of the reticular contraction.26 The reticulum can be identified to the left of midline, just caudal to the xiphoid. It will appear as a U-shaped structure, and only the wall can be seen due to the gas mixed into the ingesta. The cranial sac of the rumen will appear just caudal to the reticulum. The reticulum will have a biphasic contraction in which the first contraction is smaller, and the second completely collapses the reticular lumen as it moves dorsally. Identification of normal reticular contractions in cases of rumen distension suggest that the problem is less likely a functional motility disorder of the forestomach. A lack of reticular contractions, on the other hand, may suggest either a primary motility disorder or hypomotility due to rumen distension. Interestingly, many animals with rumen hypomotility will have reticular hypermotility, and this was particularly pronounced in cases of type 2 vagal indigestion.27 Further, imaging of this area can identify abscesses, adhesions, or fluid accumulation associated with traumatic reticuloperitonitis.

**Rumenotomy/Abdominal Exploratory**

Abdominal surgery may ultimately be necessary to accurately diagnose the underlying disease in animals with rumen distension and dysmotility. This has the advantage of being both diagnostic and therapeutic. Prior to surgery though, one must determine if the animal most likely has a type 1 or 2 vagal indigestion versus a type 3 or 4. This distinction is important as surgical diagnosis and correction of type 1 and 2 vagal indigestion is best accomplished through a left flank celiotomy and rumenotomy, while type 3 and 4 problems are best addressed from a right flank celiotomy and exploratory.

**TABLE 3**

<table>
<thead>
<tr>
<th>Location of Abdominal Distension</th>
<th>Rumen Contents</th>
<th>Rumen Chloride</th>
<th>Serum Chloride</th>
<th>Serum Bicarbonate</th>
</tr>
</thead>
<tbody>
<tr>
<td>Type 1-- Failure of eructation</td>
<td>Dorsal left</td>
<td>Gas</td>
<td>Normal</td>
<td>Normal to mildly decreased</td>
</tr>
<tr>
<td>Type 2-- Failure of rumen outflow</td>
<td>Dorsal left, ventral right</td>
<td>Fluid</td>
<td>Normal</td>
<td>Normal to mildly decreased</td>
</tr>
<tr>
<td>Type 3-- Failure of abomasal outflow</td>
<td>Dorsal left, ventral right</td>
<td>Fluid</td>
<td>Increased</td>
<td>Moderate to severely decreased</td>
</tr>
<tr>
<td>Type 4-- Partial failure of pyloric outflow/proximal intestinal obstruction</td>
<td>Dorsal left, ventral right</td>
<td>Fluid</td>
<td>Normal to increased</td>
<td>Mild to moderately decreased</td>
</tr>
</tbody>
</table>

**Treatment Options**

Treatment of the most these disorders associated with rumen distension and dysmotility will commonly require a rumenotomy or abdominal exploratory as discussed above. A few principles of therapy applicable to any of the above disorder are discussed below.

**Emergency Treatment**

Emergency treatment of severe rumen distension may be necessary even prior to complete evaluation. As the rumen becomes distended, the animal’s ability to breathe is reduced as the rumen impedes normal movement of the diaphragm. Passage of a large diameter orogastric tube should always be one’s initial consideration as this will allow passage of accumulated gas or fluid without the risk of peritonitis associated with rumen trocarization. A surfactant, such as poloxalene, can be administered at this time if there is any suspicion of a frothy bloat. Trocarization of the rumen can be performed in cases of extreme respiratory distress or if passage of an orogastric tube is not possible. A self-retaining, screw-in trocar is best, but no matter what type is used, owners should be made aware of the significant risk of peritonitis.

**Transfaunation**

For any of these diseases, there is likely to be an associated disruption of the rumen microbial populations. This disruption could be due to pH changes following rumen acidosis or due to prolonged anorexia in cases of vagal indigestion. Correction of the underlying cause of the motility disorder is key, but transfaunation with normal rumen fluid can speed the animal’s return to normal productivity by replenishing the microbial populations. Drenching via ororuminal tube an adult bovine (or adding directly to the rumen during a rumenotomy) with 10-16 L of fresh rumen fluid appears to be clinically effective. Similarly, transfaunation of 1-4 L of fresh rumen fluid in sheep and goats can re-establish normal microbial populations.
Conclusions
Rumen distension and dysmotility (most commonly hypomotility) are often found together in clinical cases. A thorough physical exam to determine the location of the rumen distension, assess the rumen contents, and careful auscultation of rumen contraction patterns will commonly provide the examiner with a relatively short differential diagnosis list. From here, rumen fluid analysis, ultrasound of the reticulum, and blood chemistry analysis can further guide surgical planning. Based on these findings, the practitioner can then make an informed decision concerning the surgical approach--left flank rumenotomy for rumen acidosis, type 1, or type 2 vagal indigestion or a right flank exploratory for type 3 or 4 vagal indigestion.

References:
Oral Fluid Therapy
Oral fluid therapy is the mainstay in treatment of dehydration in neonatal calves. Oral rehydration solutions can be used as a stand-alone treatment in mild to moderate dehydration or combined with IV fluid therapy in cases of more severe dehydration. Goals of oral fluid therapy include correction of dehydration, acidosis, and electrolyte abnormalities and provision of energy. There has long been a theoretical admonition to avoid feeding milk with oral fluids as inhibition of the abomasal milk clot could be detrimental. Several studies have now demonstrated that this risk is overstated, and that calves can continue to gain weight in spite of diarrhea if fed milk. Ideally, milk feedings are continued as usual with administration of oral fluids spaced equally between the feedings. Oral rehydration solutions should contain at least 90 mmol/L of sodium to provide adequate osmolarity to adequately rehydrate the calf. In order to maximize sodium absorption and subsequent water absorption, it should also contain an amino acid (glycine is most common), volatile fatty acid (acetate or proprionate), and glucose (ratio of 1:1-3:1 with sodium). Each of these molecules have co-transporters with sodium so inclusion of them in oral fluids will increase sodium and water absorption. Sodium concentration should not exceed 130 mmol/L and glucose should not exceed a 3:1 ratio with sodium to maintain an appropriate osmolarity of the solution.

Other electrolytes, including chloride and potassium should also be included. Chloride concentration should be less than that of sodium so that the strong ion difference is increased to provide greater alkalinizing ability. Potassium should also be included as it is commonly lost in diarrheic calves. The optimal level of both of these electrolytes is unclear at this point.

An alkalinizing agent should be included in all oral solutions for calves. Historically, sodium bicarbonate was included but it has significant drawbacks when compared to acetate and proprionate. Acetate and proprionate do not alkalinize the abomasum, increase absorption of sodium and water, and will provide additional energy when metabolized. Further, they have been shown to be as effective as bicarbonate.

IV Fluid Therapy
Intravenous fluid therapy is often critical to the success in treating calves with diarrhea. Any calf that is greater than 8% dehydrated, cannot stand, or has lost its suckle reflex should receive IV fluid therapy. Goals of fluid therapy in diarrheic calves include correction of hypovolemia, acidosis, hypoglycemia, and electrolyte abnormalities.

Isotonic fluids that are commonly used in calves include 1.3% sodium bicarbonate, lactated Ringer’s solution, and other alkalinizing fluids including Normosol and Plasmalyte. Calves with a severe acidosis (pH<7.2) commonly have a D-lactic acidosis which is very slowly eliminated by the calf. As lactated Ringer’s solution contains a racemic mixture of L and D-lactate, it should be avoided in these calves, and administration of 1.3% sodium bicarbonate is recommended. Adding 150ml of 8.4% sodium bicarbonate or 13g of sodium bicarbonate to 1L of sterile water will create isotonic sodium bicarbonate. Typically 1-4L of this solution is required to correct the acidosis and dehydration. This can be administered over several hours through a temporary jugular catheter. Balanced electrolyte solutions more slowly correct acidemia and should only be used in mild cases or
after initial correction of a severe acidosis. Many diarrheic calves will be hyperkalemic, but have a total body deficit of potassium. Therefore, addition of potassium chloride to IV fluids is not recommended without measuring serum potassium levels first. If this is not possible, IV potassium should not be given, and this deficit can be corrected using oral fluids. In an anorexic calf, dextrose can be added to lactated Ringer’s solution to create a 2.5% solution by adding 50ml of 50% dextrose to a 1L bag of LRS. This can be administered at a rate of 2-4ml/kg/hr.

To avoid the use of a short-term IV catheter, hypertonic solutions can be used in combination with oral fluids to provide sustained fluid therapy. Hypertonic saline (7.2%) at 4-5ml/kg IV over 5 minutes can be administered, and, when followed by oral fluids, provides equal benefit to continuous IV fluids. This will correct dehydration and decrease serum potassium. Hypertonic saline will also partially correct acidosis as improving renal perfusion will allow for increased excretion of D-lactate. Yet it is unlikely to correct a severe acidosis which is commonly found in diarrheic calves. To overcome this problem, recent work has focused on the use of hypertonic sodium bicarbonate solutions (5% or 8.4%). Initially discouraged due to concerns over paradoxic CNS acidosis and severe respiratory acidosis, significant side effects of use of hypertonic sodium bicarbonate have not been seen even in cases of hypoventilation. Doses used range from 5 to 10 ml/kg and are typically infused over 5-10 minutes. When followed by oral electrolyte solution, hypertonic sodium bicarbonate is more effective than hypertonic saline in correcting acidosis and as effective in correcting dehydration. It appears to be safe, and can provide a convenient and effective method of correcting dehydration and acidosis in neonatal calves.
Cryptosporidiosis is a protozoal infection that occurs in most mammals and birds that is spread through the fecal-oral route. Waterborne spread is most common in humans, whereas environmental contamination is the most common source in cattle. The oocysts are infective at the time of excretion and remain in the environment for long periods of time as only extreme heat or cold will destroy the oocysts. Most disinfectants are not capable of inactivating them, though desiccation will. Infection in all species is typically most severe in the young and immunosuppressed with profuse, watery diarrhea as the most consistent clinical sign.

**Cryptosporidiosis in Calves**

Derek M. Foster, DVM, PhD, DACVIM

**Cryptosporidiosis**

Cryptosporidiosis is a protozoal infection that occurs in most mammals and birds that is spread through the fecal-oral route. Waterborne spread is most common in humans, whereas environmental contamination is the most common source in cattle. The oocysts are infective at the time of excretion and remain in the environment for long periods of time as only extreme heat or cold will destroy the oocysts. Most disinfectants are not capable of inactivating them, though desiccation will. Infection in all species is typically most severe in the young and immunosuppressed with profuse, watery diarrhea as the most consistent clinical sign.

**Cryptosporidium spp.**

In humans, *Cryptosporidium parvum* and *hominis* are the primary pathogens. *C. parvum* is zoonotic and primarily found in cattle, while *C. hominis* is exclusively found in humans. These two pathogens cause similar clinical signs and are indistinguishable on fecal exam, so they were only recently separated into different species based on PCR testing. Because of the similarities between the two organisms, many previous waterborne outbreaks in humans were attributed to contamination from dairies, yet water contamination with human sewage was the actual culprit. Other potentially zoonotic cryptosporidial organisms include *C. meleagridis* (chickens, turkeys, wild birds), *C. felis* (domestic cats), *C. canis* (domestic dogs), *C. muris* (rodents), and *C. suis* (swine).

Four cryptosporidial species are found in cattle: *C. parvum*, *C. bovis*, *C. ryanae*, and *C. andersoni*. *C. bovis* and *ryanae* are host-adapted to cattle, are not zoonotic, and are believed to be non-pathogenic. These species are found primarily in weaned calves. *C. andersoni* infects the gastric glands of adult cattle, and causes pathology similar to ostertagiasis. *C. andersoni* is only found in adult cattle and is not zoonotic.

**Pathophysiology**

Shedding of *Cryptosporidium parvum* oocysts occurs as early as 3 days of age, peaks at 14 days of age, and may continue at low levels throughout life, but clinical disease is rarely seen beyond 3 weeks of age. Once oocysts are ingested, *C. parvum* has a complicated life cycle involving multiple stages which allows it to proliferate exponentially. Multiple proteins appear to mediate attachment of the organism to the intestinal epithelium, which can be inhibited by colostral oleic acid.

*C. parvum* infection leads to a malabsorptive diarrhea due to the intestinal epithelial cell loss and severe villous atrophy that occurs. The cell loss appears to be due to an increase in apoptosis, but which cells are lost and when is tightly regulated. It is currently unclear if this regulation of apoptosis benefits the host to maintain epithelial continuity or the pathogen to maintain its intracellular location. Because differentiated, absorptive villous epithelial cells are lost, the normal secretion of the crypts overwhelms the absorptive capacity of the villi.

Villous atrophy cannot account for all of the fluid loss in *C. parvum* infections, and prostaglandin-mediated secretion and inhibition of sodium chloride absorption have been documented. PGE2 causes approximately 25% of the secretion by acting on the enterocyte, while PGI2 leads to the majority of the secretion by stimulating the enteric nervous system. Secretion due to prostaglandin production is mediated by an intracellular increase in cyclic adenosine monophosphate and calcium.
Treatment
Treatments of C. parvum remain an elusive goal in both human and veterinary medicine. Halofuginone is marketed as a preventative, and is reasonably effective if administered daily for the first weeks of life, yet does not improve clinical signs if administered after the onset of diarrhea. Decoquinate has been used as both a treatment and preventative, but has not been shown to be effective when administered in either manner. Results with nitzoxinide have been mixed. Paromomycin, azithromycin, and activated charcoal with a wood vinegar extract have all been shown to be effective as treatments, though the availability and cost effectiveness of these treatments are questionable.

Prevention
Vaccination to prevent cryptosporidial diarrhea has been a goal that has eluded researchers for decades. Routinely, research can demonstrate antibody titers in vaccinated calves or calves fed colostrum from vaccinated cows. Additional studies that demonstrate actual protection from infection most commonly challenge calves within the first 24 hours of life after administration of colostrum from vaccinated dams. The success of passive immunity early in life with the lack of published efficacy later suggests that the efficacy is related to prevention of initial infection in the intestinal lumen as opposed to absorption of colostral antibodies. Therefore, continual feeding of colostrum theoretically could prevent infection, though this is not practical to continue through 14 days of age.

The impact of nutrition on diarrhea and shedding of C. parvum remains unclear. Improved colostrum management has been associated with reduced oocyst shedding and reduced risk of diarrhea, though this link is not consistently found in other studies. Similarly, the impact of feeding waste milk, saleable milk, or milk replacer on C. parvum is unclear. Feeding of waste milk or fermented milk has been shown to be protective, while feeding of saleable milk has been associated with increased risk though this may be influenced by the husbandry standards in the developing country in which this study took place. Use of milk replacers has been shown to both increase and decrease the risk of C. parvum infection. One study demonstrated that a high protein milk replacer would reduce clinical signs of cryptosporidiosis, but free-choice acidified milk replacer had no impact.

Cryptosporidial oocysts are extremely hardy and can persist in the environment for long periods of time, particularly in moist areas and standing water. Cleaning bottles, feeding equipment, and hutches thoroughly with soap and water can remove oocysts in many situations. Most alcohol or chlorine based disinfectants are effective against C. parvum. Chlorine dioxide can be effective but requires several hours of contact time. 3% hydrogen peroxide is likely the most effective and practical disinfectant as it only requires 20 min of contact time, but no disinfectant is completely effective.

References:
Advanced Diagnostic Techniques
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Rumen Fluid Analysis
Evidence of abnormal abdominal shape, increased or decreased rumen motility that is not explained by systemic illness, reduced or abnormal fecal output, changes in rumen texture, or suspicion of rumen acidosis are all indications for rumen fluid analysis. A sample of rumen fluid can be obtained via orogastric intubation or rumenocentesis. A larger volume of fluid can be obtained orally if a weighted tube is used. This approach does have the disadvantage of potential saliva contamination that may hinder accurate pH measurement. Rumenocentesis yields a smaller volume of fluid, but enough for most analyses. This sample will not be contaminated with saliva, but can lead to abscessation at the collection site. To obtain this sample, a five inch needle is inserted in the lower left flank cranial to the stifle and directed towards the opposite shoulder. A small amount of air is introduced to clear the needle, and then 3-5 ml of fluid can be obtained.

Analysis should include visual inspection, measurement of pH, and assessment of bacterial and protozoal activity. The fluid should be olive to brownish-green in animals with a hay diet, deep green in those on grass, and yellowish-brown in those with a diet of silage and grain. A milky gray color is consistent with rumen acidosis, and greenish-black is suggestive of rumen stasis. The fluid should be slightly viscous, which will increase with saliva contamination and decrease with decreased microbial activity. The smell of rumen fluid should be aromatic if normal, and will become more acrid in cases of acidosis and putrid with prolonged rumen stasis. The normal pH should be between 5.5 and 7 with animals on high grain diets tending to be in the lower end of the normal range. Either pH paper or a portable pH meter is an acceptable method to assess rumen pH. A pH less than 5.5 is almost always due to rapid fermentation of grain, while an increased pH can be due to anorexia or saliva contamination of the sample.

Bacterial activity can be assessed by sedimentation and methylene blue reduction. Normal fluid should separate with small particles settling out and large particles floating within 4-8 minutes. A decreased time is consistent with decreased microbial activity, and an increased time is typically due to increased surface tension that is associated with frothy bloat. Methylene blue reduction is measured by mixing 1ml of 0.03% methylene blue with 10 ml of rumen fluid. It should return to its previous color in less than 10 minutes as the bacteria reduce the dye. Increased time indicates a decrease in bacterial activity.

Protozoal activity can be easily assessed by placing a single drop of fresh fluid on a slide and examining it at low power. There should be numerous large, medium, and small protozoa actively swimming in the fluid. The larger protozoa are the least hardy, so they are the most sensitive to changes in the rumen environment.

Additional analyses can be done in certain cases. Chloride measurement can be useful in determining the type of obstruction in cases of Type II or III vagal indigestion. Normal fluid should be less than 30 mEq/L, and increases are typically due to reflux of abomasal contents due to a pyloric outflow obstruction. This measurement can be made using traditional chemistry analyzers, though acetate may falsely elevate the value. In this case, it must be interpreted in light of serum chloride and bicarbonate.

Transtracheal Wash
Most routine cases of respiratory disease can be treated based clinical signs without further diagnostics. In outbreaks, it is necessary to obtain a diagnosis of a causative agent as this will impact...
control recommendations. Necropsy samples may be biased by chronicity and previous treatment; therefore, obtaining a bacterial culture from an early case is ideal. Transtracheal washes allow examination of the leukocyte population and provide samples for bacterial and Mycoplasma culture. The calf or cow is sedated with 0.05-0.1mg/kg of lidocaine. A site for the tracheal puncture is selected in the middle of the neck, ventral to the larynx, and this area is clipped, scrubbed, and infiltrated with lidocaine. A stab incision is made through the skin to allow for easier placement of the catheter. The supplies for this procedure can be purchased as a kit, or repurposed, sterile needles and urinary catheters can be used. A 12 gauge, 3 inch needle is placed between two tracheal rings and directed ventrally into the trachea. This is used as a cannula to pass a stiff catheter (approximately 50 cm, 5 French). The distance that the catheter is passed is dependent on the size of the animal as the goal is to reach the thoracic inlet and the horizontal portion of the trachea. Twenty milliliters of sterile saline is infused, and immediately aspirated. Typical yield is only a few milliliters. If no sample is obtained, the catheter may need to be repositioned cranially or caudally. This injection and aspiration can be repeated several times to obtain adequate samples for culture and cytology. The cannula should be removed first, and then the catheter withdrawn to prevent shearing of the catheter.

**Thoracocentesis**

Pleuropneumonia can be a rare sequela to respiratory disease in cattle. Other causes of pleural effusion include neoplasia, trauma, traumatic reticuloperitonitis and pleuritis. A cow in respiratory distress with decreased to absent lung and heart sounds on one side of her thorax likely has a significant fluid accumulation. These animals may also develop right heart failure due to compression of the right atrium. Thoracic ultrasound is invaluable in identifying the location, quantity, and type of fluid present. If ultrasound is not available, a sample can be taken blindly from just caudal to the elbow. Clip and prep the chosen sampling site, and infuse lidocaine into the skin and intercostal muscles. A small stab incision is made in the skin and external surface of the muscles. A 3 in, sterile teat cannula is bluntly placed into the fluid. This will take significant force and care should be taken not to damage the underlying lung or heart. A syringe should be attached to the cannula prior to placement to prevent creation of a pneumothorax if the fluid pocket is missed. A fluid sample is obtained for cytology and culture. If a large amount of fluid is present, the cannula can be removed, and a larger thoracic catheter can be placed in a similar manner to drain the fluid.

**Abdominocentesis**

Collection of abdominal fluid is useful in diagnostic evaluations of cows with vague signs of weight loss and anorexia. It is also useful in cases of suspected lymphosarcoma, peritonitis, and uroabdomen. As rapid fibrin deposition commonly prevents diffuse peritonitis in cattle, the location of abdominocentesis can have diagnostic value. The ventral abdomen just cranialateral to the udder, behind the fold of the flank on the right side is a commonly productive site for initial sampling. diffuse peritonitis, localized peritonitis due to reproductive trauma, or lymphosarcoma can be diagnosed in this area. Sampling on the left in the same area is generally interpreted in a similar manner, but obtaining a sample is more difficult due to the presence of the rumen. Abdominocentesis in the cranial left abdomen just caudal to the sternum is helpful in diagnosing traumatic reticuloperitonitis. On the cranial right side, lymphosarcoma and peritonitis due to abomasal leakage or liver abscesses can be found. The selected site should be clipped and prepared steriley. The sample can be obtained with an 18 gauge, 1.5 inch needle or a teat cannula. If a teat
cannula is used, the site should be infused with lidocaine, and a small stab incision should be made through the skin. The needles should be slowly advanced to check for fluid and prevent enterocentesis. It is not uncommon to not obtain a sample; therefore, the needle (or cannula) is left in place and another location near the previous site is chosen for additional sampling. This can be repeated multiple times until an adequate sample is obtained. Cytology is typically diagnostic, though culture can at times be of value to guide treatment.

**Arthrocentesis**
Analysis of joint fluid can be important in differentiating degenerative joint disease and septic arthritis. Selection of the site for sampling is based on ease of collection (i.e. choose the pouch of the joint that appears the most distended). The site is prepared steriley, and an 18 gauge, 1.5 inch needle is inserted into the joint. The fluid is commonly under great pressure, and will easily be obtained. If the needle is clearly in the pocket of fluid, but no sample is obtained, the joint fluid may be too caseous to pass through the small needle. In this case, the joint can be resampled with a 16 or 14 gauge needle. Cytology and bacterial and Mycoplasma cultures are commonly useful in these cases.

**Cerebrospinal Fluid Collection**
Most common neurologic diseases of cattle do not produce pathognomonic changes to the CSF, so CSF analysis is only indicated in certain cases to rule out specific diagnoses. Meningitis (most commonly in calves), salt toxicity, and cerebrospinal nematodiasis can all be diagnosed and may be ruled out on CSF analysis. Collection is almost exclusively done at the lumbosacral space. If the animal is recumbent, it should be placed in sternal recumbency with each hindleg out to either side or complete lateral recumbency. In either position or in the standing animal, the lumbosacral space can be identified as a depression that is approximately between the tuber coxae. In most cattle, a 3.5 in spinal needle is sufficient, but in larger cows and bulls, a 5 in needle is needed. In calves, a 1.5 in needle is sufficient. The skin is clipped and prepped, and the skin is blocked with subcutaneous lidocaine. The needle is introduced into the center of the depression, perpendicular to the spine, and exactly on midline. The needle is advanced until a distinct pop is felt as the needle penetrates the ligamentum flavum. Often the animal will twitch or wag its tail at this time. The stylet is then removed and fluid should flow from the needle. Fluid is collected for cytology or measurement of sodium concentration in cases of suspected salt toxicity.
Penetrating wounds in horses are an important cause of morbidity and mortality. While many of these wounds carry a very good prognosis the situations in which they occur can be dramatic and very stressful to all concerned. The veterinarian must be ready to calmly deal with the horse and also have excellent communications with the owner. Discovering which underlying structures have been penetrated and initiating appropriate and timely treatment is critical to the management of penetrating wounds. Wounds that penetrate into the thorax or abdomen may cause systemic compromise that will require stabilization before referral and recognition of systemic signs of blood loss and respiratory distress are important. The clinical examination of small or large wounds should always include consideration of the underlying structures and further investigation if there is any concern of the wounds penetrating into those deeper tissues. It is crucial to be knowledgeable about the anatomy of the region. Small puncture wounds can be just as dangerous as large open wounds. The correct identification of involvement of underlying structures is crucial to the appropriate treatment but is not always easy. However diagnostic efforts need to be made or horses can be referred to local hospitals if facilities or circumstances restrict the capabilities on the farm.

**Thoracic Wounds**

Wounds that penetrate into the thoracic cavity are rare as the thickness of the muscles and ribs is very protective. Wounds that do have sufficient force to penetrate cause significant pain and may cause respiratory or cardiovascular compromise. When the chest is penetrated the negative pressure of the pleural cavity is lost and the lung often collapses quickly away from the chest wall. In many penetrating injuries the lung is therefore undamaged (apart from high velocity wounds such as those caused by gunshot). The penetrating object should not be removed if possible until the arrival of the veterinarian to avoid worsening of a potential pneumothorax or bleeding. If the object penetrates into the heart or major vessel there are no good options for successful management unlike in the dog or other species so removal prior to hospitalization is acceptable. The lateral thoracic vein is located superficially on either side of the thorax and may be lacerated causing substantial but usually nonfatal blood loss. If the owner has already removed the penetrating object they can be instructed to place large, clean towels over the penetrating wound and keep the horse as still and calm as possible.

The penetrating object can ideally be left in place while an exam of the horse is carried out paying special attention to mucus membrane colour, respiration, and auscultation. The pain associated with the chest wound results in very shallow breathing and auscultation for the lack of breath sounds associated with a pneumothorax can be surprisingly difficult. Definitive diagnosis of pneumothorax often requires examination of the wound after removal of the penetrating object. A clinical examination can dictate if there is a strong suspicion of penetration and if significant blood loss has occurred. It is recommended to place an intravenous catheter to enable quick access for medications and administration of fluids if the horse is showing signs of fluid loss and shock.

Removal of the penetrating object should be based on location, ability to refer or not, and available transport. If referral is an option then it would seem better to leave the object in place, however from a practical point of view; a horse has to ride in a trailer and this may be potentially damaging as
the horse sways from side to side or front to back. With the trailer ride the object may be pushed farther into the wound or if it is cut at the skin surface to prevent this from happening it may become loose within the chest cavity making retrieval difficult and increasing contamination. If the object is lateral as opposed to a ventral or cranial penetrations, the recommendation would be to remove the penetrating object and pack/wrap the chest so as to seal the wound and provide pressure on any bleeding prior to shipping. The use of clean towels, plastic cling wrap, and vetwrap around the chest is often successful. Individual gauzes should not be placed in to the wound as they may become lost in the chest cavity. The presence of a pneumothorax does not need to be remedied prior to shipping if the horse is not experiencing any marked distress. If the horse is experiencing respiratory distress then a teat cannula can be placed dorsally and caudally and air aspirated using a syringe and three-way stopcock. The horse should be started on analgesics and antimicrobials as well as ensuring the tetanus status is up to date. If the horse is showing signs of shock or marked dehydration then administration of intravenous fluids (for example 5 L of lactated ringers or 1 L of hypertonic saline followed by 5L of LRS) prior to referral can have a significant beneficial effect. The possibility of abdominal involvement should be assessed either through an abdominocentesis or examination of the wound with a thorough knowledge of the location of the diaphragm.

If referral is not an option then these wounds can be successfully addressed in the field if they are simple and do not involve any important structures. Everything including placement of a teat cannula or catheter into the caudodorsal lung field should be prepared before removal of the object. A block using local anesthetic should be placed dorsally and cranially to the wound prior to any wound manipulation. Sedation should be used judiciously so as not to depress the respiratory system. The object should be removed and the wound cleaned quickly of foreign material and any loose bone fragments from the ribs. The deep layers of muscle should be apposed. The lung can then be re-inflated by removing the air through the previously placed access dorsocaudally. A drain from the chest cavity in the distal aspect of the wound can be placed if contamination appears severe. The skin can then be closed completely or partially to facilitate drainage and a bandage applied. The chest cavity can be lavaged if desired. Fluids containing potassium or calcium should not be used when lavaging due to potential irritant effects on the pericardium. Removal of blood within the pleural cavity may aid in preventing the development of pleuritis. Systemic antimicrobials and analgesics should be continued for at least 14 days and re-evaluation of the thorax prior to discontinuing medications should be performed by either careful auscultation, ultrasound examination, or bloodwork. While this approach can work in the field for simple, shallow penetrations it is recommended to refer those cases with deep wounds where closure may be difficult due to maceration of the tissues, significant contamination exists, or the potential for abdominal involvement exists. Horses with chest wounds are painful and can be reluctant to move to water and food so these should be made readily accessible. Relief of pain is important to allow normal chest expansion during breathing and the ability to move to water and feed.

The prognosis after penetrating trauma to the chest is often better than the initial trauma would indicate. With many milder forms of penetrating injury the development of severe pleuropneumonia is not common. This may be due to the ribcage protection preventing deep penetration and contamination. Some deeper injuries however have more serious prognoses and the development of pleuritis or the need for surgical debridement and management of large wounds needs to carefully taken into consideration.
Abdominal Wounds
Penetration of the abdomen is uncommon but when it does occur the need to accurately diagnose and treat the penetration is important. Unlike the thorax (which when penetrated the lung falls away from the penetrating object), the intestines or organs are more likely to be directly traumatized by wounds. If the gastrointestinal tract has been penetrated the horse requires referral and the prognosis is guarded due to food contamination of the abdomen. The penetrating object should be left in place until arrival of the veterinarian and if referral is an option the object should not be removed until arrival at the referral clinic. Careful palpation of the penetrating object to try and ascertain the direction of the object under the skin and ultrasound, if available, can help to follow the object and try to determine if the abdomen has been penetrated. An abdominocentesis should be performed as grossly abnormal or bloody samples yield extremely useful information.

Rectal palpation may also be beneficial if free gas is palpable then the abdomen has been penetrated and/or a viscus. If the object enters into the intestinal tract it should be removed under general anesthesia to minimize contamination and repair the injury immediately if the horse is to have a chance of successful recovery. The abdomen can be surgically lavaged, explored, and chest tubes or closed suction drains placed for continued therapy after surgery. In the case of splenic trauma cardiovascular support or splenectomy can be considered. Laparoscopic exploration and lavage can also be performed. If no internal organs have been penetrated the object can be removed and the wound treated normally along with abdominal lavage with the addition of an abdominal bandage for support. Antimicrobials and analgesics should be maintained for a minimum of two weeks. Due to the high frequency of penetration into the abdomen involving organ trauma the prognosis is guarded for these injuries.

Synovial Structures
Penetration of a wound into a synovial structure such as a joint or tendon sheath is especially crucial as several studies have shown an excellent prognosis with early identification and appropriate treatment whereas a more guarded prognosis with chronicity and delayed diagnosis. This is also the most common form of penetrating injuries as many wounds to the distal limb are close to synovial structures. Diagnosis of a penetrating injury to a synovial structure requires diligent clinical examination and adjunctive diagnostic techniques as penetration is often not readily apparent.

Lameness in joint infections will start 3-7 days after the initial contamination so lameness at the time of injury is not necessarily indicative of involvement. As well if the joint or tendon sheath remains open and draining lameness may never become severe. Digital exploration after appropriate aseptic cleaning of the wound with sterile gloves may yield useful information but is not very sensitive unless there is a large, direct hole. Aspiration of joint fluid can clearly establish the presence of contamination but can be difficult if fluid is draining out the wound. A more practical method of establishing communication is to distend the synovial structure and observe the wound for fluid. The site of needle entry must be distant to the injury and performed in a sterile fashion. If joint fluid can be obtain it can be kept for analysis and culture and then the joint should be distended with saline (or lactated Ringers) and the wound observed for any fluid exiting from the joint. Twenty to 60 mls of saline can be used depending on the size of the joint. If the joint is open and draining it is not uncommon to be unable to obtain joint fluid and distension of the joint is an invaluable tool in the field or the hospital to confirm penetration.
Occasionally there will be excessive swelling or cellulitis already present at the time of examination which makes access to the underlying synovial structure difficult. The practitioner can decide if they wish to attempt a joint aspirate or wait 24-48 hours for the swelling to subside while treating with local wound care and systemic antibiotics. If a sample is obtained a culture should be submitted and analysis performed. The chances of obtaining a positive culture are higher when the sample is placed in a blood culture vial. Analysis of joint fluid for total protein, cell count and percentage of neutrophils can provide valuable information – however if the sample is taken very early after penetration the total number of cells may not have had sufficient time to increase. Therefore a shift to a high percentage of neutrophils regardless of the number of cells should be interpreted as highly suspicious. In joint fluid protein >2.5 gm/dl, cell count >20-30000 cells/ul and more than 80% neutrophils is confirmation of infection.

Radiographs have been shown to be an effective method of diagnosis with gas or soft tissue defects potentially able to confirm penetration. However normal radiographs do not rule out penetration. Digital radiographs if available are most helpful as they can be interpreted immediately. The use of contrast or placement of a probe can help make the radiographs more diagnostic and these techniques are most helpful when distension of the joint with saline cannot be performed (such as with a large laceration over the joint surface) Probe placement or injection of contrast into the wound should only be performed after sterile preparation of the wound. Radiographs also help to rule out any underlying bone or joint pathology that may have occurred as a direct result of the trauma. Diagnosis of synovial structure involvement after penetrating wounds to the foot is more challenging as access to the navicular bursa and coffin joint can be more complicated. If radiographs can be obtained before removal of the penetrating object this is the optimal method of diagnosis. Referal for MRI of the foot is often the most effective and economic method of diagnosis.

If there is any doubt about synovial involvement, and referral is an option, it is recommended to refer to facilitate early diagnosis and treatment. Treatment of penetrating wounds includes treatment of both the wound itself and the synovial structure. Systemic medication should consist of broad spectrum antimicrobials and anti-inflammatories and for 7-10 days. While treatment at the farm is possible with early, minimally contaminated wounds referral is recommended in all cases where economics permit. The prognosis for resolution of infection from wounds is very good when treatment is initiated early. Treatment at the farm should consist of systemic antimicrobials and local antimicrobial therapy following needle lavage if possible. Treatment at the referral hospital consists of arthroscopic lavage and debridement, intravenous medications and intra-articular medication delivered through a variety of methods such as a constant rate infusion catheter, regional perfusions or direct intra-synovial injection. The prognosis after effective treatment is often 80% of higher for full return to function. Wounds of the foot and tendon sheaths may be slightly lower and certainly chronicity and resistant bacteria on culture will diminish the prognosis significantly.

Suggested Readings:
Wounds in horses are relatively common especially in the distal limbs but they can also occur around the head. In the distal limb the main concerns for an examining veterinarian are the potential involvement of underlying synovial structures or flexor tendons and in the long run, limiting any excessive granulation tissue. With wounds of the head the principles of wound management are the same but there are differences in healing and potential involvement of underlying structures. There is excellent blood supply in the head and this means that wounds tend to heal well and infection is combated more readily than in the lower limb of a horse. Wounds in this location are not prone to excessive granulation tissue. However the head also contains many important structures that are only protected by a thin layer of skin. There is very little protective muscle or fascia under the skin. Involvement of the eyes, nerves, sinuses, teeth and general trauma to the respiratory passages can be critical.

Initial examination should involve the whole horse and assessment of the wound with the underlying structures in mind. Involvement of the eye, the sinus, skull fractures, trauma to the nerves or respiratory compromise and dental involvement should all be considered. A general examination should always be performed before addressing the wound to ensure there are no other less obvious traumas or problems. Additionally this allows the veterinarian to assess the mentation of the horse prior to sedation and addressing the wound. Changes in mentation are important to note as a reflection of the potential for trauma to the brain- either directly or by swelling and edema. With head wounds a close examination should be performed of the symmetry of the head (evaluate for nerve damage), palpation of the column of air from each nostril, and an ocular exam. An oral exam may be needed as well. After ascertaining if there is any underlying involvement the wound can be approached as for any wound. Lavage and debridement of necrotic or severely contaminated tissue can be performed with sedation and local anesthesia injected around the wound edges. Care must be taken to prevent any fluids from entering into the eyes particularly any antiseptics used for scrubs (usually recommend cleaning with very dilute betadine and saline rather than chlorhexidine and alcohol as this is better should any fluids enter the eye). The blood supply is very good to the soft tissue of the head so healing is usually quick and infection can be combated readily. Ventral drainage of wounds can be difficult but should be attempted to be established in all cases- this may include drainage through the sinus itself. Clostridal myositis has been reported in the masseter muscle after blunt trauma and wounds in this area should not be completely closed. Leaving a portion of the wound open to heal by second intention, the placement of drains and/or releasing incisions can all accomplish good drainage and minimize the risk of trapping some infection inside deeper tissue. Primary repair of the majority of the wound will help minimize healing time and can be used to cover open sinus fractures after sinus lavage.

With most head wounds the use of anti-inflammatory medications are recommended to aid in analgesia and reduce swelling. The use of antibiotics depends on the wound and the clinical judgement of the attending veterinarian. If good ventral drainage is established during repair and there are no underlying pockets which could accumulate infection then systemic antibiotics may not be warranted. Extensive muscle damage, ocular or sinus involvement would be an indication for a course of broad spectrum antibiotics with topical ocular antibiotics if necessary. The tetanus vaccination status should, of course, be checked and updated if necessary.
Generally head wounds are left unbandaged as it may be difficult to place a bandage without the potential to slip into the eyes or over the nostrils. For wounds with lots of drainage the bandage may also act to hold the discharge against the wounds causing maceration of the tissue edges. However for some wounds such as those that are repaired with a flap of skin over an open sinus a bandage may be useful in preventing subcutaneous accumulation of air. A mesh stockinette can be helpful with holes cut for eyes and ears or there are commercial masks available to protect the eyes which should be used for ocular trauma.

Ocular involvement is normally readily apparent and the more difficult part of the exam involves the decision on how extensive the damage is and whether or not the eye will heal or should be removed as part of the laceration repair. If the cornea is severely lacerated with loss of intraocular components then removal as part of the laceration repair is indicated. Occasionally severe swelling of the eyelids can make identification of the exact structures involved difficult or impossible. If doubt is present then the eye should be preserved and treated with topical antibiotics, ice packs, and systemic anti-inflammatories and re-evaluated in a few days time. If the orbital rim is fractured then fragments (or foreign bodies) may need to be removed or elevated to restore the integrity of the orbit and referral may be recommended. CT scan can be invaluable to assess the bony orbit and thus referral is recommended if the eye appears affected or sunken.

Tachypnea or increased respiratory effort indicates potential respiratory compromise and should be examined and addressed before the wound. Tachypnea may be simply due to pain and distress but may also indicate trauma directly to the airway or indirectly through soft tissue swelling of adjacent structures. Medications such as NSAIDS and steroids can be administered intravenously to help quickly reduce swelling in cases of severe nasal or pharyngeal edema. Acute administration of steroids at an anti-inflammatory dose does not appear to adversely affect healing of the primary wound. In horses with respiratory compromise sedation should only be used if necessary and in small doses so as not to compromise the respiratory system any further. A temporary tracheotomy should be placed if there is any concern about the horses’ ability to breathe. This will reduce the anxiety and calm the horse as well as ensuring a patent airway throughout diagnostics and the initial few days of healing. The horse should be examined for signs of trauma to the proximal larynx, trachea, and esophagus (eg.subcutaneous emphysema, ptyalism) if the wound involves this area. This will require endoscopy and radiographs so referral is recommended after establishing a patent airway if these diagnostics are not available. Rupture or even blunt trauma to these structures can result in infection and can be difficult to heal successfully without intensive management.

Neurological deficits can be observed as specific cranial nerve deficits directly from the wound and/or signs of intracranial edema or hemorrhage from trauma. Abnormal mentation is a sign of intracranial swelling and commonly presents as marked depression or potential recumbency and seizures. Therapy includes large doses of anti-inflammatories (NSAIDS and steroids), intravenous fluids and mannitol or hypertonic saline to reduce swelling. Specific nerve deficits also warrant treatment with anti-inflammatories although if the nerve is obviously severed then the utility of this is doubtful. The prognosis for recovery with an obviously severed nerve will be poor; however most of the cranial nerve deficits with head wounds can be secondary to swelling and improve with time. An electromyelogram can be performed in the longer term if necessary to try and evaluate the nerve more fully. Signs of facial nerve trauma are the most common with deviation of the upper lip and
nostrils. Horses with severe depression and abnormal central nervous system signs may be referred for more diagnostic tests (CT scan or MRI) and intensive management. The prognosis will depend on the level of damage.

Any significant trauma to the head can fracture the bones and enter into the sinuses as there is little soft tissue covering to absorb the energy of an impact. Severe bleeding can sometimes be encountered with trauma to the frontal sinus and ethmoid region. Bilateral epistaxis can be remarkable but is usually self-limiting. The horse may require some intravenous fluids if bleeding was severe. The sinus should be lavaged and loose pieces of bone fragments removed. Lavage can be performed with any balanced electrolyte solution. Additives are not recommended as they can be irritating to the nasal mucosa. If skin can be closed over defects into the sinus it should be performed as this is beneficial even if there are areas of underlying bone loss. Free, denuded pieces of bone should be removed as these may form sequestra. Mild displacement does not necessarily need to be corrected. If there are severe depression fractures, then the bone can be elevated back into position and left to heal. Often the bone does not need fixation but if necessary it can be fixed in place using wire sutures through the bony fragment and adjoining bone or through regular sutures in the surrounding soft tissue. Sinus fistulas can result if the hole into the sinus cannot be closed and so every attempt to close the wound should be made including making skin flaps from adjoining areas. These can be performed as part of the initial repair or as a secondary closure after some healing of the original injury has taken place. Once a sinus fistula has formed the skin around the edges tends to grow down the defect in the bone instead of across. This tissue must be elevated and given sharp edges to help fill in the defect and heal across the space or a transposed skin and periosteal flap can be created.

Fractures of the rostral maxilla and mandible are more commonly treated with a surgical approach than fractures into the sinuses. Fractures of the caudal mandible or head will be treated in varying ways depending on the configuration and location- for example fractures involving the orbit often need to be reduced while non-articular fractures of the vertical ramus of the mandible may or may not require repair. Fractures of the rostral maxilla and mandible are often open fractures with disruption of skin and/or oral mucosa. Despite this they commonly heal very well after surgical stabilisation with few exceptions. When tooth roots are exposed the teeth will be left in place and removed later if necessary as a surprising number do not require removal. Common methods of repair for rostral fractures include intra-oral wiring, external fixators and small plates. Stabilization results in rapid return to eating and comfort.

In summary head wounds are treated in a similar fashion to wounds in any other area but have the potential for underlying involvement of structures specific to the head that can result in the need for increased emergency treatment (fluids, tracheotomy, enucleation) or referral for more intensive diagnostics and care. After repair horses should be monitored closely for several days to ensure they are able to eat, drink and breath normally. These concepts will be demonstrated in this lecture through a series of cases addressing each problem or combinations of multiple issues.
Causes of Nasal Discharge in Geriatric Horses
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The diagnosis and treatment of nasal discharge in horses is a common problem presented to equine veterinarians. The diagnostics that can be done in the field are very efficient but somewhat limited and thus response to treatment can be used as a diagnostic test. The list of potential differential diagnoses changes slightly with age and this helps the veterinarian rule in or out the common problems before considering advanced diagnostics. Lower airway disease can cause a unilateral nasal discharge (although bilateral nasal discharge is more common) and needs to be ruled out in all ages of animals with a accurate history, physical exam and good thoracic auscultation (plus/minus ultrasound and bloodwork). However, this proceedings paper will focus more on the upper airways causes of unilateral nasal discharge.

Clinical examination of the horse with nasal discharge begins an accurate history. The length of time the horse has had the discharge, the age, the time of year, if the discharge is always on the same side, the quality of the discharge, if there is any exercise intolerance, coughing, other horses affected, problems eating, change in feed, trouble with the bit, history of trauma, medications given, and other relevant information such as time of the year and travel history play into formulating an initial differential diagnosis list. All these historical facts provide clues as to the eventual diagnosis. The clinical examination should include careful examination of the external features of the head. The presence of asymmetry or ocular changes such as ocular discharge, exophthalmos or endophthalmos may reflect changes on the inside of the sinus. The cranial nerve functions and presence or absence of muscular atrophy should also be noted. Percussion of the sinuses is not particularly sensitive but may be supportive evidence of fluid or a mass in the sinus. Placing a hand in front of each nostril can detect differences in airflow if a nasal or sinus tumour is obstructing normal airflow. An oral exam is always a critical part of the examination of a horse with nasal discharge. Any abnormalities in the mouth such as fractured teeth or large mucosal pockets may point to an infection originating in the oral cavity. Older, geriatric horses, in particular, are prone to significant dental issues.

Unfortunately, the quality of the nasal discharge does not greatly help in distinguishing between different etiologies. The exception to this is bloody discharge which is commonly seen with trauma, guttural pouch mycosis or ethmoid hematomas. With trauma to the head and sinuses it is surprisingly common to have little external signs or history of an observed trauma. Fracture of the bones may or may not be found on careful palpation. The amount of blood exiting by the nose can be impressive to the owner but is normally not life threatening. Historically the horse may have one episode of severe bleed and then several days of light drainage becoming more serosanguinous as a sinus filled with blood drains slowly out the nose. Trauma to the head without fractures generally only requires supportive treatment unless severe neurological deficits are present. Fractures of the bones can also be treated medically if the bone is not severely displaced. Displaced fractures over the sinuses or fractures involving the mandible and maxilla should be referred for surgical treatment to improve the comfort and function of the animal. Ethmoid hematomas are a benign growth in the ethmoid region into either the nasal passage or sinus. The discharge is commonly small in quantity, chronic and bloody. Often the owner will report the bloody discharge occurs after exercise or turnout. Ethmoid hematomas are diagnosed with either endoscopy or sinoscopy or CT scan of the head and can be treated in a variety of different methods. There is a risk of recurrence after treatment of approximately 40-50%. Treatment modalities include surgical excision, laser excision or
ablation, or repeated injections of formalin. The best choice depends on the location and ease of access as well as the facilities or instruments available. Guttural pouch mycosis often results in one severe bleed which the horse commonly survives and then subsequent bleeds which may be fatal. The mycosis causes the bleeding by eroding through the wall of one of the major vessels in the guttural pouch- internal or external carotid or maxillary arteries. Treatment should consist of stabilizing the horse as necessary and referral to a surgical facility for ligation or embolization of the affected arteries. The prognosis is good although the condition is serious and can be fatal before surgery or have surgical complications due to variation in neurovascular structures.

Nasal discharge of a purulent nature can be caused by many different problems. Purulent discharge may be caused by primary sinusitis, foreign bodies (grass awns), fungal rhinitis, tooth root abscesses, sinus cysts, or nasal or sinus tumours. Sinus cysts and congenital tumours are more common in very young foals while cysts, tumours and tooth problems are common in geriatric horses. Even in older horses care should be taken to ensure there is no risk of strangles being implicated in the presence of purulent nasal discharge as an initial step. Foul odors may be associated with tooth root abscesses or necrotic tissue surrounding a tumour. Most nasal cysts and tumours have a purulent discharge associated with them as they are surrounded by infected granulation tissue and prevent normal drainage. A good oral exam may help identify tooth problems such as fractures but especially in geriatric horses it may be difficult to pinpoint the tooth causing the problem. Therefore the question of how to differentiate between the very different potential causes is the main challenge facing the practitioner. Radiographs of the head can be successfully obtained in the field but can be difficult to interpret- a soft tissue mass may look identical to a sinus completely filled with pus. Tooth roots also require significant change to appear abnormal on radiographs so they are not very sensitive for teeth. However the radiographs can help identify sinus involvement, fluid lines or tumours that are bone or tooth densities (tooth origin tumours or osteosarcomas, ossifying fibromas). Deformation of the head is also more common with either sinus cysts or tumours than primary sinusitis or even tooth root abscesses.

Primary sinusitis will often respond to a course of antimicrobials. In this case the response to treatment can be helpful in determining if referral for advanced diagnostics is required or not. For sinusitis a 2-3 week course of antimicrobials will often resolve the problem. If however, there is an underlying problem, the discharge may resolve while the horse is on antibiotics but will often return soon after the antimicrobial therapy is stopped. If the discharge fails to fully resolve then further diagnostics should be performed. These often include trephination, sinoscopy, radiographs, endoscopy, biopsy or CT scan. Trephination and sinoscopy may allow direct visualization of the structures in the sinus, tooth roots and/or biopsy or any masses. CT scan is also very sensitive for detection of tooth root problems particularly when it is difficult to distinguish which of two adjacent teeth are the cause of the problem.

Treatment of the problem and the prognosis varies greatly. Tooth root problems are best addressed with oral extraction and flushing of the sinus. Teeth may need to be repelled from the sinus although this is associated with a higher rate of complications (trauma to adjacent teeth, bony fracture). Oral extraction is preferred. A plug is usually placed between the oral cavity and the sinus and a common complication it that this may become prematurely dislodged or leak causing a recurrence of the sinus infection. The sinus may need prolonged lavage if contamination is severe. The horse will also require more diligent oral care after removal of a tooth to ensure the opposite tooth does not over
grow. However, despite the complications the prognosis for the horse is very good. Older horses can also have intermittent sinusitis as the teeth grow out and may have some intermittent leakage of bacteria around the tooth. This may simply need repeated antibiotic therapy. Cysts in either very young or old horses also have a good prognosis with surgical removal of the cystic lining. The prognosis associated with tumours however varies greatly and thus biopsy is recommended. Most tumours, even benign tumours such as ossifying fibromas, carry a risk of recurrence. Dental origin tumours such as cementomas appear to carry a good prognosis while squamous cell carcinomas, osteosarcomas and adenocarcinomas are more likely to recur in a short time period. The biopsy is recommended to give the owner as much information as possible when deciding on whether to perform surgical removal of the tumour. Aspirates of local lymph nodes and examination of the horse for metastases is recommended. If radiation therapy is available it appears to be a successful adjunctive therapy to prevent recurrence while local chemotherapy is more problematic in the cavity of the sinus. When owners are willing to have adjunctive therapies performed even in older horses the risk of recurrence can be reduced. Radiation therapy in horses appears to be relatively benign with little to no reported negative side effects.

Fungal rhinitis is also recognized to have a risk of recurrence although therapy can be successful. Most commonly therapy is a combination of local debridement and topical antifungal application and systemic antifungal medications. Systemic antifungal medication can be cost-prohibitive in horses although both amphotericin B and oral fluconazole can be given relatively cheaply. The use of amphotericin B has been shown to be effective although carries a significant risk of secondary renal damage. However, it can be used successfully in combination with careful monitoring. Systemic fluconazole is also used in an oral form and while monitoring the bloodwork frequently is recommended no significant side effects have been noted in relatively small numbers of cases. The duration of therapy is long lasting several months and the fungal rhinitis may result in permanent changes to the normal interior architecture of the nose. Successful treatment is possible but owners need to realize the duration necessary and risk of recurrence.

In summary nasal discharge can be a diagnostic challenge for the veterinarian. The complexity of the sinuses make advanced diagnostics necessary when an underlying cause is present. Response to treatment is a valid method of determining if an underlying cause is present to warrant the cost and potential referral for more advanced diagnostic modalities. Although nasal discharge is common to many different problems the history, age and clinical examination of the horse may help to narrow the list of potential diagnoses. Only in the case of bloody nasal discharge is the quality of the nasal discharge itself helpful in determining the cause. Case studies will be used to demonstrate the various etiologies of nasal discharge in older horses and the appropriate treatments.
Lameness in geriatric horses can be due to trauma or the result of normal aging and accumulative wear and tear on articulations, bone, and soft tissues after an athletic life. The perception of treatment of orthopaedic injuries in geriatric horses may be different from young horses. The goal of management depends on the nature of the problem but should always involve considerations of the quality of life and chances of successful resolution rather than solely the consideration of age. The definition of what exactly is the definition of the term geriatric in horses varies slightly from one study to the next but in general is thought of as including horses over the age of 18. There are studies suggesting that the numbers of geriatric horses are increasing likely due to improvements in preventative health- vaccinations, deworming, and nutrition. Management of orthopaedic problems in geriatric horses can be successful with many tools available to the practitioner to improve and extend the quality of life.

**Arthritis**

Lameness arising from arthritis is the most common reason for loss in the horse world. However it can often be successfully managed with a variety of tools. The joint is a complex organ composed of many parts, each of which is integral to the function of the whole. The concept of the joint as a whole is important to the management of arthritis. Cartilage, joint fluid, the joint capsule, ligament and tendons, and subchondral bone all play a role in maintenance of a healthy joint. If one of these parts is not functioning normally than that can lead to dysfunction of the whole organ and a cycle of arthritis from mechanical and biochemical factors. Ligaments and muscles help to support and stabilize the joint. In geriatric horses that have had long periods of rest without exercise the rehabilitation and slow return to exercise are especially important to allow these stabilizing elements to regain function before stress is placed on the system. As well, maintenance of muscle strength in geriatric horses should be considered as part of the long term plan for arthritis management rather than complete rest. Complete box rest can be very difficult in older horses as, while it may be required for the primary problem, the loss of muscle support may make previously insignificant arthritis an important reason for not returning to function.

The subchondral bone lies directly underneath the cartilage and changes in the subchondral bone from overuse or trauma are reflected in changes and degradation of the overlying cartilage. Obviously osteochondral fragmentation requires arthroscopic treatment to remove the fragments that will continue to cause arthritic change if left untreated. However, arthroscopy to remove osteophytes and arthritic changes is not necessarily recommended and may result in more trauma to the joint. Consultation with a local surgeon is recommended if surgery is to be considered as part of the treatment protocol. Appropriate lameness examination and diagnostics of problems involving the bone, joint and tendons should be performed as part of the examination process. Specific diagnoses will greatly help in determining the correct treatment. For example a synovitis from an acute overload will have a different treatment plan than a horse with a chronic pre-existing arthritis. In one the goal is remove the inflammation and return the joint to normal before resuming exercise, while in the other the goal is to prolong the comfortable use of the horse and retard the progression of the arthritis. Treatment of the clinical signs of joint inflammation and lameness without appropriate diagnostics to determine any underlying problems will potentially worsen the long term
outcome for that individual. Physical causes of arthritis require appropriate treatment to stop the formation of arthritis or limit its’ development (fracture fixation, arthroscopy or rest).

Damage to cartilage can be difficult to diagnosis on radiographs and increasingly ultrasound has been used along with MRI. Direct trauma and fragmentation or flaps of cartilage need to be removed arthroscopically and the area debrided to bleeding subchondral bone. This allows the movement of cells and growth factors to the surface for healing. Osteochondral fragments or substantial change in the bone can indicate trauma to the overlying cartilage as well. However, often in geriatric horses those decisions may or may not have been made earlier in life and the results of those decisions or traumas are what the practitioner is left to cope with. Direct healing of cartilage continues to be difficult although there are some surgical techniques such as microfracture of the subchondral bone that can be performed or implantation of stem cells by injection or in a carrier such as collagen glue can be attempted. These techniques are becoming more popular are with future and ongoing studies may prove their efficacy. Currently there is no proof that stem cell therapy in the joint results in a better cartilage healing. They may have a more important role with soft tissue injuries within the joint. So although there is some hope for future methods to restore cartilage these are not widely available at this time.

The joint capsule and synovial fluid function to lubricate the joint as well as participate in the balance between degradation and synthesis of cartilage. Inflammation with the joint fluid not only causes pain but directly increases the production of enzymes that cause the breakdown of the cartilage matrix and fluid. Synovitis alone can be treated and the inflammation resulting from pre-existing arthritis can be treated or managed by attempting to reduce the inflammation. This will reduce the inflammation- associated degradation of fluid and cartilage matrix and/or increase the synthesis of normal joint fluid or cartilage matrix. Increasing the synthesis of normal fluid and thus cartilage matrix can be attempted in the form of treatment with hyalurronon intra-articularly or intravenously. Hyaluronan is a normal component of joint fluid and has many functions including lubrication, steric hinderance of inflammation, and positive feedback to the cartilage and joint capsule. When administered intravenously, orally, or intra-articularly there is evidence that is helps to reduce inflammation and clinical signs of arthritis. The reduction of inflammation and thus degradation can be achieved through the use of polysulfatedglycosaminoglycans (PSGAGs), steroids, nonsteroidal antiinflammatories (NSAIDS) or Interleukin receptor antagonist protein (IRAP). There have been multiple studies on the efficacy and potential drawbacks of various steroids when administered in the joint. All reduce inflammation while it appears that triamcinilone has the least potential for negative side effects although the studies have mixed results. The use of steroids is practical from an economic standpoint as well as effective in improving the clinical signs of arthritis. The use of intramuscular or intra-articular (in combination with an antibiotic) PSGAGs have proven anti-inflammatory effects. Intraarticular injections of autologus conditioned serum or IRAP is also commonly in use these days with proven anti-inflammatory effects. Interleukin-1 is a main promoter of inflammation within the joint and this medication concentrates the natural antagonist using the horses’s own blood. There have been no reported negative side effects although it is more expensive than other medications. This medication may not only alleviate the inflammation but have positive disease modifying effects. In dogs and humans platelet rich plasma is commonly used in the joint to treat arthritis and may have benefits although it is less common in equine practice.
The use of systemic NSAIDS may play a short or long term role in the management of lameness in geriatric horses. If long term use is envisioned then clear guidelines for the owner need to be established so excessive use is avoided. As well monitoring periodically for potential side effects (right dorsal colitis= decreased total protein, softer manure, gastric ulceration or renal disease) should be performed and newer COX-2 selective NSAIDS such as firocoxib considered if side effects are seen or economics permit. Topical NSAIDS in the form of topical diclofenac can also be used to reduce inflammation locally within an articulation without systemic side effects.

Thus, treatment of arthritis in geriatric horses may comprise some of the above interventions either medical or surgical but should also include a longer term plan for the horse. Conformation and shoeing defects should be corrected as these can place abnormal forces on the joints and soft tissues. Long term stall confinement should be avoided if possible and if necessary the return to exercise should be slow and controlled. A realistic plan for the continued athletic future should be discussed including potentially reducing the frequency and intensity of exercise to prolong the useful career.

Arthritis that becomes non-responsive to medical management can be treated with surgical arthrodesis or facilitated ankylosis as if the joint is eliminated by fusion the pain and dysfunction is also eliminated improving the quality of life. In some joints the horse may return to be an athlete-the pastern and lower hock joints- while in other joints- the fetlock and carpus- the goal is to remove pain and improve quality of life with no potential for an athlete. Some joints in adult horses may not be fused surgically and do not have the option for management in this form (stifle, hip, elbow, shoulder). The lower hock joints may be induced to fuse in a variety of different methods from surgical facilitated ankylosis (drilling) to injection of alcohol. These horses can continue to athletic for many years although subtle changes in the function of the entire hock joint may result in arthritis of the upper joints that may eventually limit the length of the career. The pastern joint can be successfully fused surgically resulting in approximately 60% of front limb pastern arthrodeses and 80% of hindlimb affected horses returning to athleticism. Surgical arthrodesis is the most effective method although facilitated fusion through repeated and multiple injections of alcohol has been described but is used only when surgical fusion is not an option. Surgical fusion of the carpus or fetlock is solely for improved comfort or animals used for reproduction. Fusion of the carpometacarpal joint enables horses to return to function but not when the upper two carpal joints are involved.

Other causes of lameness in geriatric horses can also occur with trauma or chronic foot problems being common. Caudal heel pain or navicular disease can be managed in much the same way as arthritis as described above with appropriate shoeing and exercise changes as well as treatment of the navicular bursa or coffin joint with medications. With the advent of MRI knowledge has been gained that indicates there are many soft tissue structures in the foot which may cause the same clinical signs as osteitis of the navicular bone. If economics permit accurate diagnosis of the exact cause of the chronic foot pain will aid in directing therapy from management of pain and continued exercise to periods of rest for soft tissue injuries. A neurectomy can be considered but the owners should be aware that the effects do not last forever and without advanced diagnostics such as MRI then the risk of rupture of the deep digital flexor tendon (which may be the primary lesion) is unknown. Laminitis is more and more being attributed to metabolic disease in the older horses such
as Cushings or metabolic syndrome and all efforts should be made to diagnose and treat those underlying causes in conjunction with pain and foot management.

Trauma causing fractures in older horses do occur and geriatric horses are certainly capable of healing a fracture with appropriate care and surgery if desired. Geriatric horses do heal well if more slowly than a foal and may be more calm with confinement and treatment. However, the prolonged confinement necessary for many fractures (3-4 months) may result in muscle wasting and stiffening of other joints resulting in more difficulty returning to full exercise. Rehabilitation is especially important and due to changes in muscle and other joints from lack of use and confinement the horse may not return to full usage. Case examples will be used to demonstrate fractures that yield a good prognosis in older horses.

Tendon injuries also occur in older horses either through trauma, prolonged overuse or lacerations. While not proven in studies the clinical consensus is that healing of tendons is less good in geriatric horses with more scarring and possible contracture. This would seem to suggest that along with the standard treatments for tendon injuries, early mobilization and biologic therapies to reduce scarring may be indicated in older horses. The use of stem cells and platelet rich plasma locally in torn tendons has early promise for an improved quality of healing and, if economics permit, may be especially useful in older horses. Once contracture has occurred the problem is very difficult to manage successfully and is often recalcitrant to surgical or medical interventions.

In summary geriatric horses can be treated in a similar fashion to younger horses in the diagnosis and treatment of lameness conditions. The most common problems are arthritis, caudal heel pain, laminitis or trauma. Long term maintenance of health and comfort need to be the goals with potential limitations to the frequency and intensity of exercise. Studies have shown that problems in geriatric horses tend to be under-diagnosed and lack appropriate treatment. However, when appropriate care is provided, many geriatric horses can continue to have long, useful, and comfortable lives.

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Angular and Flexural Limb Deformities in foals
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The treatment of angular deformities is a topic of debate. The recognition, accurate diagnosis and treatment options are discussed in this presentation using cases to demonstrate the principles. Angular limb deformities are those that cause the limb to deviate medially or laterally as opposed to flexural deformities that result in hyperextension or flexion of the limb. Angular deformities are divided in varus or valgus deformities. Varus deformities are those with the distal limb deviates inwards and valgus are those where the distal limb deviates outward. Angular and flexural deformities can arise from either congenital or developmental origins and thus diagnosis and treatment can be divided into neonatal foals (the first few weeks of life) and older foals (1-8 months).

Angular deformities in Neonates
Premature or immature foals may have incomplete ossification of the small carpal or tarsal bones. Some foals, particularly miniature foals, have soft tissue laxity which can include laxity of their collateral ligaments and may present with angular deformities. With both of these problems foals present with angular deformities which can be corrected with manual manipulation. The limbs can be straightened but return to their deformity with weight bearing. Therefore, palpation and manipulation of the limbs is an important diagnostic tool. It is also highly recommended to take a sample dorsopalmar and lateral radiograph of a carpus or tarsus as the presence and degree of incomplete ossification has a huge effect on the prognosis and treatment.

With incomplete ossification of the small carpal and tarsal bones the goal of treatment is to maintain limb alignment and reduce weight bearing allowing the bones time to ossify. As these foals are often premature there may be other intensive management required and severely affected foals can be difficult to manage successfully. A mild decrease in ossification can be treated with restriction to a box stall whereas more severe cases require the foal stand as little as possible and with the support of splints or casts to prevent crushing of the incompletely ossified bone. The bones will ossify with time but this can take 1-2 months and exercise must be severely restricted during that entire time. In the front limbs incomplete ossification of the small carpal bones will result in crushing in a medial to lateral direction whereas in the hindlimbs incomplete ossification of the small tarsal bones results in crushing in a cranial direction resulting in a flexed appearance to the tarsus. Application of splints or casts should be placed to maintain the correct straightness of the limb medially to laterally in the forelimb and to prevent flexion in the hindlimb. Splints and casts should start just distal to the elbow/stifle and finish just proximal to the fetlock. Commercial splints are available that can be easier to quickly apply and remove (such as Dynasplints). However, as all four limbs are affected the maintenance of splints can be rotated between limbs and help may be required to stand and nurse frequently. Prognosis is good for mild cases with intensive management but with severe incomplete ossification the chance of success diminishes drastically. In the longer term foals that end up with greater than 30% crushing the prognosis for an athlete will diminish.

The management of foals with laxity of the peri-articular tissues and normal ossification is conservative therapy to strengthen the tissues while reducing any abnormal trauma to the bones. Daily walking for 10 minutes to strengthen the foal and stall rest to reduce excessive exercise is recommended. Occasionally hoof extensions can be applied.
In older foals the deformity can be cause by either disparate growth at the level of the growth plates, crushing from previously unrecognized incomplete ossification or soft tissue laxity. Diagnostic examination once again includes manipulation of the limb to see if it can be made straight as with soft tissue laxity or if the problem persists due to bony abnormalities. Once again radiographs of the worst affected limb are highly recommended as disparate growth at the level of the growth plate and crushed small carpal or tarsal bones carry different treatment options and prognosis. If the carpal or tarsal bones are crushed from a previous problem with incomplete ossification then an assessment of the athletic future needs to be made and if there is any potential for improving the function of the limb. Manipulation of the growth plate is often unrewarding in these cases but occasionally may be of limited use.

Disparate growth at the level of the growth plates is more common in older foals. Disparate growth can be caused by unbalanced nutrition (trace minerals), excessive exercise or overload, or trauma to the growth plate causing closure of one side prematurely. Most commonly affected are the carp II (distal radial growth plate), tarsi (distal tibia growth plate) and fetlocks (distal cannon bone growth plate). Diagnosis requires diligent observation of the limbs. Often rotational deformities are present and so the limbs must be observed from the correct angle to determine the presence of angular deformities. Most common is mild outward rotation of the limb and a toe-out position which resolves slowly as the chest muscles develop. Careful observation of the foal from all angles and also watching the foal walk towards and away from the veterinarian help in accurate assessment of the limb. The time to effect a correction is determined by the amount of time the growth plate remains active. Much of the growth from the distal cannon bone is completed by 3-4 months of age and so treatments for fetlock deformities should be performed in foals 1-2 months of age. The growth from the distal radial growth plate is active up to 6 months of age and so treatments for carpal deformities should be treated at 3-4 months of age. Tarsal deformities should be treated around the same period with growth in the distal tibia being present rapidly up until 4 months and slower after that time. Commonly conservative therapies (including periosteal stripping) are used during the rapid growth phase and surgical intervention to cause growth retardation is used at the end of the rapid growth phase after conservative management has failed. The difficulty in managing angular deformities is deciding when and if the deformity needs to be treated. Some degree of angular deformity is normal in foals.

Conservative management consists of a variety of options but all should include restriction of exercise and hoof trimming or application of extensions. These two treatments alone will resolve the majority of deformities. Other conservative options include periosteal stripping or shockwave. Periosteal stripping is often included as a conservative management as it is minimally invasive with few risks as over correction does not occur. Based on recent experimental work showing that trauma or inflammation in the periosteum proximal to the growth plate causes feedback to the growth plate to stimulate growth some practitioners are trying multiple pricks with a needle in the periosteum. This technique is economic but unproven. Shockwave is another unproven technique based on a clinical study in England that recorded some improvement when the convex side of the growth plate was treated with shockwave. Again this treatment is not evidence based at this time. Surgical Correction: There are several accepted methods of retarding growth with varying configuration of implants. It is very important that these implants be removed before overcorrection- repeated examinations and radiographs may be required and the implants should be removed before the limb is straight as some correction will continue after removal.
There are a variety of other angular deformities. Windswept foals are those that are born with a combination of angular deviations in the hind limbs with one limb being valgus and the other varus. This is commonly due to ligamentous laxity and not a problem with bony growth. Treatment consists of conservative management- hoof extensions and restricted exercise. Radiographs should be obtained to ensure complete ossification of the cuboidal bones. If the foal is able to stand and nurse, the problem will commonly improve drastically in the first month of life. However, if the foal has trouble standing and nursing the chance of secondary complications such as sepsis are high. Miniature foals often have angular limb deformities. Most can be managed by hoof extensions and occasionally surgical treatment if required. Because there is no need for these foals to be athletic some degree of deformity can be acceptable. However, dwarf miniature foals can have serious problems with ligamentous laxity causing subluxation of the articulations. These may be managed again with supportive hoof care and occasionally arthrodesis of severely affected joints. However, owner counseling needs to be an important part of care as these may have other deformities such as deviated nasal septums.

Angular deformities are to some degree normal in foals from birth to several months of age. The difficulty for the veterinarian is to determine which foals fall outside the normal range of deviations and need some intervention in the form of conservative or surgical management.

Flexural limb deformities are a separate problem that can occur by itself or in combination with angular deformities. Flexural deformities can occur either as a laxity that causes hyperextension in the limb or a “contracture’ that causes a flexion of the limb. It is important to note that except in adult horses after an injury contracture is not an accurate word as the soft tissues have not contracted- they have just not grown or formed as long as the bones resulting in a relative shortness. Pain is likely a large part of the etiology in acquired deformities and physitis is commonly present as well.

Flexural deformities can be present at birth and result from a variety of fetal factors such as intra-uterine positioning, fetal insults (toxins, maternal sickness, placentitis) or maturity at delivery. The exact etiology of these problems is not entirely understood. Mild laxity is very common in the first few days of life and most foals improve without therapy in first few days of life. The laxity can involve one or both of the flexor tendons and can involve both the front and hind limbs. Some neonatal foals have a more severe laxity which does not resolve as quickly or is severe enough to cause injury during the time required for resolution. For these cases the treatment for the laxity consists of physical therapy in the form of controlled exercise. Small paddock turnout on grass or stall rest with daily walking is recommended. The foal can injure skin on palmar/plantar surface of fetlocks or heel bulbs. With these foals, along with the controlled exercise and turnout, one may need to protect the skin with very light bandage covering. Heavy bandages will increase the laxity so light, small bandages may be used with care during exercise. Addition of glue-on shoes with heel extensions can be a very useful adjunctive therapy. Any shoes applied to the hooves of foals should be changed frequently and removed as soon as possible as they can constrict the normal hoof growth.

Neonates may be born with the opposite problem and have hyperflexion deformities. This is more common in the front limbs either at the level of the fetlock or carpus or both. The foal is unable to
extend the limbs and severity can vary from mild to that which interferes with the ability to stand and nurse appropriately. There are a variety of treatments depending on the severity. The most common treatment is bandaging with or without intravenous high dose oxytetracycline administration. For a foal of normal size and in good health a dose of oxytetracycline can be given and can be repeated once every other day. The mechanism of action of oxytetracycline is not fully elucidated but it affects the internal structure of the ligaments and tendons so they are more easily elongated during normal weight bearing. It does have significant potential for kidney damage and is contraindicated in dehydrated foals. Foals should have been observed to nurse and urinate normally before use. As well, oxytetracycline should ideally be diluted into small amount (500-1000mls) of fluids as it is locally irritating to vein in a concentrated form. If repeated doses are desired the creatinine should be monitored. The improvement will be seen in the first 24-48 hours after administration.

Bandages in young foals result in rapid relaxation of soft tissues and can be readily applied. Bandages should be changed daily and monitoring for any potential bandage related problems must be diligent. Occasionally the foal may need to wear splints to fully resolve the contracture. In many cases referral to a hospital for monitoring during this period is recommended unless the care at the farm is constant. Splints should be applied for short intervals and then removed – for example: 12 hours on and 12 hours off. Splints are potentially dangerous if not monitored closely. Wounds caused by pressure of the splint/bandage are common and fractures a potential concern if the splint slips into the wrong place. Splints can be placed on the palmar aspect of the limb. If the hyperflexion is localized to the fetlock and below, the splint can be placed from just below the carpus to the ground with extra padding behind the pastern. Splints are placed on top of well padded bandages. If the hyperflexion is localized to the carpus, the splint needs to be placed on the palmar aspect of the limb from just below the elbow to just proximal to the fetlock. The bandages and splint should not include the fetlock and below if the fetlock is unaffected as this will cause unwanted laxity. Splints should not start or end in the middle of a long bone to minimize the risk of the foal breaking its leg. The process of elongating the soft tissues is a painful process and so foals will benefit from analgesics and this will facilitate correction. If the problem is severe then the foal may require intensive care to support feeding and prevent infection and may succumb to secondary problems such as sepsis so referral is recommended. Surgery is not commonly used for neonatal flexural deformities as it has not been effective. If the problem is severe at birth (carpi are flexed 90 degrees without any ability to extend further manually) then the prognosis is not good and euthanasia may be an option. For most mild contractures the prognosis is good with early management and bandaging.

Flexural deformities in young foals are acquired as a result of developmental factors- likely a mix of genetics and nutritional factors including trace minerals and energy. Acquired deformities are in the form of a ‘contracted’ flexural deformity and may be as the bones grow at a faster pace than the soft tissues or as pain from physisis or growth causes reduced weight bearing. Pain is a significant contributor to the etiology and must be addressed as part of the solution. Ideally these deformities need to be recognized and treated early to facilitate response to conservative medical therapy and prevent the need for surgery. There are two common forms: ‘club feet’ (flexural deformity of the distal interphalangeal joint with involvement of the deep digital flexor tendon (DDF)) and an upright fetlock confirmation or dorsally knuckling at the fetlock and pastern (flexural deformity of the fetlock and implication of the superficial digital flexor tendon (SDF)).
Deformity of the distal interphalangeal joint: The initial treatment consists of decreasing energy intake and slowing growth by eliminating grain from the ration or removing from high quality pasture. Exercise is important to continue to encourage proper function and joint angulation but excessive exercise may cause pain. Therefore, an exercise or turnout regime should be formulated based on the individual horses’ temperament and facilities available. Hoof trimming is somewhat controversial as some advocate lowering of the heels and extension of the toe through sequential trimming to try and return to a more normal configuration while others advocate a mild elevation of the heel to relax the pull of the DDFT on the third phalanx and then slowly returning to normal as the condition resolves. Simple sequential trimming of the heel as a sole treatment is not recommended as it reduces the weight bearing surface and causes more rapid wearing of the toe so should only be combined with a toe extension. When a toe extension is applied it should be filled or braced against the dorsal hoof wall to prevent frequent stumbling and stress causing flaring of the dorsal hoof wall. An important part of therapy is the need to maintain comfort during treatment so the foal will bear full weight and exercise in a controlled fashion. If the deformity fails to respond fully then surgical treatment options can be pursued. These consist of a surgical tenotomy of the check ligament of the DDF. This functionally lengthens the tendon. Cutting of the distal check ligament does not affect the foals’ future athletic ability. In severe cases a tenotomy of the DDF itself can be considered. This will correct the problem but should be considered more of a salvage procedure. Some of these horses may become able to be used as riding horses but the number able to do that is unknown.

Deformity of the fetlock: Initial treatment again consists of decreasing energy intake and slowing growth as before. With mild flexural deformity of the fetlock the foot should be trimmed to encourage break over and the heel may be slightly raised to allow laxity in the SDF and encourage the fetlock to extend. Controlled exercise with appropriate pain management is a crucial part of treatment. For more severely affected foals consideration should be given to the use of splints and/or surgery. Surgery consists of tenotomy of the superior check ligament to functionally lengthen the SDF tendon. If surgery is not followed by rigid adherence to pain management and exercise then the condition may return. Occasionally the distal check ligament may also be cut if it is felt to be contributing to the problem and the deformity is more severe.

In severe cases the flexor tendons themselves may be cut but this is a salvage procedure and is not commonly necessary. The prognosis for successful resolution if the horse is permanently knuckled forward at the fetlock is not good and care should be taken to address the problem aggressively before that point.

Carpal hyperflexion can occur in fast growing foals and can usually be treated by reduced exercise and pain management. Most foals resolve without further therapy but occasionally a period of time in bandages or splints to encourage increased laxity may be needed. Pain management is again crucial to allowing the foal to exercise normally and prevent further contracture due to pain.

In summary flexural deformities can occur in both neonates and older foals in the form of laxity or hyperflexion. Most of these problems can be resolved with medical therapy while surgery may play a role in older foals with flexural deformities. Tendon laxity generally carries a good prognosis but with
hyperflexion the prognosis varies from excellent for mild problems to guarded for more severe affected foals.
Management of Septic Joints in Foals
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Septic joints in foals are a problem that every practitioner deals with for both large and small breeding operations. The region affected may include just the joint, the joint and adjacent bone or growth plate, or just the growth plate. Unless a wound has been incurred these infections are the result of bacteraemia. It has been shown recently that most foals have transient bacteremia at some point. Growth plates and joints appear to be predisposed sites for the infection to occur after bacteremia. The underlying cause needs to be identified and treated in foals (unlike in septic joints in adults which is commonly from a wound or iatrogenic). Additionally early identification of a septic joint and/or adjacent bone is key to successful treatment.

Some septic joints in foals may be successfully managed on the farm while others may require referral depending on the severity of the infection, the facilities and management on the farm, as well as the time constraints of the veterinarian.

Clinical presentation
Clinical signs of a septic joint in foals include effusion, edema and pain on palpation, lameness or stiffness, and reluctance to stand or increased time lying down (as many foals may have more than one joint affected they may not show overt one limb lameness). Lameness in foals can be difficult to identify and more commonly the foal is noted to spend more time recumbent or be more depressed than normal by the owner. Presentation with an overt lameness in one limb is less common and so examination of any depressed or increasingly recumbent foal should be focussed on identification of any septic structure. Foals in the first few months of life are vulnerable to septic joints or growth plates. Careful palpation of all joints should be performed as there is commonly more than one joint affected. Palpation will enable the vet to distinguish between infections of the joint versus infection of the growth plate. Infection in the joint will present with effusion whereas infection of the growth plate presents with swelling and edema as well as pain on palpation but not a significant effusion within the joint itself. Differentiation can be difficult but often a high degree of clinical suspicion will be present after palpation and then can be confirmed with further diagnostics. If both are affected usually the joint effusion will be more obvious. Ultrasound can be used to see if fluid is present in the joint or around the growth plate. Once a joint infection is suspected then an optimal work-up would include a radiograph to obtain a baseline view of the bones and growth plate and an aspiration of the joint fluid for cytology and culture of any suspicious joints. Radiographic signs of infection will lag behind clinical infection so baseline radiographs should be repeated in approximately one weeks time if clinical response to treatment has been slower than expected. Radiographs are relatively insensitive for detection of bone infection unless large changes are present. Ct scans of foals with septic joints is invaluable in early detection of accompanying osteomyelitis. Knowledge of the normal appearance of the joints in neonatal foals is important as in some joints (eg. stifle) the bony surfaces are normally irregular. Infection will show up as lysis and good quality radiographs are essential. Samples of joint fluid and/or fluid from the growth plate should be submitted for culture. Samples of joint fluid are obtained after a sterile prep and aspirate of joint fluid. This can be done using an 18-gauge needle. If the infection is suspected to involve the growth plate then often a culture can be obtained by placing an 18 gauge needle into the growth plate and aspirating. Sometimes purulent fluid or blood can be obtained which may be cultured. Recent work has revealed the chance of obtaining a positive culture may be enhanced by placing the
joint fluid into haemoculture vials. Obtaining some joint fluid and placing in a purple top EDTA tube and submission for cytology will reveal if infection is present if the joint fluid is not grossly abnormal. In adults total protein is normally less than 2.5 gm/dl and cells less than 500/dL with infection being present if cells are mostly neutrophils and greater than 20-30000 cells/dL. However in foals the response to infection may be less well developed so cell counts greater than 5,000 cells/dL is strongly suspicious of infection and the shift to greater than 80-90% neutrophils is significant even in low cell counts. Routine bloodwork such as a complete blood count and fibrinogen should be routinely performed along with a SNAP test for diagnosis of failure of passive transfer if applicable. Changes in the complete blood count and fibrinogen can aid in the diagnosis of concurrent disease and in response to therapy. One study has found that fibrinogen can aid in the differentiation of joint versus physeal infection with bone infection being more common with a fibrinogen over 800mg/dl and joint infection alone being seen with fibrinogen levels under 800 mg/dl. Caution should be used in interpretation of these studies with the clinical impression and judgement always being foremost.

Examination for other foci of infection, including palpation and ultrasound of the umbilicus, careful auscultation of the lungs and taking a blood culture (as well as cultures of the joint fluid), is recommended due to the bacteraemic origin of the problem. Some foals are no longer bacteraemic at the time the joint infections are recognized and have no other signs of infection but a full examination should always be performed as concurrent diseases may affect the prognosis in a negative fashion compared to simple joint infection(s). The most common concurrent diseases are those affecting the umbilical structures, pneumonia, and diarrhea.

Common bacteria that cause joint infections in foals differ from those in adults due to the different etiology. Bacteria such as streptococcus, salmonella, E coli and Enterobacter are more common than in adults and bacteria such as salmonella have been associated with a negative prognosis. The most common joints affected in multiple studies are the tarsocrural joints and stifles. All other joints can be affected including more difficult to diagnose joints such as the coffin joint and hip joint.

**Referral or treatment at home?**

All foals with the suspicion of a septic joint are excellent candidates for referral as the ability to provide aggressive care is easier at a hospital. However, some early infections in otherwise healthy foals may be treated at the farm if economics or circumstances dictate. If the foal is systemically sick and requiring intensive care for concurrent systemic problems such as diarrhea or pneumonia then referral should be strongly considered. Referral earlier in the disease process will save on expense in the long term. If the infection involves both the joint and adjacent bone (epiphysis) then referral is recommended to allow surgical debridement of the bone along with joint lavage which can be performed arthroscopically and needs to be performed in all cases even acute infections. If the source of the lameness cannot be definitively diagnosed because an uncommon or difficult to access joint (eg hip) is involved then referral is recommended. If the foal appears to be otherwise healthy or with a mild umbilical infection/patent urachus only then some joint infections can be successfully treated at the farm with a combination of systemic and intra-articular antibiotics and joint lavages. Infection involving the growth plate is commonly treated using regional limb perfusions or direct antibiotics injection which can be performed at the farm but may need surgical debridement if they fail to respond. Generally mild joint sepsis and/or growth plate infection will respond quickly with rapid resolution of clinical signs- if the foal fails to improve clinically after the first few days of
intensive local and systemic therapy then referral is recommended for more aggressive treatment. Failure to respond can be from a number of different sources; a resistant bacteria, bone involvement, or ineffective lavages of the joint.

**Treatment**

All foals should be started on systemic and local antibiotic therapy regardless if concurrent disease is present or not. Selection of antibiotics should be based on knowledge of the common bacterial isolates as well as what is feasible on the farm. However, in general broad spectrum antimicrobials until culture results are available are recommended. One of the most common isolates is E Coli or other gram negative bacteria unlike adults. Optimal antibiotics would include a combination of a penicillin or cephalosporin combined with an aminoglycoside. For example, ceftiofur could be given systemically and amikacin given intra-articularly if the infection is confined to the joint, or ceftiofur and gentamicin systemically and amikacin locally. Other choices include trimethoprim-sulfa, oxytetracycline or others based on culture results. Some doses of antibiotics vary depending on the age of the foal so it is best to consult with your local referral center or medicine specialist if possible. Foals are physiologically distinct during the first few months of life and so pharmacokinetics are different than adults. For example, the dose of ceftiofur can range from 5-10 mg/kg twice to four times daily. Common and practical is a twice daily dose of 5 mg/kg given intravenously or intramuscularly. In a referral setting, if the foal is systemically sick, then serial monitoring of blood results is required to ensure the ongoing health of the kidneys.

If the joint fluid is grossly abnormal a lavage and intra-articular injection of antibiotics can be performed at the same time as the first joint aspiration. All joint aspirations and lavages should be performed in a sterile manner which require the foal to be still and restrained. With sick foals this may be easier than with foals that are no longer systemically ill. Sedation and restraint are commonly sufficient, anesthesia is not normally required. Common recommendations for sedation of neonatal foals include using acepromazine and butorphanol intravenous injections or diazepam. If alpha-2 agonists such as xylazine are used the dose should be extremely low as foals are more susceptible to the cardiorespiratory depression and are less capable of metabolizing the drugs. Doses should be based on the size and age of the foal as well as the behaviour and ability to physical restrain them. With young foals the Madigan squeeze technique is often sufficient to provide lateral recumbency and restraint which can then be combined with a small amount of local anaesthetic for joint lavages. Lavage can be performed using through and through needle lavage. Along with sedation some people recommend instilling 5-10 ml of lidocaine into the joint and then waiting 5-10 minutes before performing the lavage. This may not be necessary and may prolong the time of sedation but may be considered if a foal appears particularly reactive. Needle size should not be too small. Commonly 18-gauge needles can be used but the larger the better so 14 or 16 gauge needles are optimal and a minimum of two should be used. Efforts should be made to make sure all of the joint is lavaged and not just one aspect of one side- in the hock joint for example needles should be placed dorsomedially and dorsolaterally but also in the palmarolateral joint pouch. Balanced electrolyte solutions are best for lavage without any additions and quantities of a minimum of 1 litre with up to 5 litres with a pressurized bag. If the joint alone is involved then intra-articular antibiotics are an effective method of obtaining high local concentrations of antibiotics at the site of infection and should be instilled after the lavage has completed. Commonly amikacin (150- 250mg) is injected as resistance is uncommon, similar small doses of ceftiofur or gentamicin can be used as well. If there is any bone or physeal involvement then regional limb perfusions are an effective method of
obtaining high concentrations both in the joint and in the bone. Regional perfusions are performed by placing a tourniquet proximal to the joint or bone affected and injecting antibiotics into a vein distal to the tourniquet. The tourniquet is left in place for around 20 minutes and can be painful so commonly the foal will require sedation for that period of time. With young foals it is important for the concentration of the drug not to be too high as there have been anecdotal reports of irritation to the tissue and occasional skin necrosis if the concentration is high. A guideline has been to have a third of the systemic dose diluted into around 20-50mls saline- commonly reported in foals are doses of 150-250 mg amikacin or 400-600mg gentacin. Growth plates can also have antibiotics injected directly into them using a large gauge needle such as a 16 or 14-gauge needle. Doses are similar to those used for intra-articular injection. Local instillation of antibiotics performed every 2 days is sufficient and decreases trauma to the joint capsule.

Along with antibiotics other medications include anti-inflammatory medications and anti-ulcer medications if warranted. Anti-inflammatory medications may enable a painful foal to stand and nurse but should be administered judiciously to minimize potential side effects such as gastric ulceration. Concomitant administration of omeprazole or other anti-ulcer medication used to be standard if anti-inflammatory are used although the current theory is that removal of the acid stomach barrier may increase infections. Additionally, most ulcers that occur are glandular or around the pylorus and so recommendations currently include only the use of sucralfate as a ‘bandaid’ for any ulcers and not general anti-ulcer therapy.

Lavage for septic joints and local antibiotic therapy with treatment of any systemic disease are the cornerstones of treatment. Response to therapy should be immediate and resolution reached quickly. With early detection of a septic joint without systemic disease one lavage and 1-2 doses of local antibiotics with systemic therapy is often sufficient. All cases that are complicated or do not respond should be referred for more aggressive therapy. Improvement of the clinical signs is the most reliable indicator of improvement as repeated needle lavages and antibiotics will continue to cause some inflammation in the joint so the cell count should decrease but will often not return to normally acutely.

Prognosis
Reports on the outcome of septic arthritis and/or osteomyelitis are usually from university or private practice settings so may be confined to those cases unable to be treated on the farm. Reports carry a range of prognosis from 30-80% with most of the reports around 70-80% for survival. The athletic careers of foals who had had septic arthritis is again variable and dependant on bone involvement and many other variables. Negative prognostic indicators include the presence of salmonella, multiple joints, bone and joint involvement together, and the severity of concurrent diseases. Crucial to a good prognosis is aggressive and early treatment.

References:
Introduction
The online Business Dictionary describes a team as “a group of people with a full set of complementary skills required to complete a task, job, or project”. Team members must: 1) operate with a high degree of interdependence, 2) share authority and responsibility for self-management, 3) remain accountable for the collective performance, and 4) work toward a common goal and shared reward.

A veterinary team is comprised of a group of people working with the goal of providing exceptional patient and client care. When a veterinary team has a strong sense of mutual commitment to this care, a synergism is created, which results in performance that is greater than the sum of the performance of the individual team members.

Effective vs. Ineffective Teams
Cohesive teams are those that have clear measurable goals, adequately trained team members, labor divided among the participants, and effective communication. Communication has an impact on all members of the veterinary team and if there is ineffective communication, everything else will be adversely affected. There is research in the human healthcare field to suggest that successfully functioning teams with effective communication lead to better quality of care with improved patient outcomes including decreased post-operative pain and improved post-operative function, shorter hospital stays, improved patient satisfaction, and enhanced job satisfaction. Conversely, ineffective human healthcare teams more often exhibit miscommunication, which leads to medical errors, job dissatisfaction, emotional exhaustion, increased staff turnover, and compromised patient care.

Authority and Structure
Power and authority always exist, but the team must understand who the authority (or authorities) are that influence what takes place on the team. It is useful for team members to know what decisions they can make without having to consult an external authority.

The team must establish and maintain an enabling structure or a way of working that facilitates the team’s efforts to deliver their services. An enabling structure includes: 1) norms that the team establishes and maintains for itself; 2) composition of the team itself (i.e., who is on the team); and 3) tasks that the team will take on.

For team members to maintain motivation, there must be feedback and appreciation. There also should be the realization that no one alone could do the work of the team. While tasks are sometimes better done alone, for the team to work, members must acknowledge that the task is bigger than they are and that they need their teammates.

Problem-Solving Within the Team
People must communicate effectively if the team is going to function efficiently and problems are going to be solved. Problem solving requires the exchange of information, processing the information to make it mean something, and then making choices about action, further exploration,
or the choice to abandon the problem. If problem solving communication is to be effective, it requires some underlying attitudes among members of the team.

Basic respect: This does not mean that team members must agree, but it does mean that each team member must approach others from the standpoint that they are doing the best they can.

Curiosity: Each team member should maintain an attitude of curiosity about what the other person is thinking and feeling, as well as a desire to truly understand their thoughts and feelings.

Authenticity: The information each team member shares about their thoughts or feelings should be real and accurate. Each team member needs to be counted on for their honesty and authenticity in all situations.

**Essential Components of a Successful Team**

Trust takes time to develop. New members of a team can struggle to gain the trust of others, especially if the team is well-established and the roles within the team have been longstanding. It is important that team leaders provide new employees with opportunities to complete important tasks and recognize their efforts publicly to help gain the trust of the team. Team members must also trust that everyone on the team has good intentions (i.e., giving the benefit of the doubt).

A shared mental model is created within a team when each team member shares their awareness in clinical situations. This allows everyone to “be on the same page” by communicating their understanding of what is happening. Otherwise, misunderstandings and assumptions leading to oversight and error can occur. A shared mental model can be encouraged by asking open-ended questions to gather information from team members in a way that is inviting and respectful. Other helpful tools include reflective listening and empathy statements, which send the message that team members are indeed listening and appreciate the other person’s perspective.

Non-verbal awareness is also very important. Non-verbal messages often carry more weight than verbal messages. Each team members must strive to ensure that their body language is congruent with what they are saying so that mixed messages are avoided. Also, when engaged in conflict with a team member, keep body language as open and neutral as possible. This will help to ensure that people remain less reactive and more proactive. Team members do not want to appear aggressive or dominant, nor submissive. When non-verbal behaviors are open and accepting, team members are likely to consciously or unconsciously mirror that behavior, which can put people in a much better position to resolve the conflict constructively.

**Barriers to Communication**

Communication within the team is a constant challenge because of many complex issues and complicating factors. For example, power and status related to position, credentials, and designated authorities can influence team dynamics. Gender and generational differences also influence communication styles, as do differing personalities.

Another barrier to effective communication is the discomfort felt during highly charged or difficult discussions when emotional defenses might be evoked. This can occur when sharing feedback or speaking honestly about something that colleague might not want to hear. Team member instinct
during these situations is often to avoid any interpersonal discomfort and instead ignore problems to avoid disagreement.

Conflict avoidance occurs when people self-censor in order not to “hurt someone’s feelings” or “rock the boat”. When people seek harmony with the group at all costs, disaster can occur. It is important that people feel comfortable to share and offer feedback or suggestions when they feel the team could benefit.

**Workplace Conflict**

Conflict is inevitable in any team due to differences in clinical knowledge, work approaches, values, opinions, and personalities. Addressing and resolving conflict is important to team effectiveness and providing safe and quality patient care. When conflict is handled poorly and the focus is on the people involved instead of the issue at hand, the team can become divided and patient care can suffer. Poorly handled conflict also results in erosion of team morale.

Because conflict causes personal discomfort, individuals sometimes will choose to get along at all costs. This is referred to as artificial harmony and is the equivalent of paralysis. Team members are so reluctant to cause a problem that no forward progress is made. Ideally, teams should accept that conflict is part of being a team and that while there will be some personal discomfort during the exchange of ideas, problems can be resolved in a timely manner and new thinking stimulated within the team.

Very often, people believe that failure to reach consensus is actual failure. But allowing team members to express their opinion and be heard by their peers facilitates buy-in in the final decision. Even when the final decision is not the choice of each team member, they are more accepting of the choice because their voice was heard.

**Information Conflict**

Information conflict involves differing ideas, views, and opinions. It can also occur if there is a disagreement about the content of a decision. During these situations, team members should use arguments that are precise and as factually oriented as possible. Arguments that persuade primarily based on emotion should be avoided. When an argument turns into a personal win-or-lose “word war”, team members are no longer concerned with resolving problems rationally. When team members become defensive, they concentrate more on defending one point of view, rather than openly evaluating other views. Defensive attitudes prevent team members from gaining new insight and understanding.

To solve problems effectively, inquiry and accurate information should be shared. All team members should feel welcome to contribute what they know, feel, and value. Team members should also be aware of timing including not wasting time or introducing issues when there is no time to work on them. Time is a precious resource and should be regarded as such by all team members. Open body language and open-ended questions are important so that team members feel encouraged to provide information freely. Nodding heads, keeping an interested facial expression, and saying “go on, I’m listening” are good ways to get the most out of these interactions.
Personal Conflict
Personal conflict can stem from interpersonal incompatibility and is not usually task-oriented. Tension, annoyance, and animosity are common and arguments can end up resulting in problems accomplishing team tasks. Disruptive behavior among staff should be actively discouraged. Practices should have guidelines for acceptable behaviors to assist staff in better identifying, reporting, and managing behaviors that disrupt client and patient care. Types of disruptive behaviors include condescending language and voice intonation, impatience with questions, reluctance or refusal to answer questions or telephone calls, strong verbal abuse or threatening body language, and physical abuse.

DISH Script
The DISH script can be useful to communicate effectively during all types of conflict and is most effective in resolving personal conflict. The mnemonic is as follows:
- Describe the specific situation.
- Identify your concerns about the action using “I” statements.
- Suggest other alternatives.
- Highlight the benefits of shared problem solving with the goal of reaching a shared agreement. Keep in mind that the potential consequences should be stated in terms of the impact on establishing team goals and striving for success.

When using the DISH script, it is important to time the discussion soon after the conflict occurred. Likewise, the problems should be framed in terms of the personal experience and lessons learned. As much as possible, use “I” statements rather than “you” statements, which can be perceived as blame. Focus on what is right, rather than who is right to refrain from making it personal. A private location that is not in front of the client or other team members is also important to allow both team members to focus on resolving the conflict rather than saving face. By focusing on the issues, concerns, or behaviors, the discussion avoids personalizing or targeting individuals. Try to limit discussion to a single incident and not a pattern or sequence of events, which will help to keep the discussion focused. Finally, work on win-win in the sense that team unity and quality of care are dependent upon coming to a solution that all team members approve of.

Conciliatory Gestures
A conciliatory gesture is a statement made which exposes the team member’s vulnerability in the spirit of taking accountability in the conflict discussion. This is a powerful way to change the course of an adversarial discussion. Frequently, when one person uses a conciliatory gesture and it is matched by one from the other team member, this signals the end of me-against-you and the beginning of us-against-the problem. However, it is very important that these be delivered in a genuine way without sarcasm or conflicting non-verbal messages, which will have the opposite effect. Examples of conciliatory gestures include apologies, owning responsibility, conceding, or self-disclosing.

Helpful Resource: Veterinary Team Brief – Team Communication articles
https://www.veterinaryteambrief.com/topics/team-communication
Understanding Personality Types
Jolene Watson, RVT, MBTI® Certified Practitioner

Introduction
The Myers-Briggs Type Indicator® (MBTI®) is a versatile assessment of personality type and is utilized by 80% of fortune 100 companies! It describes preferences for interacting with others, gathering information, making decisions and organizing our lives. The Myers-Briggs Type Indicator® can help people make business, career and personal decisions. Clarity Coaching & Development utilizes various MBTI® customized reports to facilitate corporate and individual personality assessments focusing on stress management, time management, goal setting and effective networking etiquette.

Extrovert Preferences
Key Words: Action, Outward, People, Interaction, Many, Expressive, Do-Think-Do
People who prefer Extraversion like to focus on the outside world; they direct their energy and attention outward and get energized by interacting with people and taking action.

Characteristics associated with people who prefer Extraversion:
Drawn to the outside world
Prefer to communicate by talking
Work out ideas by talking them through
Learn best through doing or discussing
Have broad interests
Tend to be sociable and expressive
Readily take initiative in work and relationships

Introvert Preferences
Key Words: Reflection, Inward, Privacy, Concentration, Few, Quiet, Think-Do-Thinking
People who prefer Introversion like to focus on their own inner world; they direct their energy and attention inward and are energized by reflecting on their own and others’ ideas, memories, and experiences.

Characteristics associated with people who prefer Introversion:
Drawn to their inner world and prefer to communicate in writing
Work out ideas by reflecting on them
Learn best by reflection and mental “practice”
Focus in depth on a few interests
Tend to be private and contained
Take initiative selectively—when the situation or issue is very important to them

Sensing Preferences
Key Words: Facts, Realistic, Specific, Present, Keep, Practical, What is
People who prefer sensing like to take in information that is real and tangible—what they perceive using the five senses. They pay close attention to what is going on around them and are especially attuned to practical realities.
Characteristics associated with people who prefer Sensing:
Oriented to present realities
Factual and concrete and focus on what is real and actual
Observe and remember specifics and build carefully and thoroughly toward conclusions

Intuitive Preferences
Key Words: Ideas, Imaginative, General, Future, Change, Theoretical, What could be
People who prefer intuition like to take in information by seeing the big picture; they prefer to focus on the relationships and connections between facts. They look for patterns and are especially attuned to seeing new possibilities.

Characteristics associated with people who prefer Intuition:
Oriented to future possibilities and trust inspiration
Imaginative and verbally creative
Focus on the patterns and meanings in data and remember specifics
Move quickly to conclusions and follow hunches
Want to clarify ideas and theories before putting them into practice

Thinking Preferences
Key Words: Head, Distant, Things, Objective, Critique, Analyze, Firm but fair
People who prefer thinking like to decide things by looking at the logical consequences of their choice or action. They want to mentally remove themselves from the situation so they can examine the pros and cons objectively. They enjoy analyzing what’s wrong with something so they can solve the problem. Their goal is to find a standard that applies in all similar situations.

Characteristics associated with people who prefer Thinking:
Analytical and as such use cause-and-effect reasoning
Solve problems with logic
Strive for an objective standard of truth
Reasonable
Can be “tough-minded” yet fair—want everyone treated equally

Feeling Preferences
Key Words: Heart, Personal, People, Subjective, Praise, Understand, Merciful
People who prefer feeling like to decide things by considering what’s important to them and to others involved. They mentally insert themselves into the situation to identify with everyone so they can make decisions that honor people. They enjoy appreciating and supporting others and look for qualities to praise. They create harmony and treat each person as a unique individual.

Characteristics associated with people who prefer Feeling:
Guided by personal and social values and assess impacts of decisions on people
Strive for understanding, harmony, and positive interactions
Compassionate and may appear “tenderhearted”
Fair—want everyone treated as an individual
Judging Preferences

Key Words: Organized, Decision, Control, Now, Closure, Deliberate, Plan

People who prefer judging like to live in a planned, orderly way. They want to make decisions, come to closure, and move on. Their lives tend to be structured and organized, and they like to have things settled. Sticking to a plan and schedule is very important to them, and they enjoy getting things done.

Characteristics associated with people who prefer Judging:
Scheduled and like to organize their lives
Systematic and methodical
Make short- and long-term plans and like to have things decided to avoid last-minute stress

Perceiving Preferences

Key Words: Flexible, Information, Experience, Later, Options, Spontaneous, Wait

People who prefer perceiving like to live in a flexible, spontaneous way, and want to experience and understand life rather than control it. Detailed plans and final decisions feel confining to them; they prefer to stay open to new information and last-minute options. They enjoy being resourceful in adapting to the opportunities and demands of the moment.

Characteristics associated with people who prefer Perceiving:
Spontaneous, flexible, casual and open-ended
Adapt, change course
Like things loose and open to change and find last-minute pressures energizing

Source: Psychometrics Canada Ltd.- Personal Impact Report

| CHARACTERISTICS FREQUENTLY ASSOCIATED WITH EACH TYPE |
|-----------------|-----------------|-----------------|-----------------|
| ISTJ             | ISFJ             | INFJ             | INTJ             |
| Quiet, serious, succeed by being thorough and dependable. Practical, matter-of-fact, realistic, and responsible. Decide logically what should be done and work toward it steadily, regardless of distractions. Take pleasure in making everything orderly and organized—their work, their home, their life. Value traditions and loyalty. | Quiet, friendly, responsible, and conscientious. Committed and steady in meeting their obligations. Thorough, painstaking, and accurate. Loyal, considerate, notice and remember specifics about people who are important to them, concerned with how others feel. Strive to create an orderly and harmonious environment at work and at home. | Seek meaning and connection in ideas, relationships, and material possessions. Want to understand what motivates people and are insightful about others. Conscientious and committed to their firm values. Develop a clear vision about how best to serve the common good. Organized and decisive in implementing their vision. | Have original minds and great drive for implementing their ideas and achieving their goals. Quickly see patterns in external events and develop long-range explanatory perspectives. When committed, organize a job and carry it through. Skeptical and independent, have high standards of competence and performance—for themselves and others. |
| ESTP             | ESFJ             | ENFP             | ENTP             |
| Flexible and tolerant, they take a pragmatic approach focused on immediate results. Bored by theories and conceptual explanations, they want to act energetically to solve the problem. Focus on the here and now, spontaneous, enjoy each moment that they can be active with others. Enjoy material comforts and style. Learn best through doing. | Outgoing, friendly, and accepting. Exuberant lovers of life, people, and material comforts. Enjoy working with others to make things happen. Bring common sense and a realistic approach to their work, and make work fun. Flexible and spontaneous, adapt readily to new people and environments. Learn best by trying a new skill with other people. | Warmly enthusiastic and imaginative. See life as full of possibilities. Make connections between events and information very quickly, and confidently proceed based on the patterns they see. Want a lot of affirmation from others, and readily give appreciation and support. Spontaneous and flexible, often rely on their ability to improvise and their verbal fluency. | Quick, ingenious, stimulating, alert, and outspoken. Resourceful in solving new and challenging problems. Adept at generating conceptual possibilities and then analyzing them strategically. Good at reading other people. Bored by routine, will seldom do the same thing the same way, apt to turn to one new interest after another. |
| ENFJ             | ENTP             | ENFP             | ENTP             |
Report
BEFORE YOU SPEAK, THINK
T - Is it true?
H - Is it helpful?
I - Is it inspiring?
N – Is it necessary?
K – Is it kind?
Always use the ‘PLATINUM RULE’ - Treat others the way they want to be treated.

CUSTOMER BILL OF RIGHTS:
1. KNOW ME
2. VALUE ME
3. UNDERSTAND MY NEEDS
4. TREAT ME WITH RESPECT
5. VALUE MY TIME
6. MAKE IT EASY
   - Taking your customer care to the next level, Nadji Tehrani

DECISION MAKING:
- E- Share information and discuss
- I- Reflect, then talk
- S- Identify facts and realities
- N- Generate possibilities
- T- Analyze by likely outcomes
- F- Evaluate by values and relationships
- J- Make a plan
- P- Be open to changing the plan
Managing Team Conflict
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Compassion Fatigue
It is emotional, psychological, spiritual, and physical exhaustion. It necessitates a caregiving relationship within which there is an exchange of empathy, emotions, and information, along with a strong desire on the part of the caregiver to help alleviate the suffering and pain.
Source: Figly, 2006

Compassion Fatigue results from the process of dispensing care and burnout results from work related stress: excessive, prolonged, and unrelieved work-related stress.
Source: Pines and Aaronson, 1998

Burn out may require changing jobs or careers
It disturbs the ability to think clearly, modulate emotions, feel effective, or maintain hope. Healthy self-care and work-life balance along with team, practice and professional support together can enable the successful management of compassion fatigue
Source: Stoewen, 2006

In the Grip/ Acute Stress
In the grip is based on Jungian psychology and is about the out-of-character selves we encounter from time to time, particularly in response to fatigue and stress. Experiencing being in the grip of something strange and unfamiliar tends to alarm us because of its “Jekyll and Hyde” character. We may then be forced to reexamine the essence of our character and personality. In the grip explains our seemingly aberrant, abnormal experiences of ourselves and others.

In our normal, everyday activities we spend more time doing some things and less time doing others; we enjoy and have more energy for doing what we like than for doing what we don’t like. These same kinds of energy differences reflect the way energy is distributed among our four preference areas (1) using energy, (2) gathering information, (3) coming to conclusions, and (4) relating to the outside world. In the type approach to personality, we each experience one pole of each area as more comfortable and natural as the opposite, and therefore tend to use it more and enjoy its use.

Inferior Function
The inferior function is our least favored part of our personality! It is largely unconscious- we don’t direct or control it. Its unconscious energy erupts and takes over our personality when our conscious energy diminished sufficiently. We often remain unaware of the change in ourselves until the experience is over.

In the Grip/Inferior Function Reactions
These reactions may appear exaggerated or extreme- like a caricature of that type; the person may come across as childish, touchy and easily angered.

Triggers
1. Fatigue - When we are physically tired due to overwork, lack of sleep, or excessive activity, our energy is depleted and unavailable to deal with everyday events.

2. Illness - When we are ill, our bodies are out of balance. The extra stress and strain we experience saps our strength, causing low energy and fatigue.

3. Physical or psychological stress - People of different personality types identify differing sources of both positive energy and stress, and these energizers and stressors tend to be consistent with the attributes of their personalities.

4. Alcohol or mind-altering drugs - These lower our level of consciousness by decreasing our control of physical reflexes, social inhibitions, and the like.

5. Life transitions - These are likely to be accompanied by out of character experiences.

For some people, the ongoing stress becomes the norm in their lives, and they are largely unaware that they are responding to a now habitual situation in an out-of-character manner!

**During midlife the inferior function may emerge in two ways:**

1. The person may become quite interested in and excited by activities and pursuits that held little interest earlier in life. Career, relationships, outside recreational pursuits, and hobbies may be experienced with a good deal less enthusiasm, interest and commitment.

2. The person may adopt some opposite, extreme, and grossly out-of-character behavior that both the person and others may see as “irrational.” So-called midlife crises falls into this category, and may be seen in drastic career changes, dropping out, or having affairs.

EXTROVERTS- Can become aloof and shut off communication with others.
INTROVERTS- Might speak out loudly and often which is very out of character for them.
SENSORS- May become unfocused and stop paying attention to details. They can become paranoid and look for meaning in trivial things.
INTUITIVES- Might focus on details and lose focus of the real vision. They search for the ultimate purpose while feeling lost.
THINKERS- May have emotional outbursts and fear losing control of their feelings. They may become passive aggressive.
FEELERS- Can become very critical and tough to themselves and others and then feel extremely guilty. Despair can take over quickly.
PERCEIVERS- Will feel incompetent and anxiety will take over. They look for meaning in trivial events and withdraw from others.
JUDGERS- Express anger and may have outbursts of emotion and become very scattered.

**What is the Purpose?**

It is actually an opportunity for growth and self-development. It aids the psyche in regulating its energy so that growth and adaptation can proceed in a natural way.

According to Jung, It is normal for us to be passionately interested in some things to the neglect of others. When a behavior or attitude is severely one sided, the opposite energy in the unconscious becomes equally extreme. Eventually it erupts in an exaggerated and disruptive way. Eruptions of the inferior function often compensate for overuse of one’s preferred functions.

It may simply be a warning that we’re doing too much of something. Or a person may become aware through such an experience that he or she is overtired or stressed and needs to slow down, get more relaxation, or take steps to reduce stress.
It may force us to acknowledge an important feeling, habit or way of thinking that is unconsciously influencing our perceptions and actions. This may promote a change in self-concept that can encourage new approaches to ourselves and important life issues.

**IT IS IMPORTANT TO USE THE INFERIOR FUNCTION TO INCREASE OUR EXPERIENCE AND COMFORT WITH THEM FOR EFFECTIVE TYPE DEVELOPMENT.**

Source: ‘In the Grip-Understanding Type, Stress, and the Inferior Function’- Naomi L. Quenk

Empathetic Listening
55% NONVERBAL
38% TONALITY
7% WORDS
Pay full attention to the speaker; even if you don’t agree, seek to understand them.

Always use the ‘PLATINUM RULE- Treat others the way they want to be treated.

Questions to Discuss in Terms of Workplace Stress
1. What are your strengths?
2. What areas do you commit to working on (areas for improvement)?
3. What are ways to get back to balance that you know work for you?
4. How can others help you?
5. How can others make it worse?
6. Who is in your support network?

Top 5 Regrets
1. I wish I'd had the courage to live a life true to myself, not the life others expected of me.
2. I wish I hadn't worked so hard.
3. I wish I’d had the courage to express my feelings.
4. I wish I had stayed in touch with my friends.
5. I wish that I had let myself be happier. -Bronnie Ware

Rule of Three
The rule of three, law of three, or power of three is a writing principle that suggests that things that come in threes are funnier, more satisfying, or more effective than other numbers.

**Three points per email (include a subject line and priority status)**
**Three items per priority list**
**Three options for clients to choose from**
Toxic Veterinary Teams
Marie K. Holowaychuk, DVM, DACVECC, Critical Care Vet Consulting

Introduction
Lack of collaboration among team members, ineffective management, and work stress are associated with voluntary turnover and job dissatisfaction in the human healthcare field. Similarly, toxic attitudes negatively affect veterinary team function and a toxic work environment occurs when toxic attitudes in the workplace are ignored or not addressed. Consequently, toxic attitudes can lead to relationship and task conflicts, which are negatively associated with team satisfaction and performance. It is imperative that toxic attitudes and environments are recognized and addressed, to foster wellness in the veterinary workplace.

Impact of Toxic Attitudes on the Veterinary Team
The presence of people exhibiting a toxic attitude has a pronounced negative impact on the success of the veterinary team. A toxic attitude can manifest in many forms such as disrespect, resistance to change, avoidance of conflict, lack of motivation, chronically negative demeanor, always wanting to be the “go to person”, and incompatible personalities.

Negative behaviors include leaving tasks for other people to complete, being persistently pessimistic, being excessively critical of others, and demeaning fellow team members. Negative people are also more likely to express irritation, anxiety, and insecurity and affect the emotions, moods, and attitudes of the rest of the team in a disproportionate manner.

Another common contributing factor is dealing with people having a “that’s not my job” attitude or refusing to perform certain tasks that are reasonably considered part of their duties. For example, technicians not wanting to answer phones or do laundry, or receptionists not wanting to clean kennels or hold animals, as well as veterinarians not wanting to see clients or take phone calls during lunch breaks. When team members refuse to assist with duties or tasks, others consider it a control or ego issue and that they are not contributing properly as a team member. Very often these situations stem from a miscommunication or lack of understanding about what the other person is doing. For example, the “front” staff might not realize that the “back” staff are busy performing treatments or assisting with diagnostic tests, rather than sitting idle and leaving jobs for other people to do. Additionally, the “front” staff might feel unappreciated for their duties in dealing with clients or answering phones or that “back staff” do not understand all the work that they are doing.

Mood polluters are team members with a negative attitude that can be temporary (e.g., in response to a difficult client or situation) or chronic. Sometimes the presence of a mood polluter in the team can bring the other team members closer together, if they feel they can share their experiences, empathize with each other, and function well together despite the situation. However, sometimes the impact can be negative if other team members feel persistently disrupted by the behavior (e.g., if a team member always leaves “on time” even when an emergency is on its way, rather than staying late to help the rest of the team leave at a reasonable time).

Wanting to be the ‘go to’ person is considered toxic to other team members who consider it frustrating when an individual wants to “stay in charge” or in a position of power, by monopolizing certain tasks or duties or not sharing certain knowledge. This can become even more damaging to
the team if that individual does not have the appropriate skills or training to complete the task (e.g., a clinic manager insisting on doing the ordering, but with limited knowledge and experience to appropriately communicate with suppliers). If others feel that they could do the job more effectively or efficiently, this can create resentment or irritation.

Additionally, labeling certain team members as “lead technicians” or “senior veterinarians” can create a sense of unfairness among the team, especially if the role is not clearly communicated to the other team members. For example, if a technician is designated as the “lead tech” but without proper acknowledgment, unfairness results from the expectation to perform managerial duties without compensation. Alternatively, the other technicians might perceive that individual is choosing not to perform technical duties in lieu of other less technically challenging tasks.

While assigning staff members into certain roles can be beneficial when those individuals want or expect to be the “go to person” because they feel their knowledge and experience qualifies them for the job, this can create team conflict if they avoid delegating duties because they do not feel others can complete the tasks, or if others feel resentment when seeing another team member “elevated” to a different role.

Personality issues can lead to toxic environments if an individual’s personality does not fit in with the rest of the team. Most people would agree that a team member’s personality is more important than their technical skills, as the latter can be taught while the former is unlikely to change. More importantly, when people violate important interpersonal norms by making hurtful comments, behaving rudely, or embarrassing people in front of others, this “deviant” behavior results in distrust by the rest of the team and distracts team members leading to time wastage.

**Solutions to Toxic Attitudes**
Responses to negative behavior include attempting to change the person’s behavior, removing the person from the workplace, or protecting oneself through defensive behavior. If motivating the individual to change the behavior or removing the person from the workplace is not possible, a toxic environment will result. Other employees might become defensive to protect themselves; such behaviors can include lashing out, seeking revenge, providing distraction, or withdrawing from the team.

Dealing with negative behaviors is a leadership responsibility; if leaders do not address the situation, there will be persistent dissatisfaction within the team. Therefore, these behaviors cannot be ignored; rather, the instigator must be confronted, given the opportunity to correct the behavior, and given consequences if the behavior does not change. Veterinary leaders might need additional training in communication and leadership skills to address these negative behaviors effectively.

A solution to the “that’s not my job” sentiment includes having written job descriptions and consistent training, as well as accountability and flexibility. Having clear responsibilities and structure is helpful for staff members to know their duties, but it is important that they are also willing to step in and “pull their weight” or “go the extra mile” if the need arises.

Dealing with mood polluters can be difficult as changing a person’s behavior is often unsuccessful. Therefore, selecting for team members with a positive attitude is preferential to selecting for people...
with specific technical skills. Additionally, dismissing team members with a negative attitude can improve clinical atmosphere and team efficiency dramatically. Also, team building activities and acknowledgment of positive team work can be helpful in diminishing negativity.

While go-to people may be perceived as negatively impacting the team, in some cases they might not disturb the group’s effectiveness. In fact, if they have the skills and knowledge to complete their tasks, they might help the team more effectively achieve its goals. However, if the rest of the team is impacted by this behavior, it must be addressed by outlining expectations and showing the individual the benefits of sharing responsibilities and tasks with co-workers.

A solution to personality issues might be a clinic choosing to always do working interviews, carefully screening references, as well as allowing staff input with hiring decisions, to ensure personality compatibility within the team. Also, disclosing to the new hire that they will have to be flexible, adaptable, and “fit in” with the team, or they will be dismissed. Additionally, rather than selecting for certain personality traits, it might be more effective to capitalize on the positive attributes that each personality type brings to the workplace.

When people see themselves as individuals rather than part of a team, they might think and act more selfishly, rather than working cooperatively. By addressing uncivil behavior, veterinary clinics can reduce unproductive time (i.e., spend worrying about an interaction), as well as staff turnover. Reviewing hiring and training practices and diligently monitoring and recording problems will help to prevent and reduce workplace incivility.

Likewise, to alleviate problems caused by instigators of discourteous behavior, veterinary owners and managers can set zero-tolerance expectations for inappropriate workplace behavior, document and address instances of these behaviors, and avoid hiring anyone that has exhibited these behaviors in the past. Feeling respected and respecting others are keys to cooperation.

Impact of a Toxic Environment on the Veterinary Team
The presence of a toxic work environment also has a pronounced negative impact on the success of the veterinary team. A toxic environment occurs when there is broken communication and tension between staff members. This can occur due to employees lacking confidence, skills, or knowledge, employees not feeling appreciated, difficulties coping with staff turnover, dealing with conflicting demands, mishandling conflict, or not holding chronically negative or hostile people accountable for their behavior.

When an individual lacks confidence, skills, or knowledge it impacts the rest of the team as co-workers lose trust in the individual’s abilities and might feel the need to check on the co-worker, which can lead to frustration or annoyance. Likewise, individuals might feel annoyed with new staff members not being able to perform duties, complete tasks, or make decisions with confidence, which can lead to employees being disrespectful to each other. This tends to translate into groups of employees being discourteous to each other (e.g., technicians vs. receptionists), rather than to members within their own group.

Not feeling appreciated by clients, veterinarians (as technicians), or co-workers contributes to a toxic environment and is felt most frequently be technicians. This typically results from a lack of awareness of what other people do, as well as a lack of respective for an individual’s capabilities.
This leads to frustration that an individual’s education, knowledge, and skills are not recognized, as well as resentment if individuals trained on-the-job are given the same duties or pay as others who have received formal schooling.

Coping with turnover due to clinic expansion or covering for leave (e.g., maternity) is stressful for permanent employees, especially those who are resistant to change. This is probably because of the uncertainty of the new staff member’s personality, skills, and abilities, which can have a large impact on the permanent employees.

When team members are not abiding by the same clinic rules or policies it can undermine an individual’s credibility and lead to a toxic environment. For example, if a veterinarian makes an exception for a client (i.e., “changes the rules” during a situation) after a technician has advised the client it would not be possible due to clinic policy, this can have negative consequences and remove the feeling of functioning as a team.

When team members are not held accountable for their actions, the lack of consequences can create a hostile environment and interpersonal tension in the clinic. For example, if a veterinarian is constantly fighting with staff or making negative comments about co-workers, but is not reprimanded despite complaints from other team members, the lack of consequences for bad behavior can decrease motivation in other clinic staff and enhance negativity in the work environment.

When staff members are expected to perform tasks beyond their scope of practice, or tasks that are unrealistic given personnel or facility limitations, the unrealistic expectations can lead to a toxic environment. For example, asking technicians to “police” new veterinarians or remember all the subtle nuances between each veterinarian’s preferred way of practicing, can be perceived as overly high expectations. Likewise, when veterinarians are expected to multi-task and perform conflicting demands (e.g., having to help take radiographs rather than researching differential diagnoses for the patient), client service and animal safety can suffer. These situations can lead to stress, burnout, and a toxic environment.

Conflicting demands also occur when team members receive conflicting messages from two or more people, which leaves them unsure about what they should do. Likewise, when a clinic is very busy or under-staffed, individuals can feel overwhelmed with not knowing which tasks should take priority. This is exacerbated in environments with poor communication when people are unaware of what other team members are doing.

Resentment can be created with an individual feels they are extremely busy trying to manage conflicting demands, while perceiving that others are not doing as much. These feelings can also occur when team members are attempting to juggle management and clinical duties, or balance work duties with personal/family time. Ultimately, multiple competing demands can create a toxic environment when the result is responsibilities not receiving the necessary time and attention. Lack of leadership can occur with absentee owners or managers, as well as ineffective leaders. It is a source of frustration that leads to resentment and confusion among team members. Sometimes when there are multiple owners or a lack of structure, confusion and frustration can occur because communication falters and decisions are inefficiently made.
Solutions to Toxic Environments
There are many ways to show appreciation to veterinary team members including providing small prizes or notes for staff that do something exceptional, reading thankyou letters from clients during staff meetings, encouraging people to provide positive feedback to other coworkers, and hosting appreciation events such as parties or team excursions.

Coping with turnover can be enhanced by recognizing that new staff members can bring refreshing change into a hospital team and that sometimes delegating duties to others can alleviate responsibilities on a single person and have benefit for the entire team.

Rather than changing the rules for clients, it should be emphasized to the client that an exception is being made for them that is against hospital policy, to support what they were already told by another team member. This avoids undermining the other team member and ensures that everyone remains on the “same team” rather than being self-serving.

Lack of consequences can be rectified by ensuring that people are being held accountable for their actions when they negatively affect other team members. Additionally, unrealistic expectations and conflicting demands can be solved by staffing appropriately so that everyone can focus on their own duties and maintain work-life balance. Clinics with higher non-veterinarian to veterinarian ratios function more effectively through a higher net practice income than clinics with lower ratios. Therefore, managers should review work hours and staffing levels to ensure team members are not feeling overwhelmed.

A lack of leadership might be improved by ensuring there is a formal organization structure, consistent communication, and effective leadership skills.
Performance Reviews
These should be done every 3-6 months to create an open line of communication between staff and management. If an employee does not feel safe, instead of dialogue, they will resort to violence or silence according to the book ‘Crucial Conversations’ by Kerry Patterson.

Top Two Reasons People Quit
1. RELATIONSHIP WITH THEIR DIRECT SUPERVISOR/MANAGER
2. ‘PERCEIVED’ LACK OF APPRECIATION

Score Cards are a great way to keep track of employee engagement; every leader/manager should know the goals of all employees. Each employee should list, in accordance of priority, their top 10 needs/wants in their workplace. Once this is known opportunities for continuing education can be researched. Some examples of common needs/wants are included below.

Please email me at jolenewatson@live.com if you would like a copy of the actual Excel template for the score card which will tally up the score out of 100% in terms of workplace satisfaction.

1. Work-life balance
2. Flexible work schedule
3. Continuing education/travel
4. Encouraging work environment
5. Ability to specialize in the industry
6. Wage and benefits

Delegation and job sharing is another great topic at performance reviews; have each team member list their daily/weekly/monthly tasks and then have them rate how happy they are in each area including how qualified they feel. Are there employees that enjoy tasks others find stressful? It is important to cross-train in the case of absences; however, it only makes sense to delegate according to preference when possible to increase engagement/loyalty. Some examples of common tasks in the veterinary industry are (see picture on next page):

1. Radiography
2. Dentistry
3. Bookkeeping
4. Office Management
5. Surgery
6. Nutrition Consultations
7. Phlebotomy
8. Emergency Care

EXAMPLE OF A DELEGATION EXCEL TEMPLATE:
Appreciation Styles

I use these results in my couples coaching sessions as well as my corporate workshops in relation to appreciation styles in the workplace.

1. WORDS OF AFFIRMATION- Words, both oral and written, can be used to affirm and encourage those around us. Some people prefer personal one-on-one communication, while others value being praised in front of others (but it is important to know that a lot of people do not like to receive public affirmation in front of a large group.)

2. QUALITY TIME- Personal, focused time and attention with their supervisor is highly affirming for some. But others enjoy different types of time — “hanging out” with their coworkers, working together as a team on a project, or just having someone take the time to listen to them. And the type of time desired can differ significantly depending on whether it is with colleagues or with their supervisor.

3. ACTS OF SERVICE- Assisting in getting a task done can be extremely encouraging to a colleague. Helping a teammate “dig out” from being behind, working collaboratively on a project that would be difficult to do alone, or just working alongside with them on a task, are all ways of showing appreciation for their efforts.

4. TANGIBLE GIFTS- The key to an effective gift in the workplace is the “thought,” not the amount of money spent. Taking time to notice what your colleagues enjoy (chocolate, coffee, cashews), observing their hobbies and interests (sports, books, crafts) and buying them a small related gift shows that you are getting to know them as a person and understand what is important to them.

5. PHYSICAL TOUCH- This appreciation style is more geared towards marriage in terms of holding hands etc. In a workplace people who prefer physical touch are often the ‘huggers’ which can be very uncomfortable for a person who prefers to shake hands. A simple pat on the back can be uplifting for them.

It is extremely important to understand that each person has preferred appreciation styles; in order to motivate others unique approaches should be taken.
The website has multiple quizzes that can be beneficial in personal and professional relationships:

1. Love Languages for Children
2. Anger Assessment
3. Couples/Singles Appreciation Styles
4. Apology Styles

Assignment

1. Write down 10 team building ideas after brainstorming with the entire staff.
2. Write down 5 ideas for self-appreciation (example: going to the gym for acts of service and/or quality time).
3. Feel free to have your children and/or partner fill in the questionnaire if you wish.

Appreciation Tips

EXTROVERT REFERENCES:

1. Accept and encourage their enthusiasm.
2. Respect their independence.
3. Compliment them in front of others when possible.
4. Allow them to talk things out with others.
5. Let them shine!

INTROVERT REFERENCES:

1. Respect their privacy.
2. Get them lots of time to think things out.
3. Give them advance notice of staff meeting agendas.
4. Don’t interrupt them.
5. Let them observe first in new situations.

Appreciation Tips Continued

SENSING REFERENCES:

1. Give them standard operating procedures for new tasks.
2. Be practical and realistic.
3. Allow them to immediately apply what is communicated.

INTUITIVE REFERENCES:

1. Give them many options and possibilities.
2. Anticipate that they will appreciate change in their job positions.
3. Use Metaphors when explaining ideas and/or concepts.

THINKING REFERENCES:

1. Be calm and reasonable.
2. Provide honest feedback.
3. Let them analyze and critique changes.

FEELING REFERENCES:
1. Be supportive, nurturing and interested in others.
2. Let them connect with others and create a harmonious environment.
3. Appreciate their efforts often.

JUDGING PREFERENCES:
1. Be decisive and share decisions with them.
2. Provide clear expectations and guidelines.
3. Allow them time to organize their workspace.

PERCEIVING PREFERENCES:
1. Allow them flexibility in their schedule.
2. Provide a wide range of options when possible.
3. Take an easygoing approach to change.

Source: ‘Introduction to Type and Communication’ - Donna Dunning
Practical Options for Workplace Wellness
Marie K. Holowaychuk, DVM, DACVECC, Critical Care Vet Consulting

Introduction
Resilience is a person’s ability to properly adapt to stress and adversity in the form of family or relationship problems, health problems, or workplace and financial worries (Wikipedia). It is a quality that allows people to be knocked down by life and come back stronger than ever, rather than letting failure overcome them and drain their resolve (Psychology Today). Resilience allows for longevity of veterinary care providers in the profession and is reliant upon adequate self-care and wellness in the workplace.

Definition of Wellness
Wellness means living with a healthy balance of mind, body, and spirit that results in an overall feeling of wellbeing. It is more than health; it is a conscious, self-directed, and evolving multidimensional way of life. To achieve wellness, the body must be fueled, the mind engaged, and the spirit nurtured. There are many dimensions or pieces of wellness that need attention for people to fully flourish, but there does not have to be balance among all dimensions. It is important to recognize that wellness does not occur passively; it requires active awareness, acceptance, and commitment through choices made every day.

Components of Wellness
Wellness dimensions, often organized as wellness wheels, encompass aspects of a person’s wellness. Each piece interacts with another to result in physical and mental health and wellbeing.

1) Physical wellness is not just the absence of illness, but rather maintaining a thriving lifestyle. This dimension of wellness includes adopting health habits such as routine medical exams, immunizations, safety precautions, appropriate sleep hygiene, a balanced diet, regular exercise, and care for minor illnesses. It is also about avoiding or minimizing risky behaviors such tobacco, drugs, or excessive alcohol consumption. Most importantly, physical wellness is about understanding the body’s warning signs and discovering which healthy habits allow a person to feel better according to the lifestyle and level of mobility and fitness.

2) Spiritual wellness involves seeking and having a meaning and purpose in life, as well as participating in activities that are consistent with one’s beliefs and values. It is more than prayer and belief in a higher being; it is the development of a deep appreciation for the depth and expanse of life and natural forces that exist in the universe. A spiritually well person seeks harmony with the universe, expresses compassion towards others, and practices gratitude and self-reflection.

3) Emotional wellness encompasses optimism, self-esteem, self-acceptance, and the ability to experience and cope with feelings independently and interpersonally. Emotional wellness includes: practicing self-care; fostering inner resources and resilience; finding unique and healthy ways of coping with stressors; creating satisfying relationships; empathizing with others; being realistic about expectations and time; accepting and being aware of feelings; having capacity to manage feelings; knowing limitations; and knowing when to ask for help.
4) Intellectual wellness encourages participating in mentally stimulating and creative activities. Improving intellectual wellness can happen in and out of the veterinary profession. It is the ability to think critically, reason objectively, make responsible decisions, and explore new ideas and different points of view. It also emphasizes lifelong learning and inspires curiosity. It includes performing stimulating mental activities, expanding knowledge and skills, sharing gifts with others, pursuing personal interests, and keeping up on current issues.

5) Financial wellness includes having a healthy relationship with money, using skills to balance expenses and spending, making informed financial decisions and investments, setting realistic goals, and learning to prepare for short- and long-term financial needs or emergencies. Part of this dimension includes awareness that everyone’s financial values, needs, and circumstances are unique.

6) Social wellness focuses on contributing to the community and enhancing personal relationships with people. This dimension encourages taking an active part in improving communities, connecting with others, establishing supportive social networks, developing meaningful relationships, and creating safe and inclusive spaces. It allows forming interdependent relationships based on mutual commitment, trust, and respect.

7) Occupational wellness involves preparing for and participating in work that provides personal satisfaction and life enrichment that is consistent with a person’s values, goals, and lifestyle. This dimension includes taking a thoughtful and proactive approach to assessing personal satisfaction and performance in one’s work. To fulfill this dimension, a person should contribute unique gifts, skills, and talents to work that are both personally meaningful and rewarding.

8) Environmental wellness inspires people to live a lifestyle that is respectful of their surroundings. It involves understanding the dynamic relationship between the environment and people, as well as recognizing that people are responsible for the quality of the air, water, and earth. In turn, this affects the social, natural, and built environments that affect a person’s health and well-being. The environment plays a big role in personal wellbeing and includes the social (e.g., bullying, racism), natural (e.g., nature, climate), and built environments (e.g., living conditions).

Achieving Wellness
The health and wellbeing of veterinary professionals depends on their ability to care for themselves. As such, wellness is achieved by ensuring that individuals adhere to self-care strategies and goals within each of the wellness dimensions. Wellness is also fostered by a healthy organization culture and efforts of the leadership team to prioritize and protect employee wellbeing.

Responsibility for Wellness
There are a growing number of researchers and mental health professionals who believe that veterinary care providers and other caregiving professionals have a moral obligation to not only help patients, but more importantly, help themselves. Simply stated, if veterinary professionals do not care for themselves, eventually they will not be able to look after others. Ultimately, it is the individual’s responsibility to adhere to self-care. However, there are many ways that the employer can help to facilitate and prioritize wellbeing for employees.

Standards of Self-Care Practice
The Green Cross Academy of Traumatology is a non-profit organization of trained traumatologists and compassion fatigue service providers who put together standards of self-care guidelines that care providers are encouraged to abide by. The principles declare that it is unethical not to attend to self-care as practitioners, because sufficient self-care prevents harming those being helped. Ultimately, it a person’s own responsibility to care for his or herself and no situation or person can justify neglecting it. In other words, the duty to perform as a care provider cannot be fulfilled if there is not a concomitant duty to self-care.

Specific guidelines state that every care provider, regardless of his or her work role, has a right to wellness associated with self-care. In more detail, all veterinary professionals deserve a restful sleep and physical separation from work that sustains them in their role. Likewise, every veterinary professional deserves emotional and spiritual renewal, both in and outside of the work environment.

Role of Veterinary Team Members
Preferably, every veterinary professional should make a formal and tangible commitment to self-care, which is a written, public, specific, and measurable promise of self-care. The self-care plan would include set deadlines and goals connected to specific activities of self-care. The plan must be attainable, accompanied by commitment, and monitored by advocates of self-care. The plan could include strategies for the following:

1) Letting go of work when not working and embracing rejuvenation activities that are fun, stimulating, inspiring, and generate joy.
2) Acquiring adequate rest and relaxation tailored to interests and abilities and that normally result in rest and relaxation.
3) Practicing daily stress reduction methods that effectively manage stress during working hours and off-hours recognizing that they will probably be different.

Veterinary professionals must also exercise self-restraint with regards to what and how much they consume (e.g., alcohol, drugs, stimulants) since these can compromise their competence as a veterinary professional and affect their overall health and wellbeing. In addition, veterinary professionals must seek out and remember appreciation from clients and peers that sustain them emotionally and spiritually. They must also find colleagues that are committed to monitoring a person’s self-care efforts so that there is accountability and support.

Role of Veterinary Leadership
Toxic attitudes negatively affect veterinary team function and a toxic work environment occurs when toxic attitudes in the workplace are ignored or not addressed. Consequently, toxic attitudes can lead to relationship and task conflicts, which are negatively associated with team satisfaction and performance. It is imperative that toxic attitudes and environments are recognized and addressed, to foster wellness in the veterinary workplace.

Dealing with negative behaviors is a leadership responsibility; if leaders do not address the situation, there will be persistent dissatisfaction within the team. Therefore, this behavior cannot be ignored; rather, the instigator must be confronted, given the opportunity to correct the behavior, and given
consequences if the behavior does not change. Veterinary leaders might need additional training in communication and leadership skills to address these negative behaviors effectively.

A toxic environment occurs when there is broken communication and tension between staff members. This can occur due to employees lacking confidence, skills, or knowledge; employees not feeling appreciated; difficulties coping with staff turnover; mishandling conflict; or not holding chronically negative or hostile people accountable for their behavior. Effective leaders are important for resolving toxic work environments by ensuring adequate employee training, expressing appreciated to employees, taking measures to reduce staff turnover, successfully handling conflict, and holding people accountable for their actions.

Effect of Culture on Veterinary Wellness
Culture includes the people and ethos of the organization. It is how employees work together and towards a common purpose. It is dictated by the leaders and the vision they communicate and is the intention behind every interaction. Culture surrounds everyone and shapes their behavior tremendously. The heart of effective cultures is engagement; if employees are disengaged at work, it is unlikely that they will engage with wellness. In other words, if employees do not feel supported by their organization, managers, or co-workers, they are unlikely to engage in any wellness initiatives on a meaningful level.

Leaders must value employees, understand the importance of organizational culture, and recognize that wellbeing is a piece of the total employee experience. Wellness efforts that run counter to the underlying culture have the potential to foster feelings of skepticism and resentment.

At a minimum, employees must have their basic needs met to have a strong culture. Specifically, employees must: 1) have what they need to do their job; 2) feel appreciated and respected; 3) feel connected to one another; 4) feel like they have opportunities for growth; and 5) feel inspired to work towards their higher purpose.5

Nudges and Cues in the Veterinary Workplace
Research suggests that employees in companies that manifest a culture of health are three times more likely to engage in their own health and wellbeing compared to companies that do not.

Nudges are environmental prompts that make health and wellbeing choices the easier choices. Examples include signage, healthy options in vending machines, equipment, and intentional workplace design. In the veterinary practice environment, nudges could include workout rooms, nap rooms (for 24-hour hospitals), standing/treadmill desks, on-site health clinics (e.g., flu vaccines), and visual reminders to stretch/breathe between appointments/surgeries.

Cues are cultural prompts that make it normal (or abnormal) to engage in certain behaviors. Examples include policies, organizational values, rituals, communication, encouragement, recognition, and modeling (especially by leadership). In the veterinary practice environment, cues could include fitness programs and wellbeing information for new hires, walking meetings, mandatory breaks and vacation, hospital-wide stretch times, wellbeing tips in employee communications, and behaviors such as leaving work on time and not sending work emails on the weekend.
Practical Ways to Foster Workplace Wellness

Accountability: It is imperative that veterinary team members hold each other accountable for their self-care goals. For example, if a team member’s goal is to not work on weekends to spend more focused time with family, then the rest of the team could try to support that goal by not contacting the team member on the weekend. Likewise, if a team member is trying to get to yoga twice weekly, if they communicate this to their co-workers, everyone can try to help that person leave in time to get to their yoga class.

Group Challenges: It builds camaraderie and accountability to set goals for the entire veterinary team. For example, each month a practice could pick one wellness dimension to focus on and select a goal for everyone to strive towards. This goal could be tracked using electronic software or a chart posted in the break room of the hospital. Examples of group-related self-care goals in each of the wellness dimensions are as follows:

AVMA Workplace Wellness Initiatives

Participants in the 2015-2016 AVMA Future Leaders Program devised an initiative to improve wellness in the veterinary workplace. The toolkit is available on the AVMA website (www.avma.org/workplacewellness) and has 5 components: 1) a 5-minute video discussing the high risks of depression, suicide, and substance abuse in the veterinary profession; 2) a list from which employees can choose 4 healthy behaviors to improve workplace wellness (i.e., nutrition, physical fitness, mental well-being, and fun challenges); 3) discussion scenarios to stimulate dialogue regarding barriers to workplace wellness (e.g., lack of participation, indifference, time pressures, solo practitioners); 4) regular (2 per month) engaging discussions regarding wellness hurdles or check-ins with team members to briefly (< 20 minutes) allow people to listen and support each other; 5) designating a ‘wellness champion’ for the workplace to serve as an ambassador and resource to support a culture of wellness in the workplace via communication of initiatives, collection of feedback, and support of the organization’s goals.

Online Resources:

2. AAHA’s Guide to Veterinary Team Practice Wellbeing: https://www.aaha.org/professional/resources/healthy_workplace_culture_initiative.aspx
SUNDAY, JULY 8, 2018.

Companion Animal: Senior Cat Preventative Care and Management
Preventive Care Programs for the Aging Feline: Let’s go to the doctor!
Kelly St. Denis

Advances in medicine over the last few decades, along with the increasing popularity of the cat, have given us a new world of senior cats needing ongoing medical care. While we recommend annual preventive care for cats to our clients, senior cats need a minimum of biannual care. Opportunities for improving quality of life with or without quantity of life are multifold when caring for senior cats. This session will focus on ways to improve the senior life through the development of preventive care programs that provide frequent, attentive interaction with the client and patient.

Learning Objectives
1. Gain an understanding of the bodily changes associated with aging in cats
2. Attain ideas for Senior Preventive Care Strategies and how to implement them

This session is appropriate for: Veterinarians & Veterinary Technicians
Preventative Care Programs for the Aging Feline: ‘Let’s go to the Doctor!’

As cats reach their senior and geriatric years, our focus on their health needs to intensify. By now we have hopefully established a working relationship with our client(s) over the years of the patient’s life. While clients will be aware of their cat’s age in years, they may not recognize that those years are moving closer to the senior benchmark. What being senior means to their cats is also a puzzle. Clients often don’t understand exactly what changes to expect with their cat’s advancing years. The role of the veterinary team is to prepare, educate and support the client, while providing the best preventive care to senior patients. Above all, the goal of the veterinary team and the client should be to avoid patient suffering.

The American Association of Feline Practitioners (AAFP)/AAHA feline life stage guidelines define a mature cat as 7-10 years of age, a senior cat as 11 to 14 years of age, and a geriatric cat as 15 years of age and older (Vogt et al, 2010). For the purposes of this lecture, all cats over the age of 7 years will be collectively referred to as senior, unless otherwise noted. Once a cat reaches the age of 7 or 8 years, they should be seen for a healthcare assessment more frequently than once a year. For the healthy, this can be every 6 months, but for those with chronic health conditions, a move to healthcare assessments every 4 months will be beneficial. To further emphasize this need, consider that each year of an adult cat’s life might be equivalent to about 4 to 6 years of an adult human. This means that biannual health visits for senior cats is equivalent to a human visiting their doctor every 2-3 years. More frequent visits for senior cats are beneficial, allowing detection of disease early, thus improving the success of therapeutics and increasing quantity and quality of life (QOL).
TOP REASONS SENIOR CATS NEED TO SEE THE DOCTOR EVERY 6 MONTHS

- Changes can occur rapidly
- Many disease conditions begin to develop at this age
- Body weight, body condition score (BCS) and muscle condition score (MCS) changes can be detected early
- Cats are masters at hiding illness
- Owners may not recognize subtle changes as being significant
- Early disease detection often results in more successful disease treatment or management

The Aging Feline Body
The Subtle Signs of Sickness
Many practitioners are aware of the ‘subtle signs of sickness’ in cats. We understand that cats are masters at hiding illness. We understand and seek to help our clients recognize what subtle changes can mean with regard to feline health. In addition to being a sign of disease, we also have to recognize these subtle changes as evidence of possible pain and/or cognitive decline.

1. Inappropriate Elimination Behavior or Litter Box Use
2. Unexplained Weight Loss or Gain
3. Changes in Interaction
4. Changes in Grooming
5. Changes in Activity
6. Signs of Stress
7. Changes in Sleeping Habits
8. Changes in Vocalization
9. Changes in Food and Water Consumption
10. Bad Breath

Recording Normal Behaviors
Clients should consider starting a journal to highlight the normal patterns of behaviour of their cat. Timing of eating, elimination behaviors, sleep, and play, when documented, will act as an excellent resource when attempting to identify changes. This type of recording should begin as early as kitten hood. For the clinician, this journal can also assist when end-of-life decisions have to be made. Quality of life discussions are difficult at best. Knowing the level of changes that have occurred in behavior patterns over time help the client to come to terms with end-of-life decisions.

Disease Concerns
Senior and geriatric patients are at increased risk of disease in general. Risks of conditions such as chronic renal disease and hyperthyroidism increase with age. Older patients are also at increased risk of neoplasia, hypertension, cardiac issues and of course, degenerative joint disease (DJD). Dental disease and pain are common.
Pain and Degenerative Joint Disease
A variety of diseases and conditions will predispose the senior patient to pain. Even if the patient is apparently healthy, the consideration of pain secondary to DJD is critical. DJD should always be on the clinician’s radar when examining senior cats. Pain related to DJD can be a significant concern. Clients may be observing mobility changes, stiffness and possibly lameness. There are many therapeutic options available to the clinician. These should be selected based on the patient history, health status, MDB and the actual source of pain. The author frequently prescribes gabapentin as a pain medication for cats with arthritis, debilitating disease and dental disease. While daily, long-term use of non-steroidal anti-inflammatory drugs (NSAID) are frequently off-label in the feline species, these can be beneficial as an adjunct therapy. Other pain medications that the practitioner may consider for various types of pain include buprenorphine, tramadol, or amantadine. Many neutraceuticals are available for feline arthritis. These include omega fatty acid supplements, glucosamine and chondroitin, injectable polysulfated glycosaminoglycans (Adequan) and injectable pentosan polysulfate sodium (Cartrophen). Environmental management including provision of easy access food, litter and sleeping areas are beneficial to the DJD patient.

Nutritional Needs
In the early senior years, up to 11 years of age, a cat’s energy needs will decrease by 3% per year. However, at the age of 12 and up, the energy needs actually increase. As cats age, they become less efficient at digesting food, in particular fats and proteins. Senior & geriatric feline patients can be susceptible to unexplained weight loss, including natural muscle wasting (sarcopenia). Dietary palatability is a major concern at this age, as olfactory function and taste bud function diminish. It is critical to regularly ensure that the patient is consuming sufficient calories to meet their maintenance energy requirements (MERs). The veterinary team should have a good understanding of the senior, mature and geriatric veterinary diets available, and should be able to promote these to the client.

Cognitive Changes
Behavioral problems in the geriatric cat may be explained by the presence of disease and pain. Treatment of the disease, and/or treatment of pain will often resolve behavioral changes. Howling may be observed in some cases of hyperthyroidism, as well as patients with hypertension. Changes in elimination, including soiling outside of the litter box can occur with conditions such as diabetes mellitus, renal disease, lower urinary tract disease, hyperthyroidism and neoplasia. Pain can lead to many changes in behavior including, but not limited to, elimination issues, irritability, increased sleeping, howling, decreased grooming and decreased mobility. Regular clinical testing as well as pain management will help identify disease and pain-related causes of behavior changes. In some cases, cognitive dysfunction syndrome (CDS) may be the primary source behind the behavior changes noted. Although there are no specific diagnostic criteria for CDS in cats, ruling out other causes and treating for pain will help the clinician form a presumptive diagnosis. Cognitive dysfunction signs in cats can include disorientation (time or space), altered learning and memory, house soiling, altered interactions with the client, activity changes, sleep pattern changes, alterations in appetite, and/or decreased grooming. Vocalization may also occur.

Unique Environmental Needs
The senior and geriatric feline will have changes associated with their five senses, as well as reductions in strength and mobility. These changes will impact environmental needs. Changes in play items and structures, sleeping areas and litter boxes will need to be considered. Litter box locations and wall heights will need to be addressed. As arthritis needs are addressed with pain management and other care, environment changes will reduce the stresses on the musculoskeletal system. Mental stimulation in the form of interactive play, food puzzles and a variety of toys keep the senior mind sharp with the goal of slowing cognitive decline.

**Setting up Senior Preventive Care Programs**

Preparing the client for the upcoming senior years is an important part of implementing senior care programs. It is recommended to start the discussion early, remarking on how well the young adult cat is doing, what is going right, and how things start to change around 7 years of age. At 6 years of age, consider discussing future need for diagnostic testing and biannual visits. A senior care preventive package will make increased visits and laboratory testing more appealing to the client. There are many excellent resources available for clients including pamphlets from the AAFP on senior care as well as information at the Cat Healthy site and The Cat Community (See Resources). In preparation for the senior years, the veterinary team should:

- Guide clients in what to be aware of at home (exact intake, activity, sleeping, grooming etc)
- Encourage clients to quantitate and record the daily food intake
- Prescribe analgesic trials if DJD pain is a viable concern
- Prepare clients for the costs of associated laboratory testing and when these tests are recommended
- Offer discounts for full workups or develop prepaid programs that include complimentary nail trims and/or one complimentary emergency examination/consultation
- Pre-book 6 month visits at checkout
The Senior Care Prevention Package

| More frequent visits | q.4 months for cats with chronic health conditions  
|                      | q.6 months for healthy senior cats  
| Detailed history     | nutrition, actual intake, drinking  
|                      | mobility  
|                      | changes in behaviour  
|                      | elimination patterns  
|                      | vomiting, diarrhea, coughing, sneezing  
| Meticulous physical examination | thorough PE, weight, BCS, MCS  
|                          | include retinal assessment  
|                          | assess for cervical nodules (thyroid)  
|                          | pain assessment  
| Minimum Database       | Clinical chemistry, CBC, Total thyroid 4, Urinalysis, blood pressure, FeLV/FIV  
| Pre-booked visits      | Scheduled prior to checkout!  
| Package discounts or other offers to consider | Prepaid packages 6-12 months in advance  
|                          | Discounted lab fees  
|                          | Complimentary nail trims  
|                          | Complimentary emergency examination  

The Consultation

Increasing age comes with an increased risk of disease. Regular monitoring for evidence of problems includes a detailed history, physical examination and collection of a minimum data base (MDB). Every consultation should include determining weight, Body Condition Score (BCS) and Muscle Condition Score (MCS). These values should be compared to previous values in order to identify trends. The physical examination should be meticulous and thorough. Thorough evaluation of the patient’s eyes, and in particular the retinas, is something that is often overlooked. The retinas can provide early indication of hypertension, and thus their status should be assessed and recorded with every senior feline visit. Observations of unexplained changes in weight, BCS, MCS, behavior, appetite, drinking, elimination behavior and grooming need to be addressed by the client and clinician in a timely fashion.

The Minimum Database

The clinician should evaluate a MDB for every senior patient. The MDB includes a total thyroid, clinical chemistry, complete blood count and urinalysis. Urinalysis may results may indicate the need for further testing such as urine culture and/or urine protein creatinine ratio. Where renal disease is identified, thorough IRIS staging will target therapies and improve prognosis as well as quality of life.

Minimum database

| Mature Cats (7-10 years) | Senior/geriatric (>10 years) |
### Evaluating the Data

With each visit, a large amount of data will be collected from the history, PE and MDB. These should be summarized and evaluated by the clinician, and discussed with the client. Charts trending MDB numbers should be updated with each visit, allowing early identification of negative trends. For example, upward trends in creatinine from low normal (IRIS stage 1) to high normal (IRIS stage 2) along with a drop in urine specific gravity will alert the clinician to progressive chronic kidney disease. A report card can be provided to the client, summarizing the findings and giving an overall health evaluation or score. This type of report will have value to the client both for the money spent as well as understanding their cat better. Nutritional reports will help ensure that appropriate daily intake is met, thus reducing the risk of malnutrition and weight loss.

### Key Points

1. The senior years come earlier than a lot of clients realize
2. Clients need to be prepared for the senior years with an understanding of what to expect in their aging cat
3. Preventive care programs for seniors should include biannual visits
4. Preventive care programs improve the veterinary-client, the veterinary-patient bond and the client-patient bond. This reinforces a team approach to maintaining patient QOL.

### Resources:

How Do I Know if my Cat is in Pain?  https://www.catvets.com/guidelines/client-brochures

Degenerative Joint Disease in Cats  https://www.catvets.com/guidelines/client-brochures

Friends for Life: Caring for Your Older Cat  https://www.catvets.com/guidelines/client-brochures

Mature & Senior Cats: 8+ years  
References:

The aging process can cause many changes in the senior cat. From decreased digestion and metabolism to musculoskeletal changes, the senior feline body demands a unique dietary approach. Nutritional support and body condition management is a critical component of senior preventive health care. The session will review the changes in nutritional needs of senior cats with an overview on those conditions that predispose to weight loss. Metabolic and resting energy requirements and how to implement them in clinical practice will be examined. In particular, the session will focus on identifying and managing the most challenging case, the cat with unexplained weight loss.

Learning Objectives
1. Learn how to trend body weight, as well as assess and trend body and muscle condition scoring (BCS and MCS).
2. Learn how to determine metabolic energy requirements and how to implement an individualized nutritional program

This session is appropriate for: Veterinarians & Veterinary Technicians

The Nutritional Needs of Senior Cats
As cats age, their caloric and nutritional needs change. Early on in the aging process, from 7 to 11 years of age, a cat’s energy needs will decrease by 3% per year. However, at the age of 12 and up, the cat’s energy needs actually increase. A number of factors make the senior or geriatric feline patient more susceptible to weight loss. This includes changes in their digestive tract, appetite, cognition and the presence of disease and/or pain. Changes can occur rapidly, making senior biannual care and the development of nutritional support programs critical to the health and well-being of the senior feline patient.

Changes in Digestion
There is a poor understanding of changes in digestion associated with age in the feline species. Much of the information we do have is extrapolated from human medicine. It is evident that cats do experience a decline in digestibility of energy and other nutrients. Changes in appetite are a major concern in senior cats. A reduction in appetite can be associated with oral disease including oral pain, periodontal disease, and oral neoplasia, as well as difficulty with prehension and/or swallowing. It is assumed that cats will experience a reduction in olfactory and taste bud function as they age, also negatively impacting appetite. As a result, dietary palatability is a major concern in this age group. Esophageal functional changes noted in humans are likely to occur in cats, including a decreased clearance of esophageal contents that predisposes to dysphagia and gastroesophageal reflux. Although GI transit times have been shown to be similar between young (3.0 +/- 0.9 years) and senior cats (11.6 +/- 1.4 years), there is a larger variability in total transit time between individual senior cats. Rapid transit times may predispose to mal-digestion. Slower transit times may result in bacterial overgrowth and stool dehydration, potentially predisposing to constipation. Reduced colonic motility is an additional potential factor in the development of constipation. As cats age, they also become less efficient at digesting fats and proteins.
Maintenance Energy Requirements

Ensuring that the patient is consuming sufficient calories to meet their maintenance energy requirements (MERs) is a mainstay to senior nutritional support programs. The maintenance energy requirements (MER) are those calories required to supply both resting energy requirements (RER) as well as calories needed to support age or a diseased state. In cases where weight loss is required, the MER may be less than the calculated RER. The average RER for cats is about 70 kcal/day/kg metabolic body size. The RER can be estimated based on the patient’s target weight by the equation:

\[ \text{RER (kcal/day)} = 70 \times (\text{body weight in kg})^{0.75} \]

OR

\[ \text{RER (kcal/day)} = 30 \times (\text{body weight in kg}) + 70 \]

### Maintenance energy requirements (0.8 to 1.6 x RER)

<table>
<thead>
<tr>
<th></th>
<th>Maintenance energy requirements (0.8 to 1.6 x RER)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Average, neutered, healthy adult</td>
<td>= 1.2 x RER (most pets are neutered)</td>
</tr>
<tr>
<td>Intact adult</td>
<td>= 1.4 x RER</td>
</tr>
<tr>
<td>Active adult</td>
<td>= 1.6 x RER</td>
</tr>
<tr>
<td>Obese prone</td>
<td>= 1.0 x RER</td>
</tr>
<tr>
<td>Weight loss</td>
<td>= 0.8 x RER</td>
</tr>
<tr>
<td>Critical care</td>
<td>= 1.0 x RER</td>
</tr>
<tr>
<td>Weight gain</td>
<td>= 1.2-1.4 x RER at ideal weight</td>
</tr>
<tr>
<td>Geriatric</td>
<td>= 1.1 x RER</td>
</tr>
</tbody>
</table>


Body weight, Body Condition & Muscle Condition Scoring

Practices should develop a recording system for body weight, body condition score (BCS) and muscle conditioning score (MCS). Trends in these values should be observed, with individual patient assessment scheduled at regular intervals throughout the year. While the patient may be seeing the clinician every 4-6 months, it may be useful to have the patient visit with a registered veterinary technicians every 2-4 months for assessment of body weight, BCS and MCS. Trends in these values can be the first indicators of disease or failure to successfully control known diseases. The World Small Animal Veterinary Association has developed easy to use guidelines for BCS and MCS. Alternative scoring systems are available, including those outlined by AAHA. Practices should choose one set of standardized charts for all employees to follow, in order to generate as much consistency as possible. The images below are available in pd format and can be printed and framed for use in the veterinary consultation room.
Body Condition Scoring

Body condition scoring is useful for identifying obese patients that will require weight loss as well as those patients that are poorly conditioned for their body size. When used in conjunction with MER and body weight, the BCS is a valuable tool to determining dietary recommendations. Clients should be directly involved in assessment of their cat’s BCS by providing visual aids in the consultation room. This facilitates a better understanding of the patient status and needs, thereby improving compliance. The BCS should be recorded at every visit.

Muscle Condition Scoring

Muscle condition scoring is useful in cats for early detection of loss of muscle mass as an indicator of disease, as well as for monitoring cats with chronic diseases. Evaluation of muscle mass includes visual examination and palpation over the temporal bones, ribs, lumbar vertebrae, and pelvic bones. The clinician should also note those cats exhibiting overcoat syndrome, which is a loss of muscle mass in spite of obesity and elevated body condition score. The appearance of these cats can sometimes be deceptive, as they continue with elevated BCS in the face of diminishing muscle condition. Clients should be directly involved in assessment of their cat’s MCS by providing visual aids in the consultation room. This facilitates a better understanding of the patient status and needs, thereby improving compliance. The MCS should be recorded at every visit.

Weight loss in the Senior Cat

Monitoring trends in body weight, BCS and MCS allow the veterinary team to identify negative trends early. Unexplained weight loss can commence in the feline species as early as 1-2 years prior to disease onset. When body weight loss is identified and not intentional, the cause will need to be...
identified. Since clients rarely record, measure or report dietary intake, the first goal is to ensure that the cat is taking in enough calories. Ensuring adequate intake is critical in senior and geriatric patients to promote health and proper immune function, as well as to avoid situations of muscle wasting and cachexia due to insufficient protein intake. Competition for food, disease, pain or cognitive dysfunction may be contributing factors to reduced intake.

**Approach to Weight Loss in the Senior Cat:**
- Determine food fed (name, flavor, type, manufacturer, caloric content)
- Determine Amounts OFFERED and Amounts EATEN
- Ensure other pets are not eating patient’s food
- Compare to MERs
- Obtain complete history
- Perform complete PE
- Obtain complete MDB
- Pursue unidentified differentials
Medical conditions causing weight loss in the senior cat

- Impaired digestion
- Systemic disease: chronic kidney disease, hyperthyroidism, neoplasia,
- Gastrointestinal disease: intestinal parasites, inflammatory bowel disease, pancreatitis, intestinal mural lymphoma, other neoplasia
- Cardiac disease
- Infectious disease
- Oral disease: dental disease, oral neoplasia, EGC, pathologic fractures, other
- Cognitive dysfunction

**Nutritional Support Programs**

A nutritional consultation includes assessment of body weight, MCS and BCS, as well as accurate review diet type and actual intake. Nutritional consultations should be completed by one or two designated veterinary technicians in the practice. Staff should arrange and encourage communication by whatever means the client is most comfortable, including telephone, email or text messaging. Staff should encourage clients to ask questions and express any concerns as they arise. This type of ongoing support is important for both body condition management as well as management of disease. Conversations with the client may help the veterinary staff identify issues that the client has not noticed or has not considered important enough to report.

Each clinic should develop its own program for monitoring and supporting the individual nutritional needs of patients. Registered veterinary technicians are excellent resources for this type of program, and can often handle managing these programs, reporting to the clinician as concerns arise.

Biannual visits with the veterinarian will help detect body condition or body weight changes, but regular visits in between these times should also be considered. A visit every 2-4 months with the veterinary technician for a weigh-in, BCS, MCS and nutritional assessment should be considered.

**Key Points**

1. Senior and geriatric cats have unique nutritional needs
2. Digestion, body and muscle condition change with age
3. Weight loss and associated causes are best identified early
4. Body weight, BCS and MCS should be recorded and trended with every visit
Resources:
https://www.wsava.org/sites/default/files/Body%20condition%20score%20chart%20cats.pdf
https://www.wsava.org/sites/default/files/Muscle%20condition%20score%20chart-Cats.pdf

References:
Cognitive Dysfunction in the Senior Cat: Why did I come into this room?
Kelly St. Denis

As cats age, they are at increased risk for a number of common diseases. Many health conditions in the senior cat can change behaviour, either through direct effects on the body and/or through pain. Helping clients sort out changes in behaviour includes identifying or ruling out disease, identifying and treating pain and finally identifying what part of the behaviour changes are occurring as a result of senile changes in the cat’s brain. Age-related cognitive dysfunction has not been well studied in the cat, but the last two decades have witnessed a growth in the information available to us. This session will focus on helping the clinician diagnose cognitive dysfunction in the senior cat, with a review of our current understanding of the condition in cats, the associated clinical signs, available therapeutics and environmental management.

Learning Objectives
1. Understand what behavior changes are expected with age and disease, and what changes might be related to cognitive dysfunction syndrome
2. Understand options for care when a patient is diagnosed with cognitive dysfunction syndrome.

This session is appropriate for: Veterinarians & Veterinary Technicians

Cognitive dysfunction syndrome (CDS) is a neurological condition that is well recognized in aging dogs. As we have increased our understanding of this condition in dogs, it has become clear that this degenerative disease also occurs in aging cats. The disease is characterized by gradual cognitive decline associated with degenerative changes in the brain. As many of the clinical signs associated with CDS overlap with those of common feline disease, diagnostic testing to rule out disease is an essential step in diagnosing CDS.

Identifying Signs of Disease
Many practitioners are aware of the ‘subtle signs of sickness’ in cats. We understand that cats are masters at hiding illness. We understand and seek to help our clients recognize what subtle changes can mean with regard to feline health. In addition to being a sign of disease & pain, we also have to recognize some of these subtle changes as evidence of possible CDS.

The Subtle Signs of Sickness
11. Inappropriate Elimination Behavior or Litter Box Use
12. Changes in Interaction
13. Changes in Activity
14. Changes in Sleeping Habits
15. Changes in Food and Water Consumption
16. Unexplained Weight Loss or Gain
17. Changes in Grooming
18. Signs of Stress
19. Changes in Vocalization
20. Bad Breath
**Cognitive Changes**

Behavioral problems in the geriatric cat may be explained by the presence of disease and pain. Treatment of the disease, and/or treatment of pain will often resolve behavioral changes. Howling may be observed in some cases of hyperthyroidism, as well as patients with hypertension. Changes in elimination, including soiling outside of the litter box can occur with conditions such as diabetes mellitus, renal disease, lower urinary tract disease, hyperthyroidism and neoplasia. Pain can lead to many changes in behavior including, but not limited to, elimination issues, irritability, increased sleeping, howling, decreased grooming and decreased mobility. Regular clinical testing as well as pain management will help identify disease and pain-related causes of behavior changes. In some cases, cognitive dysfunction (CD) may be the primary source behind the behavior changes noted. Although there are no specific diagnostic criteria for CD in cats, ruling out other causes and treating for pain will help the clinician form a presumptive diagnosis. Cognitive dysfunction signs in cats can include disorientation (time or space), altered learning and memory, house soiling, altered interactions with the client, activity changes, sleep pattern changes, alterations in appetite, and/or decreased grooming. Vocalization may also occur.

**Diagnostic Criteria**

There are no specific diagnostic criteria for the diagnosis of cognitive dysfunction (CD) in cats. In many instances, it is a diagnosis of exclusion. Diseases that can contribute to behavioural changes must be ruled out, or under control with appropriate therapeutics. Clinical signs consistent with CDS should be present. The mnemonic DISHA is used as a means of diagnosing CD in dogs (Disorientation, Interactions, Sleep-wake, House-training, Activity). This may be of some use in the diagnosis of feline CD.

<table>
<thead>
<tr>
<th>Clinical Signs of CDS#</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Disorientation</td>
<td>Spatial disorientation</td>
</tr>
<tr>
<td>Interactions</td>
<td>Changes in interactions with family members, other pets, visitors*</td>
</tr>
<tr>
<td>Sleep-wake</td>
<td>Alterations in sleeping patterns, sleeping locations</td>
</tr>
<tr>
<td>House-training</td>
<td>House soiling</td>
</tr>
<tr>
<td>Activity@</td>
<td>Increases or decreases in activity levels, changes in activity patterns, restlessness, repetitive behaviours; wandering</td>
</tr>
</tbody>
</table>

FOR CATS ADD: Excessive Vocalization

Altered responses to stimuli

Decreased self-hygiene

Alterations in Appetite

- Common finding@
- Increased, decreased responses, anxiety, irritability
- Rule out medical problems, pain
- Increase or decrease in appetite

#adapted from Landsberg et al 2016

*most common presenting complaint in prevalence study in 11 to 14 year old cats

@most common presenting complain in prevalence in 15-21 year old cats (Gunn-Moore et al 2007)
Therapeutics
Therapies for CD are not well-studied in the feline species. Selegiline (Anipryl) has been utilized for the treatment of CD in cats. Nicergoline (Fitergol), Propentofulline, (Vivitonin), oxazepam/orazepam/clonazepam, buspirone and fluoxetine have also been used. Omega fatty acid supplements and other antioxidants may be beneficial. Environmental adjustments should be made to accommodate patient needs. Pain management is a mainstay component of care in senior and geriatric cases of CD.

Unique Environmental Needs
Senior and geriatric cats have unique environmental needs. This mainly stems from their likely reduced mobility secondary to arthritis, but can also be related to cognitive dysfunction either as a primary or secondary (to other disease) problem. The senior patient may no longer feel comfortable running to the basement to use the litter box. Placement of boxes throughout the house is recommended. A high walled litter box may be viewed as a painful challenge to be avoided. Use of low entry or low walled, uncovered litter boxes is recommended. Assistance with access to higher furniture such as beds and window sills can be accomplished with steps or platforms to reduce necessary jumping heights.

Key Points
1. Cognitive dysfunction syndrome is a recognized condition in aging cats
2. Diagnosis of CDS is primarily based on excluding medical causes of related clinical signs
3. Therapeutic trials are needed to evaluate the efficacy of various pharmaceuticals in feline CDS
4. Environmental management and mental stimulation are key aspects of therapy

References:
2. Landsberg G, Denenberg S, JA Araujo. Cognitive dysfunction in cats: a syndrome we used to dismiss as ‘old age’. JFMS 2010: 12 (11); 837-848
For cats that are diagnosed with CKD, it is critical that practitioners and clients work together to stage and monitor the disease. We now have many tools at our fingertips to improve both quality and quantity of life in the feline CKD patient. None of these tools is more powerful than the ability to stage and monitor our patients through the International Renal Interest Society (IRIS) staging guidelines. This session will review the guidelines in their latest form, including the relevance of SDMA testing to the CKD patient. This seminar will provide a practical approach to diagnosing and monitoring the feline CKD patient.

Learning Objectives
1. Understand IRIS staging including the significance of creatinine and SDMA
2. Understand IRIS sub-staging including how to interpret proteinuria and blood pressure changes

This session is appropriate for: Veterinarians

Decline in kidney function can result from a variety of causes including pyelonephritis, amyloidosis, polycystic kidney disease, neoplasia, nephrotoxicosis, hydronephrosis and chronic glomerulonephritis (Scherk, 2011). Although acute insult can lead to chronic kidney disease (CKD), age seems to be the only major, consistent risk factor associated with chronic renal insufficiency (White, 2011).

Mature cat visits ideally include a complete physical examination/consultation as well collection of a minimum database (MDB) every 4 to 6 months. A minimum database for mature cats includes a full clinical chemistry, a total thyroid test (TT4), a complete blood count, a urinalysis and a blood pressure (BP) series. Blood urea nitrogen (BUN) and creatinine have traditionally been the go-to serum values for diagnosis of kidney disease. Early diagnosis can be challenging utilizing only these values, as azotemia does not develop until there is 75% loss of nephron function. The BUN can be influenced by factors other than renal disease, including dehydration, dietary protein content, gastrointestinal bleeding and hepatic insufficiency. Creatinine is a more reliable indicator of glomerular filtration rate (GFR). However, creatinine can be influenced by muscle wasting and by dehydration. Routine screening of these values can assist the clinician in documenting upward trends in these values.

Symmetrical dimethylarginine (SDMA) measures the methylated form of the amino acid arginine. This is a by-product of protein degradation excreted by the kidneys, which increases when there is approximately 40% loss of kidney function. This analyte can be impacted by dehydration. Symmetrical dimethylarginine is not a stand-alone test and should always be interpreted in light of patient status as well as other laboratory findings. Elevated SDMA in the absence of any other evidence of renal disease should be re-evaluated in a reasonable time frame.

It is recommended that urine samples be collected by cystocentesis whenever possible, and tested immediately in the clinic laboratory. Free-flow collection will result in contaminants from vulvar or preputial skin including bacteria, epithelial cells and potentially immune cells. Changes in pH may...
also occur in voided samples as they are exposed to the external environment. This will make interpretation of the results more challenging, and interfere with necessary culture and sensitivity testing. Delays of less than 15 minutes in assessment of the urine can lead to false changes such as increased bacteria and the presence of struvite crystals. The latter phenomenon is exaggerated in concentrated urine (Sturgess et al, 2001).

Samples taken from the patient for cystocentesis should have a new needle placed on the syringe, with 0.5 mL injected immediately into a sterile, red top tube. This sample should be labeled and placed immediately into the fridge for possible urine culture and sensitivity testing. Urine samples with a low urine specific gravity (USG; less than 1.035) should be submitted for urine culture regardless of sediment activity. Even in cases with ‘quiet’ sediment, dilute urine results in an environment that is predisposed to infection. In this dilute environment, occult infection may be present. If there is sediment including blood, white blood cells and/or bacteria, a urine culture is also indicated. Samples prepared for urine culture at an outside laboratory should be kept in the red top tube in the fridge.

The urine specific gravity (USG) can be impacted by age, diet and hydration status. Determination of USG should be based on a refractometer rather than relying on the urine chemistry stick as these can be inaccurate. The value should be interpreted in light of the patient status. Generally speaking values less than 1.035 are considered to be low. Urine specific gravity varies throughout the day, such that a single low USG is not reliable evidence of a loss of concentrating ability (Scherk, 2011).

Table 1 Urine Specific Gravity Varies with Age & Diet (Adapted from Scherk, 2011)

<table>
<thead>
<tr>
<th>Age or condition</th>
<th>Expected USG</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>4-8 weeks of age</td>
<td>1.020-1.038</td>
<td></td>
</tr>
<tr>
<td>8+ weeks of age</td>
<td>Up to 1.080</td>
<td>Denotes age at which full concentrating ability is reached</td>
</tr>
<tr>
<td>Dehydrated, normal renal function</td>
<td>&gt;1.040</td>
<td>Diet dependent (wet vs dry)</td>
</tr>
<tr>
<td>Canned food only, normal renal</td>
<td>&gt;1.035</td>
<td></td>
</tr>
<tr>
<td>function</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dry food only, normal renal</td>
<td>&gt;1.035-1.040</td>
<td></td>
</tr>
<tr>
<td>function</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Inability to concentrate urine</td>
<td>1.008-1.012</td>
<td>Nephrons no longer able to modify glomerular filtrate</td>
</tr>
<tr>
<td>Isosthenuria</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dehydrated/unknown renal function</td>
<td>1.007-1.039</td>
<td>Suggestive of renal insufficiency with or without azotemia</td>
</tr>
</tbody>
</table>

International Renal Interest Society (IRIS) http://www.iris-kidney.com
For cats that are diagnosed with CKD, it is critical for practitioners to develop and promote a relationship with clients that will allow continued monitoring of the disease, including disease staging. The application of human IRIS staging guidelines to the study of feline renal disease has dramatically advanced our ability to tailor our patient therapy, thereby improving quantity and quality of life. In addition to the MDB as discussed above, imaging is likely to be beneficial. Currently the creatinine is the main data parameter used to guide IRIS staging in cats. More recently, SDMA has been added to staging tables. Looking carefully at Table 2, one can clearly see that cats with creatinine values within the normal range may be in the process of developing kidney compromise. It is for this reason that clinicians should not merely scan blood work for values outside of the normal range, but rather the creatinine should be trended in a patient-specific chart. This will allow a more clear view of what might be progressing in the individual patient. All CKD cats in IRIS stage 3 and 4 were once in IRIS stage 1 and 2.

Table 2. IRIS Staging Guidelines

<table>
<thead>
<tr>
<th>Stage</th>
<th>Renal Azotemia</th>
<th>Creatinine</th>
<th>Clinical signs</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Non-azotemic</td>
<td>&lt;140 µmol/L</td>
<td>Absent*</td>
</tr>
<tr>
<td>2</td>
<td>Mild</td>
<td>140-249 µmol/L</td>
<td>Mild or absent</td>
</tr>
<tr>
<td>3</td>
<td>Moderate</td>
<td>250-439 µmol/L</td>
<td>Moderate</td>
</tr>
<tr>
<td>4</td>
<td>severe</td>
<td>&gt;440 µmol/L</td>
<td>Severe</td>
</tr>
</tbody>
</table>

Adapted from AAFP 2015 Dru Forrester, DVM, MS, DACVIM & Jane Robertson, DVM, DACVIM Chronic Kidney Disease: Making the most of early diagnosis

Table 3. Subclassifications of IRIS staging: Proteinuria

<table>
<thead>
<tr>
<th>Urine Protein:Creatinine Ratio (UPC)</th>
<th>Substage</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;0.2</td>
<td>Non-proteinuric (NP)</td>
</tr>
<tr>
<td>0.2-0.4</td>
<td>Borderline proteinuric (BP)</td>
</tr>
<tr>
<td>&gt;0.4</td>
<td>Proteinuric (P)</td>
</tr>
</tbody>
</table>

 Taken from AAFP 2015 Dru Forrester, DVM, MS, DACVIM & Jane Robertson, DVM, DACVIM Chronic Kidney Disease: Making the most of early diagnosis

True proteinuria in cats is a known marker of poor prognosis in renal disease (Syme, H.M. et al, 2006; Syme, H.M., 2009). If proteinuria is established on the chemistry stick in the absence of active sediment, the sample will need to be submitted for a urine protein creatinine ratio (UPCR). As the sensitivity and/or specificity of the protein chemistry stick may be low, cats with a documented history of CKD should have routine assessment of UPCR ratios. The result should be used to direct therapy with medications to reduce the loss of protein into the urine. Ratios over 0.4 are significant and therapy is needed. If there is active sediment in the presence of proteinuria on the chemistry
stick, and the UPCR is very high (>0.5), then the value may be significant and therapy may be indicated.

Blood pressure changes can be impacted by and/or impact the renal state of health (Brown, 2011). Sixty-five to 100% of cats with hypertension have evidence of reduced renal function (Jepson, 2011). The gold standard for blood pressure assessment in any species is central venous catheter assessment. Blood pressures can be measured non-invasively either by Doppler or oscillometric methods, but accuracy varies and can be poor. An understanding of the method used, the individual machine and each individual patient is key to reducing misinterpretation of readings. Patient stress can be a limiting factor in blood pressure assessment. Proper use of pain management in advance, as well as following cat friendly practice and handling guidelines will significantly reduce stress. Therapeutics for hypertension should only be prescribed once the blood pressure has been confirmed to be elevated in the presence of target organ damage (TOD). Target organ damage includes changes in kidney parameters, retinal health, cardiovascular parameters and neurological health. The latter is often manifested as unexplained changes in behavior.

Table 4. Subclassifications of IRIS staging: Blood pressure

<table>
<thead>
<tr>
<th>Systolic BP (mmHg)</th>
<th>Diastolic BP (mmHg)</th>
<th>Substage</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;150</td>
<td>&lt;95</td>
<td>Minimal risk (N)</td>
</tr>
<tr>
<td>150-159</td>
<td>95-99</td>
<td>Low Risk (L)</td>
</tr>
<tr>
<td>160-179</td>
<td>100-119</td>
<td>Moderate Risk (M)</td>
</tr>
<tr>
<td>&gt;180</td>
<td>&gt;120</td>
<td>High Risk (H)</td>
</tr>
</tbody>
</table>

Taken from AAFP 2015 Dru Forrester, DVM, MS, DACVIM & Jane Robertson, DVM, DACVIM Chronic Kidney Disease: Making the most of early diagnosis

Therapeutics, Monitoring and Maintenance
With the utilization of IRIS staging, the clinician gains significant ground in combatting chronic renal disease in cats. The data collected for the purpose of IRIS staging allows a tailored, individual approach to patient therapy. Early identification and management of CKD, before creatinine levels are elevated above normal give the practitioner the ability to slow the progression of disease, thereby improving quantity, and more importantly, quality, of life.

All CKD cats in IRIS stage 3 and 4 were once in IRIS stage 1 and 2.

Therapeutics are discussed in the subsequent lecture, Feline Chronic Kidney Disease Part 2: Therapeutics

Key Points
1. Creatinine values should be trended for changes based on IRIS Stage, not so-called ‘normal’ ranges for creatinine
2. IRIS staging plays a valuable role in assessing patient status and choosing therapeutics
3. Every cat in IRIS stage 3 and 4 was once in IRIS stage 1 and 2
4. The clinician has the right tools to slow the progression of CKD and thereby improve quantity and quality of life

References:

As we move forward with a greater, more detailed understanding of feline CKD, our options for treatment have become seemingly endless. This session will continue with the topics discussed in Part 1, focusing on therapeutic options. An emphasis will be placed on choosing the right therapies to manage patient and client quality of life.

Learning Objectives
1. Learn how to tailor therapeutics to individual patient IRIS staging and sub-staging
2. Gain knowledge on available therapeutics for CKD, their utility and how to apply these with consideration for quality of life

This session is appropriate for: Veterinarians

With the utilization of IRIS staging, the clinician gains significant ground in combatting chronic renal disease in cats. The data collected for the purpose of IRIS staging allows a tailored, individual approach to patient therapy. This leads to increased quality of life as well as longevity. Client compliance is critical, both in administration of medications as well as regular monitoring. Monitoring of renal patients every 4-6 months is recommended. Full IRIS staging is recommended with each visit, but is adjusted based on the patient status and the client’s financial situation.

Table 1: Survival time by IRIS Stage

<table>
<thead>
<tr>
<th>IRIS Stage</th>
<th>2b*</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Median Survival (days)</td>
<td>1151</td>
<td>778</td>
<td>103</td>
</tr>
<tr>
<td>Range (days)</td>
<td>2-3107</td>
<td>22-2100</td>
<td>1-1920</td>
</tr>
</tbody>
</table>

*2b Creatinine of 203-249 µmol/L

Taken from AAFP 2015 Dru Forrester, DVM, MS, DACVIM & Jane Robertson, DVM, DACVIM

All CKD cats in IRIS stage 3 and 4 were once in IRIS stage 1 and 2.

Making the Most of Early Diagnosis
Early identification and management of CKD, before creatinine levels are elevated above normal give the practitioner the ability to slow the progression of disease, thereby improving quantity, and more importantly, quality, of life. Some of the many factors that may require management in CKD include:
1. Quality of Life: Pain
2. Nutrition & Hydration
3. Appetite
4. Blood pressure
5. Proteinuria
6. Phosphorus and Calcium
7. Urinary Tract Infections
8. Serum Potassium
9. Anemia
A major consideration for quality of life is the identification of multiple pharmaceuticals that may benefit the patient. The clinician will be wise to consult the client and determine what the client and patient can tolerate, thus avoiding poly-pharmacy situations where the client-patient relationship may be negatively impacted. The clinician will need to prioritize therapy. This may be based upon an understanding of factors associated with poor prognosis in CKD:

- Azotemia
- Hyper-phosphatemia
- UPCR >0.2 (0.4)
- Decreased Hgb and/or HCT
- Leukocytosis

1. Pain Management
   Above all else, mature cats should be assessed for pain. Changes noted by the client at home, observations within the examination room and findings on PE or evidence of pain during sample taking will allow the clinician to identify specific areas of concern. Having said this, the majority of mature cats will in fact be experiencing some degree of arthritis related pain. A client may not perceive changes suggestive of pain in their feline friend. Day to day changes may be subtle in their progression, eluding the client’s observations. Some clients may excuse away the changes, citing age as a factor, but not recognizing that the main impetus behind behavior changes is likely to be pain. Monitoring normal patterns of behavior will help detect changes that may be occurring as a result of pain. The presence of dehydration can be linked to pain (extrapolated from human studies). The author has observed pain and windup pain in severely dehydrated renal patients.

In a study by Lascelles et al (2010), cats between the ages of 6 months and 20 years of age were randomly selected for radiograph study. Ninety-one percent of the cats had radiographic evidence of degenerative joint disease (DJD), with equal frequency in all age groups. This type of study provides convincing evidence that DJD should not be ignored in cats, and that pain related to DJD can be a significant concern.

Treatment for pain is essential. Analgesics should be prescribed, and multimodal analgesia should be considered. Injectable products such as cartrophen or adequan are also beneficial to these patients. Additional pain medications such as buprenorphine, amantadine and non-steroidal anti-inflammatory drugs (NSAIDs) may also require consideration. Studies showing long term safety of NSAIDs meloxicam (Gowan et al, 2011) and rodenocoxib (King et al, 2016) are available. Calcium channel antagonist gabapentin is an additional useful choice for analgesia in the senior patient. Healthy senior patients may be prescribed gabapentin at an initial dose of 15-20 mg/kg POq12h. In debilitated cats, a starting dosage of 5-10 mg/kg POq12h is recommended. This medication is safe for use in all diseased states and the initial side effects may be sedation and/or mild ataxia. After 2-4 weeks, side effects abate. The cat will be more comfortable and return to normal behaviour.

2. Nutrition & Hydration
   Dietary changes recommended for cats with renal disease will vary depending on the IRIS staging results, patient status and in some cases, clinician preference. Many renal specific diets are available for use. As obligate carnivores, the limitation of daily protein intake can lead to utilization of endogenous muscle as a protein source. In the past, this had lead to the concern that muscle wasting may ensue with the use of renal diets. However, renal diets do not contain
protein levels below those minimal levels recommended by the American Association of Feed Control Officials (AAFCO) and the National Research Council (NRC). Instead, they have reduced levels compared to maintenance diets. Key changes in these diets that make them valuable in early IRIS stages include restricted phosphorus, high energy density, highly digestible protein sources, anti-inflammatory omega fatty acids (OFA-3), and vitamin B12. The diets also have increased palatability, reducing previous concerns about their acceptance by CKD cats. Improving hydration status in renal patients is generally considered to be beneficial to renal function and overall patient health. Increasing water intake may be a key factor in improving renal function and overall patient hydration status. Indirectly this can reduce pain from dehydration and constipation. The transition to an all canned food diet is an excellent choice, although patient food consumption is critical, and changes should be made slowly if necessary. Adding 1-3 tsps of additional water to canned feedings will increase the amount of water ingested, thereby further improving hydration status. Where appetite is poor, appetite stimulants may be necessary. Alternatively, feeding tubes can provide a safe, alternative way for clients to administer food, water and medications in the advanced CKD patient.

The administration of subcutaneous fluids is a common recommendation. Clients are usually able to learn this procedure for administration at home once to several times weekly, or may elect to bring the patient in for regular treatments. Addition of 20-40 mEq/L of potassium chloride to the fluids should be considered in the case of hypokalemia and/or where regular (daily) SQ fluids will be administered. Caution should be taken with SQ fluids volumes if the clinician is concerned about cardiac compromise in the patient, or if the patient has known heart disease. Balanced electrolyte solutions such as plasmalyte or LRS should be used. Sodium chloride (0.9% NaCl) fluids may also be used.

3. Appetite
As CKD progresses, appetite may be negatively impacted. Causes of Inappetence/anorexia in the CKD patient include nausea, general malaise, circulating uremic toxins, pain, dehydration and/or secondary diseases. Previously, increased acidity in the stomach was thought to occur in feline patients with CKD, necessitating the use of antacids. A 2014 study demonstrated that while gastrin concentrations were significantly higher in CKD cats when compared with non-azotemic controls, increased concentrations were not associated with gastric ulceration (McLelan, SM et al 2014). The study concluded that appetite in CKD cats was impacted by uremic toxins, rather than increased gastric acidity. The author no longer utilizes antacids in the management of feline CKD. Loss of appetite should be managed based on identified or hypothesized causes as noted above.

- Treat identified causes of Inappetence
- Treat pain
- Treat Nausea:
  - MAROPITANT 2 mg/kg PO q24h
  - Ondansentron (Zofran) 0.1-0.15 mg/kg PO or Slow IV q6-12h
  - Mirtazapine 1.88 mg/cat PO q24-72h
  - Mirtazapine 1.88-3.75 mg Transdermal q24-72h (Benson et al, 2017)
- Treat anorexia/inappetence:
  - Mirtazapine 1.88 mg/cat PO q24-72h
  - Mirtazapine 1.88-3.75 mg Transdermal q24-72h (Benson et al, 2017)
  - Cyproheptadine: 1-2 mg/kg PO q12-24h
4. Management of Hypertension
Identification of systolic blood pressure (BP) values over 150-180 mmHg, may indicate the need for hypertension-controlling drugs. Initial BP values should not be interpreted lightly. The inaccuracy of the Doppler and oscillometric BP methods should be taken into consideration. The BP testing should be repeated, and the patient assessed for evidence of target organ damage (TOD). Amlodipine besylate is a calcium channel blocker recommended as the first choice for feline hypertension. It is the most effective at controlling blood pressure in the feline species. The patient should be started on a dosage of 0.625 mg per cat orally once daily in the evening. The BP should be re-evaluated no later than 3-5 days following introduction of therapy. Adjustments should be made as needed, with continued BP rechecks every 3-5 days until a stable dosage is found. Some patients will have partially or uncontrollable hypertension with amlodipine (maximum dosage 2.5 mg/cat PO q24h), and may require additional medications. Benazepril (Fortekor TM) as a sole agent is not effective in the control of hypertension. The newly available drug from Boehringer Ingelheim, telmisartan (SemintraTM) may be effective at controlling hypertension at higher doses, but has yet to be evaluated for further benefit to hypertensive cats. Both drugs may be helpful as adjunct agents when doses of amlodipine are insufficient to control hypertension.

5. Proteinuria
Elevations in the urine protein creatinine ratio (UPCR) indicate abnormalities with the renin angiotensin aldosterone system (RAAS). These changes alter intra-glomerular pressures and result in protein loss into the filtrate/urine. The use of benazepril has been recommended in the past. In the short term this medication may reduce proteinuria. Over time, the RAAS can escape the control that benazepril exerts, resulting in resumed proteinuria. The development of telmisartan (SemintraTM) has provided an alternative that is more targeted and not likely to lead to escape mechanisms over time. Results from studies evaluating the benefits of telmisartan in early CKD in advance of proteinuria are pending.

6. Phosphorus and Calcium
Patients identified with elevated total calcium, elevated ionized calcium and/or elevated phosphorus may require phosphorus-binding agents to reduce phosphorus levels. The use of agents such as aluminum hydroxide can be challenging, as palatability may be challenging. The use of phosphorus binding agents containing calcium should be minimized unless serial monitoring of ionized calcium can be pursued. Calcitriol is a drug that is recommended frequently in renal patients. It’s primary indication for use is following diagnosis of renal secondary hyperparathyroidism. Calcitriol is the active metabolite of 25-hydroxycholecalciferol. Both calcitriol and parathyroid hormone (PTH) play a role in calcium metabolism. Calcitriol also modulates PTH activity at the transcription level. Cats with CKD may have impaired production of calcitriol, thus leading to renal secondary hyperparathyroidism. In these cases, the use of calcitriol, with regulated serum phosphorus levels, may benefit the patient in the short and long term. Some experts recommend low dose calcitriol supplementation as a means of improving quality of life in all feline CKD patients. More detailed studies on this mode of utilization are warranted (Sparkes et al, 2016).
7. Urinary Tract infections
The indiscriminate use of antibiotics in the absence of evidence of urinary tract infection (UTI) is not recommended. Criteria for the identification of UTI should include the presence of WBC and bacteria on urine sediment, the culture of specific bacteria and occasionally ultrasound evidence of renal parenchymal changes suggestive of infection. Antibiotics should be selected based on urine culture and sensitivity patterns. A repeat urine culture mid treatment is valuable, although may not be indicated with every patient. A repeat urine culture 4-6 days following cessation of therapy is critical.

In cases where urine culture is negative, but a low USG exists in the face of renal disease, ultrasound is recommended. Evaluation for renal parenchymal changes indicative of occult renal infection will assist the clinician in pursuing antibiotic therapy. In these rare cases, antibiotics that are broad spectrum and/or valuable for common sources of renal infection should be selected. These include clavulanic acid/amoxicillin or fluoroquinolones (enrofloxacin, marbofloxacin or orbofloxacin). The utilization of the newer, four-quadrant fluoroquinolone pradofloxacin (VerafloxTM) is not recommended. This latter should be reserved for very particular situations and should generally only be used based on sensitivity patterns and an absence of other options based on those sensitivity patterns.

8. Hypokalemia
Patients who exhibit even mild decreases in potassium levels in their serum require supplementation with potassium gluconate. The majority of body potassium is held in the intracellular or interstitial space. The serum potassium represents only 2% of body potassium. Therefore, any decrease noted in the serum is significant of a major decrease in the overall body stores.

9. Anemia
Chronic kidney disease can lead to a reduced production of erythropoietin. The result is a reduced production of new red blood cells from the bone marrow. As aged RBC are removed from the circulation over time, anemia of renal (and chronic) disease results. Some patients will also have iron deficiencies reducing production of new RBC. Evaluation of iron levels with consideration for supplementation is needed. These patients may also require injectable erythropoietin or darbopoietin to stimulate bone marrow production of RBC.

Key Points
1. IRIS staging of the feline patient allows identification of concurrent issues that can be treated with the aim of reducing comorbidities and slowing the progression of disease
2. Every patient in IRIS stage 3 or 4 was once in stage 1 and 2. Early identification of disease will improve quality and quantity of life.
3. Many options exist for therapy of CKD patients. These therapeutics should be chosen based on appropriate diagnostic testing and their relative importance to the patient.
4. Therapeutics should be prioritized and an effort made to avoid poly-pharmacy, which can damage the client-patient bond.

References:

Kelly St. Denis

Blood pressure assessment should be an integral part of a senior cat’s healthcare. Despite this, many veterinarians struggle with this procedure. The time required to take blood pressure in a cat may not be practical in the appointment setting, the cat may not be cooperative, the clinician may not be comfortable with the equipment available. Finally, understanding collected readings can be challenging at best. This session will review the causes and consequences of hypertension in the feline species, but will focus on the art of taking blood pressure in the cat. Therapeutics and diagnostic algorithms will provide the attendee with the resources necessary to implement blood pressure monitoring as part of routine senior care in their practice.

Learning Objectives
1. Understand the feline friendly approach to obtaining blood pressure readings
2. Understand how and when to treat cats for high blood pressure

This session is appropriate for: Veterinarians & Veterinary Technicians

Causes & Effect
Systemic hypertension is a common, but often missed medical problem in the feline species (Taylor et al, 2017). Feline systolic blood pressure (sBP) increases with age (Bijsmans et al, 2015), as does the risk for systemic hypertension (Jepson, 2011). Hypertension can be divided into three categories. Primary or idiopathic hypertension occurs where no underlying disease is present. Idiopathic hypertension occurs in 13-20% of cases. Secondary hypertension is that in which an underlying disease is present which is directly causing or predisposing to hypertension. Lastly, white coat hypertension is a real and documented phenomenon in the feline species (Belew et al, 1999).

Secondary Hypertension
Chronic Kidney Disease is commonly associated with hypertension, with 19-65% of cats with CKD developing hypertension at some time. The reverse is also true in that unrelated hypertension can predispose to azotemia as a result of elevated systemic blood pressures and their impact on the glomerular filtration rate (GFR). Azotemia may be found in up to 74% of hypertensive cats. As a secondary cause of hypertension, hyperthyroidism is also common. Ten to 23% of hyperthyroid cats may be found to be hypertensive at the time of diagnosis with hyperthyroidism. Once euthyroid, 25% or normotensive, hyperthyroid cats may develop hypertension. Primary hyperaldosternonism (PHA) occurs as a result of excess aldosterone production independent of its regulator, angiotensin II. Forty to 60% of cats with PHA are hypertensive. Other less common secondary causes of hypertension include diabetes mellitus, pheochromocytomas, and pituitary hyperadrenocorticism.

White Coat Hypertension
Stress and anxiety can impact systemic blood pressure in the feline species. Stresses associated with the trip to the clinic, handling by staff and cuff placement and inflation can all contribute to temporary elevations in sBP. In a 1999 study evaluating the white coat effect on sBP in cats, the magnitude of the white-coat effect tended to decrease during office visit, but did not disappear.
Interestingly, the mean increase in sBP was found to be significantly greater in cats with renal insufficiency. White coat hypertension is recognizable, avoidable and/or can be overcome when one approaches blood pressure measurements following the guidelines of Feline Friendly® practice (Rodan et al, 2011).

**Clinical signs of Hypertension**
As is often the case with humans, the clinical signs of hypertension often go unnoticed in the feline species. This is further compounded by the fact that cats are masters at hiding illness. Clients often miss the subtle, early signs of sickness in their pet cats, and hypertension is no exception. Clinical signs identifiable in practice are often associated with damage or changes to those organs most sensitive to hypertension. These are referred to as target organs. Target organ damage (TOD) is identifiable in the retinas, the heart, the brain and the kidneys.

<table>
<thead>
<tr>
<th>Retinas</th>
<th>Heart</th>
</tr>
</thead>
<tbody>
<tr>
<td>50% of hypertensive cats</td>
<td>Increased SVR can increase LV wall stress which may result in concentric LV hypertrophy (LVH)</td>
</tr>
<tr>
<td>can develop at sBP of 160 mmHg+</td>
<td>Gallop sounds, possibly murmurs, arrhythmias</td>
</tr>
<tr>
<td>Tortuosity of retinal blood vessels</td>
<td>Rare sequelae: heart failure, aortic dissections</td>
</tr>
<tr>
<td>Hemorrhage of retinal blood vessels</td>
<td></td>
</tr>
<tr>
<td>Cotton wool patches- detachment of retina</td>
<td></td>
</tr>
<tr>
<td>Vitreal hemorrhage</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Kidneys</th>
<th>Brain</th>
</tr>
</thead>
<tbody>
<tr>
<td>External elevations in SBP can impact internal pressures leading to glomerular hypertension and damage</td>
<td>Neurological signs in 15-46% of hypertensive cats</td>
</tr>
<tr>
<td>Azotemia occurs in up to 74% of hypertensive cats.</td>
<td>Disorientation, seizures, ataxia, depression and vestibular signs</td>
</tr>
<tr>
<td>Kidneys impact blood volume and BP through pressure natriuresis and RAAS</td>
<td>Many DDx</td>
</tr>
<tr>
<td>19-65% of cats with CKD will develop hypertension at some time</td>
<td>Presumptive Dx: signs improve with treatment</td>
</tr>
</tbody>
</table>

**Candidates for monitoring and Screening**
The most common need for systemic BP assessment is in cats 7 years of age or over. Since blood pressures increase with age and ageing cats are at increased risk of developing hypertension, this should be regarded as the high-risk group requiring routine, biannual sBP monitoring. At the same time, this group should be assessed for TOD. Cats with evidence of TOD, including blindness, and patients with diseases associated with 2˚ hypertension
will also need sBP assessment. All cats under anesthesia should be subjected to continuous sBP monitoring. Once a cat is diagnosed with hypertension, ongoing sBP monitoring is necessary.

**What You Need: Scientific Equipment**

Gold standard assessment of BP includes direct methods by central catheter or radiotelemetric implant. Direct methods are impractical and invasive. Indirect methods utilizing the Doppler or oscillometric (traditional, high definition) are readily available to clinicians, practical and non-invasive, but are highly inaccurate. For this reason, knowledge of the machine, development of a standardized protocol and assessment by a limited number of trained staff is critical (Gouni et al, 2015). Interpretation of the results based on the patient status, patient stress level and evidence of TOD is also critical.

The following should not be overlooked:

**Cuff Selection**
- 40% circumference of limb
- Snug fit

**Cuff Placement**
- Forelimb, tail, hind limb
- Over artery-area where inflation tube enters
- Roughly on same plane as heart

**Standardized Protocol**
- Minimum of 6 readings
- 1 minute between readings
- Drop first reading
- Average remainder
- Experienced individuals only assigned the task of obtaining blood pressure readings

**What you need: Feline Friendly environment**

**Choosing the Right Setting**
- In carrier/out of carrier
- Exam room vs. Treatment vs. ICU
- WARM environment
- QUIET environment
- DARKENED environment
- Minimize people present: with or without client, experienced personnel, cat friendly personnel

**Analgesics**
- Most analgesics will alter BP
- Many cats >7 have DJD
- Minimize handling
- Consider low dose gabapentin 6-10 mg/kg

**Establishing criteria for therapeutic decision making**

Before determining if a cat is truly hypertensive, the clinician must be sure of having measured the sBP carefully according to the following:
• Measure sBP according to feline friendly practice protocols
• Measure sBP according to manufacturer directions
• Measure sBP according to a standardized protocol

According to the panel for the ISFM Consensus Guidelines on the Diagnosis and Management of Hypertension in Cats (2017), the diagnosis of hypertension can be made in the four distinct circumstances. A single occasion is defined as one visit obtaining a series of readings based on the standardized protocol.

### Diagnosis of Feline Hypertension

<table>
<thead>
<tr>
<th>Indirect SBP</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥150 mmHg</td>
<td>Indirect SBP is ≥150 mmHg on a single occasion, and there is clear evidence of ocular or neurological TOD. Note: if clinical signs do not respond appropriately to adequate antihypertensive therapy, the diagnosis should be reassessed and other potential causes of the signs investigated.</td>
</tr>
<tr>
<td>≥160 mmHg</td>
<td>Indirect SBP is ≥160 mmHg on at least two separate occasions, and there is evidence of TOD including ocular, neurological, cardiac or kidney damage.</td>
</tr>
<tr>
<td>≥170 mmHg</td>
<td>Indirect SBP is ≥170 mmHg on at least two separate occasions, and the clinician does not consider ‘white coat hypertension’ is likely to be the cause.</td>
</tr>
<tr>
<td>&lt;150 mmHg</td>
<td>Indirect SBP is &lt;150 mmHg, but there is clear evidence of active ocular TOD. Note: cats should be monitored carefully. If there is any doubt about the diagnosis of hypertension, the need for long-term therapy should be reassessed by trial withdrawal of therapy once stable, and monitoring of BP and clinical signs.</td>
</tr>
</tbody>
</table>

### Therapies and Therapeutic Targets

1. Amlodipine besylate (Calcium channel blocker): This is a potent peripheral arterial dilator and is the drug of choice in feline systemic hypertension. DOSAGE: 0.625-1.25 mg/cat PO q24 or DOSAGE: 0.125-0.25 mg/kg PO q24h. Reassess sBP in 3-5 days the maximum amlodipine recommended dosage is 2.5 mg/cat q24h.
2. Benazepril (ACE inhibitor): This agent is not effective alone in the control of systemic hypertension, but may be beneficial as an adjunct to refractory treatment with amlodipine. The medication should not be added if the patient is dehydrated. DOSAGE: 0.5-1.0 mg/kg PO q24h
3. Telmisartan (Angiotensin receptor blocker (ARB)): This agent may be effective in controlling systemic hypertension at high doses but further studies are needed. Telmisartan may be beneficial as an adjunct to refractory treatment with amlodipine. LABEL DOSAGE: 1.0 mg/kg PO q24h
4. Atenolol (β-blocker) tachycardia, hyperthyroidism DOSAGE: 1.0-2.0 mg/kg PO q12h
5. Propanolol: (β-blocker)
6. Enalapril: (ACE inhibitor)
7. Ramipril: (ACE inhibitor)
8. Spironolactone: Indicated in cases of hyperaldosteronism
Therapeutic Goals
The therapeutic goals in the management of feline hypertension are simple. Prescribed medications should effectively control sBP with the goal of decrease TOD. In cases of secondary hypertension, identified primary diseases should be treated, controlled and/or cured.

Key Points
1. Hypertension is a well-recognized but under-diagnosed condition in cats
2. The clinical consequences can be severe and irreversible
3. Routine screening and monitoring is key
4. Available therapeutics are effective and safe

References:
Companion Animal: Diagnostic Imaging

Radiographic interpretation of the Thorax in the Small Animal Patient: Part One
  Eric Herrgesell, DVM, MS, DACVR

Paper not available at time of online posting.

Radiographic interpretation of the Thorax in the Small Animal Patient: Part Two
  Eric Herrgesell, DVM, MS, DACVR

Paper not available at time of online posting.

Thoracic Case Discussions of the Small Animal Patient, Part One
  Eric Herrgesell, DVM, MS, DACVR

Paper not available at time of online posting.

Thoracic Case Discussions of the Small Animal Patient, Part Two
  Eric Herrgesell, DVM, MS, DACVR

Paper not available at time of online posting.

Thoracic Case Discussions of the Small Animal Patient, Part Three
  Eric Herrgesell, DVM, MS, DACVR

Paper not available at time of online posting.

Thoracic Case Discussions of the Small Animal Patient, Part Four
  Eric Herrgesell, DVM, MS, DACVR

Paper not available at time of online posting.
Companion Animal: Rehabilitation
Introduction to Rehabilitation—How can PTs Find Things That DVMs Miss?
Janice L Huntingford, DVM, DACVSMR, CVA, CVPP, CCRT

Overview
Veterinary rehabilitation is the fastest growing segment of veterinary medicine. It is however, not all about laser and underwater treadmills but rather is based on a new diagnostic algorithm. This algorithm is based on the application of human physiotherapy techniques to the canine.

The focus of canine rehabilitation is on soft tissue rather than on bones and joints. Treatment goals are functional, designed to optimize movement and quality of life for the patient. Rehabilitation professionals complete many hours of coursework and hands-on training. They perform in-depth evaluations, manage treatment, and objectively measure progress. They use specialized tests to look for tendinopathies and soft-tissue abnormalities, and precise instruments to measure joint rotation, range of motion and detect gait abnormalities.

Rehabilitation requires a DVM to become highly skilled in manual therapies including joint mobilization, therapeutic stretches and exercise. Physioballs, therapy bands, rocker/wobble boards, Cavaletti poles and treadmills are used routinely to increase strength, coordination and flexibility. Rehab professionals are trained to use physical modalities such as ultrasound, laser and electrical stimulation. As more practices have invested in therapeutic pools and underwater treadmills, hydrotherapy is also an option for many patients.

History of Canine Rehabilitation
Human physiotherapy, or physical therapy, is a relatively new medical discipline which began in Sweden in the early 1800s; modern physical therapy was developed in Britain in the early 1900s. Physiotherapy was initially focused on the treatment of polio victims but after the outbreak of World War I the field concentrated on helping to restore mobility to injured soldiers. The first school of physiotherapy was established at Walter Reed Army Hospital in Washington D.C.1

Interest in the veterinary derivative of human physiotherapy began initially in the 1970s with the publication of a book called Physical Therapy for Animals: Selected Techniques by Ann Downer, PT. Shortly thereafter, national presentations to such groups as the American College of Veterinary Surgeons (ACVS), American Physical Therapy Association (APTA) and the American Veterinary Medical Association (AVMA) led to increased interest in Canine Rehabilitation. Interest in Canine Sports Medicine began at about the same time with publication of Canine Sports Medicine and Surgery and with the formation of the International Racing Greyhound Symposium associated with what was then known as the Eastern States Veterinary Conference (now VMX)). Rehabilitation was a frequent topic at this conference and was expanded to include all sporting dogs. Today, physical rehabilitation is becoming common in small animal practices and these exercises and modalities are used not only for recovery from orthopedic and neurological conditions, but also for wellness care and preventive medicine in the form of weight management and maintenance of muscle strength and conditioning, particularly for athletes and geriatric animals. Current practices combine the knowledge and skills of veterinarians, veterinary technicians, human physiotherapists, and physiotherapy assistants. Formal training and accreditation is now available for veterinarians and...
veterinary technicians, and advanced specialty training is available through the American College of Veterinary Sports Medicine and Rehabilitation (ACVSMR).

With advances in surgery, and with an increasing number of owners who regard their pets as family members, demand for rehabilitation for veterinary patients is increasing. Postoperative care focusing on decreasing pain, inflammation, and joint stiffness while improving mobility and quality of life may result in shorter hospital stays and improved patient and client well-being. Rehabilitation for the veterinary patient with orthopedic or neurological disease has as its overlying principle a return to normal mobility and neuromuscular function. The goals of rehabilitation are first and foremost to relieve pain and ameliorate discomfort while insuring that the patient’s condition does not worsen, particularly in the light of spinal instability or surgical repair. Once pain control has been adequately addressed, therapists can focus on restoring normal muscular function, minimizing muscular atrophy and other secondary problems, and improving and restoring mobility.

Rehabilitation therapy emphasizes return to function and maximum recovery from the disease process or surgical procedure, as well as the improvement of overall well-being, rather than the treatment of a specific disease or diagnosis. Postoperative orthopedic or neurological surgery patients are among the many patients that benefit from physical rehabilitation. Other patients include those with chronic osteoarthritis, severely debilitated patients, oncology patients, obese patients (to aid in weight loss), canine athletes and working dogs (to enhance condition or performance), and patients with orthopedic or neurological disease that require non-surgical management. Improvement in pain control, joint range of motion (ROM), muscle strength and coordination, balance and proprioception, and decrease in contracture and fibrosis are some of the objectives of physical rehabilitation. Return to function and recovery are achieved through the use of the many different modalities and techniques.

The Rehabilitation Evaluation
An ideal rehabilitation evaluation involves a thorough history, physical examination, and a complete orthopedic and neurological examination, followed by a specific rehabilitation examination. Historical data of interest to the rehabilitation professional include onset and duration of the problem, body part involved, trauma or activities associated with the injury, current pain score, lifestyle and exercise habits, weight loss or gain, and type of surgery (if any) performed. A general physical examination focusing on any pathology affecting the cardiovascular, neuromuscular, pulmonary, endocrine/metabolic, or integumentary system is critical because all of these systems can affect muscular performance. It is also important to evaluate the patient’s psychological state and willingness to perform any exercises, as well as the experience of the owner or handler.

Tips and tricks

1. Observe the dog when he/she is unaware you are watching them. I prefer to talk to the client and ignore the dog for the first few minutes. I will take the history and observe the dog in a relaxed state. Often this involves treats—we use lots of liver treats and peanut butter bowls to gain the patients trust. We evaluate spinal range of motion and ability to sit and stand while giving treats. Often the owner feels we are only petting and treating the dog. The more the pet trusts you, the more likely it is you will find out what’s going on.
2. Take a thorough history. Signalment may be helpful if you understand the orthopedic/neurological diseases of the breed and age you are dealing with. This helps to create an initial list of differential diagnoses. Remember common conditions are common!

3. Understand the owner’s goals and the problem as they recognize it. It is important to know why the owner has come to see you and what they want out of the visit. The goal of the owner of a master hunt test candidate is different from the goal of the owner of an aged Bulldog, although both may have the same problem.

4. Focus on the comfort of your patient not yours. We examine all our patients on the floor—even very small ones. For the tiny one we eventually move to a table but only when the patient is comfortable. 95% of our exams are done on the floor. If the patient is comfortable on the floor we transition from petting to palpation. Keep one hand on the patient at all time as this makes the transition into the exam seamless. Initial palpation involves assessing for muscle atrophy, asymmetry, joint swelling, muscle tenderness and areas of heat.

5. Remember dogs have 2 sides—that should be symmetrical. If the patient is comfortable standing with you on the floor behind him, you can palpate the hind and fore limb simultaneously. This is a great advantage as it allows you to compare both sides and detect small differences in swelling, effusion or heat.

6. Watch for your patient’s response. Palpate gently while an assistant holds the dog (protect yourself) but observe subtle signs of pain—shift in weight, a change in respiratory pattern, a look in his eyes, pupil dilation, or an increase in muscle tension. Palpate all limbs and assess ROM.

7. Don’t skimp on your physical exam and take all previous diagnosis with a grain of salt. Don’t overlook the rest of the body. The dog may have a fever or Horner’s syndrome and you will not know if you do not look. If you have been asked to evaluate a hind leg for a cruciate problem, that does not mean you need to ignore the rest of the dog or take that diagnosis at face value. If you do not look thoroughly you will miss things like neurological disease or bone tumors!

8. Do a thorough Gait Evaluation and use a lameness scale. (See Table 1) Decide if the gait is normal, lame (orthopedic problem) or ataxic (neurological problem) Do a proper sit test—does the dog sit squarely. Be sure to video the dog at all gaits. If the lameness is subtle and not apparent on that day, have the owner video at home. Put the dog over obstacles and around cones, up and down stairs at different paces. Often the lameness will become obvious when the dog walks outside or at a slow walk. The use of gait evaluation equipment is nice but not always necessary.

9. Use the Acronym CREPI when evaluating joints (courtesy of Dr. Ross Palmer, CSU) This stands for crepitus (C), range of motion (R), effusion (E), pain (P), instability (I). Some patients will need to be sedated to properly evaluate all joints.

<table>
<thead>
<tr>
<th>Grade</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No detectable lameness at any gait</td>
</tr>
<tr>
<td>1</td>
<td>Barely perceptible lameness</td>
</tr>
<tr>
<td>2</td>
<td>Mild or inconsistently apparent, weight bearing lameness</td>
</tr>
<tr>
<td>3</td>
<td>Moderate, obviously apparent, weight bearing lameness</td>
</tr>
</tbody>
</table>
Severe, predominantly weight bearing lameness

Severe, predominantly non-weight bearing lameness

Table 2

<table>
<thead>
<tr>
<th>Joint</th>
<th>Specific Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elbow</td>
<td>Direct palpation of medial compartment—just distal to medial epicondyle</td>
</tr>
<tr>
<td></td>
<td>Supination/pronation with carpus and elbow at 90 degrees</td>
</tr>
<tr>
<td>Shoulder</td>
<td>Biceps tendon test—flex shoulder, extend distal limb and palpate bicipital</td>
</tr>
<tr>
<td></td>
<td>groove Abduction angles (MSI)-shoulder in extension not flexion</td>
</tr>
<tr>
<td>Stifle</td>
<td>Cranial drawer—landmarks patella, lateral fabella, tibial tuberosity,</td>
</tr>
<tr>
<td></td>
<td>fibular head Tibial thrust (index finger over patella and patella tendon on</td>
</tr>
<tr>
<td></td>
<td>tibial tuberosity. Flex tarsus and compress tibia. Patellar luxation—easiest</td>
</tr>
<tr>
<td></td>
<td>to check for when stifle is in extension</td>
</tr>
<tr>
<td>Hip</td>
<td>Luxation check—triangle thumb pinch</td>
</tr>
<tr>
<td></td>
<td>Ortolani (often sedated)- Support pelvis, push dorsally with leg in</td>
</tr>
<tr>
<td></td>
<td>neutral position to subluxate, then abduct the limb to feel for “clunk” of</td>
</tr>
<tr>
<td></td>
<td>reduction= Ortolani Sign</td>
</tr>
<tr>
<td>Lumbosacral disease</td>
<td>Direct palpation and extension of hind limbs, raising tail</td>
</tr>
<tr>
<td>Iliopsoas Muscle Strain</td>
<td>Direct palpation of insertion on lesser trochanter of femur</td>
</tr>
<tr>
<td></td>
<td>Hip extension with traction and internal rotation of femur</td>
</tr>
</tbody>
</table>

**Neurological examinations**

Questions to ask yourself when doing abbreviated exam
1. Ambulatory or not?
2. Cranial nerve exam
3. Sensation
4. Postural reactions—tests of weakness or ataxia
   - Hopping, wheelbarrowing, cavalettis or stairs
5. Conscious Proprioception
6. Spinal reflexes
   - Myotatic Reflexes-patellar reflex hypo vs hyper
   - Flexor Reflexes—Crossed Extensor
   - Dermatomes
   - Panniculus

Lesion localization is important. The following is a list of common neurological conditions that can be seen by the rehab practitioner.
ACUTE
Disc disease – Type I & Type III
Discospondylitis
CCVM – Wobbler
AA - instabilities
FCE/FCL
Peripheral Nerve injuries
Plexus injuries
Neuromuscular Diseases —
Polyradiculoneuritis, Botulism, Tick Paralysis

CHRONIC
Disc Disease Type II
Degenerative Myelopathy
Cauda Equina Syndrome
Spondylosis
For many years chronic pain in dogs and cats was managed with a single pharmaceutical agent if and when the clinician determined the animal was suffering. Recently it has been realized that pain is a very complex process and involves signaling molecules, pathways, substances, receptors and transmitters with different modes of action. It is unrealistic to think only one pharmaceutical could be effective in eliminating chronic pain. It is equally unrealistic to think that drugs alone can manage pain effectively for the life of the animal. A multimodal and individualized approach to the treatment of pain in the geriatric dog is necessary and the most effective approach. It is important to discuss several things with owners once they understand that their pet has chronic pain. For example, degenerative joint diseases are not curable, so the goals are to keep the pet comfortable and enjoying a good quality of life; this requires a financial and time commitment. Achieving specific goals will most likely require some trial and error which will be result in some triumphs and disappointments for the owner.

**Assessment**

Assessment of long-term pain relies heavily on owner input; several questionnaires are available such as the Glasgow University Health-Related Dog Behaviour Questionnaire and the Canine Brief Pain Inventory (University of Pennsylvania) which identify some key indicators of chronic pain in dogs including, but not limited to decreases in mobility, activity, sociability and curiosity and increases in aggression, anxiety, daytime sleeping and vocalizing. In cats questioning the owner about the cat’s mobility, activity levels, grooming habits and general demeanor can provide very helpful information. When a cat soils outside of the litter box, chronic pain should be considered in the differential diagnosis list. The feline musculoskeletal pain index (FMPI) for the evaluation of degenerative joint disease-associated pain in cats is a useful and validated tool which is frequently updated and available for download at: https://cvm.ncsu.edu/research/labs/clinical-sciences/comparative-pain-research/clinical-metrology-instruments/ These tools are valuable for tracking efficacy of treatment and disease progression and are helpful to owners when difficult discussions regarding quality of life and euthanasia are required.

**Pharmaceutical, Nutraceutical and Dietary Therapy**

These subjects are well covered elsewhere but it is important to note that geriatrics require lower doses of many pharmaceuticals do to organ dysfunction and metabolic changes. Nutraceuticals such as Omega 3 fatty acids, Undenatured Collagen Type 2, Pentosan, Glucosamine and Avocado Soy Unsaponifiables are all important to help control pain in geriatrics.

**Environmental Modifications and Assistive Devices**

Simple environmental modifications can have a positive effect on old painful patients. Raising food and water dishes, putting down yoga mats, area rugs or carpet to reduce slipping, installing ramps and using baby gates to limit dangerous areas are all good ideas for household modifications. A foam bed or other soft area to lie on can cushion old joints. Harnesses, slings, booties, power socks, braces and orthotics are all examples of assistive devices that can be used. Sometimes carts and wheel chairs are also necessary.
**Acupuncture**

Acupuncture can be used to relieve pain, cause an autonomic response, increase the rate of nerve regeneration, and cause surgical analgesia. There are multiple theories as to how acupuncture works in humans and animals alike. It is important to understand that no one theory explains all the different effects of acupuncture. Just as research is continuously being done to further develop western medicine, additional research is being done with both human and animal acupuncture to further our understanding of this ancient healing art. The most current theories are: 1) The Gate Theory; 2) Endogenous Opioid Theory; 3) Autonomic Nervous System Input Theory; 4) Humoral Theory; 5) Bioelectric Theory; and 6) Traditional Oriental Medicine Theory.

**Physical Rehabilitation**

The goals of rehabilitation include the restoration, maintenance and promotion of optimal function and quality of life as they relate to movement disorders. The majority of rehabilitation therapeutics involves manual therapies including joint mobilizations, and therapeutic exercises. Equipment used on a regular basis in veterinary rehabilitation includes physioballs, therapy bands, rocker/wobble boards, cavaletti poles and land treadmills. Hydrotherapy equipment can include pools, resistance pools and underwater treadmills. Modalities such as hot and cold therapy, laser, electrical stimulation, shock wave therapy and therapeutic ultrasound can also be used. Regenerative medicine with platelet rich plasma and stem cells is now also a part of rehabilitation and pain management.

**Thermal Therapy** - The effects of thermotherapy are vasodilation with secondary increased local circulation, decreased pain, relaxed muscle tone, reduced muscle spasm, increased tissue extensibility, increased cellular metabolism, and increased local tissue oxygenation. Heat is generally used to reduce pain from arthritis, trigger points and muscle spasms, and to prepare tissues for exercise or stretching.

Cryotherapy can be applied via ice bath, ice massage, ice pack, vapocoolant gel, or circulating ice compression units. The beneficial effects of cryotherapy include vasoconstriction; reduced cellular metabolism; decreased nerve conduction velocity, and decreased production of pain mediators, leading to analgesia; reduction of edema and decreased muscle spasm.

**Manual Therapies**

Massage is soft tissue massage and soft tissue mobilization. Massage can decrease excessive tissue tension by aiding in removal of chemical substances in soft tissue that activate chemical nociceptors. Soft tissue massage can also, by the Gate Theory, reduce pain by stimulating large rapidly conduction fibers, selectively closing the gate against smaller pain fiber input.

Joint mobilizations - a manual technique used to assess a joint and improve its movement (arthrokinematics). Joint mobilizations improve joint lubrication, modulate mechanoreceptors, and decrease sensory input thus relieving pain. Therapeutic glides are ranked Grade I to V using the Maitland Mobilization Scale.

Passive range of motion (PROM) is defined as therapist assisted placement of the joint through its normal range of motion. These exercises are very important for neurological patients and post surgical patients to reduce pain and edema, maintain functional range of motion and improve joint
lubrication. Neurologic patients are at risk of contractures of tendons and ligaments. PROM mitigates this condition. In neurological patients PROM works best after heating the tissue with hot packs to warm up the muscles. Do each joint of the affected limb in flexion and extension approximately 10 times before entire limb is flexed. This avoids pain and spasm.

Many manual therapies can be taught to owners, so that therapy can be done in the pet’s home environment. This is also beneficial for strengthening the human-animal bond and also allows the owner to be part of the treatment team.

**Laser**

LASER is Light Amplification by Stimulated Emission of Radiation. By definition, a laser must be collimated and monochromatic. Penetration of laser energy is determined by the wavelength, and many wavelengths are patented. The physiological effects of laser stimulation include accelerated cell division via mitochondrial stimulation, increased leukocyte phagocytosis, stimulation of fibroblast production, enhanced synthesis of ATP, and angiogenesis. Treatment with laser is indicated for pain management, control of inflammation, and tissue healing.

**Electrical Therapy**

Electrical stimulation (ES) can affect the sensory and the motor nerves. Indications for ES include wound healing, pain control/relief, reduction of inflammation, muscle re-education, reversal of atrophy, and strengthening. Electrotherapy works at the cellular level to cause excitation of nerve cells, changes in cell membrane permeability, and stimulation of protein synthesis, osteosynthesis and fibroblast formation. On the tissue level, electrotherapy causes skeletal muscle and smooth muscle contraction. On the segmental level, electrotherapy facilitates muscle-pumping action, which improves joint mobility and circulatory and lymphatic drainage. ES can be either Transcutaneous Electrical Nerve Stimulation (TENS) or Neuromuscular Electrical Stimulation (NMES).

**Pulsed Electromagnetic Field Therapy (PEMF)**

PEMF has been used in humans in Europe for a number of years and by equine practitioners in North America. Its use in small animals has increased with the development of affordable and portable field devices. Mats containing coils that generate the field or portable loop devices are most commonly used. PEMF has been used to reduce pain, inflammation, the effects of stress on the body, platelet adhesion, improve circulation, and help with cell regeneration. It is used in non-healing fractures to accelerate bone repair and improve wound healing. With the proper field intensity and frequency, treatment with PEMF appears to be disease-modifying. The stimulation of TGFβ may be a mechanism by which PEMF favorably affects cartilage homeostasis. Through calcium-calmodulin-dependent pathways, PEMF may also increase nitric oxide activity. A study with rats showed the animals receiving PEMF exposure, had an increase in tensile strength of up to 69% at the repair site of the rat Achilles’ tendon at 3 weeks after transection and repair compared with non-stimulated control animals. Further studies exist for this modality.

**Sound Therapy**

Extracorporeal Shockwave therapy (ESWT) has been applied to painful OA lesions in veterinary practice, including hip and elbow dysplasia and supraspinatus tendinopathy with excellent pain relief results being reported. ESWT works by releasing a sudden high-powered shock wave resulting in
tissue modulation in a very focused depth of tissue. This modality does require deep sedation or anesthesia as the treatment is uncomfortable for the patient, however the patient experiences pain relief immediately post treatment, which can last for days to weeks. The mechanism behind the pain-relieving function of ESWT is thought to be due to increased serotonin activity in the dorsal horn, and descending inhibition of pain signals.

**Therapeutic Exercises**
Therapeutic exercise contributes to pain management through Exercise Induced Hypoalgesia (EIH) which results from activation of the opioid system with beta-endorphin release from the pituitary. It is also believed that exercise can activate large afferents and that mechanical hypoalgesia is induced by repeated low load exercises regardless of exercise mode. Exercises are used for stretching, strengthening, balance, proprioception, flexibility, endurance and muscle re-education.

**Regenerative Medicine**
Stem cell and PRP (Platelet Rich Plasma) can be used for pain management. Progenitor cells are present in almost every tissue that are self-renewing, able to become different tissue types and signal other cells to come in and repair tissue. Adipose derived and bone marrow derived mesenchymal stem cells are used. Benefits are more likely due to growth factors. PRP contains growth factors as well. This is a wide topic and only gets a brief mention here.

**References:**
Further Reading:

AAHA/AAFP 2015 Pain management Guidelines
The Role of Lasers in Rehabilitation
Janice L Huntingford, DVM, DACVSMR, CVA, CVPP, CCRT

It is estimated that 20% of all veterinary practices in North America have lasers. Laser therapy has been used for a number of years particularly by equine practitioners but in the last few years, this modality has been embraced by the small animal practitioner.

Why have lasers become so popular? Here are a few reasons:

1. Clients desire a non-pharmacological option for treatment of pain
2. Increased awareness and availability of rehabilitation services
3. Increased education and research on lasers in general
4. Development of products that have protocols that give consistent results
5. “Safe” lasers allow veterinarians to easily delegate laser treatments to auxiliary staff.

WHAT IS LASER THERAPY?
LASER stands for Light Amplification by Stimulated Emission of Radiation. LASER therapy uses light to stimulate tissues and create physiologic effects. The effect is called photobiomodulation. Photobiomodulation is a photochemical process that occurs when laser light (or any other light for that matter) interacts with cells and causes stimulation or some other chemical change. Photosynthesis or the production of Vitamin D by sunlight are natural examples of photobiomodulation. Many studies exist on the effect of laser on cells in vitro and some in vivo. Most of these studies are in humans not veterinary species.

According to research lasers are said to cause the following effects on cells:
1. Increase angiogenesis
2. Normalize calcium channels
3. Cause axonal sprouting
4. Stabilize cell membranes
5. Affect mitochondria to produce cytochrome C oxidase which in turn leads to formation of ATP (ATP is responsible for tissue healing, pain reduction and decreased inflammation).

When lasers interact with cells, some of the effects occur: include increased ATP production in the cells; enhanced Na-K pump function; enzymatic activation; macrophage activity; cell proliferation; release of growth factors by fibroblasts; proliferation of T and B lymphocytes, and production of extracellular matrix.

HOW DOES IT WORK AND WHAT DO THE TERMINOLOGIES MEAN?
The three main physiologic effects that have been proven include pain relief, reduction of inflammation, and increase in circulation to promote healing. There are components to laser equipment that lead to their ability to create these effects and they are important factors to consider when using laser therapy. These components include the wavelength of the laser, power of the probe, frequency, and dosage.

The wavelength determines the depth of penetration of the laser light into the tissues and it is measured in nanometers (nm). Wavelength is the range from the blue (400 nm) to the mid infrared...
(1100nm) can result in photobiomodulation. The longer the wavelength, the deeper the penetration (depth of absorption of the light). For example, a 600 nm laser penetrates 0.5 to 1 cm, while a 900 nm laser penetrates up to 5 cm. Depth of penetration is one of the most critical elements in laser treatment. Wavelengths from 600 nm to 1100nm are optimal for penetrating tissue. The range of wavelengths that each laser has is called the “therapeutic window”.

When light interacts with tissues, several things can happen: absorption, reflection, scatter or refraction or transmission. Reflection occurs when light hits the tissue and bounces off having no effect on the tissue. If the laser is 2 cm off of the skin, 20% of the light energy is lost due to reflection. If pressure is utilized, penetration of the laser energy is significantly increased. Significantly less reflection occurs because the act of physically pushing the blood out of the tissue causes a reduction in hemoglobin in that tissue coupled with the fact that stretching the skin decreasing the melanin concentration. One study showed coupling with pressure increased transmission rate by 51.5% compared with using contact without pressure, and 92.6% compared with using a 2-mm skin-diode distance. Another way to decrease reflection is to hold the probe perpendicular the tissue you are treating. The greater the angle, if you are treating off the skin, the more the reflection.

Transmission occurs when the light energy passes through the tissue to a deeper level.

Scattering or refraction occurs when energy passes through tissue but changes direction as it goes through causing effects in the tissue adjacent to where treatment takes place. This amount will vary with each laser. Absorption occurs when the energy stops within a cell and where it has the most effect. If too little energy is put into the tissue there can be no effect or if too much energy is placed into the tissue, detrimental effects can occur.

Penetration depth is defined as the depth after which the intensity of laser light is reduced of a factor = $e^x$ ($x = 2.718 \sim 3$) according to the Lambert Beer Law. From the definition the penetration depth depends on wavelength and tissue type; it is not dependent on laser power (W) and laser intensity at the tissue surface (W/cm2). When energy put into tissue creates heat, that energy is not utilized by the cells for the effects we look for from laser therapy (angiogenesis, removing fibrous tissue, decreasing edema, relieving pain.)

The major chromophores that absorb light and prevent light penetration are melanin, hemoglobin and water. Melanin has very high absorption particularly for wavelengths less than 830 nm—so dark skins absorbs more light. Light from longer wavelength (>1300nm) is strongly absorbed in water so penetration is less. Long wavelength lasers are used for surgical applications. Wavelengths in the 630–660 nm range, 808–830 nm range, 904–905 nm range, and 970–980 nm range are commonly used in therapeutic lasers. Darker skin or coated patients will have deeper penetration with higher wavelengths. If you have a lower wavelength, scanning with lower power will allow you to get the desired effect without heating the tissue. Anytime a dark coat or dark-skinned patient is treated the area should be monitored for overheating. The patient will not feel the tissue heat until there is a significant change in temperature, and by that time, tissue damage has occurred. Hemoglobin is least absorbed in the 630–830 nm wavelength with the most absorption, causing least penetration, in the 900 nm and above range. Therefore, if your laser is in the 900–1,000 nm range, compression
of the tissue will be important to allow for best penetration as compressing the tissue causes an ischemic effect by pushing the blood/hemoglobin out of the area.

The power of the probe determines the time to deliver the energy and it is measured in milliwatts (mW). The higher the mW, the shorter the time is required for a therapeutic dose to be delivered. The frequency is the rate at which the laser diodes are on and off and it is measured in Hertz (Hz). The dosage is the amount of energy that is desired to produce a therapeutic effect and it is measured in Joules (Joules = power [mW] × time [seconds]). 1 Joule is equal to 1 W (1000mW) × 1 second; therefore, a 1000mW laser delivers 1 Joule in 1 second of time. The current recommendations of laser dosing are 2 to 6 Joules/cm² for superficial penetration and 8 to 16 Joules/cm² for deep penetration (World Association for Laser Therapy http://www.walt.nu).

There are four classes of laser as determined by the number of milliwatts of power. Class 1 lasers have less than 0.5 mW of power; an example is a garage door opener. Class 2 lasers have less than 1 mW of power; an example is a laser pointer. Class 1 and 2 are considered non-therapeutic lasers. Class 3 and 4 lasers are considered therapeutic lasers and they are currently on the market for veterinary use. Class 3 lasers are further divided into 3A and 3B. Class 3A lasers have 1 to 5 mW of power while Class 3B have 5 to 500 mW of power. Class 4 lasers have greater than 500 mW (0.5 W) of power.

The most common indications for laser therapy include treating pain associated with degenerative joint disease; IVDD; acute and chronic sprains, strains, tendonitis, and bursitis; acute and chronic otitis and gingivitis; and skin lesions including hot spots, anal gland ruptures, lick granulomas, and clean and contaminated wounds. More recently lasers have been used over urethras to unblock blocked cats and ferrets and used intraoperatively to improve blood flow to intestines that had been compromised post foreign object ingestion. Lasers can also be utilized to stimulate acupuncture points in patients that cannot tolerate needles. The effect is not as large as with needles so this should never take the place of needling for convenience, only when needles are not an option.

CONTRAINDICATIONS TO LASER THERAPY
1. Not directly into the eyes (wear protective eye wear)
2. Caution around metal surfaces
3. Caution around open growth plates, active hemorrhage and neoplasia

THINGS WE REALLY DON’T KNOW
1. How effective it really is in animals—there are lot of laser studies but still a paucity of double blind placebo-controlled studies in animals.
2. Dosages for different conditions—this is being studied
3. Dosages for different units
4. Frequency of treatment for different conditions and different animals
5. Long term effects of laser

INTERESTING SCIENTIFIC REPORTS
In the last 3 years there has been an exponential increase in research likely due to the popularity of laser therapy as a treatment modality since it is relatively safe and easy to use.
Hagiwara found that low-level laser therapy produced an analgesic effect in inflamed peripheral tissue that was transiently antagonized by naloxone (opioid antagonist). This study demonstrated that low-level laser therapy produces analgesic effects in a rat model of peripheral inflammation. They also revealed an additional analgesic mechanism of low-level laser therapy via enhancement of peripheral endogenous opioids. These findings suggest that low-level laser therapy induces analgesia by enhancing peripheral endogenous opioid production.1

Rizzi investigated the effects of low-level laser therapy on nuclear factor kappa B (NF-kB) activation and inducible nitric oxide synthase (iNOS) expression in an experimental model of muscle trauma. Rats underwent injury to the gastrocnemius muscle produced by a single impact blunt trauma. A low-level gallium arsenide (Ga-As) laser at 904 nm, 45 mW, and 5 J/cm2 was applied continuously for 35 seconds. Researchers concluded that laser therapy reduced the inflammatory response induced by trauma and was able to block the effects of reactive oxygen species (ROS) release and the activation of NF-kB.2

Byrnes aimed to demonstrate that 810 nm light can penetrate deep into the body and promote neuronal regeneration and functional recovery. Adult rats underwent a T9 dorsal hemisection followed by treatment with an 810 nm, 150 mW diode laser (dosage = 1,589 J/cm2). Axonal regeneration and functional recovery were assessed using single and double label tract tracing and various locomotor tasks. The immune response within the spinal cord was also assessed. They found that laser therapy, with 6% power penetration to the spinal cord depth, significantly increased axonal number and distance of regrowth (p<0.001). Laser also returned aspects of function to baseline levels and significantly suppressed immune cell activation and cytokine/chemokine expression. Their results demonstrate that laser therapy delivered transcutaneously improves recovery after spinal cord injury.3

Draper performed a prospective study to determine if low-level laser therapy and surgery for intervertebral disk herniation encouraged ambulation faster than surgery alone. Thirty-six dogs with acute paraparesis/paraplegia due to acute intervertebral disk herniation were evaluated and given a modified Frankel score. Dogs with scores 0 to 3 were included in the study. Dogs were assigned to the control group (1) or the laser treatment group (2) based on alternating order of presentation. All dogs underwent surgery for their herniated disk. Dogs in group 2 were treated postoperatively with low-level laser therapy daily for five days, or until they achieved a modified Frankel score of 4. A 5 × 200 mW, 810 nm, cluster array laser was used to deliver 25 W/cm2 to the skin. All dogs were scored daily by the investigators using the modified Frankel scoring system. The time to achieve a modified Frankel score of 4 was significantly lower (p = 0.0016) in the low-level laser therapy group (median 3.5 days) than the control group (median 14 days). They concluded that low-level laser therapy in combination with surgery decreases the time to ambulation in dogs with T3-L3 myelopathy secondary to intervertebral disk herniation.4

Looney and Huntingford6 performed a study to determine the therapeutic dose of PBMT in dogs with chronic elbow degenerative joint disease. This study proved that the dosage we have always used (4-8 J/cm2) is too low and the therapeutic dose for Elbow OA should be 15-20 J/cm2.
References:
5. Pryor B and Millis D. Therapeutic Laser in Veterinary Medicine in REHABILITATION AND PHYSICAL THERAPY. VET CLINICS OF NORTH AMERICA, SMALL ANIMAL PRACTICE. Jan 2105; (1):45-56.
The Facts
Research conducted by the Association for Pet Obesity Prevention (APOP) found that approximately 58 percent of cats and 54 percent of dogs were overweight or obese in 2015. Obesity is the number one chronic health concern in our canine and feline companions. Obesity is defined as >20% over ideal body weight. Overweight is defined as >10% over ideal body weight.

For a 10 lb cat, that cat is overweight at 11 lbs and obese at 12 lbs!
For a 50 lb dog, that dog is overweight at 55 lbs and obese at 61 lbs

Obesity predisposes dogs and cats to diseases such as diabetes mellitus, heart disease and osteoarthritis. Dogs and cats that obese do not live as long as normal weight dogs.

60% of all dogs and 90% of aged cats have osteoarthritis and most of these pets are obese!

For pet parents, food often equals love and happiness. Many feel that if their pet is overweight he or she is happier. However these pets are not happy—evidence shows these pets are sad. Obesity has become an epidemic for people and for pets. It is not just a cosmetic problem—it is a health problem. We are getting fatter, our animals are getting fatter and many are suffering from painful OA. Painful OA can equal euthanasia in our patients. If we wish to reduce the deaths from euthanasia we have to deal with the pain of OA and the best way to do that is to start with dealing with pet obesity.

Pathophysiology of Obesity
Fat cells historically were considered to be depots of energy that was stored in times of plenty to be used in times of need. However, newer research has shown that adipose tissue is not an inert tissue, but rather it releases a variety of adipokines that drive the chronic inflammatory response in peripheral tissues, thereby exacerbating many disease processes.

Adipose tissues produces adipokines which are cytokines (cell signalling proteins) that are produced by adipocytes. Some more familiar adipokines are leptin and adiponectin. Leptin is the cytokine that when produced inhibits food intake. Lean animals have moderate levels of leptin. However obese animals have greatly increased leptin levels but also have leptin resistance. Adiponectin, another cytokine, increases sensitivity to insulin but it decreases as leptin increases. What we see, then, is that animals with more adiposity have decreased levels of adiponectin and decreased insulin sensitivity. Others are pro-inflammatory cytokines produced by adipose tissue include Tumour
Necrosis Factor Alpha, MCP-1 (monocyte chemotactic protein-1), IL-6, and IL-1. Macrophages migrate into adipose tissue as adiposity increases due to adipose tissue hypoxia. The macrophages then are the source of the reactive oxygen species (ROS) and they drive the oxidative stress response. 1,3,7,8,9

KEY POINT—an obese individual is in a chronic state of inflammation as well as oxidative stress.

Commonly Associated Co-Morbidities of Obesity 4-9

Arthritis (40% dog, 37% cat)
Skin issues
Diabetes
Heart Disease
Exercise Intolerance
Surgical Risk
Decreased Immune Function
**Decreased Life Span—Purina Longevity study***

Purina Longevity study showed that obese dogs live on average 2 years less than a normal weight dog

Causes of Obesity

Genetic issues—lean and obese dogs are metabolically different and are expressing different genes. What up regulates or down regulates genes? Food and nutrition can affect the expression of these genes—this is called nutrigenomics.

Overeating—this is common in our patients 1% overconsumption = 20% overweight in middle age—10 extra kibbles per day can cause a cat to be obese

Age Effects—middle age—most weight gain

Gender—Neutering—this decreases metabolism by 25-30%

Lifestyle and Exercise—less exercise, more obesity

BCS—each level is 10-15% increase/decrease in fat content

OA and Obesity

OA is defined as normal stress on an abnormal joint or abnormal stress on a normal joint—the latter would be the case with obesity. Excess weight and excess inflammation cause joint injury (remember obesity is a disease of chronic inflammation). Inflammation releases the inflammatory cascade from the phospholipid membrane.

Three stages of OA3,4,6,8
Stage One

1. Imbalance in the anabolic and catabolic processes in the cartilage
2. ECM degrades and water content increases
3. Size of aggrecan molecules in matrix decreases
4. Structure of collagen network is damaged which leads to increased stiffness of cartilage.
5. Macrophages in the synovium produce TNF alpha, IL-1Beta, IL-17,IL-18—all pro-inflammatory. These affect the chondrocytes and activate the MMPs and aggrecanase which break down the matrix.

Stage Two

1. Chondrocytes proliferate and increase metabolic activity—produce more MMPs to try repair damage—decreased TIMP. Chondrocytes express COX-2 and produce Prostaglandin E2. This enhances the degradation of aggrecan and Type II collagen
2. Cell clusters form to try and repair damage but catabolism eventually takes over.

Stage Three

1. Repair cannot keep up with damage and cartilage is lost. Chondrocytes produces nitric oxide (NO) synthase which cause progressive cartilage loss. NO inhibits matrix synthesis, activates MMPs and apoptosis.

What does a practitioner do? These patients are painful and will not exercise.. how do we overcome this? Can nutrition help very much? The answer is yes!

Research shows that as little as 6% weight loss improves lameness.

How do we help our patients?

1. Calculate the RER for the ideal body weight.
2. Feed RER x 0.7 for dogs and RER x 0.8 for cats
3. Feed that measured amount—gram scale or cup—gram scale is better
4. Use a therapeutic weight loss food with high protein
5. Do not use an over the counter or maintenance food—you will be short on protein.

RER= ideal body weight kg ^0.75 x 70

Ideal weight loss diet will have increase protein, moderate/high finer, low to moderate fat, isoflavones, be palatable—ideally have added vitamins—L carnitine, flax seed, EFA
For arthritic patients diets like Metabolic+ Mobility are ideal for EFA from fish oil and low calorie.

TIPS FOR SETTING UP A WEIGHT LOSS CLINIC IN YOUR HOSPITAL

1. Discuss nutrition and weight management for every patient.
2. Calculate the dog or cat’s current intake—include all treats.
3. Calculate calories for weight loss using formula above and use a weight loss diet. Show client why a maintenance diet or an OTC weight maintenance diet will not help (often too low in protein—dogs need minimum 2.75 g/kg during weight loss). Dogs with food sensitivity—may have to add novel proteins and adjust diets.
4. Encourage clients to weigh food for their pets.
5. Keep animal pain free while losing weight so that exercise can be encouraged. (NSAIDS, glucosamines, Omega 3)
6. Exercises—weight loss is 80% nutrition and 20 % weight loss—my favourite exercises are aquatic based—swimming and underwater treadmill..slow walks that later incorporate hills, cavalettis, uneven terrain, sand, leaves, deep grass—all good for strengthening and conditioning.
7. Start slow and work up
8. Monitor dog or cat weekly
9. Consider adding Omega 3 fatty acids to diet (100 mg EPA and DHA/kg)—this fights the chronic inflammation but don’t forget the calories.
10. Consider using a Whistle or other activity monitor.
11. Consider sponsoring a “Biggest Loser” competition—Pet Obesity Awareness Day October 12, 2016
12. Get to know a nutritionist for those overweight pets on special diets or homemade diets

**Good references for setting up in clinic programs:**

Purina Project Pet Slim Down https://www.projectpetslimdown.com/

Slim Fit (Royal Canin) https://vet-royalcanin-ca.force.com

Hill’s Healthy Weight Protocol http://www.hwp.hillsvet.com/

ACVN—College of Veterinary Nutrition http://www.acvn.org/nutrition-resources/

Association for Pet Obesity prevention-www.petobesityprevention.org/

**References:**

Therapeutic exercises are a crucial component of any patient’s rehabilitation program regardless of problem or diagnosis. Exercises need to be tailored to the individual patient taking into account the age of the patient, physical condition, condition treated, and resources available. Exercises can be used: for weight loss; to improve a patient’s range of motion; to improve muscle strength, weight bearing, flexibility, balance and proprioception; to improve aerobic capacity (endurance) and performance; and to decrease pain and improve healing in the post operative patient. Therapeutic exercises as part of a home exercise program allow the owners to become involved in their pet’s recovery and often strengthens the bond with a disabled pet.

The Role of Exercise Physiology
Exercising animals rely on skeletal muscles to perform the tasks that they are asked to perform. Skeletal muscle performance is dependent on muscle fiber type. Traditionally, muscles are classified as Type 1 (oxidative, or slow twitch) or Type 2 (glycolytic or fast twitch) (Armstrong, et al., 1982). Postural muscles (stabilizer muscles) such as the quadriceps femoris are capable of slow and sustained contraction and contain more Type 1 fibers than the muscles like the gracilis which contain more Type 2 and are speed and power (mobilizing) muscles. Type 1 muscle fibers have been thought of as the endurance muscle fibers (found in dogs who run long distances like sled dogs) and Type 2 as the sprinting muscle fibers (for sprinting dogs such as Greyhounds). However, in reality, when compared to humans, all dogs have a high oxidative capacity in all their muscles and are adapted for endurance activities (Wakshlag et al., 2004). Certain breeds, however, such as Greyhounds, do have more Type 2 muscle fibers making them better sprinters.

When muscles are immobilized, such as in casts or splints, muscle strength decreases rapidly with as much as 50% of strength lost within the first week. With disuse, postural muscles that contain more Type 1 fibers atrophy more than the mobilizing muscles containing Type 2 fibers. With geriatric sarcopenia, more Type 2 (power and strength) muscle fibers atrophy. This is an important consideration in designing an exercise program for an athlete with muscle loss due to injury versus a geriatric patient with age related atrophy (Appell, 1990).

Muscle contractions can be described as having two variables: force and length. The force is either tension or load. Load is the force exerted on the muscle by an object and muscle tension is the force the muscle exerts on an object. Isometric contractions occur when muscle tension changes with no change in muscle length. Isotonic contractions occur when the muscle tension remains the same but the muscle length changes. In canine exercise, we are concerned with isotonic contractions. Isotonic contractions occur as either concentric or eccentric contraction. Concentric contraction occurs when the muscle contracts, tensions in the muscle increases and shortening occurs. An example of this would be a human weight lifter performing a biceps curl. Eccentric contraction occurs when the muscle contracts but lengthens because the tension generated in the muscle is insufficient to overcome the load pulling down on the muscle. Think of the weight lifter slowly releasing the biceps curl and extending his elbow to put down the weight. The eccentric contraction is controlling the movement—it is the natural braking force that occurs during movement (Gillette et al, 2014).
Eccentric contractions tend to preferentially strengthen Type 2 muscle fibers whereas concentric favor Type 1 fibers.

In an exercise program, generally, concentric exercises are performed first to help accustom the muscle to movement. Eccentric exercises are added later as these have the potential to cause damage to the muscle and delayed onset muscle soreness but they help developed greater strength. A balanced program between concentric and eccentric muscle contractions is desired (Saunders, 2007).

**Principles of an Exercise Program**

According to McCauley and Van Dyke there are 5 variable parameters in any exercise program:
1. Frequency of work done (multiple times per day, daily or weekly)
2. Speed/intensity
3. Duration of work (time or number of reps)
4. Environment (terrain, footing, substrate)
5. Impact (low, high or no impact)

As the patient heals the frequency, intensity and duration of the exercise is increased to further challenge the patient and to strengthen the muscles. It is usually safe to increase the activity by 10 to 15% each week as long as the patient does not experience an increase in pain or a loss of function (Millis et al., 2014). It is important to monitor the patient for signs of fatigue—lagging behind, refusing to do exercises, panting, laying down, a spade shaped tongue, drooping tail and ears, elevated heart rate and muscle trembling. If these signs occur, one more rep should be requested from the patient to preclude avoidance behavior. The patient’s behavior the next day should be noted and the exercise program adjusted if necessary (McCauley et al, 2013)

**Control, Supportive and Assistive Devices**

Harnesses, safety vests and leashes should be used to control the patient during exercise. Harnesses that do not restrict shoulder motion are used for rehabilitation. For patients that are not ambulatory or ataxic, front and back end harnesses are used to assist both the patient and the therapist. Booties are used if the surfaces are slippery or if the dog is weak and dragging of the feet may be a problem. Doing exercises over balls, or foam rollers, over peanuts or while in a sling can be very useful for neurological patients.

**Walking**

Controlled leash walking is an excellent therapeutic exercise. Generally it is easy to do, dogs enjoy it. Health benefits include increased endurance, strength, cardiovascular fitness and good mental stimulation. Unless the dog is confined for safety and only allowed outside for bathroom privileges, most dogs can start walking soon after surgery. Leash walks generally consist of 5 minutes 2 to 3 times daily at first and each week progress by 1 to 5 minutes per walk depending on the advice of the surgeon or the experience of the therapist. During inclement weather or cold winters, land treadmill walking may be preferable to walking outdoors. Treadmill walking although it provides exercise, weight bearing and strengthening does not give the dog (or owner) any mental stimulation (Saunders, 2007)
Specific exercises are broken up into several categories

1. Balance, and Proprioceptive Exercises
2. Core strengthening
3. Hind Limb Exercises
4. Forelimb Exercises
5. Neurological Rehabilitation Exercises
6. Exercises for Cats

Balance and Proprioceptive Exercises
Cavaletti Course—The goal is to walk over the poles without touching them. Generally start with 5 minutes twice daily.

Balance Boards or Balance Discs—These are used for balance, proprioception, and strengthening legs. Place the weakest legs on the board or all 4 paws. Control the patient with a harness and rock the board back and forth.

Weave Cones—These are generally used to improve proprioception and increase core strength. Objects, normally 6-8, are lined up about 1-1/2 to 3 feet apart (depending on patient size) and the dog is weaved in and out of the objects.

Figure Eights
This exercise is also done with cones and is used to increase balance, coordination, spinal range of motion and for weight shifting. It is completed by walking the outline of the number “8” around 2 cones. The figure eight normally is twice the height of the dog and the activity should be performed a slow speed. The slow speed allows for an increase in spinal range of motion. This should be done a few minutes at a time to avoid dizziness.

Blocks—These are used in developing proprioception and core muscles. The animal places one paw on each block in a standing position and holds the stance for increasing periods of time. The blocks can be moved closer to midline or both paws can be on one block. Alternatively peanut balls can be used to simulate a plank exercise with the dog holding the plank for a period of time.

Balance Beam—This is a length of plywood described above that the dog walks along and sits on while maintaining his balance or posture. The goal is to have the dog walk along and not step off the beam.

Trampoline, Cushions, Air mattress—These uneven surfaces help improve weight bearing as well as proprioception. Be sure to use a harness to control the patient so he or she does not lose balance and fall.

Rhythmic Stabilization—Weight shifting is used to increase weight bearing and balance. Weight shifting should be done while the dog is standing. Place your thumbs over the dog’s pelvis bones and your hands down their sides. Slowly sway side-to-side making sure both legs are weight bearing. Do not use enough force to cause the dog to lose his balance.
Ball Work
Ball Work — this increases proprioception, allows for advanced strengthening and balance and increases core and trunk stability. Most exercises can be performed on a theraball. Once the dog has mastered exercises on solid ground, balls can slowly be introduced.

Core Strengthening
Crawling
This exercise is great for core strengthening, improving spinal mobility, range of motion and leg strengthening. It can be done anywhere and with anything; dogs can crawl under chairs, beds, boxes, agility tunnels or they can just learn to crawl along the floor. The higher the “crawling tunnel” that is created, the easier the exercise is for the patient.

Sit Up and Beg
This helps core strengthening, and hind leg strength. Initially have the dog sit squarely and then get up onto their back legs as though they were begging. Treats will help make this easier to teach. This exercise should not be done if the dog has back or hip issues since too much pressure will be placed on those already injured areas.

Diagonal Leg Lifts
This exercise helps with core strengthening but also balance, weight shifting and leg strengthening. The stance is achieved by lifting one leg off the ground along with the diagonal leg. Both legs are to be lifted at the same time and minimal support to the limbs should be provided so the dog has to balance. Hold this pose for 10 to 30 seconds and repeat 8 to 10 times.

Hindlimb Exercises
Sit to stand
This exercise helps improve strength in the pelvic limbs, particularly in the gluteal and hamstring muscles. When performing this exercise it is important to assure that the dog sits squarely and when he stands that he uses both pelvic legs to propel the body up, not the front limbs. This exercise can be repeated over and over for increased strengthening.

Backwards Walking
This exercise helps strengthen the pelvic legs (particularly the hamstring muscle group), increase balance, coordination and proprioception. This exercise is easier to start with the dog. This is an important exercise to teach all dogs and puppies as at some time in their lives, all dogs will need more hindlimb strength. A backwards walking session should be 5 to 10 minutes twice daily.

Incline Walking
Incline walking helps build muscle in the rear legs due to the weight being shifted towards the back. This exercise should be started with shallow inclines and then steeper inclines can be added when the pet is comfortable and adjusting well. Zig zag hill walking can also be incorporated wherein the patient walks in a zig zag pattern across the hill up and down.

Loving on Stairs/Couch
This exercise is used to improve rear leg weight bearing and strength, range of motion and hip extension. This causes the dog’s weight to be shifted to the pelvic limbs thus the weaker back legs have to support the majority of the body’s weight.

**Ladder Walking**
This exercise strengthens the legs, improves range of motion, balance, coordination and proprioception. The exercise is performed with a ladder placed flat on the ground. This particular exercise is similar to cavalettis but can change in difficulty. Initially the dog walks forward over the rungs but can be taught to walk backwards and sidestep through the ladder. This exercise should start at 5 minutes twice daily and work up in frequency.

**Front Limb Exercise**
High 5 salute
High five salute- helps with range of motion and strengthening of front leg.

**Wheelbarrow**
Wheelbarrow helps build strengthen the front limb and the core. This may seem like an easy exercise but it is actually quite challenging and needs to be done slowly. Do not do this exercise in a patient with spinal issues, shoulder problems or carpal hyperextension.

**Stairs**-
Descending stairs- helps build muscle in the front legs since the majority of the weight is forced to the front half when walking down stairs.

**Play Bow**
This exercise helps increase forelimb strength and flexibility and promotes core strengthening.

**Digging**
This is a good exercise to help strengthen the front limbs, improve ROM, proprioception and core strength.

**Neurological rehabilitation exercises**

**Assisted standing**
This exercise is important to build and maintain muscles needed for balance, proprioception, and locomotion. These muscle quickly become atrophied if not used as frequently occurs with paresis. A therapy ball, rolled towel, cushion, foam roller or other device (depending on the size of the dog) is placed under the dog’s abdomen.

**Weight shifting**
Neurological patients need to practice balancing on 3 limbs. To perform this exercise the therapist places dog in standing position and lifts one limb. When dog starts to sway limb is replaced. All 4 paws are rotated through this exercise.
Proprioceptive Neurological Facilitation (PNF) patterns
PNF patterns mimic the dog’s running motions and other normal functions of daily living (scratching, digging). PNF patterns help with ROM, stimulate the neural pathways and help redevelop new axonal pathways for movement. To perform a PNF pattern for running, lay the dog on his side and mimic the running pattern. The therapist should use one hand to mimic the ground contact at the appropriate part of the gait cycle. (Edge-Hughes, 2007)

Tapping
Tapping over a muscle belly can elicit muscle contractions and stimulate the neural receptors (Muscle Spindle Fibers and Golgi Tendon Organs) in muscle and tendon. This should be done for 3 to 5 minutes a few times a day as part of the nursing care for animals recovering from paralysis.

Tensor Bandaging, Thundershirts or Snuglis
The principle of using tensor bandages or thunder shirts for neurological dogs is to connect the front and back end of the dog and create body awareness. The slight pressure that the wrap puts on the body helps with neurological co-ordination. Many of these dogs can wear such shirts or wraps throughout recovery.

Tactile Stimulation
This is beneficial to provide stimulation to the superficial receptors in the patient’s skin. Brushing the dog, tapping, pinching, or using a vibrator provides additional sensory stimulation to increase input into the nervous system. (Edge-Hughes, 2007)

Mental Stimulation of Neurological Patients
The therapist should be aware that the patient’s mental state plays a big role in recovery. Dogs can become depressed if they can not move, get outside or interact with their family. Dogs can be mentally stimulated by taking them outside or “walks” in a wagon or stroller, moving them around the house frequently if they are unable to move, giving them new treats or squeaky toys..anything that can engage the patient will be mentally stimulating and speed recovery.

References:


What is a nutraceutical?
A food (or part of a food) that provides medical or health benefits, including the prevention and/or treatment of a disease.

What is a dietary supplement?
Product taken by mouth that contains a dietary ingredient intended to supplement the diet or a substance produced in purified or extracted form which, when administered orally to patients, aims to provide them the necessary elements for their structure and normal function to better their health and wellbeing.

Nutraceuticals are used by a large number of veterinary clients for osteoarthritis and are one of the fastest growing areas of supplementation for pets.

Goals for a nutraceutical to relieve OA pain:
1. Decrease in inflammatory prostaglandin (PGE2).
2. Decrease the production of Pro MMP 2 & 9 and active MMP 2 and 9 (the enzymes responsible for degradation of cartilage).
3. Increase the inhibitor of MMP (TIMP-2) to help restore proper balance between these enzymes.

Evidence based nutraceutical use:

Fish oil--Omega 3 DHA and EPA1,2,3,4

Arachidonic acid (AA) is the primary substrate for the lipooxygenase and cyclooxygenase enzymes. This fatty acid is derived from dietary sources and stored in phospholipids of the cell membrane until needed. AA is a member of the omega-6 fatty acid family. AA can be partly replaced in cell membranes by the omega-3 fatty acid Eicosapentanoic Acid.

The difference between omega-6 and omega-3 fatty acids centers on the location of the first double bond in the carbon chain, occurring either at the 3rd or 6th carbon from the methyl end. While mammalian cells can elongate and desaturate fatty acids, they are not able to form double-bonds beyond these defining bonds, so are unable to synthesize these fatty acids nor interconvert between these families. Thus, the presence of these fatty acids within cell membranes reflects dietary intake. And this can be important because the physiologic function of the 2 fatty acid families differ.

Eicosanoids are metabolically active compounds derived from 20-carbon fatty acids, usually arachidonic acid. The lipooxygenase (5-LOX) and cyclooxygenase (COX) enzymes are the rate-limiting steps in the production of leukotriene B4, thromboxane A2 and prostaglandin E2. In health, these eicosanoids serve important functions. However, in inflammatory conditions such
as arthritis, their production can be increased and their effects can be detrimental. For example, PGE2 can be increased up to 50 fold in arthritic joints. Leukotriene B4 has a potent chemotactic effect and promotes further inflammation. PGE2 and TXA2 both promote the release of tumor necrosis factor alpha and interleukin 1beta, both which promote further inflammation and, in joints, stimulate the production of matrix metalloproteinases or MMPs. MMPs are the collagen-destroying enzymes that break down articular cartilage in arthritic joints. Further, PGE2 is a potent stimulator of pain receptors, and contributes to the pain of arthritis.

Eicospentaenoic acid (EPA) also can be used by the LOX and COX enzymes to produce eicosanoids. However, when EPA is used by the COX and LOX enzymes, they produce the eicosanoids PGE3, thromboxane (TX) A3 and LTB5, which are less active and relatively anti-inflammatory compared to their counterparts produced from AA.

It has been demonstrated that therapeutic diets containing approximately 3.5% omega 3 fatty acids can decrease pain and lameness, improve weight bearing, and decrease the need for NSAIDs in dogs with OA. The primary source of omega 3 fatty acids is fish oil. Approximately 480 mg/kg of fish oil (50–100 mg/kg EPA) would be required as a supplement to match the amounts available in the therapeutic food discussed above. A recent placebo-controlled clinical trial in dogs with OA investigated the effects of a fish oil supplement added to a non-fish based food, dosed at 90 mg/kg EPA and 20 mg/kg DHA. These researchers found significant improvement in indicators of pain and quality of life when comparing the base-line outcome measures to those collected at the end of the 16-week trial. There is a high level of support for supplementation of omega 3 fatty acids.

**Mobility Diets**

All mobility diets are not created equal! Research shows that 7.5 g EPA +DHA/1000kcal diet significantly reduced symptoms of arthritis. This amount is quite unwieldy as well as likely to cause diarrhea. Other studies have shown as little as 1 to 3 g/1000kcal has clinical effect. Ideally for most dogs you would like to get up to the 100mg/kg of Omega 3 for arthritis. Here is an example:

For a 20kg dog you would like it to receive 2 g of Omega 3 total/day for arthritis. This dog would eat around 700 kcal so if feeding a 1.5 g Omega 3/1000kcal diet it would provide approximately 1 gram of Omega 3. To make up the additional gram, you would have to supplement with 2 capsules that contain 500mg of EPA and DHA combined. This is quite feasible.

**Green-Lipped Mussel**

Perna canaliculus is found in the waters around Australia and New Zealand. It contains EPA, DHA, and ETA. It is also a source of glycoproteins and GAGs. A randomized, double-blind, placebo controlled clinical trial in dogs with chronic pain attributed to OA found significant improvement in mobility and pain in those dogs treated with GLM compared to placebo. The dose used was 50 mg/kg. The anti-inflammatory effects of GLM may be derived from its...
omega-3 fatty acids content or the GAGs or the glycoproteins. This has yet to be determined but it does prove to be at least mildly effective.6,7

**Avocado/Soybean Unsaponifiables**

Avocado soybean unsaponifiables (ASU) are residues of avocado and soy oils combined in a 1:2 ratio to produce a product that has demonstrated anti-arthritic properties. Theoretically, ASU decrease the production of pro-inflammatory cytokines such as PGE-2 and TNF alpha. In a canine cruciate ligament transection model, ASU administration decreased osteophytes, improved cartilage thickness and produced more normal chondrocytes. Additional in vitro studies have shown that the combination of ASU with chondroitin is more effective in decreasing inflammatory cytokines than either product alone. There are no published controlled trials in clinical dogs with OA examining ASU alone or in combination products. However research on induced arthritis shows a positive result. Dasuquin (Nutramaxx) is the product generally used.

**Chondroprotectants**

**Glucosamine/Chondroitin**

Glucosamine is a precursor of glycosaminoglycan (GAG). When administered orally, glucosamine is 90% absorbed and undergoes biotransformation in the liver. It is then distributed to tissues and has been shown to have a tropism for articular cartilage. Glucosamine sulfate is absorbed better than glucosamine hydrochloride and may be more effective.

The mechanism of action of glucosamine has not been fully elucidated. In vitro studies have shown that when exogenous glucosamine is administered, it is utilized in the synthesis of GAGs. It has also been demonstrated that supplementation with glucosamine inhibits enzymes that are responsible for the degradation of cartilage, and the production of inflammatory mediators is decreased.

Chondroitin sulfate is a much larger molecule than glucosamine, and its oral bioavailability has been questioned. Low-molecular weight chondroitin sulfate is more effectively absorbed by the gastrointestinal tract than larger molecules. Metabolites of chondroitin sulfate are concentrated in articular cartilage. The mechanisms of action of chondroitin are: to stimulate GAG production; inhibit degradative enzymes; enhances the production of hyaluronic acid and prevent the degeneration of type II collagen within articular cartilage. Glucosamine and chondroitin sulfate are often combined in commercially available products. It appears that there is a synergistic effect when the two products are used together.

Studies demonstrating efficacious use of glucosamine/ chondroitin are few. McCarthy et al showed glucosamine/ chondroitin improved pain, weight bearing and disease severity scores (3/5 measures) but the onset of response was slower for glucosamine/chondroitin compared to NSAIDs. Moreau et al showed no change with the supplement so evidence is conflicting. In a systematic review only 13 studies were controlled and evidence was positive for
Glucosamine/chondroitin but this is a human study. The level of evidence supporting the use of glucosamine/chondroitin for pain management in dogs is low.

Dosage: Dose at 15mg/kg on the Chondroitin fraction.

**Flex-RX**
This product is a bioflavanoid that contains Baicailin and Catechin and has balanced COX and 5-LOX enzyme inhibition activity. In studies by Burnett et al it showed statistically significant improvement in pain scores when compared to Cosequin using veterinarian and owner VAS.

**Elk Velvet Antler**
Quality Elk Velvet comes from the antler at the velvet stage and contains Chondroitin Sulphate, collagen, glycosaminoglycan and pilose antler peptide. Study by Morneau showed improvement in dogs with clinical OA on force plate and by subjective analysis.

**Boswellia**
This is also know as Indian Frankincense in Ayurvedic Medicine. 4 compounds isolated have been isolated and purified. These have been found to have anti-LOX activity. This herb is found in human products Flexamine as Aflapin and Osteo-biflex as 5-Loxin. 2 Placebo controlled clinical trials in humans suggest efficacy for joint pain. In an unblinded open label Austrian study it was found to have 71 percent positive response in clinically lame dogs.

**Theracurmin**
Curcumin is found in veterinary nutraceuticals marketed for arthritis. Its utility as a natural NF-kB (nuclear factor kappa-light-chain-enhancer of activated B cells) and cyclooxygenase-2 inhibitor is documented in humans but not in dogs. However, its gastrointestinal absorption in most species appears to be poor. An extract of turmeric, the spice from which curcumin is derived, produced subjective, but not objective, improvements in dogs with arthritis. Theracurmin is a new water soluble curcumin that has shown to have advantages and may have promise in the future in dogs.

**References:**
Equine uveitis, and especially equine recurrent uveitis (ERU), is the leading cause of blindness in the horse. This disease can prematurely halt the career of a promising athlete. There are several underlying causes that can lead to uveitis and ERU. The clinical signs can be at times subtle, or obvious, on ocular examination. There are also several therapies that can help an equine eye rebound from these diseases and individualization of therapy can be instrumental to a successful outcome. In this session, we will review the clinical signs, diagnostic tests available, and current recommended therapies for uveitis and ERU.

Definitions:

Uveitis:
Single episode(s) of intraocular inflammation occur due to various causes, such as trauma and corneal ulceration. However, if medical therapy is discontinued too soon, the inflammation may return within a short period of time, such as 2-6 weeks. This case scenario is referred to as a pseudo-recurrence and can result in a misdiagnosis of equine recurrent uveitis (ERU). Stopping therapy after 14 to 21 days, when subtle signs of inflammation (aqueous flare, pinpoint keratitic precipitates) may be overlooked without careful examination, leads to the development of pseudo-recurrences. If left overlooked, these eyes mimic cases of insidious ERU and result in progressive intraocular changes ending in decreased vision or blindness. To prevent these pseudo-recurrences, it is recommended that the anti-inflammatory and immune-suppressive drugs are tapered off over a prolonged course of treatment (generally, 6-8 weeks).

Equine recurrent uveitis (ERU):
Multiple bouts of intraocular inflammation and associated clinical signs interrupted with variable periods of quiescence that require no therapy. True relapse of inflammation occurs following complete elimination of inflammatory lesions (aqueous flare, keratitic precipitates, fibrin clots, miosis, vitritis, or fundic lesions) with anti-inflammatory and immunosuppressive medication. Ensuring that a true state of quiescence is reached by gradually tapering medications over a prolonged period of time helps to accurately assess the horse’s underlying state of inflammation. Recurrences that occur frequently (every 3-4 months, or less), and require longer durations of treatment before the signs of inflammation subside, are more likely to develop debilitating ocular complications associated with ERU.

Clinically, ERU has been categorized into 3 syndromes: classic, insidious, and posterior ERU.
- Classic ERU: most common and is seen by periods of active and painful inflammation followed by a quiescent phase. Repeated attacks often result in vision deficits or loss.
- Insidious ERU: characterized by a persistent low grade usually non-painful intraocular inflammation with gradual and cumulative destruction. The continual
gradual destruction of the eye can lead to cataract formation and blindness. Appaloosa horses commonly have this form of ERU.

- Posterior ERU: distinguished by inflammation primarily present in the posterior segment (vitreous, retina, and choroid). Frequently, retinal degeneration is seen with this form of ERU. Warmbloods and draft breeds are often observed with this form of ERU.

**Pathophysiology**

There are several causes of uveitis. Blunt or perforating trauma appears to be one of the most common causes, followed closely by corneal ulceration. As for ERU, there are several reported causes which include immune-mediated disease as well as idiopathic. Leptospira infection has gained attention in the literature as a significant cause of ERU.

**Role of Leptospira in ERU**

In the United Kingdom, one study reporting on the incidence of serological and aqueous humour evidence of Leptospira was not conclusive as only 6/29 ERU horses had positive aqueous detection and no statistical differences were seen with serology between ERU horses and controls. In Germany, high association between Leptospira and ERU has been established in the literature. In Canada, the eastern section of Ontario and western Quebec is an endemic area and therefore high association is expected. Data was presented at the annual meeting of the American College of Veterinary Ophthalmologists in 2005 that supported the presence of the Leptospira organisms present in the eye of affected horses with high serological titers that resided in Western Quebec. In 1979, Leptospira interrogans serotype Pomona was reported to be enzootic in the Saskatchewan equine population; however, recent reviews are lacking.

This apparent association is not without controversy. In one study: “Serologic results did not correlate well with the presence of Leptospira DNA or organisms in the aqueous humor. Leptospira spp. are present in a high percentage of horses with naturally occurring recurrent uveitis.” As reported by Sandmeyers et al, “The association of Leptospira spp. with ERU is well-known, particularly Leptospira interrogans serotype Pomona; however, this association is not completely understood. It is theorized that for at least some horses, infection with Leptospira spp. is the inciting cause of uveitis”. Some authors debate its role in chronic recurrences but it is thought that the bacteria may have molecular resemblance with the immune system. The Appaloosas has been identified as an at risk breed with an increased incidence of vision loss in seropositive horses. This author also has seen a likely association with the Canadian horse in residing in western Quebec.

**Review of clinical signs of uveitis and ERU**

Intraocular inflammation will lead to a myriad of clinical signs. Severity of the clinical signs will mirror the severity of the uveitis present. With each episode of uveitis, the eye will succumb to inflammation and permanent damage which could lead to blindness.
Table 1: Initial clinical signs associated with ERU

<table>
<thead>
<tr>
<th></th>
<th>Classic</th>
<th>Insidious</th>
<th>Posterior</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eyelids</td>
<td>Swollen</td>
<td>Intermittent tearing</td>
<td>Normal</td>
</tr>
<tr>
<td></td>
<td>Tearing</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Conjunctiva/sclera</td>
<td>Moderate to severe congestion</td>
<td>Mild congestion</td>
<td>No to mild congestion</td>
</tr>
<tr>
<td>Cornea</td>
<td>Edema</td>
<td>Normal or mild edema</td>
<td>Normal</td>
</tr>
<tr>
<td></td>
<td>Keratitic precipitates</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anterior chamber</td>
<td>Aqueous flare</td>
<td>Subtle aqueous flare</td>
<td>± aqueous flare</td>
</tr>
<tr>
<td></td>
<td>Fibrin clot</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Hypopion</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Iris</td>
<td>Inflamed</td>
<td>Miosis</td>
<td>Miosis</td>
</tr>
<tr>
<td></td>
<td>Miosis</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Dyscoria (posterior synechiae)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lens</td>
<td>Normal</td>
<td>Cataracts</td>
<td>Normal</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vitreous</td>
<td>Liquefied</td>
<td>Liquefied</td>
<td>Liquefied</td>
</tr>
<tr>
<td></td>
<td>Hazy</td>
<td>Hazy</td>
<td>Hazy</td>
</tr>
<tr>
<td>Retina</td>
<td>Bullet hole lesions</td>
<td>Normal</td>
<td>Bullet hole lesions</td>
</tr>
<tr>
<td>Optic nerve</td>
<td>Peripapillary lesions</td>
<td>Normal</td>
<td>Peripapillary lesions</td>
</tr>
<tr>
<td></td>
<td>possible</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Medical management of ERU

There are several medications that can attenuate or resolve the clinical signs seen with all forms of uveitis and ERU. One element to remember is to not wean these medications too quickly as recurrences or relapses are likely. Pending the severity of clinical signs, one or more medications may be required and frequency will need to be adjusted to response to therapy.

Table 2: Medical therapy for uveitis

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dosage</th>
<th>Comments</th>
<th>Contraindications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Topical</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Dexamethasone 0.1%</td>
<td>q2-12 hours</td>
<td>May need subpalpebral lavage system for frequent delivery</td>
<td>Do not use with corneal ulcerations</td>
</tr>
<tr>
<td>Prednisolone acetate 1%</td>
<td>q 2-12 hours</td>
<td>May need subpalpebral lavage system for frequent delivery</td>
<td>Do not use with corneal ulcerations</td>
</tr>
<tr>
<td>Diclofenac 0.1%</td>
<td>q 6-8 hours</td>
<td>Compounded product only</td>
<td>Do not use if hyphema is present</td>
</tr>
<tr>
<td>Atropine 1%</td>
<td>q 8-48 hours</td>
<td>Give to effect and wean down</td>
<td>Do not use if signs of impaction; can slow down gut motility, especially in foals</td>
</tr>
</tbody>
</table>
### Systemic

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dose/Route</th>
<th>Duration &amp; Wean Down Note</th>
<th>Side Effects &amp; Precautions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Flunixin meglumine</td>
<td>1mg/kg IV, IM PO q 12-24h (severe cases) 3-5 days; wean down q 3-5 days as judged by clinical signs</td>
<td>Long-term use may cause gastric and renal problems, verify total protein and creatinine levels often</td>
<td>Do not use with horses with kidney insufficiency</td>
</tr>
<tr>
<td>Phenylbutazone</td>
<td>1g PO q 12-24h 3 days; then as needed</td>
<td>Long-term use may cause gastric and renal problems, verify total protein and creatinine levels often</td>
<td>Do not use with horses with kidney insufficiency</td>
</tr>
<tr>
<td>Dexamethasone</td>
<td>5-10mg/24h PO or 2.5-5.0 mg IM q 24h</td>
<td>May cause laminitis or exacerbate systemic infection</td>
<td></td>
</tr>
<tr>
<td>Prednisone</td>
<td>100-300 mg/24h PO, IM</td>
<td>See comment for Dexamethasone</td>
<td></td>
</tr>
</tbody>
</table>

### Injectable medication

<table>
<thead>
<tr>
<th>Medication</th>
<th>Dose/Route</th>
<th>Duration &amp; Wean Down Note</th>
<th>Side Effects &amp; Precautions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Triamcinolone</td>
<td>10-20 mg subconjunctival</td>
<td>Shake bottle well and inject soon after withdrawal</td>
<td>Do not give if corneal ulceration is present</td>
</tr>
<tr>
<td>Tissue plasminogen activator (Alteplase)</td>
<td>50 mcg Anterior chamber</td>
<td>Available at referral only</td>
<td>Will not treat hypopion</td>
</tr>
<tr>
<td>Gentamicin</td>
<td>4mg Intravitreal</td>
<td>Heavy sedation or GA Reserved for Leptospira cases</td>
<td>May progress cataract formation</td>
</tr>
</tbody>
</table>

**Surgical management of ERU**

### Suprachoroidal Cyclosporine Implants

Horses with ERU that are well controlled with conventional anti-inflammatory medical therapy (see above) successfully leading to a period of quiescence that remains even after medications are discontinued, are excellent candidates for suprachoroidal cyclosporine implantation. Intraocular inflammation must be well controlled and not active at the time of surgery. Leptospira-associated cases have not been evaluated with this procedure. Low recurrence rates have been reported and additional implants may be needed in the future. This is a referral procedure as the implants are only provided to boarded veterinary ophthalmologists.
**Pars Plana Vitrectomy**
The pars plana vitrectomy has seen widespread use in Europe. It is an intraocular surgery that removes the core of the vitreous with the horse under general anesthesia. Reported postsurgical complications include transient hypopyon, vitreal and/or retinal hemorrhage, retinal detachment, and cataract formation. Horses with Leptospira-associated uveitis and moderate to severe vitreal inflammation (membranes) are considered good surgical candidates. Although the vitrectomy has seen widespread use in Europe, this procedure is not routinely performed in North America.

**Prevention**

**Genetic testing**
In western Canada, 62.5% of the horses diagnosed with ERU were Appaloosas. In the USA, the Appaloosa, Quarter horse, Thoroughbred, Warmblood, Hanoverian, and the American Paint Horse have the highest ERU occurrence reported. The Appaloosas have been reported to be 8 times more likely to develop ERU and significantly more likely to go blind in 1 or both eyes, as a result of the inflammatory process, than any other breed. In Germany, Warmblood horses carrying the equine leukocyte antigen (ELA) ELA-A9 haplotype had the highest risk of ERU. This haplotype was present in 41% of the cases but was not present in the controls. Unfortunately, genetic typing for ELA to determine genetic risk for ERU is not available.

In the Appaloosa, leopard complex spotting (LP) of the coat pattern is thought to be a polygenic trait with incomplete dominance. The extent of the white patterning is theorized to be caused by additional genes. Horses that are heterozygous for LP (LP/N; 1 copy of the allele controlling for the presence of the spotting pattern and 1 copy of the wild-type allele) tend to have more oval spots of pigment in the white patterned area compared with homozygotes (those with 2 copies of LP).

The LP test is the lone commercial genetic test available for the Appaloosas. Risk for ERU based on this genetic test can be evaluated as LP/LP > LP/N > N/N. Horses at the highest risk of developing ERU are homozygous for LP. Heterozygous horses for the LP mutation are at a higher risk of developing ERU than horse without the breed-identifying white spotting pattern. Genotype with respect to LP can therefore be used to identify at-risk horses to monitor and treat for signs of uveitis. Careful monitoring and management of at-risk horses is recommended.

**Vaccination:**
In cases of Leptospira-associated ERU, vaccination may be the next step in prevention. This may be valuable especially in at-risk Appaloosas, as these horses are at great risk of blindness if infected with Leptospira. In the past, no commercially available vaccines were sold in North America but Zoetis USA has now developed and marketed such a vaccine.

“LEPTO EQ INNOVATOR® IS THE FIRST AND ONLY LEPTOSPIROSIS VACCINE FOR HORSES.”
Now you can help protect your horse with LEPTO EQ INNOVATOR®, the first and only equine vaccine to help prevent leptospirosis. LEPTO EQ INNOVATOR helps prevent leptospiremia caused by L. pomona, which could, but has not be demonstrated to, help reduce the potential risk of equine recurrent uveitis (ERU) infections, abortions or acute renal failure caused by L. pomona.*

*Currently, there are no vaccines available with USDA-licensed label claims against equine abortions, uveitis or acute renal failure due to L. Pomona”

There are no published studies to indicate the efficacy of this vaccine on different LP genotypes with respect to Appaloosas.

References:
3. Faber NA, Crawford M, LeFebvre RB, Buyukmihci NC, Madigan JE, Willits NH.
The equine eye is prone to trauma and consequently, corneal disease is commonly diagnosed in private practice. Corneal ulceration, corneal stromal abscesses and eosinophilic keratitis are common eye conditions seen in the field. Corneal ulceration can be challenging to treat in non-compliant patients or in patients with severe infection. Tricks of the trade for equine ocular exam will be presented in this session. We will also discuss the value of diagnostic tests when faced with these patients. Lastly, up to date therapies will be presented for the corneal diseases discussed during the session.

Anatomy and response to injury
The equine cornea is 0.8-1.0 mm thick, devoid of keratin, pigment, blood vessels and lymphatics but is richly innervated by the trigeminal nerve. It is covered by a tear film that contains mostly gram positive bacteria and possible fungal organisms. It is subject to trauma and responds immediately to repair itself. This repair starts with excessive lacrimation, blepharospasm, and local corneal epithelial migration. Should the resulting lesion remain or progress, with time, ingrowth of corneal blood vessels and corneal epithelial cells will start at the limbus and venture towards the corneal lesion.

In cases of corneal ulcers, corneal epithelium will start to slide and migrate over the exposed stroma within the first hour of insult and be maximal in the first 5-7 days. The corneal stroma will start to repair itself within the first 24 hours. Neutrophils and immunoglobulins will enter the cornea from the tear film and aide against infection. Should the corneal epithelium completely cover the exposed stroma, this phenomenon will halt stromal regeneration and could trap infection (stromal abscess). Mixed infections can include fungal organisms and the hyphae can reside deep within the stroma and penetrate Descemet’s membrane to get access to the anterior chamber.

Ocular examination findings
A comprehensive ocular examination will enable the veterinarian to obtain valuable information that will not only confirm the diagnosis, but also the therapeutic plan and prognosis. Prior to the ocular examination, intravenous sedation and local blocks will increase safety for the examiner and patient. Performing the auriculopalpebral block will paralyze the upper eyelid and the frontal block will desensitize the upper eyelid. These blocks are essential when dealing with a painful eye. Furthermore, a dark room is necessary to optimize the ocular examination by minimizing corneal reflection.

The use of ophthalmic dyes is common when investigating corneal disease. Fluorescein stain is routinely applied during an ocular examination and enables the veterinarian to detect absence of corneal epithelium or exposed corneal stroma. The use of rose Bengal dye is controversial as it will detect tear film abnormalities as opposed to epithelial disease. Also, used in routine
concentration, the dye is toxic to corneal epithelium and requires slit-lamp biomicroscopy for adequate visualization.

**Table 1: Ocular examination**

<table>
<thead>
<tr>
<th>Neuro-ophthalmic</th>
<th>Indications</th>
<th>Comments</th>
<th>Contraindications</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Dazzle</td>
<td>Subcortical reflex testing CN II &amp; VII</td>
<td>Challenging to test if painful eye</td>
<td></td>
</tr>
<tr>
<td>• Menace</td>
<td>Cortical response testing CN II &amp; VII</td>
<td>Challenging to test if miotic pupil</td>
<td></td>
</tr>
<tr>
<td>• Palpebral</td>
<td>Subcortical reflex testing CN V &amp; VII</td>
<td>Test if exophthalmic or buphthalmic eye</td>
<td></td>
</tr>
<tr>
<td>• Pupillary light reflexes (Direct and indirect)</td>
<td>Subcortical reflex testing CN II &amp; III</td>
<td>Challenging to test if miosis, hyphema, hypopion or fibrin present</td>
<td></td>
</tr>
</tbody>
</table>

**Ocular tests**

<table>
<thead>
<tr>
<th>Schirmer tear test</th>
<th>Chronic ulcer</th>
<th>Unnecessary if excessive lacrimation present</th>
<th>Caution with deep ulcer or descemetocele</th>
</tr>
</thead>
<tbody>
<tr>
<td>fluor escein stain uptake</td>
<td>Corneal ulceration</td>
<td>Do not over dilute stain</td>
<td>Caution with deep ulcer or descemetocele</td>
</tr>
<tr>
<td>Tonometry</td>
<td>Determine level of uveitis present</td>
<td>Tonopen or Tonovet</td>
<td>Caution with deep ulcer or descemetocele</td>
</tr>
<tr>
<td>Corneal cytology</td>
<td>Mid-deep ulcer</td>
<td>1 drop of topical proparacaine</td>
<td>Caution with descemetocele</td>
</tr>
<tr>
<td>Corneal culture</td>
<td>Mid-deep ulcer</td>
<td>Aerobic and mycotic cultures</td>
<td>Caution with descemetocele</td>
</tr>
<tr>
<td>Seidel’s test</td>
<td>Testing for corneal integrity</td>
<td>Suspect corneal rupture or perforation</td>
<td></td>
</tr>
<tr>
<td>Ocular exam</td>
<td>Magnification and fully charged light source</td>
<td>Performed in a dark room</td>
<td></td>
</tr>
<tr>
<td>Third eyelid</td>
<td>Prolapse</td>
<td>Verify bulbar side for retained foreign body</td>
<td>Caution with deep ulcer or descemetocele</td>
</tr>
<tr>
<td>Conjunctiva</td>
<td>Small branching blood vessels</td>
<td>Increased number with superficial disease</td>
<td></td>
</tr>
<tr>
<td>Scleral</td>
<td>Larger straight blood</td>
<td>Increased number</td>
<td></td>
</tr>
</tbody>
</table>
Corneal diseases

Corneal ulceration and stromal abscess
There are 3 types of corneal ulcerations: simple, indolent (or non-healing, refractory, recurrent), and complex. Ocular findings detected on examination will help clarify which type of ulcer is present. Once the type is confirmed, appropriate therapy can be prescribed. However, simple and indolent ulcers can progress to become complex ulcers and therefore timely re-evaluations are necessary.

Corneal stromal abscess are similar to complex ulcers as they both involve infection from either bacteria and or fungus. The main difference is that the corneal epithelium is intact and can serve as a barrier to therapy.

Therapeutic frequency of medication will depend on severity of disease. A simple ulcer can be treated 4 times daily whereas a complex ulcer may need topical therapy every hour with the use of a sub-palpebral eye lavage system. Oral NSAIDS should be given to help stem the secondary uveitis due to corneal disease and this uveitis should not be underestimated. Untreated or poorly treated secondary uveitis can damage intraocular structures and lead to cataract development, glaucoma or retinal detachment.

Table 2: Features of corneal ulcerations and stromal abscesses
<table>
<thead>
<tr>
<th>Description</th>
<th>Diagnostic tests</th>
<th>Therapy</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Simple</td>
<td>Superficial</td>
<td>Fluorescein stain</td>
<td>BNP QID*</td>
</tr>
<tr>
<td></td>
<td>Non-infected</td>
<td>Tonometry</td>
<td>Topical atropine</td>
</tr>
<tr>
<td></td>
<td>Acute lesion</td>
<td></td>
<td>1%⊕</td>
</tr>
<tr>
<td></td>
<td>No corneal blood</td>
<td></td>
<td>Systemic NSAID€</td>
</tr>
<tr>
<td></td>
<td>vessels</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Painful</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Fluorescein negative in 7 days but may take up to 12 days</td>
</tr>
<tr>
<td>Indolent</td>
<td>Superficial</td>
<td>Fluorescein stain</td>
<td>Corneal debridement</td>
</tr>
<tr>
<td></td>
<td>Non-infected</td>
<td>Tonometry</td>
<td>Grid keratotomy</td>
</tr>
<tr>
<td></td>
<td>Chronic lesion</td>
<td></td>
<td>BNP QID*</td>
</tr>
<tr>
<td></td>
<td>Corneal blood vessels</td>
<td></td>
<td>Topical atropine</td>
</tr>
<tr>
<td></td>
<td>Painful</td>
<td></td>
<td>1%⊕</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Systemic NSAID€</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Need to confirm non-infectious status</td>
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<tr>
<td></td>
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<td></td>
<td>Corneal sequestrum may need keratectomy</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>May take 15-20 days to be fluorescein negative</td>
</tr>
<tr>
<td>Complex</td>
<td>Superficial fungal plaque</td>
<td>? Fluorescein stain</td>
<td>Sub-palpebral lavage</td>
</tr>
<tr>
<td></td>
<td>Mid-stromal to deep</td>
<td>Corneal cytology</td>
<td>q 2-4 hour therapy</td>
</tr>
<tr>
<td></td>
<td>Descemetocoele</td>
<td>Corneal culture</td>
<td>Bactericidal antibiotic¥</td>
</tr>
<tr>
<td></td>
<td>Bacterial, fungal or mixed infection</td>
<td>?</td>
<td>Antifungal therapy£</td>
</tr>
<tr>
<td></td>
<td>Keratomalacia</td>
<td>Tonometry?</td>
<td>Serum if keratomalacic</td>
</tr>
<tr>
<td></td>
<td>Corneal blood vessels present after 4-5 days</td>
<td></td>
<td>Topical atropine</td>
</tr>
<tr>
<td></td>
<td>Obvious uveitis present</td>
<td></td>
<td>1%⊕</td>
</tr>
<tr>
<td></td>
<td>Painful</td>
<td></td>
<td>Systemic NSAID€</td>
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<tr>
<td></td>
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<td></td>
<td>May need conjunctival graft</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Guarded prognosis at best</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Uveitis can lead to cataract, retinal detachment</td>
</tr>
<tr>
<td>Stromal abscess</td>
<td>White to beige lesion within corneal stroma</td>
<td>Negative fluorescein stain?</td>
<td>Sub-palpebral lavage</td>
</tr>
<tr>
<td></td>
<td>Deep lesions are indicative of fungal infection</td>
<td>Corneal cytology?</td>
<td>q 2-4 hour therapy</td>
</tr>
<tr>
<td></td>
<td>Corneal blood vessels present after 4-5 days</td>
<td>? Corneal culture?</td>
<td>Bactericidal antibiotic¥</td>
</tr>
<tr>
<td></td>
<td>Obvious uveitis present</td>
<td>Tonometry</td>
<td>Antifungal therapy£</td>
</tr>
<tr>
<td></td>
<td>Painful</td>
<td></td>
<td>Topical atropine</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>1%⊕</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Systemic NSAID€</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>High winds may play role</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>May need conjunctival graft</td>
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<tr>
<td></td>
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<td></td>
<td>Guarded prognosis at best</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Uveitis can lead to cataract, retinal detachment</td>
</tr>
</tbody>
</table>

Note: BNP QID*: apply until fluorescein stain negative; Topical atropine 1%⊕: apply BID until pupillary dilation then as needed to maintain mydriasis; Systemic NSAID€: flunixin meglumine 0.5-1.0mg/kg SID to BID to effect; Bactericidal antibiotic¥: commercially available tobramycin,
ofloxacin, ciprofloxacin, moxifloxacin, compounded 50mg/ml cefazolin q 2-6 hours pending severity of lesion; Antifungal therapy£: 1% itraconazole/30% DMSO QID, 1% voriconazole 1% QID for a minimum 4 weeks

**Keratitis**
The 3 most common types of keratitis include: equine herpes virus type II, eosinophilic keratitis and immune-mediated keratitis. Herpesvirus and eosinophilic keratitis can be ulcerative in presentation whereas immune-mediated is usually non-ulcerative.

Table 3: Types of keratitis

<table>
<thead>
<tr>
<th></th>
<th>Description</th>
<th>Diagnostic tests</th>
<th>Therapy</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Herpesvirus</strong></td>
<td>Epiphora</td>
<td>Fluorescein stain</td>
<td>Bactericidal antibiotic¥</td>
<td>Lifelong infection</td>
</tr>
<tr>
<td></td>
<td>Central corneal</td>
<td>Tonometry</td>
<td>Topical antiviral⊕</td>
<td>Avoid topical steroids</td>
</tr>
<tr>
<td></td>
<td>multifocal punctate</td>
<td></td>
<td>Topical atropine⊕</td>
<td></td>
</tr>
<tr>
<td></td>
<td>ulcerative lesions</td>
<td></td>
<td>Topical diluted povidone-iodine TID</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>? Topical NSAID?</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Oral lysine 10-30g/day</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Systemic NSAID€</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td><strong>Lifelong infection</strong></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td><strong>Avoid topical steroids</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Eosinophilic</strong></td>
<td>Unilateral or bilateral</td>
<td>Fluorescein stain</td>
<td>Tobradex/maxitrol QID</td>
<td>Seasonal occurrence</td>
</tr>
<tr>
<td></td>
<td>Caseous mucoid discharge</td>
<td>Corneal cytology</td>
<td>Topical atropine⊕</td>
<td>Several weeks to months for resolution</td>
</tr>
<tr>
<td></td>
<td>Conjunctival hyperemia</td>
<td></td>
<td>Systemic NSAID€</td>
<td>Avoid topical NSAIDS</td>
</tr>
<tr>
<td></td>
<td>White plaque over superficial ulcer</td>
<td></td>
<td>Systemic cetirizine</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Lesion associated with limbus</td>
<td></td>
<td><strong>Consider keratectomy</strong></td>
<td>Keratectomy shortens therapy</td>
</tr>
<tr>
<td></td>
<td>Corneal blood vessels</td>
<td></td>
<td><strong>Consider Optimmune BID?</strong></td>
<td></td>
</tr>
<tr>
<td><strong>Immune-mediated</strong></td>
<td>Corneal blood vessels</td>
<td>Fluorescein stain</td>
<td>Topical optimmune</td>
<td><strong>Controversial</strong></td>
</tr>
<tr>
<td></td>
<td>Corneal cellular infiltrates lesions can be superficial, midstromal or deep</td>
<td>Corneal cytology</td>
<td>Topical tacrolimus</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>? Corneal culture?</td>
<td>Topical tetracycline</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Topical corticosteroids</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td></td>
<td><strong>Consider keratectomy and conjunctival graft</strong></td>
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<td></td>
<td></td>
<td></td>
<td><strong>Consider</strong></td>
<td></td>
</tr>
</tbody>
</table>
Note: Topical antiviral: 1% trifluridine, 0.5% idoxuridine QID 14-21 days

References or additional reading:

The equine eyelid can suffer from several diseases and conditions. Eyelid trauma is the most common eyelid disease followed by eyelid neoplasia. Neonatal entropion can also be a common diagnosis seen in practice. In this session we will review the surgical options for eyelid laceration and entropion repair. Several therapeutic options for eyelid neoplasia have been described in the literature. Consequently, we will explore and review the benefits and risks of the many therapeutic options for eyelid squamous cell carcinoma and sarcoids.

Anatomy and response to injury
The upper and lower eyelids are composed of 3 layers: 1) skin with associated glands; 2) musculofibrous layer; 3) palpebral conjunctiva. The skin contains 3 types of hairs: 1) dermal hair; 2) cilia; 3) vibrissae. The vibrissae are similar to antennas and provide tactile information via the trigeminal nerve. The skin meets the palpebral conjunctiva and forms a mucocutaneous junction, the palpebral margin. Within this margin, ducts of the meibomian glands exit. The musculofibrous layer contains a network of blood vessels and capillaries. Injury to upper and lower eyelids results in significant hemorrhage as well as edema. The musculofibrous layer also contains a substantial lymphoid drainage, which can be the primary site of metastasis of Squamous Cell Carcinoma (SCC) to regional lymph nodes.

The upper eyelid is innervated by the cranial nerve III, V, and VII. The main motor nerve is CN VII which can be blocked by performing an auriculopalpebral block. Sensory innervation is through CN V which can be blocked by performing a frontal block at the supraorbital foramen. Lidocaine 2% without epinephrine is recommended and onset of action of 4-6 minutes has been reported along with duration of 60-90 minutes. Variable paralysis of the lower eyelid occurs with the auriculopalpbral block.

The third eyelid or nictitan is covered with conjunctiva rich in with globlet cells, contains a T-shaped cartilage, and contains a lacrimal gland at its base. A large fat pad lies ventral to the nictitan and can prolapse following removal of this eyelid.

Congenital disease
Entropion disease
This eyelid condition is most commonly seen in sick foals. Thoroughbreds and quarter horses have been reported to be genetically predisposed to this condition. In adults of any breed, this condition is usually acquired secondary to eyelid trauma, loss of orbital content, or facial fractures. Secondary corneal ulceration is a common finding with these affected patients and must be treated accordingly (see corneal disease lecture).

Therapy consists of correcting eyelid position. Several materials have been reported to correct entropion and include saline, penicillin G, silicone, hyaluronic acid and liquid paraffin; however, the need for repeated injections, eyelid inflammation and risk of permanent scarring make the
use of these materials undesirable. Also, the use of antibiotics for this condition is ethically questionable.

In foals, temporary surgical repair is often warranted whereas in the adult, a permanent surgical correction is usually needed. Temporary vertical mattress tacking sutures or staples will allow the palpebral margin to be well apposed against the cornea. Non-absorbable 4-0 to 5-0 monofilament is first placed 1 cm away from the palpebral margin and the second bite comes out 2-3 mm away from the palpebral margin. The suture end along the cheek is left long for future suture removal while the suture end close to the palpebral margin is cut very short to minimize contact with the cornea. In most cases, the foals regain strength and health within the next 2-4 weeks and at that time, sutures can be removed.

Permanent correction of entropion entails a Hotz-Celsus procedure. This procedure is best done under general anesthesia. The initial skin incision is made 2-3 mm from the edge of the palpebral magin (the closer, the less tissue needs to be removed) and remains parallel to the margin. The use of a Jaeger lid plate, scalpel handle or butt end of tissue forceps will aid in stabilizing the skin and facilitate this incision. The second incision is made distal and parallel to the first with the ends joining to create a crescent. The skin is gently removed with small metzebaum scissors along with a strip of subcutaneous tissue (the amount of subcutaneous tissue varies with severity of entropion but care is taken not to puncture through the eyelid). Close the wound with 4-0 non-absorbable monofilament in a simple interrupted pattern (leave the suture end long on the cheek and the other short towards the cornea). Suture removal is in 14 days. Protective eye cup may be necessary to prevent self-trauma to the sutures.

Should systemic antibiotics be warranted, Cetiofur at 2.2 mg/kg IM BID for 5-7 days or TMS 15-20 mg/kg Per os BID 5-7 days can be prescribed. Anti-inflammatory therapy includes flunixin meglumine 1 mg/kg IV or 1.3 mg/kg Per os BID for 24-72 hours then 0.5mg/kg BID for 5 days or phenylbutazone 4.4 mg/kg Per os BID 24-72 hours then 2mg/kg BID for 5 days. NSAID therapy may need to be extended pending response to medication.

**Developmental disease**

**Ectopic cilia**

Although not common, ectopic cilia should be considered when chronic, and possibly intermittent, blepharospasm, ocular discharge and keratitis (ulcerative and non-ulcerative) are described by the owner. Ocular examination requires magnification to find translucent cilium or cilia in the upper eyelid palpebral conjunctiva, emerging approximately 5 mm from the eyelid margin. Common differentials include distichiae, burdock bristles, retained foreign bodies, keratoconjunctivitis sicca.

Temporary relief can be achieved with petroleum based opthalmic ointments. Surgical excision of the offending cilia is required for resolution of clinical signs. Transconjunctival excision can be done in the standing horse with intravenous sedation, auriculopalpebral and frontal block, and topical proparacaine. The open wound is left to heal by second intention. Recurrence is possible as not all cilia may be emerged at the time of surgery. Topical BNP TID
Acquired disease
Blepharoedema/Blepharitis
Blepharoedema of the equine eyelid can have several causes. If unilateral, the causes include: trauma (blunt of self), insect bite, foreign body reaction, and abscess. Should the clinical signs be bilateral, the causes include: self-trauma, systemic disease, equine viral arteritis, babesiosis, lymphosarcoma, influenza and allergic/hypersensitivity.

Blepharitis, inflammation of the eyelids, is most commonly seen following perforating trauma with secondary bacterial infection. Self-trauma, fungal and parasitical infection, can also cause blepharitis. Confirmation of diagnosis may require culture and biopsy. Treatment of the underlying cause will lead to resolution of clinical signs. Flunixin meglumine is recommended as a systemic NSAID in these cases.

Laceration
Due to the prominence of equine eyes, upper eyelid lacerations are commonly seen in practice. Quick assessment of the patient is recommended as time will increase eyelid edema and inflammation, making repair more challenging. Failure to repair eyelid lacerations will lead to keratitis (ulcerative or non-ulcerative) and epiphora. Prior to surgical repair, it is recommended to complete a comprehensive ocular exam to ensure that no other ocular diseases are present (i.e. corneal ulceration, uveitis...).

Preparation of the patient includes intravenous sedation, auriculopalpebral and frontal local blocks (may also need line block), diluted 1:50 povidone-iodine, and small surgical instruments (to accommodate 6-0 and 4-0 sutures). Minimal debridement of the wound edges is needed due to the extensive vascular supply found in eyelids. Removal of the palpebral margin should only be done in extreme circumstances as it cannot be recreated easily. If possible, the underlying palpebral conjunctiva should be identified and sutured closed with absorbable 5-0 or 6-0 suture in a simple continuous pattern. Care must be taken to not allow suture knots to contact the cornea; by placing tension on the suture line, inversion of the conjunctiva over the suture line will limit its corneal contact. Following completion of the conjunctival suture line, the skin edges at the palpebral margin need to be properly aligned and sutured to minimize future corneal trauma. A figure 8 suture or simple interrupted sutures are placed and the ends are braided upwards to minimize suture end contact with the cornea. Skin suture removal is in 14 days and a protective eye cup may be needed to prevent self-trauma to the suture line. Systemic antibiotic and NSAID therapy is similar to what was described earlier.

Neoplasia
Periocular neoplasia is commonly seen in practice. Differential diagnoses of eyelid neoplasia includes the following: other tumours (papilloma, melanoma, mastocytoma, basal cell carcinoma, schwannoma, adenoma, adenocarcinoma, hemangioma, hemangiosarcoma,
lymphoma), conjunctivitis (lymphoid hyperplasia, follicular), inflammatory lesions (abscesses, granulation tissue, foreign body reaction, solar induced radiation), and parasites (habronema, onchocerca, thelazia). The two most common tumours are Squamous Cell Carcinoma (SCC) and sarcoids.

Table 1: Common eyelid tumours

<table>
<thead>
<tr>
<th>Tumour</th>
<th>Age</th>
<th>Risks</th>
<th>Appearance</th>
<th>Therapy</th>
<th>Prevention</th>
</tr>
</thead>
<tbody>
<tr>
<td>Squamous cell carcinoma</td>
<td>3-26 yrs</td>
<td>Draft, Appaloosa,</td>
<td>4 types: Plaque, Papillomatosis,</td>
<td>Cryotherapy, Brachytherapy, Chemotherapy, Hyperthermia, Excision nictitan</td>
<td>Genetic testing* UV A&amp;B mask</td>
</tr>
<tr>
<td></td>
<td>~ 11 yrs</td>
<td>Paint, TB, Haflinger, UV radiation Lack of periocular pigmentation</td>
<td>Invasive, Non-invasive Upper and lower eyelid Mass on nictitan’s free margin</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sarcoid</td>
<td>3-6 yrs</td>
<td>QH, Appaloosa, Arabian</td>
<td>5 types: Occult, Varicose, Nodular (A&amp;B), Fibroblastic (A&amp;B), Mixed</td>
<td>Immunotherapy, Chemotherapy, Hyperthermia, Brachytherapy, Electrochemotherapy</td>
<td></td>
</tr>
<tr>
<td>Melanoma</td>
<td>&gt;15 yrs</td>
<td>Grey or white haired horses Lippizaner Percheron Arabian</td>
<td>Black nodule or mass</td>
<td>Excision eyelid</td>
<td></td>
</tr>
<tr>
<td>Lymphoma</td>
<td>Systemic LSA</td>
<td>Infiltration of eyelid and conjunctiva Nictitan protrusion 2nd to mass effect</td>
<td>Excision nictitan</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Genetic testing for Haflinger: https://www.vgl.ucdavis.edu/services/HaflingerSCC.php; Affected horses are termed R and normal are termed N. The R/R genotype is more likely to have SCC than R/N or N/N genotype. The R/R horses should be frequently examined for early detection, and excision of any suspicious lesions is warranted for consideration. Affected R/R horses are expected to have a higher recurrence risk and should be frequently examined. To reduce the incidence in the breed, breeding decisions should be advised so as to avoid mating R/R or R/N to one another.

There are numerous adjunctive therapies to treat periocular neoplasia. Of course, surgical excision with clean margins is the first goal, but unfortunately, due to the limitations of adnexal surgery, getting clean margins is not always possible.
<table>
<thead>
<tr>
<th>Therapy</th>
<th>Advantages</th>
<th>Disadvantages</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Topical</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5-Fluorouracil (Sarcoid)</td>
<td>SCC: Apply once a week for 5 week</td>
<td>Easily obtainable</td>
</tr>
<tr>
<td>Mitomycin-C (Sarcoid)</td>
<td>SCC and sarcoids: Apply 3-4 times/day for 7 days then off 7 days</td>
<td>Easily obtainable</td>
</tr>
<tr>
<td>Imiquimod (Sarcoid)</td>
<td>Sarcoids: Apply 3 times/week for 32 weeks</td>
<td>Resolution of 60% sarcoids</td>
</tr>
<tr>
<td>Mistletoe extract (Sarcoid)</td>
<td>3 injections/week for 15 weeks</td>
<td>Possible alternative therapy</td>
</tr>
<tr>
<td><strong>Intralesional</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cisplatin (SCC and sarcoid)</td>
<td>4 treatments at 2 week interval</td>
<td>Low risk of systemic complications</td>
</tr>
<tr>
<td>BCG vaccine (SCC and sarcoid)</td>
<td>Injection every 2-4 weeks until resolution</td>
<td>Sarcoids 59-69% resolution rate</td>
</tr>
<tr>
<td></td>
<td></td>
<td>One study: 100% sarcoid resolution</td>
</tr>
<tr>
<td><strong>Radiation therapy</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Brachytherapy (SCC and Sarcoid)</td>
<td>Insert wire with strontium beads</td>
<td>Local tumour destruction</td>
</tr>
<tr>
<td></td>
<td></td>
<td>86% success rate at 1 year sarcoid; 74% at 5 years; 98% in one sarcoid study</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Thermic therapy</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hyperthermia (SCC and sarcoid)</td>
<td>5-6 treatments at 2-4 weeks interval</td>
<td>30 second treatment</td>
</tr>
<tr>
<td>Cryotherapy (SCC &amp; sarcoid)</td>
<td>Double or triple freeze thaw cycle</td>
<td>Inexpensive</td>
</tr>
<tr>
<td>Photodynamic therapy</td>
<td>Photoactive agent injected followed by light irradiation</td>
<td>Promising results</td>
</tr>
<tr>
<td>----------------------</td>
<td>----------------------------------------------------------</td>
<td>-------------------</td>
</tr>
</tbody>
</table>

**References and additional reading:**

1. Honey bee biology
Honey bees are social insects. According to E. O. Wilson, the following three criteria are required for insects to be called truly social (eusocial): 1) caste division of labor, 2) cooperative care of brood/offspring, 3) overlapping generations (offspring stay with colony and contribute to general welfare). Under normal circumstances, honey bee colony consists of a queen, drones and worker bees. Both queen and workers are diploid females and have 32 chromosomes and drones are haploid males with 16 chromosomes.

1.1. Castes
1.1.1. Queen is the only mated egg-laying female with developed reproductive tract in a colony. She is slightly larger than a worker bees and her abdomen is elongated and only partially covered by the wings because it contains fully developed reproductive tract in addition to other abdominal organs. Three days after oviposition, a diploid larva hatches from a fertilized egg and is fed abundant amount of royal jelly during the entire larval period (5 days) after which queen cell is sealed with wax (8-9 days post-oviposition) and pupation occurs until eclosion (emergence) at 16 days post-oviposition. The newly emerged virgin queen will destroy other queen cells, if they exist, and during the first two weeks of life will fly out from hive to mate with 10-15 drones (polyandry) in the air. The more drones the queen mates with, the greater the genetic diversity of her offspring, and the greater the robustness of the entire colony [important for the overall health of colony]. The queen stores the collected sperm from drones in the sperm-storing organ, called spermatheca, for the entire life (3-4 years). Once mated, the queen does not leave the hive again except during natural swarming. There are two major roles that the queen performs. The first role is laying eggs that develop into workers, drones and queens (if needed). During the peak season, queen can lay ~1500 eggs a day (close to her own weight). The second role is production of multiple pheromones that maintain functionality of the entire colony. Pheromones are produced in mandibular (and other) glands and distributed throughout the entire colony via direct contact and trophallaxis (food exchange) - [important behavior -> spread of diseases]. Pheromones have many important effects on the colony, including suppression of ovary development in worker bees, suppression of queen rearing (swarm and supersedure cells) and enhancement of worker activity necessary for growth, productivity and overall health of the colony [1].

1.1.2. Drones are male bees whose main function is to mate with virgin queens after which the drones die. Drones are larger than worker bees but they are not as long as the queen. Three days after oviposition of a non-fertilized egg, a haploid larva hatches and is fed royal jelly during the first ~3 days and then pollen and nectar until the end of larval stage (day 9-10 post-oviposition). After pupation is complete, drones emerge at day 24 post-oviposition. During the first ~2 weeks of life, drones become sexually mature, and then fly out of the hive during the day to seek drone congregation areas where they join drones from other colonies which are
waiting to mate with virgin queens. Drones that succeed to mate with a queen will die during
copulation, while those that did not succeed to mate live for a few months during the summer
and are expelled out of hive and die during the fall. During the summer, drones are accepted by
all colonies, therefore they often drift from colony to colony - [important behavior -> spread of
diseases].

1.1.3. Worker honey bees are ‘sterile’ females that have hypoplastic ovaries. Both queen and
workers hatch from the identical fertilized eggs as diploid female larvae, and accordingly, they
have the same genetic composition. The quality and quantity of feed available to these diploid
female larvae determines their phenotypic development. Namely, if larvae are fed with
abundant quantities of royal jelly during the entire larval period, they will develop into queens
within 16 days post-oviposition; conversely, if the same larvae are fed restricted amount of
royal jelly for first ~3 days and then with pollen and nectar, they will develop into worker bees
within 21 days post-oviposition. The worker honey bees are responsible for all activities in
colony except reproduction. The honey bee workers are divided into two major types based on
seasonality and tasks: summer and winter workers. Summer workers live ~6 weeks.
Approximately the first half of life of an adult summer worker (3 wks) will be dedicated to
housekeeping tasks in hive (cleaning brood cells, nursing brood, attending to and feeding
queen, building wax comb and sealing brood and honey, processing nectar into honey,
processing pollen into bee bread, removing dead brood and adults from the hive, ventilating
and defending the colony) - [many of these behaviors are associated with spread of diseases].
The second half of an adult summer worker’s life (~ last 3wks) is dedicated to field work which
consists of collection of nectar, pollen, water, and propolis. The most intense harvesting occurs
within 2 miles but the forager bees can fly twice as far if nectar or pollen is not available closer -
[important behavior - spread of diseases]. Drifting of honey bee workers from hive to hive is
not as common as in drones; however, it exists. During the honey and pollen flow any worker
bee carrying the load will be allowed into the hive - [important behavior - spread of diseases].
There are seasonal variations in the total number of worker bees per colony. To maximize
honey production, the highest number of worker bees in a colony (50,000-70,000 bees) should
coincide with the major blooming season of nectar producing plants (e.g. canola and alfalfa in
Saskatchewan). In contrast, there are only 15,000 to 30,000 bees in a colony during the late
winter. Winter honey bee workers emerge at the end of summer and the beginning of fall
(September to October in SK). During that time there is limited need for nursing (due to
markedly reduced egg-lying activity) and foraging (reduced availability of flowering plants), so
workers are not ‘worn out’ with these demanding tasks and their life span is on average 150
days [2]. The main role of winter worker bees is to ensure that queen survives during the winter
until spring season starts and a new population of bees are generated. During the fall, surviving
drones are expelled from the colony in preparation for winter. Honey bees do not hibernate.
Instead, when the outside temperature falls, bees form a cluster surrounding the queen and
generate heat by vibration of thoracic muscles and abdomens using energy from consumption
of stored honey reserves. The density of the cluster is used as a mechanism for
thermoregulation to achieve an optimal temperature of 29˚C in the center around the queen
and ~6-8˚C at the periphery. Constant circulation of the bees within the cluster ensures
exchange of the peripheral vs central positions of worker bees to freezing of bees at the
The winter bees usually do not defecate within hive; instead, they accumulate waste within the rectum which can distend and occupy a substantial portion of the abdominal cavity. On sunny winter days when the temperature is above 6-7°C, bees will fly out of the hive for a short period to empty the waste from their rectum (cleansing flights).

1.1.4. Development stages of brood are the same for all three castes even though the total development time is different. The queen lays non-fertilized eggs in drone comb cells (which have a larger diameter than worker cells) and fertilized eggs in worker comb cells and queen cups. From fertilized eggs, diploid larvae emerge 3 days after oviposition and will develop into workers or queens depending on the larval food they are provided by nursing bees. Non-fertilized eggs will develop into haploid drones. After hatching, larvae are attended and nursed by many young nursing bees visiting each larva many times per day - [important behavior -> spread of diseases]. After completion of the larval stage (5-6 days), the comb cells containing larvae are sealed with wax (capped) and prepupal and pupal development occurs for 8-15 days depending on caste. Eclosion (emergence) of the imago (adult) stage occurs at day 16, 21 and 24 post-oviposition for queen, worker and drone, respectively. Larvae and pupae that die during the development process are removed and cells are cleaned by housekeeping worker bees - [important behavior -> spread of diseases]. In addition, worker bees have the ability to detected infected or infested pupae and remove them; this is called ‘hygienic behavior’ which interferes with disease progression (e.g. reproduction of Varroa) and improves colony resistance to disease. Hygienic behavior is a part of social immunity and contributes significantly to the overall health of a colony. This is an inherited trait and there are various queen-breeding programs that selectively enhance this behavior - [important management practice -> enhance disease resistance and colony health].

1.2. Reproduction

1.2.1. Natural propagating and reproduction of honey bee colony is accomplished by swarming during late spring and early summer. There are several predisposing factors that will result in swarming, including: 1) concentration of queen’s pheromones become too low to maintain colony cohesion – this could be due to rapid expansion of colony during the spring or decreased production of pheromones; 2) decreased/inadequate space for colony expansion and food storage – colony becomes too crowded; 3) decreased space for egg laying and brood rearing. Once the colony enters the “swarming mood”, queen cells with young queen larvae are produced. The old (mother) queen reduces egg-laying activity and her abdomen becomes smaller due to atrophy of ovaries in preparation for a swarming flight. Swarming will occur during favorable weather conditions (sunny and warm early afternoon), usually when the new queen cells are capped and the young queens are in the pupal stage. The old mother queen will leave hive with a substantial proportion of worker bees and form a transient swarm cluster, usually on a tree branch close to the original hive (less than 100 meters) until scout workers find a suitable location for their permanent new home, which could be a few kilometers away from the original hive - [important behavior -> spread of diseases]. Swarmed bees are docile
and do not exhibit defensive behavior. The remaining portion of the original colony will wait for the new queen to emerge, mate and re-establish functional order of the colony.

1.2.2. Swarming is detrimental for a beekeeping operation due to loss of bees and subsequent decrease in honey production. Accordingly, good beekeeping management practices aim to decrease/eliminate swarming by removing predisposing factors for this natural reproductive and propagating behavior. There are numerous management beekeeping techniques used for multiplying colonies and some of them use queens produced by commercial queen breeders who ship queens worldwide. Large-scale commercial queen breeding and production by comparatively limited number of companies is considered to be a threat to genetic diversity - [important management practice -> impact on colony health].

2. Seasonal cycle in honey bee biology and beekeeping
2.1. Winter
European honey bees are adapted to a temperate climate and will survive winter providing that they are healthy, have abundant food stores and proper ventilation (to prevent condensation within the hive). At the end of winter and beginning of spring (March in SK), the bee population is at its lowest and the colony will start to rear brood to replace the old population of winter bees. [Disease management note: At this stage, there is no or very little brood hence the great majority of Varroa mites are in phoretic stage – this is the most effective treatment time with miticides].

2.2. Spring
During the winter, the colony consumes just enough food to generate sufficient heat in the cluster to protect the queen. However, when egg-laying and brood rearing resumes, requirements for energy and protein rapidly increase, and consequently, consumption of both stored honey and pollen is also substantially increased. Initiation of brood rearing (during early spring in SK) is the most critical time for overwinter survival of a colony, because if the colony does not have sufficient food stores it will most likely run out of supplies and die of starvation considering the lack of external sources of nectar and pollen in the environment at this time of year. Once spring blooming commences (crocuses and willows in SK -> Apr-May), colonies start to expand rapidly. During this time, additional therapy against Nosema sp. and/or Paenibacillus larvae (American foulbrood) may be considered if necessary, to make sure that there is sufficient withdrawal time before major spring blooming (e.g. dandelions and caragana in SK -> May-Jun) if honey is to be harvested for human consumption. This is also a time period (May-Jun in SK) of intense beekeeping activity in the apiary, including, inter alia, spring inspections and clean-up, preparation for queen rearing, nucleus (replacement colony) establishments, and queen replacement. All of these activities are crucial for prevention of swarming, breeding of new queens and multiplication of colonies to be used for replacement or expansion of the operation. Under normal conditions, replacement of queens is done every second year, but this depends on management practices of each beekeeper. Nevertheless, the importance of high quality queens cannot be overemphasized, not only for optimal colony production, but also for the overall health of colony – [important management practice -> enhancement health of colony by high quality of queens]. An old or poor queen that does not produce sufficient quality...
and quantity of pheromones will compromise colony homeostasis and cohesion through alteration of several physiological and behavioral modifications in the worker bee activities such as reduced cleaning, guarding, foraging and brood care [1] that ultimately results in a weak colony and increased susceptibility to disease. Following natural instinct, the colony will try to replace the queen by supercedure but the progeny queen will still have the same poor genetics as its mother.

2.3. Summer
During the summer (end of June to August), the colony is at the peak of its strength and the majority of colony activities are centered on intense harvesting of food reserves (i.e. nectar and pollen) to be stored as honey and beebread for use during times of dearth (winter in SK). However, “clever” beekeepers exploit this prolific behavior to generate profit from “stolen” honey© using well established beekeeping practices.

2.4. Fall
At the end of August (in SK) all honey stored in honey supers (above brood chambers) is removed for extraction. Fall treatment and feeding is initiated to ensure that overwintering colonies are as healthy as possible and have sufficient food stores. The most important, and very often necessary, treatment is miticide strips for Varroa mite control. For many beekeepers in North America, metaphylactic treatment against American foulbrood is equally as important and it is also done at this time (September in SK). The third potential fall treatment is against Nosema apis and Nosema ceranae; this last treatment is recommended/applied based on infection rates determined in forager bees, or based on history of Nosema disease in this particular operation. Since beekeepers harvest the majority of colony honey stores accumulated during summer, in September, honey bee colonies are provided with abundant feed in the form of sugar syrup to ensure that colonies have enough food stores during the winter. During mid-October in SK, miticide strips are removed from colonies and colonies are prepared for overwintering (according to the local winter climate).

3. Transmission of disease
3.1. Mode of transmission of disease within a colony
A honey bee hive contains thousands of bees with biological behavior that requires close interaction (e.g. trophallaxis), direct contact (e.g. pheromone spread) and housekeeping duties (e.g. removal of dead brood and adult bees). These behaviors facilitate horizontal transmission of pathogens between individual bees. In addition, there are certain pathogens (e.g. viruses) that can be transmitted vertically from queen to progeny.

3.2. Mode of transmission of disease between colonies
Once the disease is established within a colony it can spread from colony to colony by natural or anthropogenic means.

3.2.1. Natural transmission of diseases between different colonies may be facilitated by 1) drones and workers drifting to adjacent colonies [3], 2) foragers from different colonies
foraging on the same crops [4], 3) queens mating with infected drones [5], 4) colony swarming, and 5) foragers robbing infected, weak or dead colonies. 

Robbing behavior of honey bees is the most important natural mode of transmission of honey bee diseases between colonies. Robbing is a special behavior of forager bees that find an unprotected source of honey that is collected and brought back into their hive. This behavior intensifies at the end of summer when there is reduced availability of nectar from flowering plants and increased abundance of foraging bees. Unprotected stores of honey could be available in dead colonies that died due to various diseases, hence the “robber” bees become contaminated and bring infectious pathogens back to their hives. Weak colonies are also often targeted by robber bees because their guard bee population is depleted and, consequently, easily overpowered. One of the major causes of colony weakness is disease; accordingly, pathogens from weak colonies are transmitted to healthy and strong colonies by their strong foraging population (robber bees). Thus, it is extremely important to remove dead-out colonies from the apiary and, if infectious disease is identified, destroy or disinfect equipment (frames, comb, etc.) to minimize disease spread. In addition, if infectious disease is not identified as the cause of weak colonies, weak colonies should be re-queened and/or merged to create strong colonies. It should be emphasized that robbing is not restricted to colonies in the same yard, but it can occur anywhere within the ~5 km (flight radius of forager bees).

3.2.2. Anthropogenic transmission of diseases (e.g. fomites, equipment, trade, etc.) is also extremely important. Using contaminated fomites and equipment, beekeepers can spread diseases from hive to hive or from yard to yard within the same operation if optimal biosafety practices are not implemented. Potentially devastating disease outbreaks can occur due to the sale and purchase of contaminated equipment or infected bees among beekeepers. National and international trade of potentially infected bees and products (e.g. packaged honey bees, queens, semen, honey, etc.) has been a major contributor to the global spread of honey bee pathogens during the last several decades [6] in spite of best intentions, strict regulations and high quality inspections. Migratory beekeeping practices also contribute significantly to transmission of diseases among colonies and dispersal of pathogens over wide geographical areas.

4. Treatment and prevention of diseases
In beekeeping industry, the integrated pest management (IPM) strategy is a relatively recent commonly used term for prevention and control of diseases that includes: 1) genetic selection for resistance to disease (e.g. hygienic behavior); 2) management practices to reduce incidence and spread of diseases (e.g. frequent inspection, maintenance of strong/healthy colonies, prevention of robbing); 3) physical control (e.g. destruction of infected equipment/colonies, segregation of infected colonies, “shaking” method for control of brood disease, regular replacement of equipment/frames, interruption of parasite cycles, screened bottom boards for Varroa management etc.); 4) chemical control (e.g. chemical therapy of infected colonies and disinfection of contaminated equipment).
4.1. Chemical treatments
Chemical therapy with synthetic or natural chemicals is used in the Canadian beekeeping industry against mites (Varroa and tracheal mites), fungi (Nosema apis and N. ceranae) and bacteria (Paenibacillus larvae -> American foulbrood, and Melissococcus plutonius -> European foulbrood). Miticides and antimicrobials are used as both therapy and metaphylaxis depending on disease conditions, season of production and management practices. Unfortunately, resistance to antimicrobial and antiparasitic synthetic drugs has become a big concern for beekeeping industry.

4.2. Administration of therapy
Two major routes are used to administer treatment to honey bees: 1) administration in feed (antibacterial and antifungal medication) and 2) external contact administration (direct contact between external surfaces of bees and therapeutic chemicals impregnated in plastic strips, dissolved in solution or vaporized in hive).

5. Major Diseases of Honey Bees in Canada
Short summary of those diseases for which chemical therapy is approved in Canada.

5.1. American foulbrood [7, 8]
American foulbrood is a devastating, contagious brood disease that develops rapidly, kills the colony and spreads to other colonies by robbing, drifting bees and anthropogenic modes.

Etiology: Paenibacillus larvae is a Gram-positive, spore forming, rod-shaped bacterium. Spores survive in contaminated equipment for decades. Approximately 2.5 billion spores are produced in each infected larva [8].

Pathogenesis: Larvae (up to 2-day old) ingest spores which germinate and proliferate in the intestine and subsequently spread throughout the body causing fatal septicemia.

Gross pathology: Brood frames have spotty brood pattern (shotgun brood); punctuated and sunken capping of brood cells; color of dead larvae which are positive changes from dull white to brown at which stage ‘ropiness’ test* is positive; and desiccated, dead larvae which form dark brown, brittle scales firmly adhered to the ‘ventral lateral’ wall of the brood cell (scale cannot be removed without destroying the cell wall). In advanced stages of disease there may be a strong decaying odor when the colony is opened, hence the name of the disease, ‘foulbrood’.

*‘Ropiness’ test – The large number of vegetative P. larvae bacteria within macerated dead larvae will generate a typical glue-like consistency that can be detected by the ‘ropiness’ test. A dead larva is macerated with a matchstick within a cell and then slowly withdrawn. If the macerated tissue can be drawn out and stretched more than 2 cm, it is indicative of AFB infection.

Diagnosis: Gross pathology, especially a postive ‘ropiness’ test and the presence of scales, are highly characteristic, or could be considered even pathognomonic, for AFB. Nevertheless,
submission of samples of affected brood (including scales, if present) is recommended for confirmation of diagnosis by bacterial culture and/or PCR.

Therapeutic treatment: treatment with antibiotics of clinically affected colonies is not recommended, and in some jurisdictions, prohibited (contact provincial apiculture specialist for more information). The safest approach is to burn the entire colony and contaminated equipment. Alternatively, if infection rate is low, contaminated equipment could be irradiated, and in some jurisdictions, adult bees may be salvaged by the “shook-swarm method”.*

* The “shook-swarm method” is used to salvage adult bees from colonies affected by brood disease (e.g. EFB and AFB). Adult bees with the queen are transferred/shaken into a screened box and kept in a cool place for a several ours to allow time for consumption and digestion of contaminated honey present in the gastrointestinal tract. These adult bees are subsequently transferred to a hive with new frames/foundation. This artificial method of separating of adult bees from infected brood reduces substantially the number of spores within a newly established colony, terminating, but not eradicating the disease [9]. Concurrent antibiotic therapy of the newly established colony will enhance efficacy of disease termination.

Metaphylaxis: In certain countries, antibiotics are prohibited in the beekeeping industry. In Canada and the USA, metaphylactic use of oxytetracycline (Oxytet-25, Oxysoi 62.5, Foul Brood Mix) and tylosin tartrate (Tylan Soluble) against AFB is permitted and used regularly by many commercial and hobby beekeepers. The label instructions for Oxytet-25 are as follows: “Thoroughly mix 454 g of OXYTET-25 with 3.5 kg of powdered sugar. Apply 32 g of medicated mix per colony on the outer parts or ends of the frames 3 times at 4 to 5 day intervals in the fall and in the spring at least 4 weeks before the main honey flow.” Administration of oxytetracycline in syrup is also possible but it is not practiced as commonly. Tylosin is recommended only in beekeeping operations in which Paenibacillus larvae developed resistance to oxytetracycline. Potential residues in honey for tylosin are much higher when administered during the spring than for oxytetracycline.

Integrated Pest Management (IPM): Strategies for AFB management include re-queening with hygienic genetics; routine renewal of comb in the brood chamber (20-30% per year) to minimize contamination; frequent inspection to identify early stages of disease; prevention of spread of disease by robbing, contaminated equipment or feed; destruction of infected colonies and equipment; irradiation of equipment to destroy both vegetative stages and spores.

5.2. European foulbrood [8]
European foulbrood is an often self-limiting brood disease that is a consequence of reduced/suboptimal larval feeding due to an insufficient number of nursing bees to care for rapidly increasing numbers of larvae. A deficiency of brood care and feeding is most likely to develop during vigorous spring build-up of colonies in temperate climates (usually during the first major nectar/pollen harvest) [8, 10].

Etiology: Melissococcus plutonius, a Gram-positive coccus, is the main causative agent of EFB. However, it is often isolated with other bacteria (e.g. Paenibacillus alvei, Brevibacillus
laterosporus, Enterococcus faecalis etc.) that may be secondary pathogens or saprophytes that may contribute to the typical sour odor of the infected colony as well as to “pseudoropiness” of affected brood (see above ‘ropiness’ test) [8, 10].

Pathogenesis: Larvae (less than 3-day-old) ingest food contaminated with M. plutonius bacteria which proliferate in the intestinal tract, competing with the larva for nutrients. During certain stages of colony expansion, the nursing bee population is insufficient to feed the expanding larval population, which, if infected with M. plutonius, will die due to starvation. Once the deficiency in nursing bees and larval nutrition is corrected, the symptoms of EFB will disappear [8, 10].

Gross pathology: Brood frames contain spotty brood pattern (shotgun brood); punctuated and sunken capping; color of dead larvae changes from dull white to brown at which stage tracheal network becomes visible; macerated dead larvae exhibiting ‘pseudoropiness’, but consistency of macerated larvae is granular and not as stretchable (less than 2 cm) as in AFB; and desiccated dead larvae which form dark brown, C-shaped, rubbery scales that are loosely attached to the bottom of brood cells. The presence of a ‘sour’ odor depends on the presence and composition of additional saprophytic bacteria [8, 10].

Diagnosis: Gross pathology could be used to distinguish EFB from AFB. Nevertheless, submission of samples of affected larvae is recommended for confirmation of diagnosis by bacterial culture and/or PCR. Submission of larvae affected at early stages (live larvae) will facilitate diagnosis because at early stages of infection, saprophytic bacteria are not as prevalent, and overgrowth by secondary bacteria in culture will be reduced [10].

Therapeutic treatment: Mild cases of EFB disappear once nectar flow becomes steady and/or nursing bee population is increased. Severe cases of disease can have a considerable impact on honey production due slow spring build-up of colonies and subsequent suboptimal population of foragers during the main honey flow. Heavily infected colonies (more than 50% brood affected) should be destroyed together with equipment. For low or moderate infections, therapy with oxytetracycline can be implemented (as described above for AFB) as long as an appropriate withdrawal period is observed. Nevertheless, the disease will usually recur the following year, therefore it is advised to use additional IPM strategies for prevention (e.g. shook-swarm method) [8, 10].

Beekeeping operations that use metaphylaxis against AFB are also protected against EFB in most instances.

Integrated Pest Management (IPM): Strategies for EFB control include re-queening with hygienic genetics, routine renewal of comb in brood chamber (20-30% per year) to minimize contamination, and the ‘shook-swarm method’ for colonies with low to moderate infections [8, 10].
Nota bene: These are the only two diseases of honey bees in Canada for which beekeepers will require a veterinary prescription to obtain antibiotics, because both tetracycline and tylosin are categorized by the Health Canada as medically important antimicrobials (MIA).

References:
5. Amiri, E., M.D. Meixner, and P. Kryger, Deformed wing virus can be transmitted during natural mating in honey bees and infect the queens. Scientific Reports, 2016. 6.