CLINIC TRACK TEAM
Lessons from the Recession

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The recession has made it clear to owners and managers of veterinary practices in the US that an increased focus on good business practices is critical for increased financial success. Data from the National Commission on Veterinary Economic Issues indicated total revenue growth of about 4% in US companion animal practices from 2007 to 2008 and flat growth from 2008 to 2009. (These are average figures—about 1/3 of the practices did not do as well as this and another 1/3 did better—there was a wide range of performance.) To put this in perspective, the average growth rate had ranged from 11-13% for several years prior to 2007. Transactions were down, on average, by 1% from 2007 to 2008 but an increase in the average transaction charge of about 5% bolstered revenue growth. A similar pattern was seen in 2009. It is clear that practices are going to have to work much harder than before to maintain revenue growth and improve profitability.

Both practice owners and clients are clearly more anxious about the future than ever before as evidenced by some of the responses to the NCVEI’s monthly QuickPolls. In March, 2009, the NCVEI asked: Do you think your revenue for 2009 will be? Users responded as follows:

- Greater than 2008: 30%
- About the same as 2008: 32%
- A little less than 2008: 29%
- A lot less than 2008: 9%

In May, 2009 the QuickPoll focused on clients. The NCVEI asked: Has your practice seen a change in the payment options used by clients in the last six months? Users responded as follows:

- No: 26%
- Yes, more pet insurance used: 1%
- Yes, more medical payment plans used: 12%
- Yes, more A/R or held checks: 27%
- One or more of the above: 34%

While the recession is at the front of everyone’s mind, other underlying trends also clearly indicate the need for improved business practices.

Student debt is a critical issue in the US and is also putting pressure on practice owners. The average debt of a 2009 graduate of a US veterinary college was $129,976. Approximate 89% of those graduating from veterinary school had student debt and 90% of the debt was incurred while in veterinary school. About 32% of the 2009 graduates had debt in excess of $150,000, 11% have debt in excess of $200,000, and some in excess of $250,000. The average private practice starting salary for 2009 graduates was $65,185 resulting in a mean debt to earnings ratio of 1.99. A traditional rule of thumb for borrowing to finance an education is that no more than the first year’s salary should be borrowed (i.e. a 1:1 ratio.)
More debt of any kind reduces young veterinarians' options and increased debt means they have to look more carefully at their future plans and make more intelligent choices. The practices they choose to work in will have to give them the ability to make more money than in the past. This problem belongs to anyone who is going to need a young veterinarian in the future. While employers are not solely responsible for solving this problem, salaries must improve and employers will have to provide a practice environment in which associates can be more productive and be paid more.

It is also clear that much of the revenue growth in veterinary medicine has come from fee increases. 76% of the fees included in the AAHA Fee Reference increased above the rate of inflation from 2004 to 2006.

**Some of the most significant changes included:**

- Hospitalization—50% of the fees had statistically significant increases of 7-16% above the rate of inflation.
- Anesthesia—67% of the fees had statistically significant increases of 11-24% above the rate of inflation.
- Treatment procedures—72% of the fees had statistically significant increases of 6-22% above the rate of inflation.
- Surgery—60% of the fees had statistically significant increases of 8-54% above the rate of inflation and many surgeries had increases of >20%.

Fees did not increase quite so dramatically from 2006 to 2008 but they didn’t decline either. And during these same periods, the average practice saw a decline in new clients per full-time-equivalent veterinarian from 271 in 2001 to 203 in 2007, per the American Animal Hospital Association. Active clients also declined from 1299 in 2001 to 1141 in 2007.

A study published in JAVMA in 2008 ("An Examination of US Consumer Pet Related and Veterinary Service Expenditures, 1980-2005") indicated that while inflation adjusted expenditures on pet products & veterinary services increased, the % of total households with veterinary service expenditures remained constant. Translated into practical terms, this means that the % of households spending money on veterinary services than is declining. Data from the AVMA indicates the number of owned pets has increased 16% from 2001 to 2006 and yet the % of pets visiting the veterinarian at least annually has declined. Data from AAHA indicates transaction growth in the average practice is flat.

**What are the implications of these changes?**

- Fees are a big component of the increase in revenue and ATC over the last few years.
- Fee increases are likely part of the reason for the decline in visits to veterinarians.
- Real economic growth must come from better medical care, growth in client numbers and improved productivity & efficiency.

So what’s the answer? Results from the AVMA-Pfizer Business Practices Study released in 2003 indicated that 62% of practice owners don’t use financial concepts to manage their businesses. Those that do make almost 2/3 again as much as those who don’t. Veterinarians should and must continue to increase their earnings but it can’t be through simply raising fees. They must focus on better business practices such as communicating value to clients so that they accept more recommendations and improving efficiency and productivity so that the practice becomes more profitable.

Doing well in the future is largely about going back to basics. There aren’t any magical new management techniques that will allow practices to make a lot more money with little effort. Instead, practices need to focus on fully implementing sound operational systems and management practices. “Fully implementing” is the key—many practices already focus to some extent on critical areas such as client communication and capturing charges but few really do it well. 99% accuracy isn’t good enough. According to an unnamed source on the internet, 99% accuracy means:

- 16,000 pieces of mail lost per hour
- 20,000 incorrect drug prescriptions filled per year
- 500 incorrect surgical operations performed each week
- 2 unsafe landings at O’Hare International Airport each day
- 50 newborn babies dropped at birth by doctors each day
- 22,000 checks deducted from the wrong accounts each hour
While these figures may not be entirely accurate given the nature of what you read on the internet, the point is critical. Even small numbers of errors can have catastrophic effects.

Most practices need improvement in multiple areas; it’s uncommon to see a practice that has just one huge problem, which, if addressed, will fix their profitability issues. Common revenue problems include:

- Inconsistent fee increases
- Excessive discounting
- Missed charges
- Inefficient use of doctors and staff (low production)
  - Minimum wage employees
  - Too many part-time employees
  - Poor hiring and training
  - Little correlation between compensation and production
- Inefficient use of building
- Focus on less profitable services
- Poor location
- Poor client service
- Adequate scheduling and doctor availability
- Owner compliance with doctor recommendations
- Low numbers of clients
- Poor communication of value to pet owners

Common expense problems include:

- Poor inventory control
- Doctor and staff compensation and benefits
- Expensive facilities
- Expensive equipment
- Excessive debt
- Poor management systems
- Upward creep in all expenses

Communicating value to clients is one of the most important areas practices must focus on going forward and is the cornerstone to the success of other changes. In order for clients to accept the practice’s pet care recommendations, they must first understand them and value them more than the other things they might choose to spend money on.

**Why is this particularly important now?**

- Clients are increasingly nervous spenders
- Fewer pet owners are visiting veterinarians; transactions are flat in many practices
- It is an increasingly competitive marketplace
- Veterinary care is complicated and difficult to understand
- Hospital visits can be frightening
- People take in information in different ways

Before you can communicate well, it is first critical to understand how clients judge value. An article in the Harvard Business Review studied the communication of value at the Mayo Clinic. The Mayo Clinic happens to do this extremely well while spending little money on traditional forms of advertising. The authors noted that “When a company’s offerings are hard to judge, customers look for subtle indicators of quality.” Translated, this means that clients can’t judge the quality of the medicine but will look at the aspects of your practice that they CAN understand to make a judgment about the medicine. This may not make logical sense but it’s the way it works. Clients can’t and don’t judge medicine; they judge service. And in order to get your message across, every activity, person, place, document, interaction, transaction, and communication should tell the same story—“Every patient, every client, every record, every time.”

Communication is also critical to insuring clients comply with practice recommendations. However there are other aspects of client compliance as well and this continues to be critical to good patient care and financial success. Information from the NCVEI website indicates veterinarians perform between 8-10 fecals per doctor per week. This would be approximately 2/day. Does this make sense given the number of wellness exams done in the average practice each day? Information from the NCVEI website also indicates 2 dentals per veterinarian are performed each week in the average practice. Given the number of pets that need dentals, does this make sense? The American Animal Hospital’s 2002 Compliance Study revealed that many clients did not comply with practice recommendations as frequently as practices thought they
did. While some of the responsibility for this is clearly the clients'; there were many instances in which practices did not make recommendations as they should have or did not make it easy for clients to comply. Even small increases in these figures will improve pet care and practice earnings.

**Why Don’t Clients Comply?**

- Lack of time
- Inconvenience
- Don’t see the value
- Cost
- Lack of recommendation by veterinary practice
- Barriers put in way by veterinary practice
- Lack of follow-up by veterinary practice

Cost is rarely the primary reason and its obvious from the above list that there are many things a practice can do to improve compliance. The Compliance Implementation Study done by AAHA in 2008 is an update to the original study. This study focused on practices that had made efforts to improve compliance and indicated the following:

- Best results occurred in practices that did NOT make big investments in staff or equipment but that DID get full practice team commitment and buy-in
- Many more practices feel they are now responsible for client compliance—in 2002, 60% interviewed said it was the client’s responsibility; in 2008, 60% interviewed said it was the practice team’s responsibility

In spite of all indications that improved client compliance is important both to good patient care and the financial success of the practice, only 22% of practices in 2007 audited medical records in the last year to measure treatment recommendations and only 20% audited medical records in last year for the rate of client compliance. You can’t manage what you can’t measure.

So what can your practice do to improve compliance?

- Locate resources to help you improve—www.aahanet.org and www.dvm360.com
- Pick the areas you first want to focus on—set goals that are a bit of a stretch but achievable
- Train your staff
- Review operations—do they support your goals?
- Monitor progress
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Using Financial Metrics Wisely
Karen E. Felsted, CPA, MS, DVM, CVPM

INTRODUCTION

Before discussing what data should be analyzed, it is first important to discuss why benchmarking is important. The term “benchmarking” is used loosely and some feel that other terms would be better. Regardless of what the activity is called, however, it is critical that it be performed. Review and analysis of critical financial and operational data should be performed on a regular basis. Analysis of data trends within the hospital as well as metric comparison to outside sources should both be done.

There is an old cliché that says “If you can’t measure it, you can’t manage it.” While it is easy to ignore clichés because you’ve heard them so many times, these phrases are generally clichés for a reason—they are widely applicable truths that should not be ignored. Obviously these specific words have to be taken with a grain of salt—it’s not necessary to count the paperclips on a daily basis in order to manage the purchase of office supplies but managing most areas of the hospital well means you need to measure the activities.

Improved management of the hospital is critical to better patient care and greater financial success. Quality patient care requires a sound economic foundation. A practice that is not financially successful will find it difficult to invest in the facilities, equipment, medications and staff necessary to provide the best patient care much less provide for the financial security of the practice, its owners and the staff. Results from the AVMA-Pfizer Business Practices Study released in 2006 clearly demonstrated the connection between the measurement of data and those hospitals that are financially successful. In a nutshell, the study showed that the majority of practice owners don’t use financial concepts to manage their businesses and those that do make more money. For example, only 38% of the survey respondents indicated they used financial concepts to run their businesses. The number of respondents who performed a monthly review of financial data ranged from 22% to 77% depending on the individual items analyzed. And yet the study showed that there was almost a $50,000 difference in the average earnings of veterinarians who used financial concepts to run their businesses versus those who did not.

Key Areas in Which Benchmarks Should be Reviewed

Listed below are the critical areas in which data review and analysis should be done on a regular basis. Within each key area are some data points that should be reviewed by all practices every month.

- Revenue
  - Revenue by category
  - Revenue by FTE doctor and by individual doctor
  - Revenue drivers—transactions and ATC
  - Discounts and missed charges

- Expenses
  - Doctor labor costs
  - Support staff labor costs
  - Costs of goods sold
  - Facility costs

- Profitability

- Clients
  - New clients
  - Active clients
  - Lost clients
  - Source of clients

- Fees

- Facility
  - Revenue and profits per sf
  - Revenue and profits per exam room
  - Revenue and profits for boarding and grooming areas
It will often be necessary for the practice to gather additional information beyond that discussed here. The KPIs listed in this paper are the starting points. When a trend is discovered or an area that appears problematic, further analysis will be necessary. For example, let's say that the practice owner or manager is analyzing the revenue of the practice at the doctor level and finds that the average revenue per doctor is lower than that seen in most practices and that there is a great deal of variation in productivity amongst doctors. Some of the information you may want to gather to help determine how to address this situation includes the following:

- Number of hours worked each week by the doctors—revenue variability may be a function simply of the time spent in the practice
- Support staff help utilized by each doctor—some doctors may be able to produce more because they have access to and use more support staff
- Number of key procedures (CBCs, chemistry panels, x-rays) performed by each doctor in relation to the number of transactions they generate—revenue may vary because of different approaches to cases which should be more consistent
- Measurement of client compliance with key recommendations by doctors and staff

**SOURCE OF DATA**

Where does this data come from? Your practice most likely has two sets of software used in the operational and accounting functions of the practice. The first is the veterinary practice management system which helps manage and analyze revenue collection, inventory, receivables and other revenue related information. The second system is an accounting package such as QuickBooks or Peachtree. The accounting software is used to manage the payment of bills and payroll, prepare financial statements and aid the accountant in the preparation of the practice's tax return.

"Key performance indicator" or "KPI" is a term often used for the financial or non-financial practice operating statistics or metrics analyzed: KPIs:

- Provide information about the operations of the practice not normally available in traditional financial statements
- Help explain changes seen in the financial statements
- Measure performance of the practice
- Indicate areas in which improvement is necessary

**GUIDELINES FOR EFFECTIVE FINANCIAL ANALYSIS**

**Standardization of revenue categories**

In order to compare your practice to others, it is necessary to set up your revenue service categories in a fashion similar to those commonly used in veterinary medicine and used in the published studies you will be comparing the practice to. Start with the major procedure categories such as Examinations, Surgery, Laboratory Tests, Imaging, etc. It may be helpful to have subcategories underneath the primary categories. For example, the primary surgery category will simply be called “Surgery” but you may have subcategories such as “OHE & Castration,” “Orthopedic” and “Soft Tissue.” Individual services such as “OHE-Feline” or “Castration-Canine-0-25#s” will then be listed under each heading as appropriate. All practices are a little different from one another and ultimately you will need to include all of the service codes and categories that best reflect how you operate; however, you will have a greater ability to compare yourself to others if your setup is similar to above and you will not lose any flexibility in internal analysis by staying consistent with the industry standards. Once a practice has been in business for awhile, these categories tend to get muddled. You should review them annually and make sure that procedures are included in the right category or subcategory and that there aren't too many things included in "Miscellaneous."
Consistency in the recording of information

It is important to record information in the same categories or perform calculations in the same way each time you do them in order for your numbers to be comparable over time. For example, if you started out by classifying the anesthesia used in dental procedures in the “Dental Revenue” category and then later switched it to the “Anesthesia Revenue” category, these numbers will not be comparable before and after the change and your trends analysis and related conclusions may be inaccurate. In order to achieve this comparability, it is important to carefully set up your categories and define your calculations when you set up your system and, when it is necessary to add a service code to be careful that you put it in the right place. Sometimes it will be necessary for you to make a big change; for example, to switch the dental anesthesia from “Dental Revenue” to “Anesthesia Revenue” in order to improve your analysis. This is acceptable; just remember that in the month of the change, you will lose comparability. You will regain this comparability as time goes on and you add more data.

Common basis for comparison

Comparison of raw numbers is useful to a point in financial analysis but, in general, it is necessary to do some kind of ratio analysis or use a common basis of comparison to get the best results.

Ratios can be calculated from information available in the balance sheet, profit and loss statement or from other reports available in your practice management system. Ratios are more useful in analysis and planning compared to actual numbers for several reasons:

- Relationships between various pieces of information are often more clearly apparent

- Trends over months or years can be more easily analyzed as the practice grows

- Businesses of different sizes can be more easily compared

The most common ratio used in veterinary financial analysis is that of expressing types of revenue or expenses as a percentage of total gross revenue. For example, dentistry revenue may be $30,000 in the current year. If that is compared to dental revenue in the prior year during which the practice also generated $30,000 you might think the practice was continuing to provide dental services for a similar percentage of pets in the practice. However, if the total revenue grew from last year to this year, the dental revenue as a percentage of total revenue actually declined. This means the practice may have been actually providing less in the way of dental services given the growth in the revenue.

Let’s say you compare your $30,000 of dental revenue to a study reporting this information for the veterinary industry, and that study told you that the average dentistry revenue in a practice was $40,000 per year. Did this tell you anything useful? Not really because you aren’t necessarily comparing yourself to a practice of similar size. However, if you calculate your dentistry revenue as a percentage of total gross revenue (say 3%) and compare that to outside metrics that tell you that the average practice has dental revenue of 4% of gross revenue, then you know that you are doing less well than the average practice in this area. This kind of ratio analysis is critical to accurate comparison over time or between practices of different sizes.

Another frequently used common basis for comparison is the calculation of doctors on a full-time-equivalent basis. This makes it possible for you to compare your numbers to practices that have different numbers of doctors or to compare the figures in your own practice from year to year as you add doctors. A single FTE doctor is one who works 40-45 hours per week, 48+ weeks per year. If you have a doctor who works 30 hours/week 50 weeks per year, this is 0.75 of a FTE doctor. If you have a doctor who works 40 hours per week but started July 1, 2007 then for 2007, this will be a 0.50 FTE doctor.

Comparison to industry studies

There are a number of readily available sources of industry metrics that you can compare your practice to. The most common are:

- The National Commission on Veterinary Economic Issues website (www.ncvei.org) is available for AAHA and AVMA members both in the US and Canada. There is no charge for any of these services and your data is stringently privacy protected. This is the most current data available.

- Financial & Productivity Pulsepoints (available for purchase from the American Animal Hospital Association)

- AVMA Business Measures Study (available for purchase form the AVMA)

- Benchmarks: A Study of Well-Managed Practices (available for purchase from Advanstar)

- CVMA
Key factors to remember when comparing your practice to published data:

- Is the data meant to represent an average practice or “best practices”? Different studies have different purposes.

- Is the data reliable? Reviewing the methodology section of the reports will give you a feel for how many practices responded and how comparative the data is. It is also important to understand how certain calculations were performed in order to know if that metric is comparable to your practice. For example, if you define the number of active clients as any client who has ever been to your practice and a study defines it as a client who has been in your practice within the last twelve months, then your data is not comparable to that particular study. As data is cut into ever smaller units (for example, by region or state), sample sizes get smaller. While smaller sample sizes may not be perfectly representative of the profession, you can still gain some useful information from them as long as you are careful about the reliance you place on the information.

- No study will be perfectly comparable to your practice. For example, you may be comparing 2008 data to a study with 2006 data. You may include different items in the measure of personal doctor revenue production than the study does. Or there may be a small number of respondents to a particular question in a study that you are interested in. This doesn’t mean the study is useless to you. You can still gain some useful information from them as long as you are careful about the reliance you place on the information.

- Just because you are different from the comparative practices doesn’t mean there is something wrong with your practice. All practices are unique in one way or another and, occasionally, the published benchmarks may not be the standards you want to shoot for. However, if your numbers are significantly different, it is strongly recommended that you investigate why and determine if you have a financial or operational problem that needs to be resolved. In most cases, if your numbers are significantly different from the various studies, you have a problem.

- Comparison to industry averages can be very useful in identifying potential problems in the practice, but comparison of your own data from the current period to previous periods is better for identifying less obvious changes and monitoring corrective action.

Using the information discovered

Identifying trends or problem areas is not enough. It is critical that you investigate the changes or the potential problem areas and determine if action is needed to correct an issue.

At first you may not know how to interpret these reports or trends. Sources of information include the following:

- If you used the NCVEI benchmarking website, there is a “Recommended Treatment” section for each benchmarking tool which gives you some basic information about improvements you should consider in your practice. The Resource Library has additional studies, articles and other information.

- AVMA
- AAHA
- Dvm360.com
- VetPartners (formerly the Association of Veterinary Practice Management Consultants and Advisors (www.vetpartners.org)
Are You as Profitable as You Think?
Karen E. Felsted, CPA, MS, DVM, CVPM

The Brakke Management and Behavior Study empirically demonstrated the importance to practice owners of understanding the finances of their practices and how few owners really do. Bottom line: The majority of practice owners don’t understand financial terms and those that do, make more money. Only half of the group understood “pre-tax profits” and “cash flow.” Only 10-20% of the respondents could choose the correct definition of the other terms in a multiple choice format. And it makes a big difference in earnings—Male owners who answered three or more questions right had personal incomes of 7% greater than those who didn’t—female owners who answered three or more questions right had incomes that were 19% higher than those who didn’t.

In addition to the obvious impact on current cash flow, profitability also is a critical determinant of practice value. Historically, practice owners have assumed (and with good reason) that when they decided to sell their practices there would be buyers ready to purchase them and willing to pay a good price. In other words, they have assumed there was value in these businesses that could be transferred to someone else. Of course, there have always been a few practices for which this assumption didn’t hold true. A buyer couldn’t be found or what buyers wanted to pay wasn’t remotely what the seller thought the practice was worth. Typically these practices have been easy to identify and had several traits in common. They tended to be smaller practices with owners who had not focused much on the business side of things. Often the facility and equipment were old and the doctors hadn’t kept up with the changes in medicine as much as perhaps they should have. These practices had little profit in them and, because the bulk of practice value is determined by profitability, the practices had little value. Fortunately there weren’t too many of these practices.

However, in the last few years, the number of practices with no or little value has been increasing—to the point where the Veterinary Valuation Resource Council of VetPartners (formerly the Association of Veterinary Practice Management Consultants and Advisors) coined the term “No-LoSM practice” to describe these practices. More and more practices, when appraised, did not have the value that would normally have been expected. And, in almost all cases, the owners of these practices were totally unaware of the problem. Some of these practices had traits in common with the practices that have historically had little or no value. They were small practices with a low level of profitability and couldn’t keep up with changing client demands regarding service, quality of medicine, advanced technology and improved facilities. The other practices with no or little value, however, were a surprising group. On the surface, these practices would appear to be doing very well. They are located in very attractive facilities, practice good medicine, have all the latest equipment and a large support staff, offer comparatively high compensation and benefits to their employees and, in the owners’ eyes, cash flow is strong. However, practice value is largely based on profits and the very factors that make these practices look attractive on the surface are those that are reducing profitability.

Understanding the profitability of a practice is one of the most important concepts necessary to manage a veterinary hospital well. Profitability is the one single number which tells you how you are doing financially. Calculating the true operating profits of a practice is not a simple task. None of the standard financial or management reports a practice usually gets show this figure. Neither the taxable income from the tax return nor the net income from the profit and loss statement represents true profitability. This doesn’t mean those reports are improperly prepared; it simply means the reports required by your taxing authority or accounting standards for small businesses weren’t designed to determine profitability. No one report will give a practice all of the financial information it needs to make intelligent operating decisions; unfortunately, the report that seems to be prepared least often is the one that calculates true practice profitability. Because practice owners and managers aren’t used to getting this kind of information, they generally don’t know what the true profitability of their practice is. The first time many owners realize their true profitability is when their appraiser talks to them about it.

Operating profit is the difference between the operating revenues and expenses of a practice. Operating revenue and expenses include only items normally and necessarily seen in the day to day operations of the practice such as fees for professional services and drugs and medical supplies expense. These items should be stated at fair market value rates. For ease of comparison with other practices, the profit margin is generally stated as a percentage—this is calculated as practice profits divided by gross revenue. Some of the items that must be calculated differently to determine operating profit versus taxable income...
or net income include: practice owner payments, facility and equipment rent if these items are owned by the practice owner and leased to the practice, services provided by family members to the practice, depreciation, interest on debt and perks.

Owner compensation is one of the most significant adjustments and almost always has to be calculated differently in determining operating profits than would be done for the tax return or other reports. Owners often arbitrarily determine an amount they will be paid through their payroll system; this amount often has no correlation to the actual medical, surgical and management work the owner does in the practice and therefore the tax return or income statement looks as if the practice is more or less profitable than it really is. Tax regulations can also dictate how some aspects of owner payments must be handled and these regulations can vary by entity type. For example, in the US, owner compensation must be reported differently for a C corporation than for a partnership. A practice may appear to be more or less profitable than it really is simply because of these regulations.

So how do you calculate operating profit? Net income per the financial statements or tax return is the starting point. Various adjustments are made from there.

- Add back: depreciation, amortization and interest on debt
- Deduct the estimated average amount spent on equipment per year—purchasing equipment is a true operating expense of the practice but depreciation as determined by tax law is not the best estimate
- Determine how much the owner was paid during the year and what it was comprised of (salary, rent, etc)
- Adjust owner compensation to represent a fair compensation for medical/surgical work—20% of personal production is a good average in a small animal practice
- Adjust owner compensation for management work—management expense generally averages 3-4% of gross revenues—if you have a practice or office manager, the owner should get less than 3% of revenues as management compensation
- Adjust rent expense to fair market value if paid to owner at a rate greater or less than fair market value
- Adjust equipment lease expense to fair market value if paid to owner at a rate greater or less than fair market value
- Determine the $ amount of personal perks paid by the practice and remove this expense—perks would be items not necessary to the operation of the practice but paid by the practice generally to gain a tax advantage (examples include excess meals and entertainment, excess auto costs, swimming pool payments, personal furniture, trips to Tahiti, etc)
- Deduct the cost associated with free services provided to the practice—family members may provide bookkeeping or other services to the practice at no charge—if the practice had to hire someone to do this work, there would be a cost involved and this should be included as an expense
- Remove any true non-recurring income or expenses such as one-time insurance proceeds or expenses related to a natural disaster
- Recalculate net income
- Divide the new net income by gross revenue

The above may sound a little daunting but there are resources available to help you. The National Commission on Veterinary Economic Issues has a tool on its website (www.ncvei.org) to help you calculate your profitability. This tool is a joint project between NCVEI and VetPartners and is available to anyone who is an AVMA or AAHA member.

The resulting percentage is the true operating profit of the practice—how does it compare to other investments you have? And to other practices? 18% would be considered superior, 13-16% average, and less than 13% is below average.

If your profits aren’t what you want them to be, what can you do about it? A lack of profitability either comes from revenues that are too low, expenses that are too high, or a combination of the two. Expense management is often the easiest to understand so it will be discussed first.

What practices don’t do when making the decisions to invest in equipment, staff or facilities is to make sure that the costs will lead to increased levels of revenue and thus profits.

For example, how much space and what kind of building is really necessary to practice veterinary medicine? Operating out of the Taj Mahal can be very psychologically rewarding but may not be good financially. For example, if a practice moves into a beautiful, new facility and the rent doubles, will there be a sufficient increase in revenue (and more importantly profits) to cover this rent increase?

The same goes for the addition of staff. A doctor’s work life may be much easier and personally rewarding with 3 techs trailing behind him or her during the day, but does this doctor actually
produce more revenue with this additional support staff? If not, the cost of the staff is eating into the profitability of the practice. Other staff problems seen in practices include the hiring of low-level, minimum wage staff that can’t do the job properly, too many part-time employees and a lack of training and supervision. All of these lead to inefficiencies in getting the job done.

Declining revenue or a lack in growth of revenue is the other factor contributing to a lack of profitability. Practices often don’t focus specifically on growing revenue because it’s harder for them to determine what to do. Prior to the recession, many practices didn’t have to worry about it either; they’ve been fortunate in that the revenue has just seemed to be there. If they don’t have the 12-13% growth they did a few years ago, owners often assume that’s just because of the recession or the practice has matured or the demographics of their area are changing and that there is nothing they can do about it.

The reality is quite different, however, for many practices. Generally there is much a practice can do to keep revenues strong even if located in a demographically challenged area. For example, are fees appropriate? It’s not uncommon to see a practice that hasn’t increased fees in two years or has only increased a few of them by a small percentage. Most expenses in a practice rise annually because the providers of those goods and services raise their prices—this is true of staff costs, drugs and medical supplies and the various other goods and services used by a typical practice. If the practice isn’t raising its fees at least 5% per year, profitability will suffer even if nothing else changes in the practice.

Lack of attention to discounts and missed charges can also lead to declining revenue. Even a small amount of products or services given away by well-meaning doctors or other team members can significantly decrease revenue and profitability. Missed charges, those not deliberately given away, can also dramatically reduce the profit margin. Capturing charges is generally about having good systems in place and is essential to efficient operations. It is a rare practice who doesn’t experience these problems on a fairly regular basis.

Understanding not only the profitability of the practice but the kinds of factors that lead to this state is critical. Until the practice has an idea of the root causes of the problem, it is difficult to determine what the correct solution is. Working with a financial advisor or practice consultant may help in not only gaining a greater understanding of the issues impacting profitability but in identifying and implementing solutions.
It’s All about Implementation

Karen E. Felsted, CPA, MS, DVM, CVPM

Doing well in the future is largely about going back to basics. There aren’t any magical new management techniques that will allow practices to make a lot more money with little effort. Instead, practices need to focus on fully implementing sound operational systems and management practices. “Fully implementing” is the key—many practices already focus to some extent on critical areas such as client communication and capturing charges but few really do it well. 99% accuracy isn’t good enough. According to an unnamed source on the internet, 99% accuracy means:

- 16,000 pieces of mail lost per hour
- 20,000 incorrect drug prescriptions filled per year
- 500 incorrect surgical operations performed each week
- 2 unsafe landings at O’Hare International Airport each day
- 50 newborn babies dropped at birth by doctors each day
- 22,000 checks deducted from the wrong accounts each hour

While these figures may not be entirely accurate given the nature of what you read on the internet, the point is critical. Even small numbers of errors can have catastrophic effects.

In order to effectively implement sound business practices, the hospital must:

- Have an owner or practice manager with a strong set of financial, managerial and HR skills—the title isn’t enough
- Design efficient policies, procedures and systems for getting things done
- Have a detailed, understanding of the skills staff members must have to provide outstanding patient care and client service
- Hire effectively—find and keep the employees with the right skills and attitudes necessary to achieve the practice’s goals
- Provide high quality, effective and ongoing training programs to both new team members as well as those who have been in the practice for awhile
- Have high levels of employee retention—revolving door employees aren’t around long enough to be efficient and effective
- Delegate effectively—tasks should be done by the lowest level person who can do the job properly
- Monitor staff activities frequently—in most practices staff are always busy doing something—what they are doing, however, is the key point—is it the most important activity that should be done?
- Regularly review key metrics

Some of these key areas are discussed below.

Financially successful practices are, almost by definition, well-managed practices. It used to be possible to have a successful practice without a great deal of business skills, but this is no longer true. Clients have higher expectations in both the medical and clients service arenas, veterinarians face increased competition, and the complexity of business regulation has increased.

Most people aren’t born with a full set of good management skills. Just as it took training and practice to learn to perform an OHE, it takes similar dedication to become a skilled manager. One difficulty in veterinary medicine is that most veterinarians do not graduate from veterinary school with a solid grounding in business skills. Veterinary school curriculums are already strained by the burgeoning amount of veterinary knowledge that needs to be passed on to students and most students didn’t take business courses in their undergraduate years. Another difficulty is that many veterinarians aren’t inherently interested in business management. They went to veterinary school to learn to practice medicine, not to be a business manager. Many practices have hired practice managers to take on the business side of the practice. However, not all managers have been given the training and resources they need to do their jobs well. Fortunately, these are correctible problems.

Management duties are handled differently in different practices depending on the size of the practice, the interests of the owner veterinarians and the money available to hire managerial help. In some practices, the owner veterinarians do all of the manage-
ment work either out of necessity or because they enjoy it. In other practices, the owners have hired a full-time, experienced practice manager with the result that the veterinarians primarily practice medicine. The owners make the high-level decisions, but leave day-to-day management to those paid and trained to do it. In other practices, management duties are divided between the owners and other individuals, including support staff, practice managers or associate veterinarians. As practices get bigger and as management duties become more complex, it becomes more necessary to have highly skilled, professional managers in place. Veterinarians will contribute more to the profitability of the clinic through the practice of medicine and surgery than by being managers.

While owners certainly don’t have to be involved in the day-to-day management activities of a practice, it is critical that they provide vision and leadership to the business. In addition, they need to set the framework for the decisions to be made, direct and approve the overall activities, and support the management personnel in their responsibilities.

In order to design an efficient procedure or system, you must first identify what tasks must be accomplished in the hospital. Take a look at each area in your practice and list all of the significant tasks that must be done:

- **Reception**
  - Answer phone
  - Intake—new clients
  - Intake—on-going clients
  - Check-out
  - Etc

- **Exam rooms**
  - Outpatient visits—doctor tasks, non-doctor tasks
  - Entering charges
  - Client education
  - Cleaning
  - Etc

- **The “back”—treatment, surgery, hospitalized pet area**
  - Outpatient visits—medical procedures
  - Intake of day patients or those requiring ongoing hospitalization
  - Entering charges
  - Pet care—meds, feeding, walking, daily exams
  - Cleaning
  - Etc

- **Boarding/grooming**
  - Intake
  - Daily tasks—feeding, walking, meds
  - Cleaning
  - Etc

- **Management**
  - Scheduling
  - Dealing with employee conflict
  - Dealing with client problems
  - Inventory control
  - Financial analysis
  - Etc
The steps involved in each task should then be documented. A written procedure manual is an important tool to be used in design/improving systems and in staff training. The manual should include:

- Steps necessary to accomplish task
- Who is responsible
- When task should be completed
- Equipment, people, supplies needed
- Who will verify task is properly completed
- Priority of tasks
- Fill-in tasks

Each task should be reviewed for bottlenecks and inefficiencies. All staff members involved in the task should be involved in the review; often times those performing the task will have the best ideas about how they can be done more efficiently. Key components of efficiency include segregation of tasks, keeping people, equipment and supplies in the area the work needs to be done, reducing distractions and doing as much prep work as possible before the task itself needs to be performed.

Another component of implementation is having doctors and staff who have the skills necessary to perform the tasks. Those skills need to be specifically identified; this can be done by:

- Employee interviews/focus groups
- Surveys
- Observation
- Review of job descriptions
- Performance data review
- Knowledge tests
- Medical record review

If staff do not have all of the skills necessary to efficiently perform the necessary tasks, a training program must be put into place. A good training program takes time to design and implement, but the benefits will far outweigh the costs. Not only do staff members need training in multiple areas—medical topics, client service topics and the specifics of their job duties, but they also need training in how to prioritize their job duties and in working efficiently. This training gives employees the ability to provide better client service and allows veterinarians to delegate more tasks and concentrate on doing what they were trained to do—diagnose and treat cases. Equally importantly, staff training keeps employees interested and motivated. The more an employee knows how to do, the more interesting their job is and the more pride they have in the practice and confidence in themselves and what they do. And employees who have a greater range of skill and knowledge can be more efficiently utilized resulting in greater income to the practice. Review your training program to make sure you are addressing all of the steps below:

- Decide what kinds of training you need. Most practices need a new-hire training and orientation program (for both doctors and support staff) as well as an on-going training program. The new-hire program will usually have a standard content whereas on-going training programs are generally more flexible depending on the needs of the practice.
- Designate a veterinarian or senior support staff member as the Training Manager. Give them the time and the tools necessary to set up and manage an on-going training program.
- Make sure you have current written job descriptions to aid staff and doctors in understanding the parameters and expectations of each position.
- Decide on the critical topics. New-hire training generally consists of an orientation to the practice’s employee policies and procedures, introduction to all staff members, computer system training, OSHA training, time spent in each area of the clinic and finally, specific training in the new employee’s position. On-going training programs are generally split between business and client service topics (good telephone skills or dealing with clients’ grief) and medical topics (vaccination programs, catheter placement, etc)
- Decide which topics are important to which employee groups. Some topics (computer system use, good telephone skills) should be taught to everyone whereas receptionists generally don’t need to know how to intubate a dog.
- Get input from everyone in the practice as to what training is currently needed. Have a doctors’ meeting and see what skills they think are lacking in the support staff. Ask the support staff what lack of knowledge or skills is impeding their progress. Ask recent new hires what would have made their jobs easier had it been included in the orientation program.
- Decide on the timing of the training. Some kind of training should occur every week. Some sessions will include all staff members and others will only include one department (i.e. receptionists.) There is no ideal time for training, but pick one that is least disruptive to the practice. Many practices use the lunch hour for staff meetings and training.
sessions but this is often inefficient because it is difficult to start on time and there are frequent interruptions. Early morning works well for many practices. For example staff meetings and training are scheduled every Wednesday morning from 7am-9am. Either the practice doesn’t open until 9am that day or one person is designated to deal with the unavoidable walk-ins or emergencies. If the practice is open during this time, no scheduling of appointments, drop-offs, etc should occur and the phone should be forwarded to the answering service or machine.

- Vary the subject matter and the type of training session. Some will be traditional classroom style; others may include role-playing or hands-on practice sessions.

- Take advantage of the many resources already available in the form of books, tapes, etc. from the AVMA, AAHA and other organizations.

- Involve everyone in your practice. If you have a technician who is particularly skilled in catheter placement, have them help the less experienced staff members. If one of your doctors has a special interest in orthopedics, ask them to teach a session. Bring in outside speakers—drug company reps will often talk on topics related to their products. Someone in the practice may have a friend who works in a related area (for ex. a psychologist who can talk about grief counseling or a marketing guru who can lead a discussion on attracting more clients to the practice.)

- Share knowledge gained at outside meetings. Each participant (doctor or staff member) should discuss the most useful things learned with the rest of the practice.

Delegation is also critical to implementation of tasks; however it must be done right to be effective. This doesn’t mean just dumping any task on the first employee you can find and expecting it to be done perfectly.

**Think through first, why do you want to delegate tasks?** This will help you decide what to delegate and to whom. **Common reasons include:**

- Redistribution of the workload as the clinic grows
- Reduce higher level employee work (and stress) load
- Provide employees with more interesting tasks and opportunities
- Obtain new insights in how to accomplish tasks

**Effective delegation is an art, not a science. These steps are critical:**

- The potential delegator must get over the idea that they alone can correctly perform the task
- Determine trust level with employees before delegating
- Decide what tasks to delegate
- Match tasks with the appropriate employee
- Allocate time to do the task to the employee who is now in charge of it
- Provide correct level of control and authority
- Follow-up periodically
- Offer the right reward and recognition for jobs well done

In general tasks should be done by the lowest level person who can do the task correctly. However, in spite of this rule, some tasks should not be delegated. These include sensitive interpersonal communications, critical client matters, tasks that, by law, a veterinarian must do, the dissemination of good news, the releasing of critical information, and disciplinary tasks.
Within any group of individuals, one will find diversity that stems from differences in personal experiences, perceptions of what is seen, understandings of what is heard, abilities to manage stress and approaches to solving problems. Diversity can foster progress, innovation, advancements, best medicine practices and strong clinic teams if the differences are acknowledged and appreciated.

Differences between individuals can be based on gender, age, culture, perceptions as well as a number of additional innate characteristics:

a) preference for left or right hemisphere processing
   - left brain processing refers to the use of logic, detail, reality based information to present practical strategies
   - right brain processing refers to the use of feelings, imagination, symbols, images and fantasy to present possibilities

b) predilection for a given behaviour style
   - do you focus on data and facts or on people?
   - do you prefer to make decisions quickly or are you more deliberate in your decision-making?

c) preferred style of learning
   - auditory learners learn best by sharing in discussions, listening to a lecture or talking aloud
   - visual learners learn best by reading the information that is to be learned, watching demonstrations and taking notes
   - tactile learners learn best by physically rehearsing or practicing the skill, imitating or experiencing the lesson

Diversity within a group can have its challenges and needs to be managed if we are to gain the advantages of multiple viewpoints. After all, the wider the lens – the better the view!

**Strategies to manage diversity**

1. Create an environment of openness

   - lead by example to develop a clinic culture that encourages feedback in a safe environment
   - establish guidelines to help avoid personal or petty issues from becoming the source of team conflicts (eg., does the issue affect patient care, client service or an employee’s ability to do their job in a safe manner?)
   - provide formal forums for discussion such as regularly scheduled performance reviews and staff meetings
   - establish “core values” for the practice to set out collective standards by which all team members must abide
   - coach team members on how to give and receive constructive feedback
2. Receive and give supportive feedback

As mentioned previously, team members should be coached on how to receive and to give constructive feedback. A common misconception that is held when a difference of opinion arises is the notion that someone has to win and someone has to lose the debate. In fact, it is better to start with a common interest or common outcome that is to be achieved through the discussion. In veterinary practice, this common ground is often what is best for patient care.

The ‘golden rules’ for giving feedback:
- Give feedback close to the event
- Ask questions to ensure you have the ‘full story’ and all perspectives
- Stick to one topic (don’t string together several criticisms at one sitting)
- Don’t remind people of previous instances that were resolved
- Address issues or problems with the person who is directly able to resolve or change the situation
- Avoid blame – focus on how their actions affected you or your work
- Speak respectfully
- Do not sound threatening
- Keep it balanced
- Focus on the problem and facts, not the personality
- Don’t overstate the problem (avoid “never”, “always”, or “worst”)

Golden rules for receiving feedback:
- Welcome feedback
- Listen to criticism
- Maintain eye contact and open body language
- Restate the criticism to ensure full understanding
- Acknowledge the emotion or frustration experienced by the speaker
- View the criticism as an attempt to solve the problem versus as a personal attack
- Focus on possible solutions

3. Schedule effective staff meetings

An effective forum for encouraging new ideas and contributions to the practice is through well organized staff meetings. Unfortunately, many staff meetings are viewed as simply complaint sessions that do not result in clearly actionable decisions. Staff meetings require active leadership, advanced planning and a dependable pattern of concluding with actionable decisions.

Tips for organizing effective meetings:

a) Select a chairperson for the meeting (this assignment can be rotated to different team members throughout the year). The chairperson’s responsibilities include preparing the agenda, keeping the discussion on track and focused, summarizing and ensuring understanding, and assigning tasks

b) Before the meeting:
- seek input for the agenda
- prepare and post the agenda
- list the decisions that are to be made at that meeting
- assign who is responsible for leading each discussion point

c) Plan the agenda
- start and end time
- purpose of the meeting
- who will participate
- location
- order of the topics
- decisions to be made
- who is reporting on what

d) After the meeting
- post the decisions that were made
- post the action points and the person(s) responsible
Effective meetings also require the skilful handling of the common roadblocks to communication. This includes strategies to manage unfocused discussions and negative behaviours such as angry outbursts, the ‘meeting hog’ and the ‘interrupter’.

<table>
<thead>
<tr>
<th>BEHAVIOR</th>
<th>MANAGEMENT STRATEGY</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stray off topic</td>
<td>• identify this is occurring, refocus</td>
</tr>
<tr>
<td></td>
<td>• adopt a topic “parking lot”</td>
</tr>
<tr>
<td>The “meeting hog”</td>
<td>• “That’s an interesting point. What do the rest of you think?”</td>
</tr>
<tr>
<td></td>
<td>• “We’ve been making Mary do all the work. What do the rest of you think?”</td>
</tr>
<tr>
<td></td>
<td>• use a prop – ‘talking stick’</td>
</tr>
<tr>
<td>The “interrupter”</td>
<td>• chairperson to identify this behavior and redirect conversation</td>
</tr>
<tr>
<td></td>
<td>• use a prop</td>
</tr>
<tr>
<td>Tempers flare</td>
<td>• the chair should emphasize points of agreement and minimize points of disagreement</td>
</tr>
<tr>
<td></td>
<td>• draw attention to the objectives of the meeting</td>
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<td></td>
<td>• ask direct questions relating to the topic</td>
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Creating Teams That Work

Jayne Takahashi, BSc, DVM, MBA

In most cases, effective teams don’t happen by sheer luck. It takes a conscious effort and process to develop successful teams. This seminar will discuss factors that impact the success of a working team, parameters to help assess team effectiveness, and key elements that foster strong team communication. Much of the information presented here is based on the work of Richard Hackman of Harvard University, a leading researcher on effective teams.

To be effective, veterinary teams need to accomplish three things:

- Satisfy the needs of their clients (pet owner, other veterinary practices, breeders, farmer, etc) with products or services that are of high quality, in the appropriate quantity, and in a timely fashion.
- Grow in effectiveness as a team so that team members are personally challenged and the team increases its ability to successfully take on greater challenges.
- Contribute to the learning and well-being of individual members so they can take pride in their contributions and have a sense of self worth.

Factors affecting the success of working teams.

What are the factors that have an impact on how well a group of individuals can be brought together to form a highly functional, productive, enduring team? The following model identifies three key factors internal to the team: compelling direction, enabling structure and real teams. Externally, it is valuable to have a supportive culture that encourages teamwork with strong leadership and coaching skills. We will focus on the three internal factors.

1. Compelling direction that is challenging, clear and consequential.

- Challenging tasks encourage new learning and counters boredom, loafing and rote activities that diminish team performance and member’s interest in staying with the team. It stimulates motivation.
- Clear goals and direction for the team minimizes anxiety and frustration. It orients all members to the overall strategy and purpose.
- Consequential direction provides meaning and purpose for your team members, clients and for the practice. It is difficult to sustain interest and energy in a team if there is no apparent purpose for one’s efforts.
2. Enabling structure to facilitate the way a team's efforts generate the service or product that it desires. An enabling structure should give consideration to the task design, team norms and team composition.

Consider the design of the task that the team is to work on. The manner in which the task is constructed will determine the experience that team members have individually and collectively.

- Team members find tasks meaningful if the task requires the use of a variety of skills, if team members individually and collectively are clear about the task to be done, and if the task has individual and collective significance.

- The task should also entail a degree of autonomy for members to contribute in their own way so that every team member can experience their own sense of responsibility and pride taken in their contribution. Visible and/or reportable results of the team's efforts are important to maintain motivation. Seeing that something has been accomplished, changed or observed as a result of one's efforts is rewarding.

Consider the norms that the team establishes and maintains for itself. Norms are the behavioural expectations of a group. Every team has them. These can be established consciously but more often than not, they happen at a subconscious level. There are three essential norms that contribute to the success of teams:

- Continual scanning of the environment. Effective teams are not isolated within their own world. Members are observant and responsive to changes in their environment. For example, your patients and clients will be better served if your clinic team can address their needs with the latest in medical protocols, technology and most widely accepted communication channels. Effective teams will regularly scan their environment and invent new or adjust current approaches or protocols in response to changes in their surroundings.

- Clarity about what one must always do.

- Clarity about what one must never do.

Consider the composition of the team itself.

- **Size**: obviously a team has to consist of at least two members. Interestingly, beyond six members, membership becomes less appealing and often there is a decline in team effectiveness. Research indicates that the highest satisfaction ratings are associated with teams of four to five members – think of barbershop quartettes, string quartettes!

- **Diversity**: diversity is a large contributor to the development of successful teams. The diversity or perspectives that arise from differences in gender, nationality, training, age, experience, skills and talents can provide a rich source of ideas and approaches. Ideally, skillful management of diversity would create a comfort level for the differences while pushing the limits of current thoughts and direction. Specific strategies and skills required to manage diversity are presented in the "Managing Diversity" seminar within this workshop.

- **Skills**: every member of the team must have the skills and knowledge to contribute to the task. These include both task related skills and interpersonal skills. Members can be criticized for incompetence or social loafing if they do not possess adequate training or poor communication abilities. Dissatisfaction, loss of team cohesion and efficacy can result.

**Assessing team effectiveness**

By keeping the objectives and characteristics of successful teams in mind, an overview of team effectiveness can be obtained by using considering the ‘gains and losses’ of the team process in the following areas:

- **Team Effort**
  Is there social loafing on the team or a shared commitment to the task?

- **Performance strategy**
  Do the performance strategies of the team emphasize mindless repetition or innovation?

- **Knowledge and skill**
  Does the team have the appropriate knowledge and skill to accomplish group objectives? Is the weighting and balance between knowledge and skills appropriate?
Communicating on teams

Teams can be viewed as problem solving units where a vast amount of information needs to be shared between members. In the case of veterinary practices, much of the team’s activity is problem solving to find ways to deliver the best in client service and exceptional patient care for each individual case. The team is required to exchange information, process the information and come to a decision. Arriving at the most effective solution will depend on group’s ability maintain a common set of communication principles:

• respect one another
• understand one another by making an effort to appreciate other viewpoints
• be curious about one another in an effort to thoroughly understand the feelings and perspective of others
• honor the need for authenticity of information and expressions of feelings

Another way to depict the importance of common principles in team communication is the following model:

The underlying premise is that team communication is founded on two dimensions: the degree to which we respect information, thoughts, feelings, values; and the degree to which we respect the other members of our team. The degree of respect in each dimension can range from high to low and reflects an attitude towards the project, task and people involved. Attitudes have an impact on the way people behave and communicate which in turn, can affect the problem solving capabilities of a team and the quality of the decisions that are made.

When we have a high degree of respect for the information as well as the people on our team (the widest part of the triangle), we feel comfortable being transparent with the thoughts and feelings that we share with colleagues. It feels safe to ask and share information with each other in the spirit of honest inquiry and curiosity. More information gets exchanged here than in any other area. Because there is more information and it is the best information available, there is a greater likelihood that the best decisions will be made.

As our degree of respect decreases for the information and ideas presented by others (moving down the triangle), there is a tendency to move into a persuasion mode in an effort to sell our own ideas. It becomes beneficial to use only information that will help to build a case for our own suggestions. There is an attempt to control the information and the outcome. Others may turn to finding issues and objections with your views.

As the level of respect diminishes further, arguments and defensive behavior appear. Comments can start to become personal which triggers defensive positions and arguments. The information that is shared is based on defending one’s self and one’s belief with minimal efforts to evaluate other views. The common result is minimal problem solving and little in the way of agreement or a resolution to the problem.

When there is minimal to no respect for the information being presented or for others in the group, open problem solving does not exist. Our mission is to defeat the other person at all costs. Volume, words, looks, and language are now employed to get the other person to submit. The response to such behavior is either to fight back or to take flight by physically leaving or withdrawing from the discussion.

In summary, effective teams are found to have many common elements:

• Clearly defined tasks, roles, authority and boundaries
• Challenging, clear and consequential goals, projects and tasks
• Supportive environment to provide leadership and coaching
• Adherence to a common set of norms established by the group
• Respect for the diversity within the group
• Skillful, knowledgeable members
• Communication principles based on respect for each other

References


* The information, powerpoint presentation and notes for this seminar were adapted from the Bayer Animal Healthcare Communication Project Module 1, “Creating Teams That Work” with permission from the Institute of Healthcare Communication.
“Which Animal Are You?”
Understanding Behaviour Styles

Jayne Takahashi, BSc, DVM, MBA

Behaviour styles refer to what we do and how we do it. Everyone tends to have a preferred behaviour style, although this can be one style at work and another style in your personal life. This is NOT about personality nor is there one style that is better than another. In fact, we often need to adapt our style to suit the situation or person we are interacting with.

Behaviour styles are based on how you like to obtain your information (your focus) and how you tend to make decisions (your pace):

- **FOCUS** – like to work with facts or like to work with people
- **PACE** – you tend to make quick decisions or are deliberate in your decision-making process

**Behaviour Styles GRID**
(modified from Mentor International Sales Management and Training)
FOUR BEHAVIOUR STYLES:

ANALYTICAL or “Owls”
PACE: deliberate, reviews all information/data before making a decision
FOCUS: facts, data, technical information, details
Tend to be: systematic, reflective, organized, detailed, may appear too cautious

DRIVERS or “Lions”
PACE: make quick decisions, do not like to ‘waste’ time
FOCUS: facts, data, technical information, details
Tend to be: results/goal oriented, competitive, driven, impatient, decisive

AMIABLES or “Retrievers”
PACE: deliberate, likes to review all information, prefer to take their time
FOCUS: people, like to obtain information through social interaction
Tend to be: friendly, warm, affectionate, good listeners, team players, slower paced

EXPRESSIVES or “Dolphins”
PACE: make quick decisions, based on intuition versus facts
FOCUS: people, like to obtain information through social interaction
Tend to be: fast paced, spontaneous, talkative, may be impatient, inspirational

Understanding your own preferred behaviour style and learning to identify your client’s behaviour style can help you ‘connect’ with and relate to your clients. This can improve the way you can deliver information to each client.

Successful communication is the ability to communicate with someone else based on the other person’s comfort zone and behaviour style. You may need to temporarily tailor your behaviour style to work with the other person. This does NOT mean conforming to another person’s point of view or changing yourself. It simply means providing information in a manner that suits the behaviour style of the other person.

TIPS TO COMMUNICATE WITH EACH STYLE

ANALYTICAL or “Owls”
Do:
- be organized, business-like, precise
- provide detailed information, evidence, pros and cons

Don’t:
- be disorganized, messy, too casual or informal
- rush for a decision
- use opinion as evidence

DRIVERS or “Lions”
Do:
- be clear, specific, brief
- stick to business,
- agree or disagree with facts, not the person

Don’t
- waste their time,
- be disorganized or messy
- try to build a personal relationship
- let them have ‘control’ by providing options
AMIABLES or “Retrievers”

Do:
- be informal, take time to socialize, start with a personal comment, provide data and facts

Don’t:
- rush into business or rush decision-making
- be abrupt or vague
- don’t make a decision for them
- debate facts and figures

EXPRESSIVES or “Dolphins”

Do:
- take time to build a relationship
- be fast moving, socialize
- talk about big concepts/ideas/big picture
- ask for their opinion

Don’t:
- present a lot of facts and figures
- be cold or aloof
- press for solutions

Philosophy of a good communicator

Assume 100% of the responsibility for understanding what the other person means
Assume 100% of the responsibility for making sure the other person understands you
“That’s easy for you to say”  
Communication within the healthcare team

Jayne Takahashi, BSc, DVM, MBA

The benefits of effective communication within the clinic team can have a significant impact on team satisfaction, the client’s experience and most importantly, patient care and health outcome. This has been documented in both the human healthcare profession1,2,3 and veterinary profession4,5,6. In addition, our veterinary associations will tell us that poor communication is often at the root of most client complaints submitted to our disciplinary committees.

What factors affect team communication?

The Institute for Healthcare Communication (IHC – www.healthcarecom.org) reports the following as the most common factors affecting communication based on the results of focus groups with veterinarians, veterinary technicians and receptionists, as well as surveys of veterinary practitioners and a review of the literature:

a) Gender differences: women were viewed, in many instances, to be strong listeners and more comfortable expressing compassion for their co-workers than their male colleagues. In contrast, the men were thought to be more willing to make the difficult decisions when it comes to staff issues.

b) Power differentials: there is reluctance to confront colleagues, express ideas or disagree with colleagues as a result of one’s position within the practice. A difference in power can be perceived based on employer/employee status; owner/associate relationships and veterinarian/ATH/receptionist roles.

c) Generational differences: generalizations about the attitudes, perceptions and behaviours of generations born in specific periods have been widely studied and publicized.

» “Baby boomers” (1946 – 1964) have been described by some as “overachievers” or even “workaholics” due to a highly competitive workplace.

» “Generation X-ers” (1964 - 1981) are computer savvy and may foster a strong interest in work-life balance to counter the workaholic nature of their parents.

» “Generation Y or Generation Next” (1974 – 1980) have been influenced by the technology of the internet and are seeking an independent and innovative work style.

» These differences in style of work and attitudes can present potential communication challenges and opportunities.

d) Experience: this can refer to differences in work experience as well as life experience.

e) Communication styles: each of us has a preference for the way we like to receive information, process information and share information. A common root of miscommunication is a lack of awareness for your own style and the different style of others. There are a number of models and instruments, such as the Meyers-Briggs Style Indicator, to help us recognize these differences. A different model based on Behaviour Styles was discussed in the “Which Animal Are You?” seminar within this workshop.

Essential Components for Successful Teams

Patrick Lencioni of the Table Group Company (www.tablegroup.com) developed a model for successful teams. The IHC has adapted this model for veterinary practice. There are five essential components in developing and maintaining a successful team. This seminar will focus on the importance of trust within the clinic team and the role of constructive conflict as a foundation for successful teams.

Trust

Before team members will trust in each other they must first all agree to a few common beliefs held by all in the practice. These are often referred to as Core Values and represent the fundamental principles that determine our commitment to each other. Examples may include: a) respect others and treat them as you wish to be treated; b) be willing to teach others; c) give others the benefit of the doubt, etc.
Highly effective teams tend to:

- Work together with a “shared mental model”
- Have clear roles and responsibilities
- Engage in regular feedback (two way dialogue)
- Develop a strong collective vision for the practice

A mental model is a mental vision or picture of the relevant facts and relationships that define an event, situation or issue. Within a veterinary practice, it is critical that everyone is “on the same page” or shares a common mental model of the priorities of the practice, the expectations of every team member, the level of patient care that is provided, the practice’s core values and the awareness of differences that can occur in perceptions and understandings.

As with communication in any and all situations, the use of four core communication skills will enable us to obtain a common mental image of a discussion, event or issue and to avoid incorrect assumptions and potential errors. The core communication skills are:

a) Open-ended questions
b) Reflective listening
c) The use of empathy statements
d) Observation of non-verbal cues

A useful framework for sharing information with team members for the purpose of arriving at a common shared mental model is an adaptation of a familiar clinical tool known as “SOAP.” This clinical tool has been modified in its application to help facilitate differences in information. In this case, SOAP represents:

- Subjective – What’s the background and context of current information?
- Objective – What do we know for certain?
- Assessment – What is our assessment of the problem(s)?
- Plan - What is recommended?

Constructive Conflict

Within any team, no matter how cohesive and effective the team is, conflict is inevitable. Conflict should not be viewed as negative as it affords the opportunity to appreciate the differences in approaches, thoughts and experience. Progress and healthy change often result. The manner in which the conflict is addressed and resolved will influence the impact of the conflict on the team.

When handled poorly, conflict can divide the team, interfere with patient care and erode team morale. If conflict is ignored, team enthusiasm and initiative can dwindle. For many, conflict causes a great deal of personal discomfort and is deemed best ignored than addressed. This reluctance to manage conflict can result in artificial harmony within the practice with many unresolved issues brewing under the surface.

A starting point for managing conflict is to change the perspective that it is a negative occurrence. In contrast, constructive conflict enables healthy debate and problem-solving; can produce the best solutions in the shortest time; and stimulates new thinking that can result in progress and growth.

There are two types of conflict:

a) Information conflict: this involves differing views, ideas, and opinions. We have already discussed how our SOAP framework is beneficial for managing differences in information.

b) Personal conflict: this may stem from interpersonal compatibility and is not usually task-related. Tension, annoyance, and animosity are common consequences that can obstruct the completion of team tasks.

One strategy to help manage interpersonal conflict is the DESC model which is a mnemonic for:

- Describe the specific situation
- Express your concerns about the action
- Suggest other alternatives
- Consequences should be shared
Before having this discussion, there are some key considerations to keep in mind.

- The discussion should be timely and soon after the conflict occurred
- Work to a win-win solution
- Use “I” rather than “you” statements to minimize defensiveness and increase accountability
- Focus on what is right, not who is right
- Choose a private location for this discussion
- Limit to ideas and concepts - not personal
- Describe vs. evaluate

References


* The information, powerpoint presentation and notes for this seminar were adapted from the Bayer Animal Healthcare Communication Project Module 12, “Easy For You To Say” with permission from the Institute of Healthcare Communication.
Overcoming Common Communication Barriers

Jayne Takahashi, BSc, DVM, MBA

Great communication is not always easy. The message that we intended to relay is not always received with the same meaning. This is natural and normal. Interpersonal communication is subject to many common barriers and sources of miscommunication. By recognizing these barriers, we are better able to improve on our communication skills with family, friends, colleagues, employees and most of all, with our clients. While this discussion will focus on removing the barriers within a veterinary practice, these principles can apply to all realms of interpersonal communication.

Common Communication Barriers

- “Medical –ese.” The use of jargon and technical terminology that is not understood or is misunderstood by clients is a common occurrence in veterinary medicine.
  - Watch for non-verbal cues that suggest confusion or anxiety.
  - Use open-ended inquiry to assess the level of understanding.
  - Ask clients to re-iterate their understanding of your information.
  - Incorporate diagrams and other visual aids in your explanations.

- Choice of communication channel. Select the appropriate channel of communication for each client and situation.
  - Verbal communication. The majority of our communication is done verbally. It is easy and natural to do, however, conversations are often remembered or interpreted differently by different participants.
    - Important details should be provided in writing.
    - Use active listening skills to paraphrase your understanding of client’s viewpoints and concerns.
    - Use open-ended inquiry to ensure that you client has an accurate understanding of your information.
  - Non-verbal communication. Our body language, facial expressions, gestures, and demeanor can reinforce our words or contradict our verbal message. Non-verbal cues are more accurate reflections of a person’s true feelings. When there is a disagreement between the verbal and non-verbal message, people will believe the non-verbal message.
    - Ensure that your body language is consistent with your verbal message.
    - Watch the non-verbal cues of your client to better assess their true feelings and attitude to your information.
    - Wherever possible, try to remain at the same level as your client to maintain eye contact
    - ‘Mirroring’ your client’s body language and demeanor can put people at ease and is one way of demonstrating empathy with your client
  - Written communication. Although this form of communication is more permanent and independent of personal biases, it can still be ambiguous and subject to misinterpretation if it is badly phrased.
    - Clear and thorough documentation of client discussions, medical treatments and plans in patient medical files are essential. This is your best defense against disciplinary suits/actions.
    - Email and text messaging should not replace personal conversations as misinterpretation of the intentions is commonplace.
• Environmental barriers. These include factors such as noise, temperature, air quality, location, and the immediate surroundings (cramped/spacious, tidy/messy, etc.). Each of these can affect a person’s ability to concentrate, communicate or listen. It can also affect enthusiasm, interest, motivation.

  » The first impression of your practice and the level of medicine that will be provided is often based on what the client sees, hears and smells as they walk through the front door. Walk through your front door at least once a month to see how you would rate your practice.

  » Assess your environment for professional appearance, cleanliness, comfort and safety for both patients and clients.

  » Minimize distractions during conversations

• Organizational barriers. Position and status within an organization will affect how individuals react to each other. Associates vs. owners; employer vs. employee; veterinarian vs. AHT are examples of organizational barriers.

  » Put yourself in the position of the other person as you assess the situation.

• Perception. Everyone has multiple filters through which they process information based on heredity, life experiences, education, socio-economic differences, etc. As a result, the message can be viewed differently based on our own perceptual lenses. This gives way to viewing people on the basis of stereotypes, projecting our own viewpoints on others instead of listening to the thoughts of others and hearing only what we wish to hear.

  » Remain curious about other people.

  » Use open-ended inquiry to discover the viewpoints of others.

  » Avoid making judgments or assumptions.

  » Practice active listening skills.

  » Appreciate and understand differences in behavior styles. Modify the delivery of your information based on how the other person prefers to receive information (Refer to “What Animal Are You?” presentation in this workshop).

  » Remain open-minded and curious in your interactions. Listen to understand the other person.

• Culture. A person’s culture, background and attendant bias and prejudices can affect their reception and interpretation of a message and interfere with the communication process.

  » Identify your own attitudes, prejudices and degree of personal ethnocentrism.

  » Take time to listen to the other person.

  » Don’t jump to conclusions and make assumptions as the choice of words and non-verbal cues often differ between cultures.

  » Encourage feedback and ask questions to determine the commonalities that can form an agreed upon approach to patient care.

• Personal level of stress. Behaviour and attitude can change drastically when a person is under stress. Stress can distort the message, heighten defensiveness and change the frame of mind.

  » If you are aware that a person is undergoing additional stresses, it is helpful to acknowledge the difficulty of the time for this person.

  » Determine the person’s view of the situation or issue before moving into your side of the discussion.

• Language. Dialects or a foreign language are obvious sources of potential miscommunication.

  » Seek assistance from friends or family that can facilitate the discussion.

  » Use drawings, illustrations and other visual aids as part of your explanation.

  » Provide important information in written format.

• Attention span. Everyone has a certain amount of time when their mind is most alert. After this time span, the mind loses focus and does not take in new information.

  » Watch for signs of disengaging from the conversation.

  » Engage the person by asking questions.

• Emotions. Emotions can block both the reception of a message as well as the processing of information. It is difficult to think logically when strong emotions are present.

  » Recognize when your own emotions are coming into play and find a strategy that helps you to stay with rational thought (eg. note-taking).
» Recognize when the other person’s emotions are interfering with his/her ability to process information or to manage the discussion in a rational manner.

» Use empathic statements to acknowledge the other person’s emotions.

» Adjust your delivery of the information that best suits the situation.

Absence of feedback. Feedback lets the sender know that their message has been received. Better yet, feedback can also provide specific information as to how the message is perceived. Without this information, communication becomes a monologue rather than a two way means of sharing ideas.

» Paraphrase the message to acknowledge receipt of the information as well as to confirm a correct understanding.

» Learn to give and receive feedback in a supportive manner.

Information overload. With the time pressures of clinic appointments schedules, there is a tendency to compress too much information into a short period of time. Similarly, too much information may be offered in situations where the client is unable to cope with the information — euthanasia, delivery of bad news or stresses in their personal lives.

» Watch for signs of anxiety, confusion, disengaging from the conversation.

» Chunk the information and check for the level of understanding.

» Ask the client what type of information they prefer to receive.

Differences in learning styles. If people do not receive information in a format that they naturally prefer, they tend to switch off.

» “Why?” people want all the reasons for doing something.

» “What?” people want all the facts about it.

» “How?” people want only the information they need to get it done.

» “What if?” people are more interested in the consequences of doing it.

Listening. One of the most powerful communication skills that we can develop is the ability to actively listen. Active listening involves:

» Listen to the message, paying attention to non-verbal cues.

» Paraphrase or summarize key facts.

» Use reflective statements.

» Ask if your understanding is correct.

» Clarify the message, if necessary.

10 tips for effective listening

1. Stop talking. You cannot listen when you are talking.

2. Put the person at ease. Encourage the person to talk by creating a permissive, safe environment. Use body language and non-verbal cues to indicate your interest and verbally express your appreciation for the discussion.

3. Show the person that you want to listen. Look and act interested. Remain curious with a willingness to understand versus oppose their views. Remove distractions and focused on the discussion. Do not doodle, shuffle papers etc.

4. Be sensitive to the speakers views. View the discussion from the other person’s perspective. Use empathic statements to acknowledge your understanding.

5. Use clear and direct language suitable for the other person. Select the appropriate channel or materials for communication. If you are not sure, ask the speaker for their preference.

6. Do not interrupt the other person. Allow the other person to complete their thoughts, do not complete there sentences.

7. Avoid being judgmental. Keep an open mind and a willingness to give the other person the benefit of the doubt. Listen without an agenda or expectations.

8. Monitor and control your emotions. If necessary, ask for time to think about the discussion before expressing your views. Emotions can interfere with clear understanding of the message.

9. Learn to give and receive feedback skillfully and constructively. Avoid the win-lose attitude when presenting opposing ideas. Provide feedback in a supportive manner to avoid an argumentative tone that may people on the defensive.

10. Ask open-ended, probing questions. This shows that you are interested and helps to share a common understanding.
Managing complaints

- “TEXAS” model
- Thank the person for bringing their complaint to your attention
- Explanation – using open-ended inquiry, ask for an explanation of what has taken place
- Ask for clarification where necessary or obtain Acknowledgement from the speaker that you understanding of the situation is accurate
- Solution – work with the person to find a solution to the situation

Consider Proper Meeting Formats

Meetings can be one of the most useful forms of communication on any team. Unfortunately, these are often viewed as unimportant and a wasteful use of time. Effective meetings require an effective chairperson to lead the discussions and thoughtful preparation as to the agenda and objectives of the meeting. A discussion on how to organize a staff meeting can be found in the “Managing Diversity” seminar within this workshop.

Well organized and effectively chaired meetings can address any number of situations. Meetings can take on a variety of formats from informal to formal. Here are some considerations when choosing the type of meeting for a given situation.

- One-on-ones. Informal meetings between the team leader and each individual team member are ideal for motivating people, catching up on progress, and ensuring that any problems are identified and dealt with promptly.
- Full team meetings. These are scheduled on a regular basis so that all team members are updated on each area or task. Team meetings are useful for identifying and addressing gaps or slippage in schedules. These sessions ensure that all parts of the “big picture” come together.
- Presentations. These are more formal presentations that are useful when the same message is to be shared with the larger group as a whole. The formality of this presentation may also give added importance and emphasis to the message. Important information to key stakeholders such as project sponsors or shareholders is often shared in a more formal presentation setting.

Recognizing the common barriers to communication is the first step to minimizing their impact. By consciously incorporating the four core communication skills (open-ended inquiry, reflective listening, empathic statements and non-verbal communication) in interpersonal communication, many of the sources of miscommunication can be avoided. Continual practice of these skills will help to develop confidence to communicate effectively in any situation.
"Strangers In Crisis, Partners In Care"
Sharing difficult news

Jayne Takahashi, BSc, DVM, MBA

Communicating with clients in an emergency situation presents a number of challenges. The interaction is filled with emotion, time is limited to develop a trusting relationship, rapid decision-making needs to take place, and in most cases, this is your first meeting with the pet owner. The healthcare professional and the client are strangers to each other, yet partners in the care of the pet.

Let’s take a look at this crisis situation from the pet owner and their family’s perspective. They have entered an emergency care centre and feel like strangers in a strange land. There is a desperate need for information about their pet, the process, what happens next, who is in charge, etc. A wide range of intense emotions are being experienced and often, expressed – fear, anger, anxiety, grief, shame, guilt. This can result in interpersonal conflict between the pet owner and the veterinary team and/or the pet owner and their family. Long wait times in the clinic can exacerbate all of these responses.

From a veterinary team’s perspective, there is a high need for efficient information sharing, management of time pressures, tough decision-making and all this is done in emotionally charged situations. Personal emotions are suppressed in order to tend to the needs of the patient.

Given these two perspectives, a number of communication barriers can arise between the veterinary team and the client:

- Absence of a pre-existing relationship
- Environmental factors (time, lack of privacy, interruptions)
- Stressors on client – guilt, time, worry, financial concerns
- Stressors on veterinary healthcare team
- Cultural and language issues
- Unrealistic expectations
- Mandated reporting / informing issues
- Fee-for-service discussions

Ptacek1 reports the most common fears held by clinicians when delivering bad news include:

- Being blamed
- Lack of confidence and/or skills in delivering the news
- Client’s reaction
- Appropriate and extent of sharing emotion
- Not knowing all the answers
- Personal reaction
- Uncertainty about client’s expectations
- Destroying client’s hopes
- Embarrassment

During the initial moments with the client, it is beneficial to establish rapport, let the client know what your role is in the situation (“I will be one of the veterinarians tending to Toby”), and determine the client’s perspectives on the situation.

Even in emergency situations, there are techniques that will help to establish rapport with clients in a short period of time. Simply asking the client “how are you holding up?” can let them know that you are concerned for them as well as their pet. The four core communication can help develop healthy interactions with clients: open-ended questions, empathy statements, reflective listening and observing non-verbal cues.

Open-ended inquiry: “Tell me more about…”
- “What happened next…”
- “What have you noticed…”

Reflective listening
- Sounds like you’re unsure whether to go ahead with these tests right now.”

Empathy
- I can see this has been a painful decision for you and your daughter.”
Asking an open-ended question or making an inquiry to learn more about the client’s perspective on their visit to the emergency facility is a strategy to discover expectations – “Tell me what Digger has been doing that caused you to bring him here today.”

In many situations, another team member will have taken a history so it is important to acknowledge that you realize the client is telling their story more than once. You can include this in your open-ended question – “Sharon indicated that Toby was attacked by your neighbour’s dog. That had to be a scary moment for you. What happened?”

If the pet was brought in by someone other than the pet owner, you will need to assess the owner’s current understanding of the situation – “What is your understanding of why Millie was admitted?”

Pet owners want to know who is caring for their pet and what happens next. This is the time to be clear about your role and to orient your client as to the process of the evaluation or care of their pet. Tell them what you will be doing and what will happen next.

For example, “I will be one of the doctors who will be treating Ruby while she’s here at our hospital. I would like to spend a short time reviewing what happened and talk about the tests that we will be thinking about running for Ruby. I’ve examined Ruby and would like your permission to run some blood tests which I’ll explain. Then I’ll discuss the cost estimates for this.”

The delivery of difficult news is one of the most challenging aspects of veterinary care. For the purposes of this discussion, the definition of ‘bad news’ is any information likely to be perceived by the client as distressful, unwanted, and/or unexpected. Bad news can take on many forms:

- Unexpected death, i.e., anesthetic death
- Unexpected surgical intervention
- Chronic illness (renal failure, diabetes, cushing’s disease)
- Terminal illness, i.e., cancer
- Treatment failure
- Disease recurrence
- Extensive and/or serious injury, i.e., hit by car
- Perceived expensive treatment
- Problem perceived as offensive or embarrassing to client (e.g., fleas, ringworm, weight issues, etc.)

The underlying skills and approach to effectively work through interactions in an emergency setting or as you are delivering difficult news are essentially the same as in other interactions. Practicing the skills on a daily basis certainly increase your confidence and comfort when in a high pressure scenario. The first step is to learn to prepare yourself for these discussions. Gather the necessary information from the client related to their knowledge, expectations and degree of readiness to hear this type of information. Again, open-ended inquiry can obtain this type of information – “How do you feel Toby is doing right now?”

Prepare your news in a manner that best suits the client’s needs and abilities. Will they require additional written information or instructions? Would it be helpful to have a family member or friend to support them during the discussion or to help with the understanding of your explanation. Give consideration to the amount of information that you are delivering and the enormity of the news. Will it be so shocking that your client will be unlikely to remember the relevant points of the conversation? Perhaps he/she will require additional time for the news to “sink in.”

After you have shared the news, it is important to tend to the pet owner’s immediate emotional and informational needs. Then you can assess their understanding of your message – “I know that I am sharing some unexpected news. What are your thoughts right now?” Once you have this understanding, you can develop a plan or strategy for the next steps with your client’s input.

Reports from the human medicine literature suggest that although full disclosure of bad news may have a negative emotional impact in the short run, most individuals will adjust well in the long run. Uncertainty is a major cause of emotional distress for individuals — relief from uncertainty can, in itself, be therapeutic. Although, we cannot change the change the news itself, we can certainly have the potential to shape the experience for the client in a positive manner or we can add to their confusion and anxiety. A positive interaction can strengthen the veterinarian-client relationship and foster collaboration on the healthcare plan for the pet.
Here is one model for delivering difficult news that is based on a protocol published by medical oncologist, Robert Buckman3.

### 5 STEP PROTOCOL

**PREPARE**

**ASSESS CLIENT KNOWLEDGE & PREFERENCES**

**SHARE THE INFORMATION** (Bad News)

**ATTEND TO FEELINGS**

**FOLLOW-UP AND PLANNING**

1. **Preparation for the discussion**
   As you prepare, consider the following:
   - Ensure that you have accurate information. Bad news should not be based on preliminary results. It is better to wait for the information to be confirmed. Once your message is clear and confirmed, decide how this can be shared in understandable language.
   - Create an environment that is conducive to an open, attentive discussion with clients. Schedule ample time for the appointment and in a private space with seating, if possible. One person should be responsible for sharing the news, ideally the veterinarian who will have ongoing contact with the patient and client. The client should be offered the option to having family or other significant individuals present for emotional support. Have tissues readily available.
   - Plan for privacy. Only include veterinary team members who will be actively involved in patient care. Having more than one member of the team may be overwhelming for the client. Turn pagers and cell phones to vibrate. Instruct the healthcare team to not interrupt this appointment.

2. **Assessment of client knowledge and preferences**
   Before presenting the information that you have prepared, it is critical to assess your client’s current level of knowledge, understanding and perceptions about their pet’s condition. This can be done through the use of active listening skills, open-ended inquiry and the observation of non-verbal body cues.
   - Pay particular attention to the client’s vocabulary and the words that are used as well as, avoided.
   - Watch for body language that suggests confusion, fear, anxiety.
   - Assess how much or to what degree the information will be given. Check with the client as to the level of information they want to know – “Would you prefer to focus on the big picture or would you prefer a more detailed explanation?”
   - Through your discussion, ascertain and address unrealistic expectations, wishful thinking about the animal’s illness, or important but adverse details of the illness that are being overlooked.

3. **Sharing the news**
   Recognize that your client will need time to process the difficult news that you are about to share with him/her. The process will likely take more than one conversation as the pet owner works through the information. The nature and the amount of information that a client requests may change over time as well, so it is a good practice to continue to check on how much or the type of information your client requires.
   - Begin with a forewarning statement to prepare your client for difficult news – “I have some difficult news to share with you about the results of Misty’s blood test.”
   - Always use the pet’s name.
   - Start at the level of comprehension and vocabulary of the client.
• Pay careful attention to your own body position and nonverbal messages. It is best to be positioned at a similar eye level with your client so that eye contact can be maintained, preferably seated.

• Avoid the use of “medical-ese,” jargon and technical terminology. Continually check in with your client as to their level of understanding and watch for non-verbal cues that signal confusion. This can occur even with a client who you know has medical background. Emotional distress can interfere with information uptake.

• Avoid euphemisms such as “tumor”, “growth,” or “illness.” If the animal has cancer, then use this word. To do otherwise can create ambiguity, impair understanding and adversely influence decisions.

• Provide the information in manageable chunks. Check in periodically for the client’s understanding. Remember that your client is hearing this information for the very first time and it helps to keep the pace of your speech slower than you usually speak. They are absorbing a lot of medical information and their emotional reactions often make it difficult to process information.

• Speaking at a lower tone will serve to help reduce anxiety.

• When the news is particularly distressing, allow your client to absorb and comprehend the severity and finality of the news. The client may be silent or may question whether the news can possibly be true. This type of reaction and displays of shock or periods of silence can be a normal course of coping.

4. Attend to feelings

There is a wide range of emotional reactions to difficult news and the reaction is often a reflection of the bond between the client and the pet. After sharing the news, you need to be prepared to respond to the client’s emotions.

• It helps to look at the situation from the client’s perspective and to listen for their specific concerns. For example, as a veterinarian, you may view the prognosis for pancreatitis in a patient very good, however, the pet’s owner can only think about the neighbor’s dog that died from pancreatitis. Open-ended inquiry is helpful to obtain your client’s perspective.

• Provide time and space for the client to react. Accept and validate your client’s feelings and concerns by letting them know it is quite normal to feel this way - “It’s understandable for you to be shocked and upset about what happened to Muffin” or, “Many families have difficulty making these kinds of decisions.”

• Though it may be extremely difficult, even painful, honour and support silence. It is a signal to the client and family that you are willing to be patient and be present with them.

• Asking the family what they need and offering to be helpful are additional strategies that convey both empathy and compassion. In the event of the death of the animal, assess the client’s resources for support and readiness to leave the clinic on their own facility unaccompanied.

• Offer statements of empathy to match the emotion.
  » Shock: “Hearing these results about Sadie is clearly a shock for you.”
  » Sadness: “I wish I had different news to share with you. I can see you’re really hurting.”
  » Doubt: “I’m sure it’s hard to imagine how this can possibly be true.”
  » Anger: “I understand you’re angry about this turn of events.”

The manner in which you tend to your feelings and the feelings of your client will not only help your client through a difficult time, but it can influence your client’s beliefs and attitudes about the overall experience and your veterinary team. Research on the impact of difficult news delivered to parents regarding their child’s health showed that the parents recalled (years later) and appreciated the confidence, concern, and caring attitude of the physician. They also remembered that they were allowed time to talk and ask questions at the time.

5. Follow-up and planning

After the difficult news is shared, it is important to once again assess the client’s level of understanding of what has been discussed thus far. This is an opportunity to hear from the client about their concerns and to fill in gaps in understanding or clarify misinformation.

• Assess the client’s understanding - “We’ve covered a lot of ground and I can see this is upsetting. Let’s take a minute and review the information I’ve shared so far to see if it makes sense” or “What other information would be helpful for you at this time?”

• There is a limit to how much difficult news a client can absorb, particularly in one interaction. The veterinarian should keep to the essential pieces of information, answer the client’s questions and address concerns. If appropriate and/or possible, it may be necessary to schedule a follow-up appointment.
• Providing a message of realistic hope and support is important even if the disease or medical situation is too far advanced for curative treatment. Try to reassure the client that you will provide support (medical and non-medical) for as long as needed or if appropriate, to plan to make the patient as comfortable as possible or discuss quality death via euthanasia. Keep in mind that clients who have a clear plan for the future are less likely to feel anxious and uncertain.

• Finally, document the information (including a discharge summary, if applicable) that was given to the client in the medical record. This will ensure that consistent follow up is provided by all of the healthcare team or the patient’s primary care veterinarian.

You cannot change the news itself …

However, the manner in which you present the news can shape the client experience.

REFERENCES


* The information, powerpoint presentation and notes for this seminar were adapted from the Bayer Animal Healthcare Communication Project Module 9, “Strangers in Crisis” with permission from the Institute of Healthcare Communication.
COMPANION ANIMAL
Myths and Misconceptions in Small Animal Anaesthesia

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Although most areas of veterinary medicine have "myths" that are perpetuated over the years, veterinary anesthesia seems to have more than its fair share. Myths typically result when an anesthetized or sedated patient responds in an unexpected or unpredictable way to a drug, and due to a lack of full understanding of all of the differentials that may have caused it, the veterinarian or owner assigns a single cause. The misinformation is subsequently shared by clients, breeders, veterinarians, and clinic staff without anyone stopping to correct the misunderstanding before it is passed on to the next person. Myths may also arise from the prevailing scientific evidence of the time, and are often based on past clinical experiences of the practitioner or owner. Although owners and breeders often have their own beliefs (i.e. “I was told by my breeder that my dog is allergic to acepromazine”) and do not hesitate to share them with us, it is the myths that many veterinarians continue to follow that pertain to the clinical practice of veterinary anesthesia that I will address today.

Veterinary anesthesia has advanced by huge steps over the last 10 years as a result of careful and considerate research, both in the laboratory and in the clinical setting. We now know more that ever about the drugs that we use and their effects on patient physiology and wellbeing. Unfortunately, in most cases, myths are more easily disseminated than is this new knowledge (especially via the internet), and the result is perpetuation of misconceptions and misunderstandings that may, in fact, compromise patient safety and prevent practitioners from offering what is considered to be the current standard of anesthetic care.

In November 2003, Drs. Ann Wagner, Bonnie Wright, and Peter Hellyer of Colorado State University authored an excellent review entitled “Myths and Misconceptions in Small Animal Anesthesia” that was published by The Journal of the American Veterinary Medical Association. In it, they addressed many of the myths and misconceptions that are common to veterinary anesthesia, and in doing so explained how scientific evidence has ‘debunked’ these misunderstandings. This lecture will focus on several of the myths included in that paper, as well as others that I have come across in my practice of anesthesia and pain management over the years.

Myths:

1. Many breeds of dog are sensitive to anesthetics.
2. Preanesthetic medications should not be used because they will delay recovery.
3. Acepromazine is a bad drug and should be avoided.
4. Butorphanol is an effective and long-lasting analgesic.
5. Dexmedetomidine is the same drug as xylazine.
6. Dexmedetomidine is only useful for bad dogs and cats.
7. Induction of anesthesia with gas anesthetics is safer than with injectable anesthetics.
8. Using the oxygen flush valve to fill the rebreathing bag is safe.
9. Pulse oximeters monitor the patient’s ventilation.
10. Animals that take too long to wake up were probably at too high a vaporizer setting.
An Ounce of Prevention: What We Have Learned About Patient Management and Anaesthetic Risk

Matt Read, DVM, DACVA (Diplomate, American College of Veterinary Anesthesiologists)

Should our clients be concerned when their pets have to undergo anesthesia for surgery or other diagnostic procedures? Is anesthesia a high risk endeavor for small animal patients, or are clients generally anxious about anesthesia without good reason?

In people, the risk of death associated with anesthesia itself is believed to be approximately 1:20,000, which is very low compared to the risk that veterinary patients face in our practices. The risk of death in small animal patients has been documented to be approximately 1:1,000 patients. However, it is imperative that we remember that to the owner, when a pet dies as a result of sedation or anesthesia, no matter what the literature may recognize as preoperative risk, to those clients it really doesn’t matter how low the odds are. Each of us needs to make every effort to reduce anesthetic risk for each of our patients in whatever way that we can. As with all aspects of medicine, complications will occur with anesthesia. Our responsibility as anesthetists is to prevent those that can be prevented, to be watchful for those that are unexpected, and to be prepared to support our patients in whatever way that they need us to.

One of the most important ways to reduce anesthetic risk is to perform a thorough preoperative assessment of the patient. Anesthesia is not sleep. It is a drug-induced state that results in widespread depression of the body, and it can have effects on all of the body's systems. We know that anesthesia has the potential to compromise the patient's physiology at unpredictable times and in unpredictable ways. Unfortunately, when they occur, anesthetic crises tend to be rapid in onset and devastating in nature. Rarely are there complications of anesthesia that do not require timely intervention by the anesthetist to prevent worsening of the situation and risk of severe disturbances including patient death. Preoperative assessment provides information about the patient that can be used to maximize safety and minimize the potential for complications. The goal of every anesthetic or sedation should be to have an uneventful recovery for that patient. Often the best anesthetic is a boring one.

The effects of general anesthesia are well-known at this point in time. We use anesthesia for the beneficial effects of central nervous system depression; unconsciousness, muscle relaxation, analgesia, and the blunting of autonomic responses and the stress response to stimulation. However, with central nervous system depression come the recognized side effects of anesthesia: cardiovascular depression and respiratory depression. When alterations in these systems occur, patients are at great risk of compromise. Hypotension, arrhythmias, hypercarbia, and hypoxemia can all have profound impacts on blood flow, oxygen delivery, acid-base status, and stress responses, and are the primary causes of patient morbidity and mortality if left untreated. These potential side effects should be anticipated and supportive measures should be available at all times in case they do occur. For example, it should never be a case of monitoring IN CASE hypotension occurs, but rather, monitoring to make sure that WHEN IT occurs, we are watching for it and are prepared to intervene to correct the disturbance before it compromises the patient.

The risk of death associated with anesthesia in people is very, very low compared to the risk in animals. In the 1950's, studies showed that the risk of dying in dogs was approximately 1:90. In the 1980's, a follow-up study in the United Kingdom found the death rate in small animals had decreased to approximately 1:750 dogs and cats. In that study, the authors also documented that if there was recognized disease in the patient, the risk of death increased markedly to about 1:30 or 3%. Recently a very large study was conducted in the United Kingdom to assess the risk of dying in small animals within 48 hours of sedation and anesthesia. The Confidential Enquiry into Perioperative Small Animal Fatalities (CEPSAF) looked at different aspects of anesthesia on the risk of dying in dogs and cats and small exotic species. This study included prospectively collected data from nearly 200,000 patients over a two-year period and the authors followed these patients for 48 hours following their anesthetic or sedation. In this study, the overall mortality rate in small animals undergoing anesthesia was documented to be 1:1,000 for healthy dogs and cats. Sick dogs and cats carried much higher risk, and in this particular study, it was found that in dogs and cats that had clinical signs of disease on presentation, the risk of dying was approximately 1:70. The results of this large study definitively showed us that there is an increased risk of dying associated with 5 different factors: the
degree of clinical disease (ASA Status), age, weight, the urgency of the procedure, and the complexity/duration of the procedure.

**ASA PHYSICAL STATUS**

ASA physical status refers to a classification system that was developed by the American Society of Anesthesiologists. It is a simple 5-point system for scoring the relative physical status of a patient before any sedative or anesthetic drugs are administered. The ASA status takes into account the findings of the patient’s history, physical examination, and any pertinent diagnostic testing that has been done. It does not relate to the risk associated with planned procedure, and it should be assigned PREOPERATIVELY before we learn how the patient actually does under anesthesia.

An ASA Class 1 patient is defined as a normal, healthy patient with no signs of clinically important disease. Based on this assessment, the patient should have no reason to be at increased risk for anesthesia, and as such, should carry the lowest risk of dying under anesthesia.

ASA Class 2 patients have mild systemic compromise. These patients may have signs of compromise, are not considered to be at increased anesthetic risk. Examples might include patients with mild dehydration, azotemia, or overweight body condition (not obesity).

ASA Class 3 patients are those that have recognized systemic diseases but the diseases are not considered to be incapacitating at that time. These patients are not yet on medications for their disease, and are being monitored closely by the veterinarian and the owners to look for any sort of deterioration in health status. Examples might include patients with detectable cardiac disease that is not a threat to life (heart murmurs, arrhythmias that are not being medicated), certain endocrine diseases (stable hypothyroid), regenerative anemia, renal insufficiency, etc.

ASA Class 4 patients are those that have severe systemic conditions that are a constant threat to the patient’s health and well-being. These patients are on medications or receive other therapies to stabilize the disease and prevent further deterioration. Examples might include patients with systemic hypertension, pneumonia, cardiac diseases that require different medications to prevent congestive heart failure or arrhythmias, chronic nonregenerative anemia, chronic renal failure (that receive medications or fluids), etc.

ASA Class 5 patients are those patients that have such severe disease when presented that they are not expected to live 24 hours with or without surgery or diagnostic procedures. Examples would include comatose patients, patients with severe blood loss from uncontrolled hemorrhage, shock (sepsis, GDV), etc.

ASA status may also be assigned with the letter E to denote that the procedure is an emergency. This annotation reminds us that we may not have enough time to fully and ideally stabilize the patient before inducing anesthesia, further increasing the patient’s risk.

The results of the CEP SAF study from the United Kingdom found that ASA 1 and 2 patients carry the same 1:1,000 risk of dying. Increasing ASA status from 2 to 3 raises the risk of dying six times in dogs and three times in cats. Further increasing the ASA Status from 3 to 4 or 5 increases the risk of dying another six-fold in dogs or another threefold in cats. What this tells us is that we should always strive to be able to assess these patients before any anesthetic drugs are given. We know that the anesthetic drugs are going to have more of an effect on the body when the patient has clinical disease. This is because the disease, be it cardiac, hepatic or renal, has the potential to reduce the therapeutic index of the different drugs in terms of how they are circulated through the body and distributed, how they are metabolized and eliminated at the level of the liver and the kidney. Overall, if the drugs hit the patient harder, they may predispose the patient to greater degrees of cardiovascular and respiratory depression. We should always strive to assess risk before any drugs are given. Major associations between health status and anesthetic-related death have been reported in many species (including people, dogs, cats and horses) and all of these studies have found that pre-existing pathology may reduce the safety of the different drugs.

**PATIENT AGE**

Age also has an effect on patient risk and it was found that increasing the patient’s age raises the risk of dying in dogs. The CEP SAF study found that dogs that were greater than 12 years old were eight times more likely to die but that between the ages of 8 to 12 years old, those dogs had the same risk of dying as young adults. Increasing age also increases the risk of dying in cats. 12 years old was found to be the number that we should look for, with cats that were older than 12 years being two times more likely to die than younger animals. With advancing age, patients are likely more prone to the side effects of anesthesia (hypothermia, the depressive effects of the drugs on the cardiovascular or respiratory systems), and are more prone to prolonged recoveries from the reduced abilities of the metabolic functions of the kidney and the liver. Age is independent of ASA status, and should be considered separately from other signs of clinical disease. For example, an otherwise healthy 13 year old dog still carries increased risk because of its age, irrespective of its ASA status.
PATIENT WEIGHT

Patient weight also has an effect on anesthetic risk, as you might expect. Small patients carry a much higher risk of dying. Dogs that were less than 5 kilograms were found to be seven times more likely to die than the other dogs. Cats that were less than 2 kilograms were found to be twelve times more likely to die and if they were overweight and obese (greater than 6 kilograms for cats), they were found to be three times more likely to die. Small patients are more prone to drug overdose, especially if people aren’t weighing them accurately and are just estimating body size based on breed and age. They are also much more prone to hypothermia since they have much smaller body mass to surface area ratios and tend to radiate more heat. As well, they are typically maintained on non-rebreathing breathing circuits, which further predispose any patient to hypothermia and heat loss through the high fresh gas flows. Small patients can also be very difficult to manage perioperatively in terms of being able to safely intubate them, obtaining IV access with catheters, etc. Monitors are also more challenging to place in these small patients which often results in people not watching as closely for the side effects of anesthesia and intervening on the patient’s behalf. When a monitor is alarming, I frequently hear people explain that “he’s just small so the monitor must not be working properly”, instead of trusting the monitor and assessing the patient.

URGENCY OF PROCEDURE

The urgency of the procedure also affects anesthetic risk. There is increased risk of dying when there is no time to stabilize the patient and when the procedure is performed outside of regular office hours. With scheduled procedures serving as the baseline, if a non-scheduled procedure is undertaken, it increases the patient’s risk three-fold and for emergencies done outside of regular hours, the risk of dying is increased nine times. This finding should stress to us that we must take the time to look at the patient before anesthesia, to make every attempt to stabilize them with fluids, with warming, with analgesia, with blood transfusions, etc., prior to anesthesia in order to improve the chances for an uneventful outcome. Just because we are tired or preoccupied, or there is less technical support after hours, we cannot forget to consider all of the issues surrounding emergency anesthesia and take short-cuts with patient care. The fact that you are considering emergency anesthesia should remind you of how high-risk this endeavor is, and you should slow down your thought process to cover all the important considerations of patient care.

PROCEDURAL COMPLEXITY AND DURATION

Finally, the complexity of the procedure has an effect on anesthetic risk whereby the invasiveness and the duration of the procedure both affect risk. In very complex procedures that impose greater stressors on the body’s physiology, patients were found to be three to five times more likely to die since these types of procedures predispose to greater degrees of hyperthermia, fluid losses and physiologic compromise. When we look at the procedure itself, the degree of invasiveness has a great number of effects including the amount of pain that the patient experiences and the amount of edema that results from tissue handling. We also need to take into account the expected body position for the patient for that particular procedure, especially if multiple procedures are going to be undertaken whereby body position is going to be changed along the way. These changes in body position can have profound impacts on ventilation and blood pressure, which can be quite detrimental to the patient if we are not anticipating them in the time period of actually rotating the patient from one position to another.

LABORATORY TESTING

Lab testing is not effective in predicting anesthetic-related complications. Several studies in both people and animals have failed to support the case for routine pre-anesthetic testing in the absence of abnormalities in the patient’s history or physical examination. One recent study in the Netherlands looked at over 1,500 dog anesthetics. In that study, they found that 60% of the dogs that presented to their hospital showed no reason for being at increased ASA anesthetic risk based on their history and physical examination alone. Given those findings, the authors pointed out that 84% of those dogs would not have otherwise required any further blood work following the history and physical examination alone. However, based on the study design, blood work was performed in all of the patients to later allow them to look for potential correlations with any complications that were associated with the patient’s anesthetic. The results found that in only 8% of the dogs, the blood work would have resulted in them altering the patient’s ASA status preoperatively beyond what the history and physical examination would have already assigned to the patient. In only 1% of the patients would surgery have been postponed based on the findings, and that the anesthetic protocol would have been changed in only 0.2% of the dogs based on the blood work changes that were measured. 2% of the dogs in the study had some sort of critical incident during anesthesia and all of those critical incidents were related to the anesthetics themselves and nearly all of them occurred in dogs with so-called “normal” blood work. What this confirms to us is that blood work is not a good predictor of the potential for complications.
under anesthesia and that for many veterinarians and pet owners, we are just getting a false sense of security when blood work is confirmed to be normal. Abnormalities on blood work are usually of little clinical significance to the anesthetic itself, and generally do not prompt changes in the anesthetic management. The conclusions of this study support what many anesthesiologists already believe – the types of complications seen with anesthesia such as arrhythmias, hypotension, hypothermia and hypoventilation are simply not predicted based on blood work. The bottom line is that extensive laboratory testing is not a substitute for taking a good history and performing a thorough physical examination. Altering drugs and implementing methods for patient support will simply not cover up for shortcomings in our preoperative assessment.

In terms of anticipating what sort of patient monitoring and supportive measures we are going to need, we should be able to anticipate the common complications that are inherent with anesthesia. We know that hypotension, hypoventilation and hypothermia are the most common complications seen in domestic species under anesthesia. Certain complications are also specific to particular patients or procedures and may predispose the patient to cardiac arrhythmias, hypoxemia, blood loss and hemorrhage, or difficulty with intubation.

As a profession, we need to change our approach to providing anesthesia to our patients. The first step is to not spend as much time and energy thinking about what drugs and doses we will be using for anesthesia, and more time thinking about what WILL happen to the patient after the drugs are given. Since these studies show us that the drugs themselves have relatively little effect on patient outcome, we must move away from concentrating so much on the particular drugs and the doses, and move towards monitoring for the potential side effects of these drugs once the patient is actually sedated or anaesthetized. Anesthetic drugs rarely result directly in a patient’s death, but our inattention to their physiological well being while they are anaesthetized does. Individualized patient assessment, careful and considerate anesthetic planning and monitoring, and appropriate supportive measures are the areas that we can make the biggest impact on patient outcome.

Don’t just meet your clients’ expectations, exceed them!!

Further reading:


**Think Outside The Bottle: Anaesthetic Considerations for Patients with Disease**

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**CARDIAC DISEASE**

Many veterinarians are presented with the challenge of anesthetizing cardiac patients in their general practices. It is very common to see these types of patients in practice as many animals are born with defects or will develop heart disease over the course of their lifetime. Some are born with malformations such as ventricular septal defects and patent ductus arteriosus. Other heart diseases are more age-related and typically involve endocardiosis of the valve leaflets of the mitral and tricuspid valves that results in insufficiency at these locations. Other patients may develop aberrant arrhythmias over time (often resulting in ventricular arrhythmias), or they may develop arrhythmias relating to conduction such as 2nd or 3rd degree AV blocks.

There are several anesthetic considerations when presented with a patient with cardiac disease. We first need to determine the nature of the disease and whether the disease is congenital or acquired. This is an important differentiation because if the patient has been living with the disease for much of its life, they are typically going to be more compensated than if the disease developed recently. As well, the potential for hemodynamic consequences will differ depending on the disease and its pathophysiology when these considerations are associated with the age of the patient. Another question to ask is whether or not the patient is on any cardiac medications since several of these drugs may interact with the anesthetic agents and may cause further vasodilation and hypotension or have the potential to cause arrhythmias. It is also important to consider the procedure in terms of the nature of the procedure and what body position the patient will be put in to allow for the surgery or medical intervention. Body position can have a profound effect on cardiovascular function in many patients and it is important to anticipate what sort of effect this might have well before the patient is anesthetized. We need to consider what anesthetic drugs are available, and consider the hemodynamic effects of the different anesthetic drugs and how they may be different in a healthy versus an unstable cardiac patient. Much of what we are taught in school is based on the use of anesthetic drugs in healthy patients, so we need to anticipate how ‘routine’ side effects such as hypotension may be manifested in a patient with limited cardiac reserve. Finally, it is important to consider what is available for monitoring the vital signs of the particular patient and how we are going to assess the patient’s well-being and provide treatment or support interventions on their behalf. Cardiovascular function may be compromised during anesthesia with changes in heart rate, blood pressure and cardiac rhythm and in patients that are already compromised with preexisting cardiac disease, these changes may be even more profound.

The main goal of any anesthetic is to try and prevent any substantial changes in cardiac output and oxygen delivery to the body, and this is especially a priority for cardiac patients. Cardiac output is a measure of the flow of blood through the heart and is the product of the heart rate multiplied by the stroke volume (the volume of blood pumped each time the heart contracts). The heart is a pump and it needs to maintain a steady rate and rhythm in order to drive blood flow through the body.

There are many cardiac diseases that veterinary patients are presented with and I like to first separate these into two initial categories; patients with abnormal heart rhythm, and patients with abnormal heart sounds. Heart rhythm relates to abnormalities of electrical conduction through the heart, and heart sounds are related to the sounds that we can hear with a stethoscope that reflect the patterns of blood flow as it flows through the different heart valves. Abnormal heart sounds result from turbulence that is created from anatomical alterations of the mitral, tricuspid, aortic and pulmonic valves. Separating cardiac patients out into one of these two broad groups helps us get started with considering the appropriate anesthetic management that we will later employ.

Arrhythmias should ideally be detected on preoperative physical examination and not once the patient is already sedated or anesthetized, however certain arrhythmias can develop following sedation or anesthesia with specific drugs. On physical exam, arrhythmias may be detected with hands-on methods such as palpating peripheral pulses for irregularities or deficits, or through auscultation of heart sounds with a stethoscope. Certain monitors may also allow us to detect the presence of arrhythmias, including the use of Doppler ultrasonic monitors, pulse oximeters, and ECG’s. Disorders of cardiac rhythm can impact the ability of the heart chambers to fill adequately with blood between beats during diastole and, as a result, can affect stroke volume and cardiac output. Extremes in heart rate (profound bradycardia or sustained tachycardia) can also negatively affect cardiac output. In the case of bradycardia, profound decreases in heart rate can...
compromise cardiac output (since flow is partially dependent on rate), while in the case of tachycardia, there is simply not enough time for adequate ventricular filling during diastole to maintain stroke volume. Remember, the heart muscle itself is perfused with oxygenated blood during diastole/relaxation, so if we induce sustained tachycardia in a patient, we are not only increasing its workload (by increasing how often it needs to contract), but we are also potentially limiting its ability to stay oxygenated. This can be a serious problem in certain patients, especially those with hypertrophied hearts that have already elevated oxygen demands.

In most cardiac patients we need to aim to maintain heart rate within a “normal” range and avoid any extremes of bradycardia or tachycardia. Numerous diseases and drugs can affect cardiac rhythm. For this reason, we need to consider the drug effects in relation to the observed arrhythmia to decide whether it was anticipated or unexpected, and whether or not we need to treat it. In general, in patients with pre-existing tachycardia or ventricular arrhythmias we want to avoid or prevent changes that could further increase heart rate and the oxygen demands of the myocardium. For this reason, it is important to prevent or treat causes of pain and hypotension, and to avoid the routine use of drugs that increase heart rate such as atropine or glycopyrrolate. For those patients that present with bradycarrhythmias, we should plan to avoid situations that will further increase vagal tone and plan to incorporate anticholinergics into our anesthetic protocol to maintain heart rate in a normal range.

We can detect the presence of valvular disease with auscultation of heart sounds. Based on the presence of abnormal heart sounds, we can further categorize a patient with cardiac disease into one of two groups; patients that are susceptible to “volume overload” (those patients with insufficiency/regurgitation diseases), and patients that are faced with “pressure overload” (those patients with stenosis of their outflow tracts). When there are changes in blood flow that result from either insufficiency or stenosis at a heart valve, alterations in the flow of blood results in turbulence and abnormal sounds. Hearing abnormal sounds is the first step towards a diagnosis, and further diagnostic testing (thoracic radiography, echocardiography) may be indicated to evaluate progression of the disease and help to establish prognosis and treatment plans. When planning anesthesia for these patients, we need to consider how the heart is compensating for each type of these diseases. Insufficiency at a valve will typically result in volume overload upstream as a consequence of blood backing up from one heart chamber to another, most typically from a ventricle into the atrium, resulting in atrial dilation and enlargement. Because blood is backing up into the atria or potentially into the lungs or systemic system (including the liver), there is more blood volume for the heart to pump forward and therefore iatrogenic volume overload can be a potential consequence of poorly managed fluid therapy and anesthesia.

Volume overload diseases are relatively common in older small breed dogs and include mitral and tricuspid insufficiency/regurgitation. These diseases are typically age-related and are the result of progressive degeneration of the valve leaflets such that the valve leaflets eventually do not close completely during ventricular systole so there is regurgitant backflow of blood from the ventricle into the atrium. When the blood is ejected backwards through the insufficient valve leaflets, turbulence is generated and this is what creates the abnormal heart sounds that we can detect with cardiac auscultation. In the early stages of these diseases, fractional shortening (the ability for the heart to contract and pump blood forward) is usually increased so cardiac output is well maintained while the patient is awake. However, under anesthesia we need to aim to maintain heart rate and myocardial contractility to compensate for drug-related depression that can occur. Another consideration for patients being treated for mitral disease includes making sure that the patient is normovolemic and well-hydrated preoperatively, since diuretic drugs that these patients may be treated with can result in underlying dehydration. If the patient shows signs of dehydration, we should make every attempt to rehydrate them prior to anesthesia since the anticipated hypotension that will occur with anesthesia would be more profound in these cases. The goal for anesthesia of these patients is to encourage forward flow of blood through the heart and out to the body, as we can do this through choosing specific drugs to use and incorporating appropriate supportive techniques.

Stenotic malformations at the pulmonic or aortic valve typically results in pressure overload as a consequence, and I think of these as ‘thumb on the hose’ types of diseases. These conditions result in narrowing of the outflow tract to either the aorta in the case of subaortic or aortic stenosis, or the pulmonary artery in the case of pulmonic stenosis. In these patients, it is harder for the heart to pump the blood downstream against this increased resistance and therefore the heart muscle compensates by becoming thickened/hypertrophied. When myocardial hypertrophy occurs, the oxygen demands of the heart are increased so as part of our case management we need to prevent situations that would further increase the workload on the heart (i.e. use of anticholinergics, increased heart rate that can result from pain, hypercarbia, etc.). I also ‘lump’ patients with other hypertrophic diseases into this broad group of patients when planning my anesthetic management. Cats with HCM share some similar characteristics in terms of managing heart rate and myocardial oxygenation since they also have “big hearts”. Although the etiology of the hypertrophy is different with HCM than it is with subaortic stenosis, we need to avoid increases in afterload and sustained increases in heart rate in these feline patients as well. In addition to monitoring heart rate and blood pressure, we typically monitor for arrhythmias with an ECG to look for ventricular premature contractions and other morphology changes that can occur with myocardial hypoxia.
Another preoperative consideration for cardiac patients is fluid therapy. Fluid imbalances can be common in these patients since many of them are on diuretics. When indicated, it is important to slowly rehydrate the patient with IV fluids over several hours before anesthesia to minimize the potential for acutely fluid overloading the patient. Under anesthesia, we will often provide a maintenance rate of fluids between 5 and 10 ml/kg/hour which is lower than the typical anesthetic rates that used for stable noncardiac patients. As well, we typically use lower bolus rates of fluids in cases of hypotension as we don’t want to overload the heart with very rapid bolus of IV fluids. In unstable patients, we also measure central venous pressure (CVP) by advancing the tip of a catheter into the thoracic vena cava. By doing so, we can objectively measure the patient’s fluid status and get an idea of how the heart is performing in the face of this blood volume. We use this information to guide further fluid therapy since CVP is an objective way of determining if fluid overload is occurring.

We should also consider which cardiac medications the patient is on in order to anticipate what side effects might occur. For example, the ACE inhibitors cause vasodilation and with the concurrent dose-dependent vasodilation that can occur with the use of such drugs as acepromazine, propofol and isoflurane, the degree of hypotension induced by anesthesia would be expected to be more severe. As mentioned, diuretics can cause dehydration which needs to addressed with the use of proper fluid therapy. Beta blockers can also potentiate bradycardia and hypotension so we need to anticipate what sort of side effects these drugs may have depending on the particular patient’s status.

Ideally, we want to stabilize the patient before anesthesia. If it is an emergency or urgent situation, this may not always be possible but we should always make every attempt to stabilize these patients because they do not have the reserve that many healthy patients would. This means stabilizing the heart rate and rhythm and trying to optimize cardiac function by maintaining the use of cardiac medications up until the time of anesthesia. Although these drugs may affect blood pressure and heart rate during anesthesia, it is often better to keep a well-regulated patient on its cardiac medications, rather than altering their dose schedule and potentially making them worse postoperatively as we get them back onto their normal dosing. The anesthetic agents that we choose to use in the patient should be targeted to maintain minimal adverse cardiovascular effects, including drugs that have either short duration of action or the potential for reversibility. Acepromazine is one exception, as it has been found to have benefits in these patients even though it is neither short-acting nor reversible. There really is no best cardiovascular protocol or cardiac anesthetic protocol for cardiac patients and every case requires individualized treatment.

In terms of premedication for the patient, acepromazine is generally very well-tolerated in stable patients. Acepromazine has anti-arrhythmic properties which can be very useful in many patients, and is one of the agents that can be used to lower the required doses of the major anesthetic drugs such as isoflurane. Caution should be used when other drugs are ‘on-board’ that cause vasodilation (ACE inhibitors, etc.) since acepromazine can cause alpha1 blockade, resulting in dose-dependent vasodilation and hypotension. Due to this effect, in patients that have cardiovascular instability or shock we avoid the use of acepromazine but in otherwise stable cardiac patients acepromazine does have a useful role.

Opioids are also standard drugs used for premedication as they produce reliable analgesia and sedation with minimal to no negative cardiovascular effects. Other agents that are typically well-tolerated include benzodiazepines such as diazepam and midazolam. This class of drug has minimal to no cardiovascular effects, and is therefore a good choice for both stable and unstable cardiac patients. Anti-cholinergics such as atropine and glycopyrrolate are indicated for specific cardiac patients, but are typically avoided in animals that we want to avoid tachycardia in. In most instances, glycopyrrolate is a better choice than atropine because it doesn’t tend to drive the heart rate up to such extreme rates. In certain patients, alpha2 agonists such as dexmedetomidine can be used, but for the most part this drug is avoided in cardiac patients due to its sometimes profound cardiovascular effects (hypertension and resultant bradycardia, marked decrease in cardiac output).

All of the typically used injectable agents can be used for induction of anesthesia depending on the patient. Propofol is a short-acting, titratable drug that is well-tolerated in most cardiac patients. It does cause dose-dependent vasodilation, hypotension, and myocardial depression, but these changes are usually not severe at clinically used doses (unless the patient is in shock) and the effects are short-lived due to the rapid redistribution and elimination of the drug. Propofol is my first choice for stable cardiac patients, especially after the patient has been premedicated. Ketamine is useful for induction when it is combined with a benzodiazepine, but I find this combination is less titratable than propofol. Ketamine/diazepam will maintain heart rate and blood pressure better than propofol, so this combination can be useful in certain patients where cardiac output needs to be maintained on induction (it is my first choice for unstable patients). Thiopental should be used with caution or avoided altogether in cases with pre-existing arrhythmias, since this barbiturate can potentiate ventricular arrhythmias. Masking the patient down with an inhalational induction technique is usually not the best option since it is slower than an injectable technique, less titratable, more stressful for the patient (causing tachycardia), and results in greater cardiovascular depression than injectable drugs at a time when we don’t typically have any monitoring or supportive measures in place.
Inhalational maintenance of anesthesia can be achieved with the use of either isoflurane or sevoflurane. Halothane can cause ventricular arrhythmias and results in greater myocardial depression (decreased contractility) so it is generally avoided in cardiac patients. Total intravenous anesthesia can be provided with such drugs as fentanyl, midazolam or propofol and is a good alternative for some patients. One of the most important ways of further decreasing the levels of the major anesthetics (and therefore decreasing their dose-dependent side effects such as hypotension and hypventilation) is to use local anesthetic blocks when possible as the primary means of providing analgesia for the procedure. Every cardiac patient should receive a local anesthetic if the procedure is amenable to it (i.e. dental blocks, epidurals). If they are getting all of their pain relief from a local, we can use very low levels of the general anesthetic, resulting in a much more stable patient.

Typically, monitoring involves measuring heart rate, rhythm, and blood pressure, as well as monitoring adequacy of patient ventilation. Be ready ahead of time to support cardiovascular and respiratory function by having access to inotropes such as dobutamine (to improve cardiac output and blood pressure through increasing cardiac contractility) and anti-arrhythmics such as lidocaine for treating the occurrence of ventricular disturbances. With cardiac patients, it is very important to plan ahead as each patient is an individual and when complications arise, they will be acute and profound. As well, consider your own capabilities, comfort level, and limitations since as many of these patients can have unexpected side effects during anesthesia. Referral is a reasonable option for many cardiac patients if monitoring and supportive measures are not readily available in the general practice.

**RESPIRATORY DISEASE**

Many patients are presented with diseases of the respiratory system. Patients may have diseases of the upper airway (tracheal collapse, nasal tumour), lower airways (bronchitis, asthma), gas exchange surfaces (pneumonia, pulmonary edema), pleural space (pneumothorax, diaphragmatic hernia), or chest wall (tumours, rib fractures). All of these situations can have an impact on how easily the patient breaths and how it exchanges gases with the blood being delivered to the lungs. For this reason, we need to take into account the specific process that is occurring, how it might impact anesthesia, and how anesthesia might impact it. In most of these cases, the challenge is to not further stress the patient!

Most anesthetic drugs have the potential to cause dose-dependent decreases in respiratory function, resulting from decreased respiratory drive at the level of the brainstem. When the anesthetic drugs that we administer make the patient more tolerant to elevated levels of CO2, it breathes less (either by taking fewer breaths per minute, or by taking shallower breaths). When alveolar minute ventilation decreases, systemic CO2 levels rise and we call this hypercarbia (>50mmHg of CO2 in the expired breath or in the arterial blood). Low arterial blood oxygen levels (hypoxemia) can result from diseases that impact the gas exchange surfaces of the lungs or the relationship between ventilation and perfusion, and can be potentially life-threatening since oxygen is the body's fuel for metabolism. It is vitally important not to stress these patients because patients with pre-existing respiratory diseases often have decreased respiratory reserves (ability to compensate for drug-induced changes in breathing pattern, changes in body position, etc.) and therefore have less ability to compensate for the anesthetic-induced changes that occur. It is also important to remember the ABC's of life support since the ability to maintain a patent airway should always be the most important priority in any situation. When presented with a stressed respiratory patient, performing a thorough physical examination and obtaining preoperative laboratory data may be limited or impossible without the use of prudent premedication and/or general anesthesia. Take care with these patients!

Patient evaluation is very important. When possible, we should start by taking a complete history from the owners to uncover evidence of exercise intolerance or respiratory disease. Before handling the patient, we should assess its respiratory pattern and character through a distant exam, looking for signs of dyspnea and what body position the patient is maintaining itself in. By doing so, we are trying to differentiate whether or not the changes in its respiratory pattern occur during inspiration or expiration (suggesting whether the changes are the result of diseases of the upper airway or the lower airway). Once we put our hands on the patient, thoracic auscultation should be performed to listen for increased breath sounds, the absence of breath sounds, or abnormal breath sounds. Evidence of hypoxemia and hypoventilation should be looked at by assessing mucous membrane colour. When hypoxemia occurs, the mucous membranes will turn purple to blue, which is known as cyanosis. Diagnostic testing can include performing blood work (CBC, biochemistry), the performance of thoracic radiography to look for pulmonary changes or cardiovascular changes that might occur with thoracic or respiratory disease, trans tracheal wash for culture/cytology, performing arterial blood gas analysis to measure oxygen levels and carbon dioxide levels to assess for hypoxemia or hypoventilation, and advanced imaging or airway scoping procedures such as CT, ultrasound, or tracheoscopy/bronchoscopy to further assess the changes that are occurring with the patient’s disease.

The goals of any anesthetic procedure are to minimize stress and excitement in the patient and this is even more important in patients with pre-existing respiratory disease. In patients that are stressed from dynamic upper airway diseases such as laryngeal paralysis or tracheal collapse, the judicious use of tranquilizers can be very helpful in calming the patient and relaxing them. Once they are relaxed, they stop panting and breathe more easily. In these cases, acepromazine is my first choice. Intravenous
If the patient allows it, pre-oxygenation by placing a mask over the animal’s muzzle and nose is very helpful prior to induction of anesthesia. By allowing the patient to breathe 100% oxygen for several minutes prior to induction, we are able to fill the functional residual capacity of the lungs with oxygen, buying us time if the intubation of the patient becomes difficult and the patient becomes apneic after induction. We should also plan for a variety of complications that can occur during induction since respiratory patients are potentially some of the most catastrophic of all inductions and we need to be prepared for anything. Ideally, we want to have a rapid induction with a reliable IV agent to allow for rapid control of the airway with an endotracheal tube. Consider having an assortment of endotracheal tube sizes, intubation aids such as stylettes, the use of suction in case there are airway secretions or bleeding, the use of a laryngoscope to help visualize the airway, and plenty of light so that you can work comfortably regardless of the situation. Depending on the patient and how difficult we think oral intubation will be, we sometimes plan for an emergency tracheotomy by pre-clipping the patient’s ventral neck ahead of induction, having prep solutions present, and having drapes, a surgical blade and a surgical pack nearby. In these extreme cases, emergency tracheotomy may be the only option to quickly secure the patient’s airway.

Patient monitoring is as important as you would expect in these cases. Routine patient monitors are employed but particular attention should be taken to specifically monitor the respiratory system with auscultation, pulse oximetry (SPO2), and capnography (end tidal CO2). It is nice to have the capability to measure arterial blood gases as well. We also need to anticipate and be ready to support the respiratory and cardiovascular systems. It is very useful to have the ability to ventilate the patient either manually or mechanically. In certain patients, such as those with an open chest, the ability to ventilate the patient is a necessity. You should also think about the patient’s particular disease when you decide to ventilate them. For example, in cases of pleural space disease it is often more efficient to use short, shallow breaths since the lungs may not have the physical space to expand normally. As well, when you have an open chest it is common for the patient to develop atelectasis (lung collapse) even when you are seeming to ventilate the patient adequately. This collapse occurs as a result of absorption of oxygen from poorly ventilated areas, so it is useful to intermittently (every 3-5 minutes) “sigh” the patient to open up these collapsed areas and improve gas exchange. We should also be prepared to tap the chest by thoracocentesis prior to sedation and anesthesia if the patient has any sort of air or fluid in the pleural space. Stabilizing patients before any drugs are in the system is very important in these animals.

In terms of choosing anesthetic drugs, the judicious use of pre-anesthetics or tranquilizers such as acepromazine can be very useful in many of these patients to relieve anxiety and calm the patient. When they are more relaxed, they typically breathe better, but we still need to remember to monitor them carefully for problems. When people have problems with respiratory patients, it is usually the result of them treating the patients as they would regular patients in the clinic. Sedation and recovery are high-risk periods for these animals. Depending on the situation, if a patient is in respiratory distress it is often better to simply induce IV with a rapid-acting injectable anesthetic like propofol while they are positioned on the table with lots of personnel to help. This allows for a rapid, reliable induction that permits security of the airway. We often don’t need sedation first, but instead, can use the drugs (acepromazine, hydromorphone) that are typically given as a premedication after induction to help smooth things out once we have the airway secure.

The airway is always the priority, followed by oxygenation and ventilation of the patient (remember your ABC’s). Get the airway established with a tube first, inflate the cuff to allow for ventilation, and then you can think about other drugs that can be used. Masking these patients down is not a good idea as it simply takes too long and they often have respiratory difficulties already. Maintenance of anesthesia is usually achieved with an inhalant such as isoflurane or sevoflurane provided in 100% oxygen but total IV anesthesia with propofol can also be very useful in certain patients and promotes very smooth recoveries. Opioids and local anesthesia should be used for analgesia in these patients to help provide a balanced approach to maintenance of anesthesia as well as promoting a very smooth recovery. Typically if we provide good analgesia, we have a smooth recovery without any distress. Recovery is a high risk period for any patient, so carefully monitor them for signs of respiratory distress and make sure you are able to support them as needed. It is often necessary to support oxygenation by mask, nasal catheters, or induction chambers until the animal is breathing well and has the capacity to support itself in a natural position that will promote adequate ventilation and gas exchange.

HEPATIC DISEASE

Anesthesia for patients with clinical liver disease can be a challenge. The liver is responsible for many functions in the body including glucose homeostasis, fat metabolism, amino acid metabolism, protein synthesis, bile acid formation and excretion, host defense and inflammatory mechanisms, and drug and hormone metabolism. The liver typically receives about 20 percent of the cardiac output in the body. Thirty percent of its total blood flow comes from the hepatic artery, which supplies 90% of its oxygen. The other 70% of liver blood flow comes from the portal vein. Anesthesia may alter flow through one or both of these blood...
supplies to varying degrees, and as a result, we need to consider the impact of anesthesia on this circulation. It is important to assess liver function before anesthesia by using the physical examination to look for indications of abnormal growth rate, assessing the mentation of the patient for indications of hepatic encephalopathy, and palpating the abdomen to assess liver size and to assess for the presence or absence of ascites. Diagnostic imaging can be very useful and includes the use of abdominal radiography, angiography and ultrasonography. Blood work can also give us an indication of liver function through analysis of the serum biochemical profile and special testing. We can measure AST, ALT, alkaline phosphatase and GGT which can be indicators of hepatic and biliary damage. Pre and postprandial serum bile acids can be used as an indication of liver function, and clotting times (PT/PTT), ammonia tolerance testing and looking at the levels of protein, albumin, urea nitrogen, creatinine and glucose can also give us useful information about overall liver function. Complete blood cell counts are typically normal but in certain cases such as portosystemic shunt, a CBC may demonstrate a red blood cell microcytosis.

As most people know, most anesthetic drugs are metabolized by the liver. Metabolism is often slowed if the patient is hypothermic. Glucose metabolism is usually not a problem until we lose 80% of normal liver function but we may see hypoglycemia in cases where the patient is stressed due to anesthesia and surgery or in certain patients such as those with a portosystemic shunt.

One important consideration when it comes to choosing the anesthetic agents for patients with liver disease is a concept known as the “extraction ratio”. The extraction ratio is a measure of the ability of the liver to remove drugs from circulation and clear them from the body. The extraction ratio is the amount of drug that is extracted or removed relative to the amount of drug that is delivered to the liver. There are two broad groups of drugs – those with “high extraction ratios” and those with “low extraction ratios”. High extraction ratio drugs are those that are easily removed and metabolized once they arrive at the liver. Their elimination depends on the rate that they are actually delivered to the liver rather than the actual speed of metabolism, and as a result, elimination and clearance of these high extraction ratio drugs depends almost entirely on cardiac output and hepatic blood flow, not on liver function. Therefore, if blood flow to the liver is decreased, we will potentially see a prolonged duration of effect for these specific drugs. High extraction ratio drugs include most of the opioids, propofol, ketamine, local anesthetics and some benzodiazepines. Isoflurane is minimally metabolized by the liver (0.2%) and most is eliminated by the respiratory system so isoflurane is the agent of choice for maintenance of anesthesia. Propofol is a drug that is a good choice for patients with liver disease. One reason for this is that the total body elimination of propofol actually exceeds liver blood flow, suggesting that it is actually metabolized at sites outside of the liver. As a result, propofol is a good choice for induction and/or maintenance (as a CRI) even in patients with advanced liver disease.

Some drugs take longer to actually be metabolized once they arrive at the liver, as their metabolic pathways are more complicated and involve more biotransformation. In the case of “low extraction ratio drugs”, the elimination doesn’t depend on how much of the drug is brought to the liver by blood flow, but instead, how long it actually takes for the liver to metabolize them. In these cases, if the patient has clinical liver disease that is detected on liver function testing, we will potentially see a longer duration of drug action and more side effects. There are two main classes of drug that fall into the low extraction ratio category: the thiobarbiturates and the nonsteroidal anti-inflammatory drugs. This is the reason why we typically avoid use of thiobarbiturates and nonsteroidal anti-inflammatory drugs in patients with severe clinical liver disease.

When planning the anesthetic protocol for patients with liver disease, the overall goal is to maintain liver blood flow and oxygenation. Generally speaking, if we keep the liver happy, it will sort everything else out for us. We usually avoid using alpha2 agonists or other agents that can cause vasoconstriction in patients with liver disease because we do not want to decrease blood flow to the liver. Although some studies demonstrate maintenance of splanchic perfusion with the use of dexmedetomidine in dogs, we should still expect to see a drop in overall cardiac output so liver blood flow still has the potential to be compromised. We usually use short-acting drugs or drugs that can be potentially reversed so we can quickly assess the patients for deterioration and minimize side effects. Realistically, we rarely anesthetize patients in true liver failure in veterinary medicine, and as a result we don’t have to deal with many of the issues that are described in human medicine.

Aside from the changes that occur in the liver itself with disease, there are several problems that we can anticipate ahead of time relating to patients undergoing surgical procedures for the liver. It is very important to monitor and support body temperature in these patients as they may be in thin body condition and have small livers with low glycogen stores. As well, for procedures involving the liver, there typically needs to be a large abdominal incision which leads to greater potential for heat loss through evaporation and radiation. For these reasons, liver patients are more prone to hypothermia so we need to be more aggressive in preventing and treating this anesthetic complication. We also need to anticipate the potential for severe blood loss with liver procedures as oozing can be significant and often goes undetected. We need to plan for marked hypovolemia and hypotension by having extra IV fluids ready, as well as the potential use of colloids and blood products. Typically, hepatic surgical procedures involve cranial abdominal manipulation by the surgeon. The presence of their hands and traction by surgical instruments frequently results in inadvertent compression of the vena cava that causes decreased venous return to the heart, decreased cardiac output and decreased blood pressure. Watch
for this! As a result, these patients are prone to rapid shifts in blood pressure and need to be monitored and supported closely.

In our practice, patients that are undergoing procedures for the liver or have concurrent liver disease are premedicated with an opioid for analgesia and sedation with or without the combination of a tranquilizer such as acepromazine or a benzodiazepine such as diazepam or midazolam. Although some practitioners are wary of using acepromazine in these patients, I use it to smooth out the anesthetic and to help us to further decrease the inhalant anesthetic through a balanced anesthetic technique that is hard to achieve with the use of an opioid alone. Induction of anesthesia is preferentially made with the use of propofol or ketamine. We avoid the use of thiobarbiturates (that rely heavily on metabolism for clearance) and masking with inhalational agents (that can decrease liver blood flow through a stress response). Maintenance of anesthesia is performed with the use of either isoflurane or sevoflurane, as these drugs have the least effect on liver metabolism and liver blood flow. Halothane is not recommended for patients with liver disease as halothane is approximately 20% metabolized by the liver and therefore can be hard on the liver that doesn’t have adequate function. Finally, local and regional anesthesia should be used where appropriate to provide further analgesia to the patient. This promotes good pain management, lowers the levels of the major anesthetics (so less side effects), and results in a smooth recovery period without any significant pain or additional drugs being needed. It is important to continue to check the patient by monitoring the patient’s vital parameters (temperature, pulse, respiration, blood pressure, pain) and blood glucose. Always plan for the worst by anticipating the potential for blood loss, hypothermia and pain and look to support the liver with the use of blood pressure supportive measures and choosing specific anesthetics that will have the least effect on liver blood flow. There is no doubt that the outcome of patients with liver disease is more dependent on appropriate management and support than on the specific drugs being used.

**URINARY TRACT DISEASE**

Patients that have urinary tract diseases are frequently presented to general practitioners. These diseases may be concurrent with other issues involving the cardiac, liver or respiratory systems as patients age, or they can be primary disorders of the urinary tract such as blocked cats or ruptured bladders in patients that have suffered trauma.

We should start by taking the patient’s history and performing a thorough physical examination. Palpation of kidneys is not always possible with abdominal palpation, but hydration status can easily be assessed. Biochemical profiles can be measured to look at blood urea nitrogen and creatinine levels, electrolyte levels and CBC to look for signs of chronic anemia that might result from chronic renal failure. Urinalysis should also be performed to measure the urine specific gravity, to look at urine sediment, and to document the presence of proteinuria. Diagnostic imaging may also be indicated, including the use of radiography and ultrasonography. Finally, specific tests can also be used to measure creatinine clearance or the fractional excretion of electrolytes in selected patients. It is also important to ask about what concurrent medications the patient may be on, as certain drugs (i.e. ACE inhibitors) used to treat hypertension associated with chronic renal failure may predispose the patient to further decreases in blood pressure under anesthesia.

Azotemia is the result of accumulation of nitrogenous products of cellular metabolism and can be assessed by measuring urea and creatinine levels in the blood. Urea and creatinine are both eliminated primarily by glomerular filtration in the kidneys. Typically there must be a sizeable reduction in glomerular filtration rate before concentrations of urea or creatinine become persistently elevated, with a 75% reduction in kidney function being required before levels of these two parameters increase. Creatinine is released in a steady state into circulation whereas urea levels can be influenced by many non-renal parameters, including hepatic function, ingestion of proteins, hydration status and increased catabolism. Because of this, we usually look at creatinine more than BUN to assess kidney function, and we can relate creatinine levels back to urine specific gravity to assess whether the levels of creatinine are appropriate. Severe azotemia may result in alterations of the blood brain barrier and can affect the level of protein binding of some anesthetic drugs, making certain agents (those that are normally highly-protein bound such as benzodiazepines) potentially more active in the body.

Azotemia can be divided into three different categories – prerenal, renal and postrenal. It is important to know how to differentiate between the different causes of azotemia and how to relate these through the use of urine specific gravity and physical examination. As one would expect, the anesthetic management of each type of disease will be potentially different. Prerenal azotemia is the result of dehydration or hypovolemia and these patients must have their fluid imbalances corrected prior to anesthesia. These patients are not usually difficult to manage under anesthesia, as long as their state of hydration is corrected preoperatively. Renal azotemia is the result of the kidneys themselves being unable to function properly, and, in addition to age-related changes, can result from nephrotoxins or glomerulonephritis. These cases are more challenging to manage under anesthesia, and we should always be concerned with the potential of making things even worse with a poorly managed anesthetic event (i.e. untreated hypotension leading to hypoxic damage to the remaining nephrons). Postrenal azotemia is typically the result of urethral obstruction/tear or a ruptured bladder. In these patients, excreted wastes (urea, creatinine, potassium, acids) are reabsorbed to very high levels, and hydration status can be a problem. We need to stabilize electrolytes and hydration, while at the same time balancing the fact that any fluids put into
the body may stay there until we correct the underlying disease. These are often the most unstable urinary patients that we see (often as emergencies), and need to be handled accordingly.

Another potential complication of patients with urinary tract diseases is the presence of severe hyperkalemia. This is a common finding in patients with postrenal azotemia resulting from lower urinary tract obstructions or ruptured bladders. Since potassium acts as a break on the heart, hyperkalemia can result in severe bradycardia that is unresponsive to anticholinergics. The ECG of a hyperkalemic patient will show peaked T waves, prolonged P-R intervals and wide QRS complexes. With severe hyperkalemia, we may also see the loss of P waves entirely. Hyperkalemia may be markedly worsened by anesthesia itself and is an important cause of perioperative mortality. It is therefore best avoided. Every attempt should be made to decrease potassium levels medically to below 6 mEq/L prior to anesthesia.

The kidneys have an enormous requirement for oxygen since they are one of the most highly active organs in the body. As a result, the kidneys normally receive 25 percent of the body’s entire cardiac output. Blood flow to the kidneys is under local sympathetic control (vasoconstriction/vasodilation), and is maintained relatively constant when mean arterial blood pressure (MAP) ranges between 60-160mmHg. This means that in most healthy patients, regardless of the transient changes in blood pressure that occur over the course of the day, there are no substantial changes in blood flow and delivery of oxygen to the kidneys – they are maintained in a steady-state. However, when MAP decreases below 60mmHg for an extended period of time (which can easily occur under the depressant effects of anesthesia), there can be a concurrent decrease in renal blood flow. As a result, oxygenation of the highly metabolic kidneys can be compromised. For this reason, we need to strive to maintain our patients’ MAPs above the “cut-off” of 60mmHg since the body’s own autoregulatory mechanisms are good at maintaining blood flow to the kidneys above this level despite any subtle changes that may occur in blood pressure. This means that we need to target hydration, blood volume, our doses of anesthetic drugs, depth of anesthesia, and the potential use of other agents to maintain blood pressure, cardiac output, and renal blood flow. It is even more vital to prevent hypotension in patients that have chronic hypertension (patients with renal failure, Cushing’s disease, etc.) since they typically have more difficulty maintaining renal blood flow in the face of hypotension during anesthesia.

Anesthesia may negatively alter renal blood flow and glomerular filtration rate. These effects are usually dose dependent, so we need to aim to provide low levels of our general anesthetic to these patients. The renal responses to these changes in blood pressure are dependent on pre-existing hydration status and perioperative fluid administration. As well, stress responses during anesthesia and surgery have the potential to release catecholamines that cause alpha1 activation and reduce renal blood flow. It is therefore important for us to avoid prolonged stress responses that can result from hypercarbia and pain. Finally, auto-regulatory mechanisms may be impaired with renal disease or from the use of certain drugs. Conditions such as chronic systemic hypertension, or the use of nonsteroidal anti-inflammatories that affect the COX system and constituitive prostaglandin levels can both have negative effects on renal blood flow.

Preoperative preparation of these patients depends largely on the primary underlying abnormality. Acute and chronic renal failure patients often appear relatively stable when they are awake but frequently require aggressive intraoperative support if they are not adequately prepared for anesthesia. Urinary obstruction and ruptured bladders are emergency situations. These patients can be extremely variable in their presentation from minimally compromised to shocky or near death. Although these patients often need to be anesthetized sooner than we may otherwise like, it does not eliminate the need for appropriate preoperative stabilization. We need to decrease potassium levels and support blood volume and body temperature in these critical patients. Do not be afraid to volume-load these blocked patients if they are unstable – you can always perform cystocentesis and drain the bladder for them if it gets too big, but you will have a hard time resuscitating them if they crash when they are dehydrated and hypovolemic without prior fluid correction.

Support for any patient with renal disease involves the use of IV fluids. Fluid therapy before anesthesia is used to correct volume deficits and promote diuresis. We typically administer these fluids over several hours to avoid rapidly overloading the patient immediately preop. Further renal blood support involves the use of drugs that can increase cardiac output, glomerular filtration and enhance renal blood flow. In the past, people used dopamine infusions, however the use of this drug has recently fallen out of favour. Dopamine may cause diuresis in some patients but it doesn’t improve creatinine clearance overall. In addition, there is debate as to whether or not dopamine receptors are even present in the kidneys of many domestic species, raising doubt as to whether or not the “renal dose” of dopamine is actually having any effect. Dopamine is not readily available to us anymore, and as a result we no longer use it in our patients. Dobutamine infusions may be used to provide support of renal blood flow by increasing cardiac output. This effect results primarily from increased cardiac contractility, and can increase glomerular filtration rate and urine production so we commonly use this infusion during anesthesia of renal patients. Additionally, mannitol can be administered as a bolus followed by a constant rate infusion during anesthesia. This intervention has been shown to osmotically increase blood volume, renal blood flow, and glomerular filtration rate and urine output. This is an easy supportive measure to take, and has been shown to provide benefit to the renal patient.
As far as drugs go, we typically aim to use short-acting drugs or drugs that can be potentially reversed. We will usually avoid the use of alpha2 agonists (dexmedetomidine), nonsteroidal anti-inflammatories and halothane in renal patients due to the potential for these particular drugs to have negative effects on renal blood flow. It has been suggested to avoid the use of ketamine in cats with severe renal disease since cats excrete ketamine unchanged in their urine. It is thought that in cases of absolute renal failure, ketamine can be recycled and result in a prolonged duration of effect. In many other patients (prerenal, postrenal), ketamine can still be safely used. Other anesthetic agents are usually well-tolerated by these patients. Premedication is often achieved with low doses of acepromazine or a benzodiazepine in combination with an opioid. Remember the diuretic effects of dexmedetomidine, as you may want to avoid the use of this drug in blocked patients that may not easily be unobstructed. Induction of anesthesia can be used with any injectable anesthetic such as propofol, ketamine or thiopental and maintenance of anesthesia is typically achieved through the use of inhalationalss such as isoflurane and sevoflurane. These drugs are preferred since they do not typically result in significant decreases in renal blood flow like halothane does. These drugs do, however, have systemic effects on decreasing blood pressure so we should try to use the lowest possible level of inhalant that we can, and lower their doses using balanced anesthetic techniques (use of premeds, local analgesia).

Since we cannot easily measure the important parameters of cardiac output and renal blood flow in most clinical patients, we assume that when we maintain good blood pressure, we are also maintaining cardiac output and renal blood flow. Remember that the overall goal for anesthetizing patients with renal disease is to maintain MAP within a range greater than 70mmHg. Maintaining and supporting blood pressure involves the use of prudent preoperative and intraoperative fluids, balanced anesthesia and analgesia, and the use of blood pressure supportive measures that do not themselves cause vasoconstriction (i.e. no vasopressors). We frequently monitor urine output via catheterization and use of a closed collection system as an indirect reflection of renal blood flow and perfusion of kidneys. Urine output should typically be above 1 to 2 ml/kg/hour. If we are not achieving this level, it usually means we are not providing adequate fluid support and the patient has volume deficits that remain uncorrected.

The use of local or regional anesthesia where appropriate can greatly benefit the patient by decreasing the doses of isoflurane and sevoflurane that are used for maintenance. Always remember to use local blocks where appropriate in these patients, including dental blocks and epidurals to help you turn the vaporizer down. The recovery period should involve continued renal support and monitoring to make sure that urine output and hydration status are appropriate to the patient. The use of nonsteroidal anti-inflammatories postoperatively in these animals is controversial and really depends on the degree of renal dysfunction and the volume status of the patient. Risk benefit should be assessed by the individual clinician and this decision should be made as appropriate using all information available.

Anesthesia for patients with urinary tract diseases can be challenging, but by simplifying concepts and using appropriate decision-making before any drugs are given, patients can be well cared for. Think about the physiology of what the particular disease is causing in the patient, what you might expect to happen once anesthetic agents are in the system, what changes in cardiovascular status will occur with recumbency, the health status of the patient, and the concurrent drugs being used. It is important to think of these patients as individuals and think about what you are capable of doing for that particular patient on that particular day. Not every clinic is equipped to handle these cases so recognize your limits and consider referral when appropriate.
A is for Adnexa

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No Paper Available
B is for Brain (Neuro-Ophthalmology)

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Assess vision: menace response, dazzle reflex, cotton ball test

Blind

Pupillary light reflex

Positive

Fundic examination

Negative

Fundic examination

Assess: optic nerve, tapetal reflectivity, vessels

Normal

Abnormal

ERG

PRA

Optic neuritis, retinal detachment

Lesion: cornea, aqueous, lens, vitreous

Unable

Normal

Abnormal

Intraocular pressure

Elevated = glaucoma

Systemic work-up?

Systemic work-up?

Normal

Abnormal

PRA

Optic neuritis, retinal detachment

Optic nerve(s), optic chiasm, lesion

CT/MRI scan, CSF analysis

Cortical blindness

NEuro exam, MRI/CT scan, CSF analysis

SARDS

SARDS

Normal

Abnormal

Optic neuritis, retinal detachment

CT/MRI scan, CSF analysis

Systemic work-up?
C is for Canine Cornea
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ANATOMY

The cornea is a transparent avascular tissue that transmits and refracts light and protects the intraocular contents. It relies on aqueous humor and tears for nutrition and waste removal and eyelids and nictitans membrane for protection. Corneal thickness in the dog varies from 409 µm (axial) to 784 µm (peripheral). The cornea measures 12 to 16 mm vertically and 13 to 17 mm horizontally. The most external layer is nonkeratinized, stratified squamous epithelium, 5 to 7 cell layers thick that turns over every 7 days. The basal epithelial cells are anchored to the basement membrane by hemidesmosomes. Nerve endings are present within the epithelial cells. The normal tear film prevents keratinization of the epithelium.

The corneal stroma comprises 90% of the corneal thickness. Small diameter collagen fibrils (run parallel to corneal surface) in precise organization, with intermixed keratocytes, compose the stroma. Any disorganization of the fibrils results in an opacity (normally 99% of the light passes through the cornea without scattering).

The anterior stroma is innervated by the trigeminal nerve and these nerves traverse radially into the epithelium. Central cornea more sensitive compared to periphery. Diabetic patients have reduced corneal sensation. The cornea is relatively dehydrated (75 to 90% water) and this is maintained by the endothelium and epithelium. Descemet’s membrane is the basement membrane of the endothelial cell (thickens with age). The endothelium is a single layer of cells with tight junctions to prevent fluid entry into the stroma. The endothelial cells actively pump fluid out of the stroma. Young animals may have some ability to regenerate endothelial cells but mature animals are not able to regenerate cells. Endothelial cell numbers decrease with age and below a critical number corneal edema will occur.

CORNEAL WOUND HEALING

Epithelial Healing

With an epithelial defect the cells beside the area release attachments via hemidesmosomes to the stroma. This allows the cells to slide and fill in the defect. It takes between 48 to 72 hours for the cornea to be completely covered by sliding epithelial cells of an ulcer that encompassed the whole cornea. The healing process then thickens this epithelial cover by mitosis. The corneal epithelium is completely replaced in two weeks.

Stromal Healing

Initially neutrophils come in via chemotaxis. Keratocytes adjacent to the defect transform into fibroblasts that synthesize collagen and other extracellular matrix components that pushes the epithelium anteriorly. Collagen lamellae can be disorganized and this will create opacification. Vessels invade the stroma (takes 5 to 7 days to form and progress 1 to 2 mm per day) and granulation tissue is created. Eventually the vessels collapse and become ghost vessels.

Response to Disease

1. Edema – occurs with damage to the endothelium or loss of epithelium (glaucoma, ulceration, anterior uveitis, endothelial dystrophy or lens luxation)
2. Cellular infiltration - infected/inflamed ulcers, immune-mediated disease or neoplasia
3. Pigment – can occur with any chronic irritation
4. Scar formation – abnormal arrangement of collagen fibers
5. Vessels
6. Secondary veitis
CANINE CORNEAL ULCERATION

This is one of the most common complaints that I examine in my practice. The management of an ulcer consists of 1) determine the cause, 2) medical treatment and 3) protect and support the cornea.

DIAGNOSIS

Fluorescein sodium is a water-soluble fluorescent dye that is repelled by the hydrophobic epithelium and Descemet’s membrane but adheres to the hydrophilic stroma. Its peak excitation is in the blue portion of the visible spectrum and peak emission is in the green portion of the visible spectrum. I usually wet the impregnated strip and touch it to the conjunctiva (if it touches the corneal epithelium it will stain the area) and then rinse with saline.

Fluorescein sodium can also be used to determine if a wound leak is present. Concentrated dye is placed on the cornea and not rinsed. If aqueous is leaking through the defect a rivulet of green will be seen against a background of yellow-orange. This is called a Seidel test.

CAUSES

Thorough ocular examination may allow identification of a cause or predisposing factor.

Trauma – This is the most common cause of ulceration.
Causes include blunt or penetrating trauma, and exposure to alkali, shampoo, acids (batteries and cleaning agents) or mace. Alkaline material (soap, lye and lime) penetrates the cornea more rapidly than acids. The alkali joins the cell membrane lipids and disrupts the cells and softens the surrounding tissue. Ulcers that are located under the third eyelid should be examined for foreign body. Treatment consists of irrigation if a caustic agent is suspected (20 to 30 minutes in severe cases), prevent infection (broad spectrum antibiotic) and treat pain and discomfort to severe discomfort. Corneal ulceration may occur and chronic changes such as pigmentation, scarring and corneal vascularization may be noted. Four types of entropion can occur. A) Conformational – the most common. It is due to a disproportionate size of orbits and globe position. Breed predisposition for Shar Peis, Rottweilers, Chow Chows, Cocker Spaniels and Bulldogs. Application of topical anesthesia does not correct the entropion. Modified Hotz-Celsius to correct the simple cases is appropriate. More complicated cases may need a Brow sling (due to ptosis in Shar Pei and Chow Chow), Stades procedure (due to ptosis) or more complicated blepharoplasty procedures if ectropion is present as well. B) – Spastic – occur secondary to ocular pain, so the dog retracts the globe and then the eyelids roll in. This can occur with concomitant ulceration and is corrected with application of topical anesthesia. Treat the underlying problem and the entropion resolves. C) – Cicatricial – secondary to scar formation from trauma or chronically spastic tissue scars and rolls in. Requires releasing techniques (“V” to a “Y”). D) – Puppy – Shar Peis and Golden Retrievers. Temporary tacking sutures (vertical mattress) of 4-0 to 5-0 nylon. Recheck every 3 to 4 weeks. Sutures may need to be replaced. No definitive surgery until skull conformation near adult size.

Eyelid Mass – Typically tumors do not cause problems.

Foreign Body – Either embedded in the cornea or under the third eyelid. Corneal foreign bodies can be removed under topical anesthesia and fine forceps.

Keratoconjunctivitis Sicca – Lack of the aqueous layer of the tear film due to autoimmune destruction (most cases) of the gland of the nictitans membrane and the lacrimal gland. KCS typically present as chronic conjunctivitis (mucopurulent discharge and hyperemia) and keratitis (vessels, pigment, scar, ulceration and perforation). Treatment consists of 1) Artificial tears – I recommend human pre-
Types of Simple Corneal Ulcers

Superficial Ulcer

Clinical Signs
This is a defect confined to loss of the epithelium (relatively flat). These can be difficult to visualize without fluorescein stain. Clinical signs consist of blepharospasm, epiphora, discharge, enophthalmos, hyperemic conjunctiva, third eyelid elevation and corneal changes. Painful due to stimulation of the nerve endings in the epithelium and anterior stroma. Classified as either simple, refractory or progressive.

Treatment
If a simple ulcer topical broad-spectrum antibiotics (triple antibiotic) are used to prevent bacterial infection. Topical atropine can be used to alleviate cilary body muscle spasms. This type of ulcer should heal within one week and if this does not occur look for a reason.

Refractory Ulcer

These are chronic non-healing. Initially described in boxers but subsequently diagnosed in most breeds. Middle aged to older dogs are predisposed. Initiating cause likely trauma and the ulcer does not heal through normal wound healing mechanisms.

Diagnosis
Any ulcer in a middle-aged patient that does not heal within 7 to 14 days should be suspected to be an indolent ulcer. The ulcer is superficial (no stromal loss) and characterized by a lip of nonadherent epithelium. Fluorescein stain migrates under the epithelium and creates a halo around the ulcer. Patients are painful in the initial stages but with chronicity tend to become more comfortable.

Pathophysiology
The basic defect in the cornea in these ulcers is nonadherence of the corneal epithelium to the underlying stroma. The epithelial basement membrane and hemidesmosomes is either absent or very sparse. Under the ulcer is an acellular tissue and abnormal nerve plexus that are significant in the disease process. The acellular zone maybe a barrier to epithelial cells adhering to the underlying stroma. Suspected that abnormal healing process with delayed hemidesmosome formation. The previous list of causes of ulceration may incite an indolent ulcer. Breed predisposition may represent a form of primary corneal epithelial or anterior stromal...
dystrophy. In one study the basement membrane has been found to be abnormal under the defect in patients with indolent ulcers but normal in areas away from the defect. This acellular zone may prevent penetration of hemidesmosomes from the epithelial cells. Another study measured the protease levels in the tear film and found them to be elevated. The proteases may not allow a fibronectin network to form under the epithelial cells and allow them to make there initial attachment to the basement membrane.

**Treatment**

Resolution of the ulcer may take weeks to months. Client education is imperative when dealing with an indolent ulcer. Owners must be educated about the slow healing, multiple rechecks, different therapies, and possible recurrences. Medical treatment involves broad-spectrum antibiotics (two to three times a day) and is rarely successful in healing ulcers alone. Multiple treatment options are recognized to heal indolent ulcers. The following is a list of potential treatments:

1) **Debridement** – This is advocated in all indolent ulcers. The removal of all of the nonadherent epithelium is done under topical anesthesia (proparacaine) using a dry cotton tipped swab. Normal epithelium does not debride. It is not unusual for the ulcer to greatly increase in size with this procedure.

2) **Grid Keratotomy** – Under topical anesthesia superficial linear stromal incisions in a crosshatch pattern are made with a 25-gauge needle held tangential to the cornea. The incisions are 0.5 mm to 1 mm apart and extend just beyond the ulcer edge into normal epithelium. The keratotomy creates a channel in the acellular membrane through which granulation tissue from the stroma then spreads out on the epithelial side of the stroma to create viable tissue for the epithelium to attach to. 75-80% of ulcers will heal within 2 weeks after debridement and grid keratotomy.

3) **Diamond Burr** - Under topical anesthesia superficial stroma is removed. The burr removes the acellular membrane to allow the epithelium to attach to the normal stroma.

4) **5% Polysulfated glycosaminoglycan (PSGAG) Solution** is made from Adequan® IM (100 mg/ml) and diluted to 50 mg PSGAG/ml. Applied three times daily. Hypothesis is the PSGAG binds the proteases and allows epithelium to bind to the underlying basement membrane. I use this as an adjunctive therapy.

5) **Contact Lens** – Placed on the eye to protect and maintain apposition of the healing epithelium. Usually stay on for 1 to 4 days. I use human contact lenses.

6) **Cyanoacrylate tissue adhesive** - Topical anesthesia is applied, then the cornea is dried (cotton tipped swab). A drop of tissue glue is applied to the end of a 1 cc syringe. The drop is placed on the defect and allowed to solidify (30 to 60 seconds). Only a thin film of tissue glue is needed over the defect. Once the ulcer re-epithelializes the adhesive sloughs. Adhesive may remove abnormal basement membrane.

7) **Thermal Cautery** – Done after debridement. Multiple superficial burns in and around the ulcerated area are performed. Alters the superficial stroma to allow cells to adhere. Reserved for those cases nonresponsive to alter therapies.

8) **Superficial Keratectomy** – Performed if the ulcer is non-responsive to the above treatments. This is done under general anesthesia. The loose epithelium is debrided with a cotton-tipped swab.

A) - An incision is made around the ulcer with a No. 64 Beaver Blade or punch biopsy into the superficial stroma.

C) - The edge is then grasped with forceps (Colibri forceps are ideal) and elevated. The ulcerated area is then removed by use of the scalpel blade or a Martinez corneal dissector.

Post-operative care is the same for any ulcer. Almost 100% effective in healing ulcers within two weeks post surgery. Tramadol to alleviate discomfort for 4 to 7 days post procedure.

**CANINE NONULCERATIVE KERATITIS**

**PIGMENTARY KERATITIS**

Non-specific change that develops with long standing trauma/irritation to the cornea.

Lagophthalmos is the inability to close the eyelids normally and is common in brachycephalic breeds. Often these dogs will sleep with eyelids open and the blinking is decreased in frequency and incomplete. The axial cornea is relatively dry and prone to chronic irritation. Pigmentary keratitis is usually present. Facial nerve paralysis will prevent blinking and can be associated with KCS. If facial nerve paralysis is present a permanent tarsorrhaphy and ocular lubricants may be required if recurrent ulcers occur. For brachycephalic breeds a medial canthoplasty (pocket technique) to shorten the eyelid length and increase coverage over the eye can be used as a preventative.

A) The upper and lower eyelids are split at the margin for a depth of approximately 1 cm (creating a pocket). This splits the tarso-conjunctival tissue from the skin/obicularis oculi. The length of the split is 4 to 5 mm. The anterior strip of eyelid margin is removed. B) A flap is created by cutting the lateral extent...
of the upper eyelid tarso-conjunctival tissue, and should be about 8
mm long. 4-0 nylon (with a stent) is placed through the skin into the
lower pocket. The suture then exits the pocket, passed through the
tip of the flap, through the lower pocket, out the skin and stent. This
procedure brings the flap into the lower pocket for extra strength to
the closure. c) The eyelid margin is apposed using 5-0 nylon or viryl.

**IMMUNE KERATITIS**

**Clinical Signs**

Chronic superficial keratitis presents as a vascularized, pigmented
lesion (with chronic cases) of the anterior temporal cornea near
the limbus. With chronicity it moves centrally and can cause
blindness. The leading edge will often be white that is composed
of lymphocytes. Most commonly bilateral, but often asym-
metrical. An associated condition called plasmoma can occur
which is thickening and depigmentation of the third eyelid.

**Pathophysiology**

This is an immune-mediated disease with associated breed
predisposition and environmental factors. Most common in
German Shepherds and Greyhounds but I have seen this is most
breeds. The younger the age at presentation the more severe
the condition. Clinically more severe and more common with
increased elevation (>1400 meters). The ultraviolet radiation may
alter corneal antigens with resultant cell-mediated inflammation.

**Treatment**

This is a controllable but not curable disease requiring life long
treatment. Topical dexamethasone 0.1%, Cyclosporine 1 or 2%
and/or Tacrolimus 0.02% have been shown to be effective. Need
frequent treatment initially then reduction in frequency once
inflammation controlled. Make sure to measure tear production.

**CORNEAL LUMPS**

**Limbal Melanoma**

A dark pigmented infiltrative mass at the limbus. More common
in large breed dogs. Generally benign tumor but can be locally
aggressive especially in young dogs. Treatment is determined
by age. In old dogs if the mass is not progressing it can be
monitored. In progressing lesions or young dogs two options;
1) full thickness scleral graft or 2) diode laser ablation.

**Dermoid**

A congenital brown mass on the cornea with hairs emanating from
it, usually located at lateral limbus. Usually do not cause a problem
until greater than 6 months of age when the hairs start irritating
the cornea. Removal of the lesion with a keratectomy is curative.

**Epithelial Inclusion Cyst**

Occurs secondary to prior injury that deposits epithelial
cells into the stroma. These cells proliferate and cause a
tan cystic lesion. Usually not painful but associated vessels
can be present. Keratectomy is the treatment of choice.

**Granulations Tissue**

Pink fleshy mass the occurs with chronic injury like entropion
or refractory ulcers. Common in Shar Peis with entropion

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C is for Cornea (Feline)

Brian Skorobohach, DVM, Diplomate American College of Veterinary Ophthalmologists

**EYELID AGENESIS**

**CLINICAL SIGNS**
Tends to be a bilateral condition of the upper lateral eyelid. Concurrent keratitis, corneal pigmentation, epithelial hyperplasia and corneal ulceration may occur secondary to trichiasis and inadequate eyelid function. Keratoconjunctivitis sicca has been associated with the condition. Patients may also have concurrent optic nerve coloboma, which may cause blindness.

**TREATMENT**
Surgical procedure is determined by eyelid function. I find in most cases the corneal irritation is secondary to trichiasis. Therefore removal of the hairs with cryoepilation is effective. If eyelid function is abnormal then surgical reconstruction of the upper eyelid is warranted. This will create more eyelid coverage over the cornea. Rotational flap from the lower eyelid to the upper eyelid is the most successful surgery. The initial stage is separating the conjunctiva from the upper eyelid. A flap of full thickness skin from the lower eyelid is created by making parallel incisions, approximately 4 mm apart (depending on the defect), 5 mm from the eyelid margin. The full thickness flap is rotated to become the new upper eyelid. Sutured to the skin using 5-0 to 6-0 nylon. The conjunctiva is sutured to the flap in a simple continuous pattern using 6-0 to 7-0 Vicryl®.

**FELINE HERPESVIRUS KERATITIS**

**ETIOLOGY**
FHV-1 primary infection is associated with respiratory and ocular signs. FHV-1 replicates in the epithelium of the upper respiratory tract with involvement of the conjunctiva and cornea. FHV-1 is capable of causing corneal disease by two different processes. Firstly, the corneal epithelium can be invaded and destroyed, with resultant dendritic ulcer formation. Secondly, stromal keratitis can occur due to viral antigen localization in the stroma with subsequent immune mediated response.

Approximately 80% of cats become latently infected after exposure to FHV-1. Reactivation in 45% of these will occur and cause recrudescent clinical disease or asymptomatic shedding. Recrudescence can occur with stressors such as boarding, illness, surgery, corticosteroid therapy, etc. It is assumed that the virus is reactivated from latency to cause chronic disease. The virus is latent within the trigeminal ganglion with non-neuronal sites of latency possible.

**CLINICAL SIGNS**
Corneal diseases caused by feline herpesvirus-1 (FHV-1) are dendritic ulcers and stromal keratitis. Dendritic ulcers are superficial with a branching pattern that is pathognomonic for FHV-1 infection. They can be difficult to diagnose without the use of Rose Bengal stain, since they can be partial thickness through the epithelium. Dendritic ulcers may coalesce to form geographic ulcers and this the usual presentation. Associated signs include conjunctivitis, blepharospasm and ocular discharge. Occasionally the ulcers will deepen and result in a descemetocele or perforation if secondary bacterial infection occurs.

Stromal keratitis is evidenced by extensive vascularization and opaqueness to the cornea. The opaque-ness is due to corneal edema and cellular infiltration. Associated geographic ulcers are usually present. Blepharospasm and conjunctivitis can be moderate to severe.

**DIAGNOSIS**
On the basis of history and clinical signs, a diagnosis of acute FHV-1 infection is made. Laboratory tests to aid in the diagnosis include conjunctival cytology, virus isolation, immunofluorescent-antibody test (IFA) and polymerase chain reaction (PCR). Cytologic exam of conjunctival scrapings is used to look for intranuclear inclusion bodies, which are extremely difficult to find. Usually lymphocytes and macrophages seen with neutrophils noted in chronic infections.

IFA is a common technique used to identify FHV-1 infection from corneal or conjunctival smears. It has been shown that only 8.8% of cats with chronic conjunctivitis were positive for FHV-1. In experimentally infected cats, only 20% were positive for FHV-1 by IFA versus 95% for virus isolation. There is also the potential of false positives if fluorescein dye is used before collection of ocular samples.

Virus isolation is the gold standard. Samples need to be
collected with Dacron or cotton swabs, since alginate swabs inhibit the growth of herpesvirus. Virus isolation on clinical specimens is impractical, since delayed handling and temperature in transport affect it. Virus isolation has shown to be insensitive with chronic FHV-1 infections. PCR is a highly sensitive test, which makes interpretation difficult. At this time, no laboratory performs this test reliably.

**TREATMENT**

In the treatment of keratitis, the most effective medication is trifluridine (Viroptic® – every hour x 1 day then every 4 to 6 hours). Other less efficacious but less expensive therapies are idoxuridine (5 times/day) and vidarabine (Vira-A® – 5 times/day. Antiviral medications are virostatic, expensive and frequently irritating. Tear supplementation is often used if tear production inadequate. Oral lysine dosed at 250 to 500 mg/cat may prevent or reduce the severity of ocular infections. It prevents viral replication by competitive inhibition with arginine for incorporation into viral proteins, competitive inhibition of arginine transport and induces arginase. Corticosteroids are contraindicated in FHV-1 infections since they can make the infection worse and more protracted. If a stromal keratitis is nonresponsive to topical antivirals and no ulcers are present the addition of corticosteroids (or cyclosporine) to the antiviral therapy can be used with extreme caution. Chronic cases can be extremely frustrating to treat and the prognosis for permanent recovery is poor.

**FELINE CORNEAL SEQUESTRUM**

**CLINICAL SIGNS**

The sequestrum is usually in the axial or paraxial cornea, oval and pigmented. The pigmentation can range from amber to black. The corneal epithelium overlying the sequestrum ulcerates as the lesion becomes more heavily pigmented. Sequestrums can encompass the full thickness of the stroma but tend to affect only the anterior stroma. Associated neovascularization, perilesional edema and inflammation are common. The condition is usually unilateral, except in cats with a breed predisposition. Associated blepharospasm and epiphora with black tear staining of periocular hairs can be evident.

**ETIOLOGY**

The exact cause of this condition is unknown. Himalayans, Siamese and Persians are predisposed which may suggest an inherited stromal dystrophy. Alternatively the predilection for brachycephalic breeds may result from chronic ocular irritation due to lagophthalmia. The presence of concurrent entropion, distichiasis, ulcerative keratitis, keratoconjunctivitis sicca or viral keratoconjunctivitis also suggests the theory that sequestration may be secondary to corneal irritation. HJV-1 detection in sequestrum has been documented at 55% and 18% via PCR, 73% via immunoperoxidase and 23% via IFA.

**DIAGNOSIS**

A centrally located pigmented lesion on the cornea of a cat is pathognomonic for a corneal sequestrum. Histopathology of keratectomy specimens shows degenerative collagen with a surrounding zone of inflammatory cells with no overlying epithelium.

**TREATMENT**

The depth of the lesion and the amount of ocular discomfort dictate the management of sequestrum. If the sequestrum is not causing any ocular discomfort and there is no possibility of full thickness corneal perforation, it may be allowed to slough naturally. During this time, the cat is treated with a topical artificial tear ointment. It may take months for the sequestrum to extrude from the cornea. Surgical approach to removing sequestrums involves either a superficial keratectomy alone or with a conjunctival pedicle graft or a corneoconjunctival transposition. Recurrence may happen even after surgical intervention. There is anecdotal evidence that recurrence is less likely with conjunctival graft placement.

**EOSINOPHILIC KERATOCONJUNCTIVITIS**

**CLINICAL SIGNS**

The presentation can be variable. In the initial stages, the lesions are usually unilateral but progress bilaterally. Corneal infiltration and neovascularization of the dorsolateral and ventrolateral quadrants are most common. The lesion is typically a proliferative, edematous, white to pink vascularized plaque with superficial creamy-white deposits that are easily removed. The condition will involve the whole cornea if the condition is allowed to progress. Associated corneal erosions can be present. Cats usually have mild blepharospasm and ocular discharge. The conjunctiva is hyperemic and edematous with thickening of the eyelid margins and third eyelid. Occasionally, only the conjunctiva and third eyelid may be involved. Conjunctivitis cases may be unilateral or bilateral with degeneration and erosions on the eyelid margin and blepharospasm.

**ETIOLOGY**

The etiology of eosinophilic keratoconjunctivitis is unknown at this time. Feline herpesvirus association with eosinophilic keratoconjunctivitis has been demonstrated in two separate studies. In one, 76% of corneal scrapings were positive for FHV-1 via PCR and in the other 33% of conjunctival and corneal scrapings were positive for FHV-1 via IFA.
**DIAGNOSIS**

Corneal scrapings are obtained using the blunt end of a #15 Bard-Parker scalpel blade after application of a topical anesthetic (proparacaine). Diff-Quik stain shows variable amounts of eosinophils, mast cells, lymphocytes, plasma cells and neutrophils. The presence of eosinophils or mast cells is considered diagnostic of eosinophilic keratitis but some scrapings will be devoid of eosinophils. Histopathology, via superficial keratectomy, shows superficial corneal stromal neovascularization and invasion with eosinophils, mast cells, macrophages, plasma cells and lymphocytes.

**TREATMENT**

Corticosteroids applied topically are the mainstay of treatment. Dexamethasone or prednisolone-acetate can be used initially 4 times a day then slowly tapered over a few months as the lesions regress. Due to frequent recurrence, medications will need to be used on a PRN basis. Oral prednisone and topical cyclosporine can be used.

The other form of treatment is Megestrol Acetate 2.5 mg once daily for 7 days, then tapering slowly to 2.5 mg per week. Due to adverse reactions such as increased appetite and weight gain, behaviour change, mammary hyperplasia, adrenocortical suppression and diabetes mellitus, megestrol acetate is not recommended unless the case is refractory to steroids. I have never needed to use this treatment.

Corticosteroids are contraindicated in FHV-1 infections since they can make the infection worse and more protracted. If a stromal keratitis is nonresponsive to topical antivirals and no ulcers are present the addition of corticosteroids (or cyclosporine) to the antiviral therapy can be used with extreme caution. Chronic cases can be extremely frustrating to treat and the prognosis for permanent recovery is poor.

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GLAUCOMA - Definition [<Gr. glaukos, gray, glistening]: A group of diseases characterized by increased intraocular pressure (IOP), resulting in damage to the ganglion cells and optic nerve.

It is the most frequent cause of irreversible blindness in dogs.

ANATOMY AND PHYSIOLOGY

The aqueous humor (AH) is the transparent fluid that fills the anterior and posterior chamber. It supplies the inner cornea and lens with nutrients.

AH is formed by three mechanisms; diffusion, ultrafiltration (hydrostatic pressure difference between capillaries in the ciliary body and the posterior chamber) and active secretion. Active secretion is the most important mechanism in AH formation. Carbonic anhydrase catalyzes the reaction: CO2 + H2O ⇔ HCO3- + H+. The bicarbonate ion is followed by water into the posterior chamber.

The AH is produced at a relatively constant rate and flows into the posterior chamber. From there it moves into the anterior chamber then into the iridocorneal angle (the area between the cornea and the iris). The pectinate ligaments span the ICA.

The AH then flows either through the conventional or unconventional pathway. In the conventional route the AH flows into the trabecular meshwork then into the scleral venous plexus and into the circulation (1) and (2) (ciliary, conjunctival and vortex veins). The unconventional route (3) is independent of IOP and accounts for a small amount of the aqueous humor outflow (15% in dogs and 3% in cats). The AH flows into the stroma of the iris, ciliary body and choroid, then into the systemic circulation. The rate of AH production equals AH outflow in normal dogs.

CAUSES

The IOP elevation is due to decreased aqueous humor outflow. Decreased aqueous humor outflow is caused by either primary or secondary ICA abnormalities as viewed by gonioscopy.

Primary ICA abnormalities are: 1) Goniodysgenesis - A continuous sheet of tissue bridging across the ICA in place of pectinate ligaments. Usually a few flow holes are present; 2) Narrow Angle Glaucoma - The ICA progressively narrows and causing the IOP to rise. As the IOP increases the ICA narrows and may collapse which results in a sudden increase in IOP and; 3) Open Angle Glaucoma - Most common cause of glaucoma in humans but rarely seen in dogs. Primary glaucomas are considered to be inherited condition that is ultimately bilateral. The list below is the most common breeds that I have documented primary glaucoma. Primary glaucoma is rarely seen in cats.

BREEDS PREDIPOSED TO PRIMARY GLAUCOMA

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<td>Akita</td>
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<td>Chow Chow</td>
<td>Siberian Husky</td>
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Secondary ICA abnormalities are: 1) Anterior Lens Luxation - The lens causes collapse of the ICA. Considered to be an inherited condition in terriers, Border collie and Shar Pei; 2) Anterior Uveitis - Glaucoma occurs acutely due to blockage of the ICA with inflammatory cells and fibrin or in chronic cases by peripheral anterior synchiae or posterior synchiae (iris bombé). Most common cause of glaucoma in cats: 3) Intraocular neoplasia and: 4) Hyphema.

**CLINICAL SIGNS**

Clinical signs of glaucoma can be very subtle in the early stages and can be interpreted as simple conjunctivitis. Injection of the episcleral vasculature is a result of venous blood shunting to more superficial vessels. The endothelial cells of the cornea are responsible for maintaining the cornea as a relatively dehydrated state. The elevated pressure damages these cells and hydrostatic pressure forces aqueous into the corneal stroma causing corneal edema. Blepharospasm indicates pain from stretching the nociceptive nerves in the choroid/sclera. The iris sphincter muscle undergoes ischemic necrosis and mydriasis results. Mild aqueous flare is evident in cases due to damage to the epithelial cells in the ciliary body allowing leakage of protein into the posterior chamber. Vascular and mechanical injury to the retinal ganglion cells and optic nerve cause decreased vision/blindness.

Chronic changes include the above clinical signs as well as the ones listed below. The sclera is able to stretch with chronicity and buphthalmia is clinically noted. Due to buphthalmia the zonules holding the lens rupture and lens subluxation is noted. Usually the lens is posteriorly luxated and incomplete (subluxation – as seen by an aphakic crescent). Optic disc cupping occurs with loss of ganglion cells and mechanical compression on the lamina cribrosa. The pressure may eventually return to normal due to extensive damage to the ciliary body.

**DIAGNOSIS**

Normal IOP is between 15 to 25 mmHg based on determining accurate IOP via tonometry. The Shiotz tonometer places a known weight on the cornea and the distance that weight can indent the eye is measured. The force resisting indentation (which is the IOP) can be calculated from the calibration tables. The applanation tonometer only requires very slight touching of the cornea and aggressive pressure readings will be record elevated IOP. The condom on the applanation tonometer should be placed on snugly but not too tight. Both tonometers should be cleaned regularly.

Some cases of glaucoma will have intermittent blindness described by owners and on examination the pressure is normal. If it a case where glaucoma is suspected IOP curves should be recommended. This involves IOP checks every 2 hours for 12 to 24 hours. Some cases only have one pressure spike in 24 hours. This pressure spike over the long term can eventually cause significant retinal and optic nerve damage or turn into an acute glaucoma crisis.

Gonioscopy is performed to assess the ICA. Usually done on the “normal” eye since the cornea can be edematous or the pupil dilated, obscuring the ICA, making examination difficult. The ICA in one eye looks like the other eye.

**THERAPY**

Once the diagnosis is established the potential to restore or preserve vision must be assessed in order to plan a course of treatment. Obviously if vision is present the ability to maintain vision is very good. If vision is absent it is necessary to determine how long the pressure has been elevated (are signs of chronicity present – very, very poor prognosis). Glaucoma can cause irreversible vision loss in a matter of days depending on the severity of IOP elevation. A study has shown necrosis and apoptosis of ganglion cells in retinas affected ≤ 1 day and end-stage retinal atrophy by day 7 in primary glaucoma cases.

In cases for potential preservation of vision, medical therapy should be instituted to lower the IOP as quickly as possible. The goal is to reduce the pressure to < 10 mmHg (the lower the better). I am not happy getting the pressure < 25 mmHg. Emergency therapy for primary glaucoma cases that I use is as follows:

1) Xalatan 1 gtt - Check IOP in 30 minutes

2) If IOP < 25 mmHg - Recheck IOP 2 h
   >30 mmHg - Cosopt 1 gtt q 15 min x 4 tx and 2.5 - 5 mg/kg Methazolamide
   PO - Check IOP in 30 minutes

3) If IOP > 30 mmHg - Go to # 4 or Try IV Mannitol 1 - 2 mg/kg over 30 minutes - Check IOP in 30 minutes - probably will not work

4) If IOP > 30 mmHg - Surgery to save vision

Continue medical therapy to maintain pressure in normal range. Medical therapy is often temporary (patients become refractory) or may not be effective so long term reduction of IOP nearly always
requires surgical intervention. If surgery is an option it should be done before significant optic nerve damage has been done.

Medical therapy should also be initiated on the apparently normal eye in primary glaucoma cases since this eye is a very high risk of developing glaucoma. Prophylactic therapy has been shown to delay the onset of glaucoma, on average, to 31 months after the initial visit. Patients not receiving any medications developed glaucoma, on average, 8 months after the initial visit. Prophylactic therapy also forces the owner to examine the eye twice daily and hopefully this will allow for earlier detection of any abnormalities.

In cases of glaucoma secondary to lens luxation lens, removal is indicated on an emergency basis. Glaucoma secondary to anterior uveitis is best managed with medical therapy to control the inflammation (topical +/- oral corticosteroids) and the pressure (this can be a very unrewarding experience). If the anterior uveitis is secondary to lens induced uveitis then immediate removal of the cataractous lens is indicated.

MEDICAL THERAPY

The following is a list of the most commonly used anti-glaucoma medications, mechanism of action, side effects, clinical application, trade names and dosage recommendations:

1) Prostaglandin Analogue:
   a. Mechanism of Action: Reduce IOP by increasing unconventional outflow. Can be very effective in lowering IOP.
   b. Side Effects: Miosis (can be very pronounced), conjunctival irritation and may worsen uveitis. Do not use in anterior lens luxations since it may result in pupillary blockade and worsening of the glaucoma. This is an expensive medication.
   c. Clinical Application: Can be used for acute glaucoma (the only negative is that it can make examination of the optic nerve difficult post treatment) and long term management of glaucoma.
   d. Latanoprost (Xalatan®) - Once daily is fine for most patients but resistant cases may require twice daily regimen. Acute glaucoma cases can respond within 30 minutes.
   e. Travaprost (Travatan®) – Once daily to twice daily.
   f. Bimatoprost (Lumigan®) – Once daily.
   g. Unoprostone isopropyl (Rescula®) – Once daily.

2) ß-Blockers:
   a. Mechanism of Action: Reduce IOP by decreasing the formation of aqueous humor. Very weak IOP lowering effects.
   b. Side Effects: Miosis (very mild) and reduces ability to heal epithelial defects (therefore do not use with corneal ulcers). Contraindicated in patients with lower airway disease or heart failure.
   d. Timolol (Timoptic®) – Use the 0.5% solution not the 0.25% solution. Two times daily application.

3) Carbonic Anhydrase Inhibitors (CAI):
   a. Mechanism of Action: Reduce IOP by decreasing the formation of aqueous humor (reduces active secretion by the non–pigmented epithelium of the ciliary body)
   b. Side Effects: Topical medications are well tolerated but may exacerbate anterior uveitis (unexplained phenomena) and can cause ocular irritation. Oral CAIs cause GI disturbance and metabolic acidosis (panting resolves within the first few weeks of starting treatment).
   c. Clinical Application: Acute glaucoma and long term management.
   d. Dorzolamide (Trusopt®) - Three times daily application.
   e. Brinzolamide (Azopt®) - Three times daily application.
   f. Methazolamide (Neptazane®) – 2.5 mg/kg to 5 mg/kg orally two to three times daily.
   g. Dorzolamide + Timolol (Cosopt®) - Two times daily application. I use this more frequently than dorzolamide alone.

4) Parasympathomimetics:
   a. Mechanism of Action: Reduce IOP by increasing outflow through the conventional pathway. Opens up the iridocorneal angle thereby decreasing resistance to aqueous humor passage.
   b. Side Effects: Miosis (can be severe) and local irritation due to low pH. May exacerbate anterior uveitis. Do not use in anterior lens luxations since it may result in pupillary blockade and worsening of the glaucoma. GI disturbance can also occur.
Clinical Application: Long term management of glaucoma.

Pilocarpine (0.5% to 8%). Three to four times daily.

5) Hyperosmotics:
   a. Mechanism of Action: Decreases ultrafiltration by the ciliary body. Reduces volume of the vitreous and aqueous humor with resultant decrease in IOP. Due to the volume loss in the vitreous the iris face moves posteriorly and thereby opens up the iridocorneal angle.
   b. Side Effects: May overload cardiovascular system due to fluid overload with resultant pulmonary edema. Cerebral dehydration manifested as nausea, vomiting and altered consciousness. Avoid in patients with renal failure. Less effective if uveitis present since mannitol will leak into the vitreous and aqueous humor.
   d. Mannitol – 1 to 2 g/kg IV over 20 to 30 minutes. Works within 30 minutes to one hour. Do not give water for 2 to 3 hours.

6) Calcium Channel blockers:
   a. Mechanism of Action: Retinal ganglion cells (RGC) are injured in glaucoma due to mechanical and/or ischemic insults. The RGC release intracellular glutamate, which over stimulates glutamate receptors on surrounding RGC causing calcium homeostasis (levels in the cell increase) to be disrupted. This RGC dies and the cycle continues. Therefore once the damage begins it can be difficult to halt.
   b. Side Effects: Hypotension, therefore monitor blood pressure.
   c. Clinical Application: Acute glaucoma as a neuroprotective agent. May protect against glutamate-mediated excitotoxicity.
   d. Amlodipine besylate (Norvasc®) – 0.2 mg/kg once daily (use for the first few days).

SURGICAL TECHNIQUES TO SAVE VISION

Surgical therapy is most effective if employed before significant optic nerve and ganglion cell damage has been done. The following surgeries can be done separately or in conjunction with each other.

1) Laser Cyclophotocoagulation (laser CPC):
The theory is to destroy part of the ciliary body with subsequent decrease in aqueous humor production. The pigmented cells of the ciliary body absorb the energy from the laser (780 to 850 nm) and coagulation necrosis occurs. A contact diode laser is applied to the sclera 4 mm posterior to the limbus (approximately 30 spots using 1000 mW for 5000 ms). Complications include uveitis, cataaracts and corneal ulceration. The pressure may increase initially so frequent IOP recordings are needed immediately post-op (I will keep them in the hospital until the pressure stabilizes). Can take one to two weeks for the full effect to be appreciated. Can be very successful treatment. If the IOP spikes again it can be repeated.

2) Gonioimplant:
A shunt is placed in the anterior chamber and exits under the conjunctiva (increases aqueous humor outflow). Main complication is fibrosis over the tube under the conjunctiva, which prevents fluid outflow. Fibrosis needs to be resected and the implant will function again. The implants I place are mainly to control the IOP in the immediate post-op period after laser CPC.
SURGICAL TECHNIQUES FOR THE BLIND EYE

The following procedures are recommended only for non-visual eyes. In humans IOP over 35 mmHg is considered painful so if the IOP is greater than 35 mmHg I recommend one of the procedures listed below.

1) Enucleation:
Should be 100% successful. Procedure can be viewed in most surgical texts. Transpalpebral technique used for cases with corneal infection to decrease risk of post-operative infection. Should send eye in for histopathology to determine diagnosis.

2) Evisceration:
Can be used for chronic primary glaucoma and lens luxation cases. A 120o incision through the dorsal conjunctiva (approximately 3 to 5 mm behind the limbus). Incise sclera for 120 o (do not incise the uveal tract). Insert cyclodialysis spatula between uvea and sclera and rotate to separate. Note – there will be bleeding in this procedure. Remove uvea (should only be attached at the optic nerve), lens and retina. Place silicone ball (2 mm larger than limbus to limbus diameter of normal eye) into globe with sphere introducer. Close sclera and conjunctiva (6-0 vicryl). Complications include corneal ulcers, infection and wound dehiscence. Procedure done for cosmetic results. Do not use if secondary glaucoma suspected due to bacterial or fungal infections or neoplasia. Post-operative medications include topical ophthalmic ointment, oral antibiotics and anti-inflammatory.

3) Pharmacologic Ciliary Body Ablation:
Inject 25 mg gentamicin (do not exceed the patient’s daily dose) and 1 mg dexamethasone into vitreous (20 gauge needle inserted 6 to 8 mm posterior to limbus directed towards the optic nerve). Can try to aspirate 1 cc of vitreous prior to injection but I usually find the vitreous too viscous. Variable results, from phthisis bulbi (10%) to inadequate pressure control (effective in lowering pressure in 65% of cases). Chronic inflammation may result. May take a month for the pressure to lower. Do not use for intraocular neoplasia or infection. Less effective in primary lens luxation cases. I reserve for patients that are at risk for complications with general anesthesia.

BIBLIOGRAPHY


STROMAL ULCER AND DESCemetOCELE

CLINICAL SIGNS

An ulcer that invades the stroma of the cornea results in a facet. Stromal loss secondary to bacterial infection in most cases. If the defect reaches Descemet’s membrane it is considered a descemetocoele. Usually painful but some patients seem comfortable. These can be sterile or infected.

DIAGNOSIS

Fluorescein stain will be retained by the stroma if it is present. Descemet’s membrane is lipophilic and repels the stain therefore appears as a clear area in the center of a depression. The depression associated with the descemetocoele may retain fluorescein stain but if eyewash used to flush the defect and patient not allowed to blink the clear area will be noted. Cytology of the defect edge advised.

TREATMENT

Initially superficial stromal ulcers are managed the same as superficial ulcers. Typical antibiotics are tobramycin or moxifloxacin. If the ulcer progressively deepens adjunctive therapy is required and reassess for any underlying disorders. Cyanoacrylate tissue adhesive can be used to treat ulcers that have not progressed to a descemetocoele. Deep ulcers (>75% thickness) and descemetocoeles require reinforcement of the cornea, which can be provided by a conjunctival graft. These are ocular emergencies and require surgical intervention. Conjunctival grafts supply fibrovascular tissue to fill in the stromal defect. The redundancy and loose attachments allow the bulbar conjunctiva to be resected and relocated. Options include the pedicle graft or the bridge graft.

The conjunctival pedicle graft is illustrated below (Note: In the illustration the patient in dorsal recumbency with the surgeon located dorsal to the 12 o’clock position of the globe). Remove any necrotic debris and epithelium from the ulcer (if the graft is sutured to an epithelialized surface it will not adhere). The base of the graft will be located at the 12 o’clock position. A) I start my initial incision (with scissors) perpendicular to the limbus at approximately at 3 o’clock (use the dorsolateral conjunctiva since it is the most easily accessible). The length of this incision should be 10% larger than the lesion. B) The next incision is through the fornix-based conjunctiva and ends at the lateral most extent of the ulcer. The limbal-based incision is extended to the medial extent of the lesion. Remove Tenon’s capsule (dense white tissue) under the conjunctiva. If not removed it will cause graft retraction and dehiscence. The graft is placed over the defect and it should not contract (therefore little tension is placed on the graft). C) Graft sutured to the edge of the ulcer into healthy corneal stroma with 7-0 to 8-0 Vicryl® in a simple interrupted pattern (1 to 2 mm apart).

The main causes of graft dehiscence are aqueous leakage, melting ulcer, too much tension on the graft and a graft direction of more than 45o from the vertical.

The bridge graft has a blood supply feeding the graft from both sides. A) The graft is created by making two parallel incisions in the conjunctiva with the first at the limbus. The graft should be at least 10 mm wide to ensure adequate perfusion. Again Tenon’s capsule should be dissected off the graft. B) Both edges of the graft are then sutured to the cornea.

Post-operative care is the same for any ulcer (antibiotics, atropine, E-collar). Systemic antibiotics will now be able to reach the corneal ulcer as a blood supply has been created. Six to 8 weeks post-operatively the graft is severed from its base and this can be done using topical anesthesia alone. The graft is only adhered to the cornea where no epithelium existed. I do not cut grafts on eyes with KCS that have not responded to medications (anecdotal evidence indicates decreased ulcer risk over the area covered by the graft if blood supply left intact). Within a few months the graft turns into a scar. Depending on the size of the graft most patients have useful vision after the procedure.
MELTING ULCER

CLINICAL SIGNS
Melting ulcers have an acute onset of blepharospasm, mucopurulent discharge, conjunctival hyperemia, moderate to severe corneal edema, and gelatinous ulcerated area with yellow/white stromal infiltrate. Corneal vessels, hypopyon, aqueous flare and miosis may be present. Vision impairment may result due to severe corneal scarring, corneal perforation, endophthalmitis and phthisis bulbi.

PATHOPHYSIOLOGY
Proteolytic enzymes aid in the removal of devitalized cells and debris during normal corneal healing. Keratomalacia is a result of excessive levels of proteolytic enzymes that degrade the extracellular matrix of the cornea. Sources of proteases are the cellular components of the cornea itself, inflammatory cells and infecting bacteria, fungi and yeast. The two major endogenous proteases are matrix metalloproteinases (MMPs) produced by corneal epithelial cells, keratocytes and PMNs, and serine proteases, liberated by PMNs. Topical corticosteroids increase the effects of proteases, decrease defense mechanisms and inhibit corneal epithelialization (therefore contraindicated!)

DIAGNOSIS
Cytology of the ulcer is performed using topical anesthesia and the blunt end of a scalpel blade. Sample the thickest part of the ulcer to prevent inadvertent rupture of the cornea. The slide is stained then examined under oil immersion to assess for bacteria and PMNs (typical melting ulcer contains numerous PMNs). Antimicrobial therapy is based on initial cytology of the corneal ulcer. Bacterial (+/- fungal) culture and sensitivity is recommended. Be careful to only touch the ulcer and not the eyelids. Some of these ulcers have no bacteria present.

TREATMENT
Aggressive medical therapy is recommended for an ulcer with keratomalacia to prevent further loss of stroma. The animal may need to be hospitalized to assure proper treatment (from 1 to 7 days). An E-collar should be placed on the patient to minimize self-trauma. If trichiasis is present cut the hairs. Atropine topically to prevent ciliary body muscle spasms.

If gram-positive cocci are seen a 50 mg/ml solution of cefazolin is recommended (keep refrigerated). Ciprofloxacin is a good broad-spectrum antibiotic but resistant strains of Streptococcus sp. have been isolated from melting ulcers. Moxifloxacin is a better choice. Tobramycin is the antibiotic of choice for gram-negative rod infections. Antibiotics should be administered every 2 hours until the melting has abated.

Anti-collagenase therapy halts the melting induced by the proteases. Numerous anti-proteases have been recommended and include the following: 1) Autogenous serum – contains α2-macroglobulin, a non-specific protease inhibitor, and α1-antitrypsin, a serine protease inhibitor. These inhibit the MMPs and serine proteases, with resultant arrest of corneal degradation. Keep the serum refrigerated (discard in 5 to 7 days) and sterile (since it makes a very good bacterial growth media). Applied every 2 to 4 hours depending the severity of the lesion and frequency is reduced with cessation of melting; and 2) MMPs require zinc and calcium to be active so any metal-chelating agent will inhibit collagenolysis. Tetracycline (doxycycline), 2 to 10% acetylcysteine and EDTA can be used every two to four hours. I do not commonly use these medications. Because they have different mechanisms of action combination therapy may be beneficial. Continue treatment until ulcer starts to heal as noted by decreased blepharospasm and ulcer area decreasing.

Surgical therapy to help heal the keratomalacia is indicated if medical therapy proves ineffective or the ulcer as at risk of rupturing. Ideally the melting should be stabilized prior to graft placement to prevent dehiscence. The surgical approach is to perform a keratectomy, which removes the necrotic tissue, organisms and proteases, thereby decreasing the stimulus for further corneal degradation. Conjunctival grafts deliver a direct blood supply, including anti-collagenases and antibiotics, and support the keratectomy site. Therefore serum and other anti-collagenases can be discontinued.
CORNEAL PERFORATION/IRIS PROLAPSE

**CLINICAL SIGNS**
If the penetrating object or ulcer breaks through Descemet’s membrane the iris usually prolapses, combines with a fibrin plug, results in a temporarily seal and the anterior chamber reforms. The iris prolapse and fibrin plug appear as a red tissue elevated off the cornea in the center of the lesion. Aqueous leakage can be determined using the Seidel test. When an ulcer ruptures the owners describe the dog vocalizing and the face is noted to be wet.

**TREATMENT**
This is a surgical emergency. The clot is not disturbed until surgery. In most cases the iris is replaced into the anterior chamber unless it appears necrotic or infected then that part is excised. The iris is usually adhered to the edge of the lesion so it has to be teased off. The clot in the anterior chamber is removed. To create a seal porcine collagen (Biosist® and A-Cell®) are sutured in the defect using 8-0 Vicryl®. A conjunctival graft is then placed over the lesion so that it fully covers the collagen. Treatment is the same as after a conjunctival graft.

Some full thickness perforations (like cat claw injuries) may be self sealing and only require topical therapy to allow the defect to heal.

**CONCERNS**
The most common sequela to a perforation is anterior synechiae. If these are extensive vision will be diminished or extinguished.

Rupture to the anterior lens capsule may occur with penetrating trauma (very common with cat claw injuries). This is associated with miosis and moderate to severe aqueous flare. Failure to remove the lens with a ruptured capsule is associated with a chronic and severe anterior uveitis since the cortical material incites an inflammatory reaction. If the lens is not removed the eye either develops secondary glaucoma or phthisis bulbi.

CORNEAL LACERATION

Sharp trauma to the cornea may cause variable thickness lacerations to the cornea. Partial thickness lacerations may only require medical management. If the laceration is deeper, apposition of the edges can be done with 7-0 to 9-0 suture material in a simple interrupted pattern. Suture material may be left in place or can be removed in 3 weeks to lessen vascular response and scar formation. With full thickness lacerations, place sutures 1 mm apart and 75-80% of the corneal thickness. Once the sutures are placed the anterior chamber is re-inflated with lactated ringers or air bubble. Treatment includes topical antibiotics and atropine and oral antibiotics and anti-inflammatory. If the laceration extends to the limbus dissect the conjunctiva to assess if the laceration extends into the sclera. If it extends into the sclera this is a poor prognostic indicator.

Sharp trauma to the cornea may cause variable thickness lacerations to the cornea. Partial thickness lacerations may only require medical management. If the laceration is deeper, apposition of the edges can be done with 7-0 to 9-0 suture material in a simple interrupted pattern. Suture material may be left in place or can be removed in 3 weeks to lessen vascular response and scar formation. With full thickness lacerations, place sutures 1 mm apart and 75-80% of the corneal thickness. Once the sutures are placed the anterior chamber is re-inflated with lactated ringers or air bubble. Treatment includes topical antibiotics and atropine and oral antibiotics and anti-inflammatory. If the laceration extends to the limbus dissect the conjunctiva to assess if the laceration extends into the sclera. If it extends into the sclera this is a poor prognostic indicator.
Selection of Patients for Treatment with Psychotropic Medication

Diane Frank, DMV, DACVB

Which patients should we medicate? Drugs in behavioural medicine are generally prescribed to decrease anxiety or reactivity, thus facilitating implementation of behaviour modification techniques and in some cases accelerating rate of progress. Antidepressants can help achieve these goals if patients are carefully selected.

Anxiety

Anxiety in humans is defined as the anticipation of a future danger or threat, real or imaginary. Anxiety can be normal or a sign of an illness. This definition can also be used for animals. Most dogs and cats presented to the veterinarian are fearful or anxious. Some will remain anxious as long as they are on the table but are fine when they are back on the floor or out the door from the veterinary hospital. These patients are normal. On the other hand, separation anxiety, panic disorder, generalized anxiety, phobias and obsessive-compulsive disorders are sub-groups of anxiety disorders.

Behaviours and body language during the appointment

During a behavioural consultation, the animal is usually free to move around the room. Physical examination is done at the end of the appointment. Dogs can express anxiety (and/or fear) by panting, puffing their cheeks, crinkling their brow, yawning repeatedly, licking their lips constantly, pulling their ears backs, trembling, tucking their tail, trying to back up or escape, hiding, whining, or even seeking owner attention excessively. Many anxious dogs will have dilated pupils, will pace, and are unable to settle down and relax. Each sign is non-specific. Anxiety (or fear) can result in urination, defecation or excessive salivation. Finally aggression may also be a sign of anxiety. Similarly cats can express anxiety (and/or fear) by panting, licking their lips frequently, pulling their ears down and back, arching their back, tucking their tail, trying to escape, vocalizing, pacing, freezing or being aggressive. Each sign is non-specific.

Some animals will have increased motor activity whereas others will have decreased motor activity. Vigilance may be increased. Reactivity during the appointment may be exaggerated and may even increase over time. Exploratory behaviour of the consultation room may be absent and should be distinguished from increased motor activity. These behaviours (also compatible with anxiety) serve as baseline and can be compared with behaviours expressed during follow-up visits.

Anxiety during the behavioural consultation is not sufficient to conclude that a given animal suffers from an anxiety disorder. But clients during the appointment are educated on how to recognize subtle signs of their animal's anxiety or fear. Following the appointment they will be much more attentive to the animal's body language and behaviours. They may realize that their animal is exhibiting signs of anxiety on a daily basis in the home environment in the absence of an identifiable cause. In the latter case, their animal is perhaps suffering from an anxiety-related disorder. The veterinarian should make it a point to ask if signs of anxiety (or fear) occur when the dog or cat is in its familiar environment and determine if that anxiety/fear is appropriate for the context.

Reactivity

Excessive reactivity can also be an indication of illness. An animal becoming more and more aggressive during the appointment in the absence of any threat may be “over-reactive”. An animal becoming disobedient may in fact be “over-reactive” in that context. This animal is unable to hear (“emergency mode”) any commands. Ask a person if following a near miss car accident, he/she would be able to tell what song had just played on the radio at the time of that close call… The ears may have heard the song but the brain did not register the information, as it was not essential for survival… Dogs and cats in “emergency mode” will require medication to decrease the level of reactivity.
Videotapes

Objective baseline data are obtained from tapes. Videotapes of the animal at home may reveal signs compatible with anxiety and in some cases may even be indicative of generalized anxiety. Videotapes are essential to confirm diagnosis of separation anxiety as well as assess response to pharmacological treatment. Tapes are also very useful to identify occurrence of silent threats occurring between household pets (inter-cat or inter-dog aggression) that are often unrecognized or missed by clients.

Indications for antidepressant medication

1. Signs compatible with generalized anxiety in familiar environments in the absence of danger or threat
2. Reactivity during the appointment for behavioural evaluation increases over time without any threat to the animal
3. Excessive reactivity to benign stimuli
4. Behaviour sequence is altered (other medical conditions ruled out)
5. Behaviour is inappropriate for the context
6. Frequency, severity or duration of the behaviour is excessive for the context
7. Recovery time after an undesirable behaviour is excessive
8. Animal is in “emergency mode” during episodes of undesirable behaviour

Setting up realistic expectations and follow-up

It is very important to distinguish between client request or demand (“fix my dog”) and client expectations (time frame for improvement, amount of improvement necessary to preserve the animal-human bond, realistic expectations, etc.). It is equally important for the veterinarian to have a clear idea in terms of observable behaviour changes expected with a given psychotropic medication so that he/she can educate the owner on what to look for. Initial goal of the medication may be solely to reduce reactivity so that the dog or cat can hear the client giving instructions. If an animal stays reactive for a long time following the initial trigger (for example a thunderstorm phobic dog still shaking two hours after the end of the storm), the first goal may be to decrease duration of recovery time. Then, the next step may be to reduce frequency and duration for some of undesirable behaviours such as pacing or whining. By taking care to collect accurate baseline values (frequency, duration, presence of mental illness, “emergency mode”, progression over time, etc.) it becomes possible to 1) set realistic expectations for the role of antidepressant medication in the treatment plan and 2) objectively assess whether a given drug has the desired clinical effects for the patient.

Drugs choices and dosages

Examples of the following criteria will be given with clinical cases and video presentations.

<table>
<thead>
<tr>
<th></th>
<th>DOG</th>
<th>CAT</th>
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</thead>
<tbody>
<tr>
<td>Alprazolam (BZD)</td>
<td>0.01-0.1 mg/kg q8-12h</td>
<td>0.125-0.25 per cat q12h</td>
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<tr>
<td>Buspirone (azaspirone)</td>
<td>1.0-2.0 mg/kg q12h</td>
<td>0.5-1.0 mg/kg q12h</td>
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<tr>
<td>Clorazepate (BZD)</td>
<td>2 mg/kg q12h</td>
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<tr>
<td>Clonazepam (BZD)</td>
<td>0.05-0.5 mg/kg q12h</td>
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<tr>
<td>Clomipramine (TCA)</td>
<td>1-3 mg/kg q12h</td>
<td>0.25-0.5 mg/kg q24h</td>
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<tr>
<td>Diazepam (BZD)</td>
<td>0.5-2.2 mg/kg PRN</td>
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<tr>
<td>Fluoxetine (SSRI)</td>
<td>0.5-1 mg/kg q24h</td>
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<tr>
<td>Fluvoxamine (SSRI)</td>
<td>1-3 mg/kg q12h</td>
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<tr>
<td>Selegiline (MAOI)</td>
<td>0.5-1 mg/kg q24h</td>
<td>0.5-1 mg/kg q24h</td>
</tr>
<tr>
<td>Sertraline (SSRI)</td>
<td>1-3 mg/kg q24h</td>
<td>0.5-1 mg/kg q12h</td>
</tr>
<tr>
<td>Trazodone (atypical antidepressant)</td>
<td>1 mg/kg q12h 7 days then up to 3 mg/kg q12h</td>
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</table>
Conclusion

Behaviour consultations should be handled like any other request for professional advice. First the veterinarian must determine if the behaviour changes are within normal limits or whether these changes are compatible with illness. Differential diagnosis should include the possibility of “mental illness” if clinical signs are compatible. When mental illness is identified, pharmacological treatment can accelerate treatment response and in some cases is actually essential to initiate improvement.

References/Suggested reading


Assessing Dangerousness in Dogs

Diane Frank, DVM, DACVB

Assessing risk of injury from a dog

Dangerousness does not necessarily equate with aggressiveness. A 50 kg enthusiastic excited dog running right into an owner and knocking down or injuring that person is dangerous. On the other hand, a growling dog that has never bitten is aggressive but not necessarily dangerous. Aggression has been defined as “behaviour that leads to the damage or destruction of a target entity”. Some definitions of aggression also include the display of threats in the absence of injury. Thus aggression encompasses a wide variety of behaviours from subtle body postures and facial expressions to explosive attacks. Aggression can be an expression of normal or abnormal behaviour. Description of the behaviour sequence, context, frequency and severity of aggressive events as well as health status of the dog allows us to tease apart appropriate normal from inappropriate abnormal behaviours.

Appropriateness of the aggressive behaviour given the context

Context is also important to determine if behaviour is normal or illness-related. Behaviour can be inappropriate given the context. If I decide to randomly kick a person on the street given that this person did not even interact with me, kicking is inappropriate behaviour on my part in the described context. If on the other hand I am being mugged on the street and I am kicking my assailant, the same behaviour becomes appropriate. The context makes all the difference. A dog with an otitis bites the veterinarian (or owner). Context will be considered (painful condition, defensive aggression) when interpreting this aggressive event and this dog will likely not be labelled as a high-risk dangerous dog. A dog will bark or growl briefly at the approach of a stranger and then he will wait and watch for the response. Based on the response of the receiver (the stranger in this example), the dog will decide on its subsequent action. The behaviours will depend on the receiver’s response and the dog’s interpretation of the response. If a dog growls snarls and lunges simultaneously without any other form of warning has an altered sequence because there is no clear initiation phase. Aggression can be the result of fear or anxiety. Anxiety is defined as anticipation of a future threat or danger (real or imaginary). Anxious dogs occasionally are unable to tell the difference between real threat and absence of threat. It is therefore important to realize that some aggressive dogs may in fact be ill and suffering from an anxiety disorder.

Behaviour sequence

Behaviour is always a sequence. Observing the entire sequence is essential to determine if an animal is behaving normally or not. In the case of canine aggression we could illustrate the sequence as initial warning such as a growl (initiation), then a pause, then in some cases a bite (action), and finally immediate volitional release (end of sequence). Behaviour becomes “abnormal” or illness-related if some of the steps from the sequence are omitted or altered. A dog growling and biting simultaneously without any other form of warning has an altered sequence because there is no clear initiation phase. Aggression can be the result of fear or anxiety. Anxiety is defined as anticipation of a future threat or danger (real or imaginary). Anxious dogs occasionally are unable to tell the difference between real threat and absence of threat. It is therefore important to realize that some aggressive dogs may in fact be ill and suffering from an anxiety disorder.

Severity and frequency of the aggression given the context

A dog can bark, growl, lift its lips, snarl, snap, bite, and then latch on or release. Bites can be single or multiple, inhibited or uninhibited. Clients are questioned on the severity and frequency of the aggressive events. Severity of bite can in some cases be exacerbated by fear or pain. The veterinarian must determine whether the severity and/or frequency of the aggressive behaviours are appropriate for that given context.
Predictability of the aggressive events

The context (triggers and specific situations) and the dog’s body language are used in determining predictability of the aggressive events. If the dog exhibits only defensive aggression, the events are more predictable. Defensive aggression for the purpose of this presentation is defined as one individual “approaching or entering the animal’s space” and interacting with the animal (touching, handling); the animal reacts aggressively to the approach or physical contact. Offensive aggression for the purpose of this presentation is defined as aggression occurring without interaction (touching, handling or even looking at the animal). The aggressive animal is the one approaching the individual (victim of aggression). The trigger for the aggressive behaviour is often difficult to identify.

Size of the patient

A larger dog can potentially produce more damage.

Health status of the patient

A painful condition may exacerbate anxiety and aggression. Dogs that are behaving abnormally can be more unpredictable and therefore more dangerous.

Social environment (humans and other animals)

Relative risk for a young child unable to read and interpret canine body language can be increased as the child will not understand a warning growl.

Conclusion

Any dog can be aggressive and bite. A zero risk level of bite does not exist for a live animal. So the only way to guarantee that a dog will never bite again is to kill the dog. Assessing level of risk for a specific case requires a complete analysis: the animal, its behaviours and health status, and all interactions in its social environment. Clinical cases will be presented to illustrate various points.

References


Separation Anxiety: Are we all talking about the same cases?

Diane Frank, DVM, DACVB

Undesirable behaviours that occur when the owner is absent have been referred to as separation anxiety, separation-related behaviours or problems, isolation anxiety, separation reactions, separation anxiety syndrome, and attachment problems. Some authors suggest that there is an anxiety component, whereas others omit the term altogether or even disagree that there is truly anxiety. This presentation using video clips generated by owners, will illustrate the range of behaviours exhibited by dogs during owner absences. The importance and necessity of obtaining videotapes will be discussed.

Definition

The necessary conditions for a diagnosis of separation anxiety are physical or behavioural signs of distress exhibited by the animal only in the absence of or during lack of access to the owner. Sufficient conditions for a diagnosis of separation anxiety are consistent, intensive destruction, elimination, vocalization, or salivation exhibited only in the virtual or actual absence of the owner. Behaviours are most severe within the first 15-20 minutes of separation, and many anxiety-related (autonomic hyper-reactivity, increased motor activity, and increased vigilance and scanning) may become apparent as the client displays behaviours associated with the intention to leave.

Diagnosis

Few authors have specifically addressed whether they believe that separation anxiety represents normal behaviour or a behavioural disorder, and age of onset of the condition is rarely mentioned. Information on the actual duration and severity of signs following the owner’s departure in dogs with separation anxiety is still lacking because only 2 studies have looked at videotapes of dogs left home alone. Information is also lacking on the progression of clinical signs over time for a given patient. The diagnosis for most authors seems to rely almost exclusively on owner reports, and the actual behaviours of dogs are rarely videotaped to confirm the diagnosis or assess the response to treatment. This means that different clinical syndromes could perhaps have been grouped under the same category. For example, dogs that vocalize when left alone may be doing so in response to various stimuli in their environment and not truly exhibiting separation anxiety. This inaccurate categorization could explain the discrepancy in opinions on how to treat separation anxiety, as well as explain some treatment failures.

Importance of obtaining video clips

Some of the video clips presented will illustrate that relying on the fact that a dog has eaten treats during an owner absence is not a reliable predictor that the dog is not anxious. Other clips will show dogs vocalizing for different reasons that are identifiable only by visualizing their behaviours and body language.

Review of videotapes obtained during owner absence seems essential to confirm the diagnosis of separation anxiety in dogs. They are also useful to encourage owner compliance, because the owner can actually see the behaviours that occur before and after pharmacologic treatment. Videotapes objectively document the response to pharmacologic treatment and allow the clinician to adjust dosage when necessary.

Some authors will label dogs with separation anxiety even if the dog only exhibits signs of distress associated with a second departure or if clients arrive home later than usual. The dog is fine during the regular daily routine and does not destroy, vocalize or eliminate except if there is a change of schedule. Do we really know that these dogs are fine during regular routines without videotaping? Another added advantage of filming is that it can be used to document signs of anxiety, such as pacing and panting, during an owner’s absence. Owners may in fact be unaware that their dog is distressed (i.e. pacing and panting) during their absence if they do not see concrete evidence of distress (i.e. saliva, urine, or feces) upon their return or do not receive complaints about excessive vocalization while they are gone.
References/suggested reading


Overall KL. Clinical behavioral medicine for small animals. St. Louis: Mosby Year Book Inc, (1997); 518.
Interdog Aggression: When to Intervene?

Diane Frank, DVM, DACVB

Are all cases of inter-dog aggression normal interactions? Is obedience training sufficient or should we medicate? If so, how long should we medicate? Is it the client’s fault or the dog’s “fault”?

Body language and behaviour sequence

Behaviour is always a sequence. Important questions include asking whether aggression was triggered by specific behaviours on the part of the receiving “victim” dog. Was communication between the two dogs clear and appropriate? Description of the body language is important to assess relative roles played by fear, anxiety or predatory behaviour during aggressive events.

Context

Context is also important to determine if behaviour is normal or illness-related. A dog attacking another dog without prior interaction (or communication) is ill. A patient that is unable to make the distinction between threat and absence of threat is ill. Generally adult dogs are very tolerant of puppies. An adult dog attacking a puppy that was not interacting with the former is generally not normal!

Intensity, duration or severity

Dogs can bite without contact (snap) or bite with contact (some may or may not control pressure applied at the time of the bite). Severity of the bite has to be appropriate given the context. A dog causing serious injuries to another dog that had submitted is likely ill or presenting predatory behaviour.

Frequency

A dog challenging another dog expects a response. If the receiver submits or defers, one does not expect the challenger to repeat the same threat or sequence. If a dog repeatedly challenges another dog within the same given context and the recipient is always submitting, we are dealing with a sick dog. A normal dog will not repeat the identical behaviour over and over again to always obtain the same and only response (submission).

Normal behaviour (household dogs)

Several scenarios are possible: The first involves a young animal reaching social maturity (18-36 months) and challenging the older dog. In this scenario, a dog that has been challenged can respond 1) by immediately deferring or submitting, 2) by not submitting initially but eventually doing so if the challenge is intensified, 3) by fighting, with one dog winning and both dogs accepting the outcome or 4) by fighting, and neither dog yielding to the other. These dogs are following the rules of social canine communication and exhibiting complete unaltered behaviour sequences. These dogs are behaving normally and owner intervention will perhaps only be necessary in the last example. These cases are rarely seen in referral practice.

Abnormal behaviour (household dogs)

A different scenario is an older dog perceiving the younger dog as a threat even though the younger dog has not challenged or threatened the older one. Another scenario could involve one dog challenging the other with the latter deferring but the challenger still attacking and injuring the victim. An anxious dog “worried about everything” and simply redirecting the aggression to another household dog is another possibility. These last three scenarios are examples of illness-related behaviours of at least one of the dogs involved. In these cases, clients eventually consult because the fights become more frequent and severe.

Most inter-dog aggression cases seen at the Veterinary Hospital of the University of Pennsylvania (VHUP) Behavior Clinic occurred between housemates. Some dogs were aggressive both to familiar and unfamiliar dogs. Predatory aggression is occasionally a cause “of inter-dog aggression” between housemates or towards an unfamiliar dogs. The behavioural sequence and the history allow us to confirm that diagnosis.
Aggression to unfamiliar dogs

Aggression to unknown (unfamiliar) dogs can occur if one dog is anxious (or fearful) or because it perceives a threat or challenge. This threat or challenge may be real or perceived. The fearful dog may react spontaneously or may have had a traumatic previous experience. It may have been attacked by an unfamiliar dog or may have had inadequate socialization. Again one must detail the behaviour sequence of our patient. Was he responding to a real threat? Or was it a perceived threat that is compatible with illness? General treatment recommendations are similar to those described for fighting between familiar dogs. An anxious-ill dog may be overtly aggressive to non-familiar dogs and may be threatening the other household dogs without the client’s knowledge… Clients often miss silent signs of threats and challenge. These can include blocking access to a location, stealing objects, posturing in a ritualized display where the challenger approaches the other dog’s shoulders in a perpendicular manner (T-challenge). Video clips will illustrate some of these silent threats. The situation may in fact be worse than what the client has perceived.

Treatment

Testosterone stimulates dogs to roam and urine mark and facilitates fighting. Although castration greatly decreases roaming, urine marking and fighting between dogs and appears to be effective in about 60% of dogs, it is not a cure for all cases of inter-dog aggression. In fact most of our patients presented as a referral for inter-dog aggression have already been neutered. So how do we treat them?

Traditionally, emphasis was placed on identifying the dominant dog and reinforcing the dominant status. But ill dogs no longer communicate normally. They also perceive or anticipate dangers or threats that are real or imaginary. Not all inter-dog aggression cases are due to hierarchical conflicts. The ideal situation is to be able to visualize the behavioural sequence (directly or on video). Is each dog signalling appropriately and is each dog reading the signals properly?

Treatment of inter-dog aggression with normal dogs that are fighting is easily achieved by establishing “hierarchy”. The dog that is physically most likely to win will be fed, walked, or given attention first. If the fighting only occurs over special food items or toys, these triggers can simply be removed from the environment.

Treatment of anxious (ill) dogs includes pharmacological intervention, behaviour modification, and occasionally environmental management. Clients are instructed not to use body parts to separate fighting dogs. We are no longer talking about a dominant or submissive dog but instead we are considering behaviours that are normal or illness-related. Dogs are separated when supervision is impossible. The dog behaving appropriately (and normally) is reinforced. This dog receives everything first. He gets attention, food, treats, first. Each dog must sit before any interaction with any given person takes place. They can be fitted with head collars (Gentle Leaders®). Eventually the dogs are taught to sit in each other’s presence and are rewarded for being non-aggressive in each other’s presence. The aggressor can be medicated with clomipramine, fluoxetine, or fluvoxamine. None of these drugs are labelled for use for aggression in dogs. Complete blood cell counts and biochemistry panels are always done prior to prescribing medication for behavioural conditions. Occasionally victims also require medication. Duration of pharmacological therapy varies from case to case.

Conclusion

This approach distinguishing normal patients from ill dogs (anxiety-related disorders) can simplify decisions in terms of treatment. Questions such as which dog should be favoured or which dog should be medicated are easily answered.

References/Suggested Reading


Fears and Phobias
Diane Frank, DVM, DACVB

Definitions

**Fear** - is defined as an aversive emotional state consisting of psychological and physiological responses to a real external threat or danger. Fear is considered adaptive since avoiding or defending oneself against a threat may result in increased chance of survival. Fear can become pathological in some cases.

**Phobia** - A sudden, excessive, profound fear is labelled as phobia. The intensity of a phobia is more extreme than a fear. The signs of phobia may also persist for longer periods following removal of the triggering stimulus. In some cases, the phobia may be triggered in the absence of the initiating stimulus.

**Anxiety** - is described as the apprehension and anticipation of a future real or imaginary threat or danger. Anxiety is in fact also an emotional state associated with adaptive physiological and behavioural responses and only becomes pathological (disorder) when it is exhibited in contextually inappropriate situations or in more natural ones but to a level that impairs effective adaptive responses.

Baseline information for excessive fear or phobias

1. Signs exhibited by the animal (best to have a checklist)
2. Excessive reactivity to benign stimuli during the appointment
3. Duration of signs associated with the fear/phobia, once the stimulus has ended or in other words: time necessary for recovery after the end of the stimulus
4. Is the animal in “emergency mode” during the event?

Treatment

Treatment generally includes behavioural modification (habituation; desensitization and counter-conditioning), and medication. Again it is important to set realistic expectations with the client about their animal.

References

Obsessive-Compulsive Disorders (OCD): Do they really exist?

Diane Frank, DVM, DACVB

Definitions

**Stereotypy** - A stereotypy is a repetitive, constant, behaviour and appears to serve no obvious purpose.

**Obsession** - Obsessions are ideas, thoughts, impulsions or images generally intrusive that cause marked anxiety and distress.

**Compulsion** - is a repetitive behaviour performed in order to prevent or reduce anxiety or distress.

**Obsessive compulsive disorder (OCD)**
In psychiatry, OCD is a disorder in which a person feels compelled to perform certain actions repeatedly to alleviate persistent fears or intrusive thoughts.

**Compulsive disorders or CD (veterinary medicine)** - Some veterinarians do not think that animals can obsess so rather than referring to obsessive compulsive disorders, they will talk about compulsive disorders in animals. Compulsive disorders are defined as behaviours that are usually brought on by conflict but that are subsequently shown outside the original context. The behaviours might share a similar pathophysiology (e.g. changes in serotonin, dopamine and beta-endorphin systems). Compulsive behaviours seem abnormal because they are displayed out of context and are often repetitive, exaggerated or sustained.

Unfortunately these behaviours do not always seem to be out of context, or associated with conflict. Additionally to be labelled a true CD, the repetitive behaviour should occur in the absence of any primary dermatologic, neurologic or other medical condition. However the problem with many cases that one might consider as compulsive disorders is that there might not be a good or affordable way to diagnose an underlying medical problem and in some cases the medical condition might be difficult to treat (i.e. no good treatment available). Ultimately one is still left with trying to manage the behaviour or the consequences of the behaviour.

Examples of listed compulsive disorders in dogs

- Shadow chasing (Border collie)
- Light chasing
- Spinning (English Bull Terrier)
- Spinning/tail chasing (German shepherd)
- Lick granuloma
- Self-mutilation
- Fly biting
- Pica
- Fence running
- Flank sucking (Doberman)
- Checking (Min Schnauzer)
- Excessive licking of surfaces

Examples of listed compulsive disorders in cats

- Wool or fabric eating
- Pica
- Excessive grooming
- Hyperesthesia
- Self-mutilation
- Tail-chasing
Food for thought

There is little published scientific data on compulsive disorders in dogs and cats. Andrew Luescher reported that approximately two thirds of cases improved to the client’s satisfaction. Karen Overall also reported improvement with pharmacological treatment and behaviour modification. Improvement in both cases was defined as a reduction of frequency or duration. A decrease in one or the other of greater than 50% is considered improvement. No publications so far list complete resolution of CD with behavioural treatment. Food for thought…

This presentation will illustrate some cases that presented as CD but in fact had other underlying conditions. Results of a study on dogs exhibiting excessive licking of surfaces will be presented. A case series of fly biting dogs will also be highlighted.

References


The diagnostic tests used most often in veterinary dermatology are skin cytology and skin scrapings. Becoming proficient in these two techniques, and more importantly, taking the time to do them, will take you a long way in diagnosing patients with skin disease. Other tests may be used less frequently, but are still very useful in many situations.

Skin Surface Cytology

Why do skin surface cytology?

Skin cytology is a very high-yield, inexpensive procedure for patients with skin disease. It tells us more about what is going on with the skin, more quickly, than any other diagnostic test. Nearly all patients with skin disease, and particularly those with skin lesions, should have this test performed. It is also very useful for monitoring therapy. The primary purpose of skin cytology is to assess the presence of bacterial or yeast overgrowth or infection. Another important purpose is to assess what the skin is “doing” – particularly to detect and characterize the degree and type of inflammation.

How is cytology collected?

Cytology can be collected by direct or tape techniques. Within these two techniques, there are many variations in methodology.

For the direct techniques, material can be collected from the surface of the skin as follows:

1) impressions: directly pressing the slide on the skin several times. Although a regular glass slide suffices, adhesive-coated slides (Duro-Tak™) are preferred by some.

2) swabbing the affected area vigorously, then rolling swab onto a slide (like making an ear cytology preparation)

3) scraping the surface debris from the skin using a scalpel blade (or a broken cotton-tipped applicator, e.g. in the nail fold) and smearing the material onto a slide like buttering bread – these scrapings are superficial (do not draw blood) and no oil is used in their collection.

Heat-fixing is generally not necessary, but may help with preparations that are dry or contain little material. All direct techniques are stained with Diff Quik (fixative + 2 stains) and examined microscopically. Although the slide can be stained at lower magnification, high power (1000x) magnification and oil immersion should be used as the final step.

For the tape wet mount technique, samples are collected as follows:

1) A 3-4 cm piece of tape is pressed onto the skin several (3-4) times, sticky side down. Clear tape or Scotch® tape can be used.

2) The piece of tape is placed sticky side down on the microscope slide. It is only attached to the slide by one end in order to hold it in place. Most of the tape is resting on, but not adhering to, the glass slide.

3) A drop of blue (last) stain of Diff Quik or new methylene blue is placed under the loose part of the tape to make a wet mount. Alternatively, the slide is immersed in the purple (last) stain of Diff Quik for a few seconds to allow the stain to enter the space between the tape and the slide. The latter is simpler but contaminates the stain.

4) The back of the slide is rinsed gently.

5) The slide is blotted in a paper towel or bibulous paper.

6) The tape is examined under high power (1000x) oil immersion. Note that if Scotch® tape is used, the image will be cloudy on lower powers due to its opacity - only under oil immersion does the tape become clear.
* In alternative tape method - a dry preparation - the tape is stained with the blue Diff Quik stain, rinsed, dried with a blow dryer, placed sticky-side down on the glass slide and examined as above.

**Why the different direct techniques?**
Some of it depends on personal preference - many practitioners always prefer to use one technique. However, each one is best for certain situations and has advantages over the other techniques:

**Direct techniques:**

**Impression**
Works well for: moist, exudative lesions, pustules (open with needle), crusts (peel crust, touch skin), very greasy skin, draining lesions
Not as well for: dry or minimally exudative lesions, small areas (e.g., nail folds), awkward areas (e.g., interdigital)

**Swab**
Works well for: moist, exudative lesions, small areas (e.g., face & lip folds, nail beds), ears
Not as well for: dry or minimally exudative lesions

**Scrapping**
Works well for: large greasy lesions
Not as well for: sensitive areas, rambunctious patients, near eyes

**Tape wet mount technique:**
Works well for: greasy or dry skin, minimally abnormal skin, awkward areas, small areas, sensitive areas
Not as well for: purulent lesions, pustules, wet skin

**Direct technique or tape technique?**
Each technique provides certain advantages and none are perfect. In fact, they are complementary and it is ideal to try both the tape technique and one of the direct techniques on each patient. Here are some tips about their relative merits:

**Direct techniques**

**Good:**

1) The direct techniques make a smear that is somewhat easier to read.

2) It is a thinner preparation so you don’t need to focus up and down as much. Everything adhering to the slide, there is no movement as can be seen in a wet mount.

3) Most organisms stain deeply.

4) It is easier to identify and quantify bacteria on direct techniques - rods vs. cocci can be differentiated more easily than on a wet mount.

5) Inflammatory cells and other cells such as acantholytic cells can be identified more easily, particularly on the direct impression smears, in which they are not usually damaged.

6) These techniques make nice preparations with moist, exudative lesions.

**Bad:**

1) Picks up less material than a tape smear from many skin lesions.

2) May not obtain adequate samples from dry or minimally greasy skin.

3) Staining takes longer than with the tape preparations, as all 3 Diff Quik steps are used, and the slide must dry.
**Tape wet mount technique**

**Good:**
1) Better at picking up material from minimally exudative or dry skin.
2) Faster staining and no air-drying.
3) Well tolerated by pets in areas such as interdigital spaces.
4) An easier way to find *Malassezia* when it is present in low numbers - this is the major advantage.

**Bad:**
1) The tape technique makes a very “busy” slide with lots of material. It can be a bit overwhelming when you first look at these types of preparations.
2) Bacteria may not be as easy to see and quantify with wet-mount preparations.
3) Not as good for identifying cellular inflammation, acantholytic cells, etc.

**Interpretation**

First, assess the presence and numbers of microorganisms. Remember that recent bathing interferes significantly with cytologic assessment. Examine approximately 10 representative high power oil immersion fields (hpOIF). Record the approximate numbers of yeast per field. For bacteria, it may be easier to score them as 0 to 4+ rather than counting. Record whether they consist of rods, cocci, or a mixed population.

Yeast organisms are most often *Malassezia pachydermatis*. Cocci are most often *Staphylococcus pseudintermedius*. Rod-shaped organisms are less commonly found on skin and ear preparations and should be considered significant pathogens in most sites.

What numbers are significant? It’s difficult to establish a “cut-off”. Although *Malassezia* and cocci inhabit some areas of normal skin in low numbers, we don’t find many using cytologic techniques. On the skin, I consider one or more *Malassezia* per hpOIF significant (3 or more in the canine ear canal). However, since Malassezia hypersensitivity can be present with even lower numbers, finding even one yeast per 5 hpOIF from inflamed skin (or 1 per hpOIF in an inflamed ear canal) may warrant antifungal therapy (see Morris DO, 2008). Bacterial numbers are harder to quantitate. Finding them within inflammatory cells is significant in most cases.

Cytology is very useful for monitoring response to therapy for infections in the skin or ears. In some pets, rechecking cytology at every is warranted because the nature of infection can be dynamic and changes in therapy can be made quickly.

The next step after assessing microorganisms is to assess the cellular response of the skin. This may include the presence of neutrophils, eosinophils, macrophages, acantholytic cells, or neoplastic cells.

The key is to get familiar with cytologic techniques and to use them often; you will become more familiar with collection and interpretation very quickly.

One last note: Learn the difference between bacteria (which stain blue/purple) and melanin (similar in size, oval, but always brown/black and NOT blue/purple).

**Aspirates**

**Why do aspirates?**

Fine needle aspirates are most useful for assessing nodules, tumors, and cysts. They provide a way to assess the contents of these lesions without surface contamination.

**Interpretation**

Cytologic assessment of the aspirate yields information about the nature of the lesion. If neoplasia is suspected, interpretation by a clinical pathologist is warranted. If there is an inflammatory infiltrate, close examination for infectious organisms is recommended, and may include the use of special techniques such as acid-fast staining by a reference laboratory. Aspirated material can also be collected for bacterial or fungal culture.

**Skin scraping**

**Why do skin scrapings?**

Skin scrapings are primarily indicated for the diagnosis of mite and louse infestations. Before performing skin scrapings, consider which parasite you are looking for, as the technique will vary.

**Technique**

Skin scrapings are readily performed in most sites; however, skin scrapings of sensitive skin around the face and feet may require sedation.

Deep skin scrapings are necessary to find the deep follicular dwellers *Demodex canis* and *D. cati* mites. The skin should be squeezed before and/or during the scraping. A new (or clean dulled) #10 scalpel blade dipped in mineral oil is scraped in the direction of hair growth until slight dermal bleeding is observed. At least 3 sites should be sampled if multiple areas are affected, and each one
placed on a separate site on the slide (e.g. 2 scrapings per slide) so the site of origin can be recorded. Droplets of blood should be seen microscopically if the scrapings are of adequate depth. An additional way to look for Demodex is with a trichogram (see below).

More superficial skin scrapings are useful for Sarcoptes, Notoedres, surface-dwelling Demodex gatoi, and Cheyletiella. In superficial scrapings, squeezing the skin and drawing blood is not necessary. The aim is to collect surface scale, crust, and epidermis. Since finding these mites is always significant, the site of origin is not important and you can pool the material from multiple scrapings on 1 or 2 slides. For Sarcoptes, scrape multiple (5-10) areas, concentrating on ear margins, elbows, hocks, and any areas with excessive crust and scale. For Cheyletiella and Demodex gatoi, scrapings should be broad and superficial, and you may first apply mineral oil to the skin to aid in retrieval. If in doubt, perform both deep and superficial scrapings.

For any of these scrapings, the material is collected on a slide and covered with a coverslip. Examine the material under 40x (low power) and then 100x (medium power). Lowering the condenser increases contrast and makes scarce parasites easier to find. Scan the slide completely.

An additional test similar to skin scrapings involves looking for Otodectes mites in ear canals. Exudate is collected with a cotton swab and applied to a glass slide with mineral oil.

**Interpretation**

Although Demodex canis mites are normal inhabitants of the skin, in reality they are rarely found on skin scrapings from normal skin. The finding of any Demodex canis mites should be considered “suspicious”, but a diagnosis of demodicosis is made by finding multiple mites. Note the relative proportion of adult vs. immature mites and eggs. Record the approximate proportion of live mites and track these parameters at recheck examinations. The sites of the scrapings should be recorded so on subsequent visits, the same sites can be scraped.

Finding of one mite or egg of Sarcoptes, Notoedres, Cheyletiella, Demodex gatoi, or Otodectes suffices for the diagnosis. The same is true for lice. Fecal pellets, which are easily mistaken for skin debris, should raise suspicion for skin mites. It is very important to realize that negative scrapings do not rule out these parasites.

**Pitfalls**

Demodex canis should be easily retrieved on skin scrapings. However, it may be harder to find in scarred skin, and from sites such as the feet. It may be more difficult to find mites on certain breeds as well; the Shar pei is commonly mentioned. It is often useful to supplement scrapings with hair plucks, which can retrieve Demodex in areas that are difficult to scrape.

Sarcoptes is notoriously elusive and skin scrapings only find it in about 20-30% of cases. A treatment trial is recommended if these mites are suspected.

Cats often remove surface dwelling parasites by fastidious grooming. Fecal floats are sometimes useful for finding Cheyletiella and other parasites in this species.

**Clear tape preparation**

Clear tape preparations are used to find some of the same surface dwellers that superficial skin scrapings target, but can sample larger areas. They are useful for Cheyletiella, lice, poultry mites (Dermanyssus), and fur mites (Lynxacarus). The tape is pressed on the affected skin and fur multiple times, then placed on a slide sticky-side down with a drop of mineral oil underneath it for easier examination. It is examined like a skin scraping, with the tape acting as a coverslip.

**Flea examination**

Fleas are visible to the naked eye, but are quick and elusive. Parting of the hair on the caudal dorsum and turning the pet over can help reveal them. Flea combs can also help. Often, only flea feces or “dirt” is seen. To differentiate flea feces from soil or other debris, rub the material on a moistened white tissue; flea dirt will dissolve into a brownish red smear. Note that material collected by flea combings can also be collected and placed in fecal floatation solution to find some of the more elusive parasites such as Cheyletiella.

Flea dirt can harbor Bartonella henselae, a causative agent of cat scratch fever - avoid contact with broken skin.

**Trichogram (hair pluck)**

A forceps is used to forcefully pluck hairs from affected skin. The hairs are placed onto a drop of mineral oil under a coverslip and examined under a low power objective. Although there are many reasons to collect a trichogram, my (biased) order of its usefulness is:

To find Demodex mites in areas that are difficult to scrape.

To find louse ova (nits) or occasionally Cheyletiella ova.

To diagnose color dilution alopecia or other structural abnormalities of hair.

To look for evidence of self-inflicted alopecia, in which the ends of the hairs appear broken. This is particularly useful in alopecic cats, which can be “closet” over-groomers.
To look for dermatophyte-infected hairs, which can look fuzzy, swollen, and distorted. Note that this is neither a sensitive nor specific way to diagnose a dermatophyte infection, but can provide support for your tentative diagnosis while you await a fungal culture.

To determine the approximate ratio of hairs in telogen (resting) vs. anagen (growth) phase. Telogen hair bulbs are spear-shaped while anagen bulbs are rounded, soft, and may be bent. This takes practice and a truly forceful pluck, and “normal” ratios have not been established for pets. There is great variation between breeds of dogs, so the results are difficult to interpret.

Wood’s Lamp examination

In approximately half of Microsporum canis dermatophyte infections, apple-green fluorescence of tryptophan metabolites is seen under ultraviolet light (Wood’s lamp). No other dermatophytes of veterinary importance fluoresce. The fluorescence must be seen along the hairs, rather than on the skin. Some drugs, soaps, and bacteria may also show fluorescence but are not associated with hair shafts; they are nonetheless the source of many false positives. The lamp should be warmed up for 5 minutes before use.

Dermatophyte fungal culture

A fungal culture is indicated in any pet with possible dermatophytosis as it confirms the diagnosis and allows identification of the causative fungus. Hairs and scale from the edge of a lesion (or those fluorescing under a Wood’s lamp) are collected using forceps. Broken hairs or those which epilate easily are best. Swabbing the lesion with alcohol has been recommended to reduce contaminant molds, but is not necessary. If lesions are generalized or if asymptomatic carriers are suspected, the Mackenzie toothbrush technique is best. The hair is brushed with a new toothbrush for 1 minute, and the loose hair and scale are gently pressed onto the dermatophyte culture medium.

Fungal cultures can be submitted to a laboratory or kept in-house for at least 3 weeks. If they are kept in house, it is imperative to check them and record findings daily. DTM (dermatophyte test medium) contains a color indicator that turns from yellow to red when proteins in the medium are used. Since dermatophytes preferentially use proteins over carbohydrates, colony growth is preceded by focal color change of the medium. Contaminant molds may also eventually use proteins after their carbohydrate sources run out, so they may turn the medium red once they are well established. Thus, it is the timing of the red color change that is most useful and recording any color change daily is very important. Dermatophytes are typically incubated in a dark area at room temperature, though 25 to 30°C results in faster growth. There are various dermatophyte media available, e.g. Derma-SAB™ plates (Impact Diagnostic International, www.impactdiagnostic.com) as they are easy to inoculate and follow, and contain both DTM medium and Sabourad’s medium. In a dry climate they need protection from desiccation.

All positive cultures should be examined for colony characteristics and microscopic appearance. Dermatophyte colonies are always white, cream, or tan while gray or green colonies are contaminants. Microscopic examination should yield the typical macroconidia (Microsporum species) or microconidia (Trichophyton species); if these are not found, incubate the culture for a few more days. Microscopic examination consists of a wet mount: material is picked up from the surface of the colonies using clear tape, and placed on a drop of lactophenol cotton blue stain.

Overall, fungal cultures can be difficult to carefully monitor and interpret in-house, and submission to a reference laboratory is recommended unless the above conditions can be met.

Bacterial or deeper fungal culture

Bacterial cultures are not routinely performed in uncomplicated bacterial pyoderma cases. Cultures are indicated in cases of severe deep pyoderma, if appropriate empirical therapy has failed to resolve pyoderma, or if numerous rod-shaped bacteria are identified on cytology from the skin or ears. These bacteria tend to have a less predictable response to antibiotics. Remember that all cultures should be accompanied by cytologic examination, and culture results should always be interpreted in relation to cytologic findings.

Bacterial cultures are collected from ear canals by swabbing deep in the canal using a culturette swab. Cultures from the skin can be obtained in several ways. Ulcerated and erosive lesions are generally unsuitable. If intact pustules are present, they can be opened with a sterile needle and swabbed. Epidermal collarette lesions can be sampled by rolling a swab over the collarettes. Aseptic preparation should not be used for surface lesions. If plaques, nodules, deep pyoderma, or draining tracts are present, it is best to aseptically disinfect the surface and steriley collect samples by biopsy or aspiration. The biopsy can be sent to the laboratory in a sterile tube or transport medium (not formalin). If you suspect an unusual organism (deep fungus, Nocardia, Actinomyces, Mycobacterium), contact your laboratory for specific collection and submission guidelines. Not all organisms can be grown in a laboratory due to cultural requirements or safety concerns.
Skin biopsy

Why do skin biopsies?

Skin biopsies are very useful tests in veterinary dermatology. They are indicated when dealing with a serious, unusual, or poorly responsive skin disease. They are recommended when neoplasia, immune mediated disease, or conditions only found by histopathology are suspected. Biopsies cannot replace “derm due diligence”, most importantly skin scrapings and cytology, but they can often narrow down the list of differential diagnoses.

Techniques

If the lesions are secondarily infected, treatment with antibiotics for 1-2 weeks before biopsy is ideal. Select a variety of the most representative samples, including both primary and secondary lesions. Multiple samples (3 or more) should be submitted, as well as a normal sample of skin in most cases. In general, ulcers and erosions should be avoided; the affected but still intact periphery of these lesions is more useful. Alopecic skin should be biopsied in the center of the most hairless areas as well as in the junctional and normal areas. Depigmenting lesions should be biopsied in an area of active depigmentation (gray color) rather than the final stage (white/pink). Pustules, papules, and vesicles are all very useful primary lesions. If taking biopsies of crusted areas, make sure to include the entire crust (it can sometimes fall off during collection).

Local anesthesia often suffices for biopsy collection. If biopsies involve the face, feet, ears, or other sensitive areas, if the patient is intractable very small, or if a larger wedge biopsy is needed, sedation (in conjunction with local anesthesia) or general anesthesia may be used.

The sites to be biopsied are clipped very gently using scissors or a clipper blade held several millimeters from the skin. The lesions are marked. There should be no surgical preparation of the skin, with the exception of biopsies collected solely for cultures. This is not a sterile procedure and infection of biopsy sites is very uncommon. Approximately 0.5 to 1.0 ml of local anesthetic (usually 2% lidocaine, without epinephrine) is injected subcutaneously beneath the lesion in a fanning motion using a single needle stick. Lidocaine stings. Lidocaine injections can be rendered less painful (and perhaps more effective) by mixing a small amount of sodium bicarbonate (approximately 1 part to 10 parts lidocaine) into the syringe prior to injection. This combination is not stable.

Punch biopsies are most commonly used. A biopsy punch of appropriate size is rotated with moderate pressure in a position perpendicular to the skin, in one direction until a reduction in resistance is felt. A 6 mm punch is appropriate for most sites; a 4 mm punch may be preferred on eyelids, the nasal planum, and small footpads. A small thumb forceps is used to remove the biopsy “plug”, which is usually attached by the subcutaneous tissue, and the attachment is cut using iris scissors. The sample should be handled gently, grasping the edge of the subcutaneous tissue rather than by crushing the entire plug. Elliptical biopsies are sometimes used to remove nodules, fragile lesions such as bullae, or deep lesions involving the subcutaneous tissue. Finally, biopsy sites are sutured: a cruciate pattern using 4-0 or 3-0 nylon suffices for most 6 mm biopsies. Pets generally do not bother their biopsy sites, but consider a physical barrier such as an e-collar when collecting biopsies from very pruritic animals or from the feet. Biopsies are submitted in formalin with a thorough history.

Interpretation

Send skin biopsies to a pathologist with a special interest in dermatology. Remember that a skin biopsy can sometimes “miss” a diagnosis that is made more easily another way. Examples include most ectoparasite infestations, dermatophytosis, and Malassezia dermatitis. Skin biopsies are not always helpful in animals suspected of being allergic, except to rule out similar-appearing “zebras”. One of the most important factors aiding the usefulness of a skin biopsy is the submission of a good history, examination findings, and differential diagnoses. In addition to histopathology, biopsies can be used for immunohistochemical staining to look for various organisms or for antibody deposition in immune-mediated diseases.

Intradermal allergy testing

Intradermal allergy testing (skin testing) is performed in pets diagnosed with environmental allergies (atopic dermatitis). This test is routinely performed by dermatologists and infrequently in general practice; as it takes experience to become proficient with the test, and maintaining the test kits in practice can be expensive. The test is used only to select offending allergens for avoidance or inclusion in allergen-specific immunotherapy. It is not used to diagnose atopic dermatitis. It is not appropriate to perform allergy testing when the question is “is this patient environmentally allergic or not?” but rather when the question is “to which specific substances is this patient sensitive?”.

In this test, small amounts of commonly offending allergens are injected intradermally. The appearance of a wheal at the injection site, suggesting type-I hypersensitivity and thus the presence of IgE bound to mast cells in the skin, suggests that the injected allergen is of significance to the patient. The test is easier to perform in dogs than in cats. Allergy testing should not be used to investigate food allergies.
Serum testing for Allergen-specific IgE

Serum allergy tests use ELISA methodologies to quantitate circulating allergen-specific IgE levels. Like the intradermal allergy test, these tests are never used to diagnose atopic dermatitis, but to identify offending environmental allergens. Again, this is mostly useful if immunotherapy is to be pursued. In general, serum allergy tests do not correlate closely with intradermal allergy tests. There has been no conclusive evidence for the superiority of one test over the other, as no “gold standard” exists. However, it is known that when technical performance is used as a criterion, there are substantial variations between certain laboratories. Finally, although food-specific IgE testing is offered by many of these laboratories, these tests should not be used for the investigation of food allergy as the results can be highly misleading.

Bloodwork, urinalysis, and endocrine testing

Although it will not be discussed at length here, general health profiles are useful in some dermatology patients, particularly when ruling out concurrent or underlying disease. They are also used when long-term drug therapy is used. Endocrine testing including thyroid profiles and confirmatory testing for hyperadrenocorticism are also often used.

Elimination diet trial

An elimination diet trial is the only way to evaluate food adverse reactions in dogs and cats. Adverse food reactions can occur at any age and are rarely associated with a recent food change. Dogs and cats with nonseasonal pruritus, recurrent pyoderma, or cats with miliary dermatitis, self-induced alopecia, eosinophilic granuloma complex, and head/neck pruritus should be investigated with a diet trial. The diet trial is an 8-10 week period during which only one protein and one carbohydrate source are fed to the dog. Either a home cooked diet or a prescription diet may be used. Of the prescription diets, novel proteins (fish, kangaroo, venison, etc) and hydrolyzed proteins (soy or chicken protein) are available. Maintaining exclusivity is critical, and it is important to educate owners to the fact that:

- It may take 8 weeks to see a response.
- Any divergence from the trial can result in a failure to make the diagnosis. This includes the other pets’ food, feces, chewable supplements, rawhides, plates, handouts from well-meaning neighbors or children, and many others.

If the pet improves with the elimination diet, it is important to confirm the diagnosis of an adverse food reaction by resuming feeding of the suspected offending food. A relapse of symptoms should be seen within 5 days in most cases (sometimes immediately), but within 14 days in all cases. Some owners agree to investigate further to determine the specific allergenic ingredients. While feeding the well-tolerated diet, one ingredient from the offending diet is added for 7 to 14 days at a time to try to elicit the symptoms.

Ectoparasite treatment trial

Any pruritic dog or cat could be infested with Sarcoptes, Notoedres, Otodectes, Cheyletiella, lice, or other parasite without yielding positive skin scrapings. Similarly, a pet may be exposed to flea bites and not show evidence of fleas on physical examination. Treatment with drugs such as selamectin, imidacloprid + moxidectin, or niterypyram (fleas), or spinosad (fleas) can be very useful in ruling out parasites as a cause or contributor to pruritus. Note that not all ectoparasites are targeted by these drugs.

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Canine Demodicosis: An Update

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Demodicosis is a common inflammatory disease associated with an increase in the number of *Demodex* mites found on the skin. Our understanding of demodicosis has changed in recent years, and the prognosis for patients with generalized disease has improved with the use of newer treatments.

Demodex mites

Healthy dogs are thought to possess *Demodex canis* mites as part of their normal cutaneous flora. Mites are transmitted to neonates from the bitch within 2 to 3 days of birth. They feed on skin cells, sebum, and epidermal debris, and spend their entire life cycle on the host. Mite numbers are kept at relatively low numbers by the host and it is difficult to demonstrate *Demodex* mites in the skin of most healthy dogs. (Scott, Miller, & Griffin, 2001) The life cycle of *Demodex canis* consists of four stages: a fusiform egg, a six-legged larva, an eight-legged nymph, and an eight-legged adult.

In addition to *Demodex canis*, two less common species of *Demodex* mites have been reported in the dog. A short-bodied, “stubby” *Demodex* mite has been reported to coinfect the skin of some dogs with *D. canis*. It measures about 50% the length of the *D. canis* female and may reside in the stratum corneum rather than within hair follicles. There are no distinguishing features of history or clinical signs specific to this mite, unofficially called *D. cornei*. (Tater & Patterson, 2008) It is likely that this mite may be missed on routine skin scrapings because it is found with the more ubiquitous *D. canis*. (Chen, 1995; Chesney, 1999; Desch & Hillier, 2003)

*Demodex injai* is a large-bodied mite whose adults, nymphs, larvae, and eggs are of greater size than those of *D. canis*. Adult male *D. injai* mites are more than 2 times the length of *D. canis* males and adult females are about 50% longer than *D. canis* females. (Desch & Hillier, 2003) This mite shows a preference for the dorsal trunk of adult dogs, particularly Terrier breeds (West Highland White terriers) and their crosses. Excessive greasiness in affected skin is a common clinical sign. One report describes 10 dogs (9 Shih Tzus) with intense facial pruritus due to low numbers of this mite. (Forsythe et al, 2009) This mite can be found alone or with *D. canis*. *Demodex injai* may be under-diagnosed due to its unusual presentation and the potential for low numbers of mites to be found on skin scrapings. In one report of eight cases, none were suspected of having demodicosis at referral. (Hillier & Desch, 2002; Desch & Hillier, 2003; Robson et al, 2003)

Clinical Disease

Infection with *Demodex* mites is typically described as either localized or generalized demodicosis although there are no uniformly accepted criteria for differentiating one form from the other. One classification considers demodicosis generalized when infestation affects an entire body region, two or more feet, or produces 6 or more lesions.

Localized demodicosis usually consists of one to several small, erythematous, scaly, and often hyperpigmented areas of alopecia, most commonly on the face and forelegs. Pruritus in demodicosis is variable, and more severe when secondary pyoderma is present. Most cases first present in young dogs (3 to 6 mos) and resolve without treatment. Progression to generalized disease is rare. A less common form of localized disease is demodicosis limited to the ear canals associated with a ceruminous otitis externa. (Scott, Miller, & Griffin, 2001)

Generalized demodicosis usually starts in dogs less than 18 months of age and less often in adult dogs (over 4 years of age). The lesions are typically more severe and complicated by secondary pyoderma. Cutaneous changes in both young and older patients include comedones, papules, pustules, follicular casts, plaques, crusts, edema, and deep folliculitis and furunculosis. (Mueller, 2004) Peripheral lymphadenopathy is common. Pain, pruritus, and malaise may be present and sometimes severe. Secondary infection, often with *Staphylococcus pseudintermedius*, is common. Involvement of the feet (pododemodicosis) is common and requires longer treatment periods. (Lemarie et al, 1996) In the adult-onset patients, concurrent immunosuppressive factors may be present to allow the previously controlled mites to proliferate excessively. Identification and control of these factors, which include hyperadrenocorticism (spontaneous or iatrogenic), hypothyroidism, heartworm disease, leishmaniasis, and neoplasia, is beneficial to the successful treatment of the disease. (Scott, Miller, & Griffin, 2001; Duclos et al, 1994) Administration of corticosteroids before the onset of generalized demodicosis was identified most often as an underlying cause for demodicosis in one study. (Lemarie et al,
1996) The role of these potentially immunosuppressive factors, and whether they are truly underlying the development of the disease or simply existing concurrently with it, is not well understood.

Although demodicosis is typically considered to be non-contagious among dogs, occasional anecdotal reports suggest that close contact with a heavily infested dog can result in development of clinical disease.

Pathogenesis
Purebred dogs appear to be at increased risk of developing demodicosis, but the breeds listed to be predisposed breeds vary among reports. An autosomal recessive mode of inheritance has been proposed based on limited numbers of kennels. In addition to breed, other factors predisposing to demodicosis include age, poor nutrition, estrus, parturi- tion, stress, endoparasites, and debilitating diseases.

Although it appears that the immune system is crucial in keeping mite numbers low in normal dogs, the contribution of the immune system in allowing the development of demodicosis is still poorly understood. Puppies who exhibit demodicosis do not appear to suffer from other symptoms of immune dysfunction. Furthermore, while some dogs treated with immunosuppressive agents develop demodicosis, most do not. A mite-specific T-lymphocyte immunoincompetence of variable severity has been proposed to explain these differences. The deficiency may be exaggerated by humoral factors associated with concurrent pyoderma and high mite numbers. Our understanding of the immune factors involved in this disease is far from complete.

Since genetic factors are strongly suspected to play a major role in the disease, dogs with generalized demodicosis, their siblings, and their parents should not be bred.

Diagnosis
The diagnosis of demodicosis is usually made by demonstrating the mites on deep skin scrapings. Demodex mites are part of the normal flora but their numbers are very low and it is rare to find mites on a normal dog. If very low numbers are found, additional scrapings should be collected. The diagnosis of demodicosis is supported by finding multiple mites or a large proportion of immature mites. Skin scrapings can be performed easily on most dogs, although some patients do require sedation.

See “Improving Your Diagnostic Skills in Dermatology” in this issue for skin scraping and trichography techniques. Trichography is a good initial screening test for Demodex mites, particularly in sensitive areas. It is not uncommon for demodicosis to be diagnosed for the first time in a patient with chronic skin disease simply because skin scrapings have not previously been done.

Demodicosis is sometimes diagnosed by histopathology. If a skin scraping has not been performed prior to the biopsy, this more invasive diagnostic procedure is unnecessary. Histopathology is an appropriate diagnostic test if demodicosis is suspected but skin scrapings have been negative, for example in patients with chronic, scarred lesions (particularly pododermatitis). A biopsy is also appropriate if a concurrent dermatopathy (e.g. calcinosis cutis) is suspected.

Owners of dogs with juvenile-onset generalized demodicosis should be questioned about recent or ongoing stressors including inadequate nutrition, surgery and anesthesia, boarding, and internal or external parasitism. In older dogs, a search for concurrent or underlying sources of immune suppression should be more fruitful. Particular emphasis should be placed on a thorough drug history, including any possible oral, topical, or injectable corticosteroids. Routine clinical laboratory testing including thyroid hormone evaluation should be performed. The possibility of hyperadrenocorticism and neoplasia should be considered in adult dogs with generalized demodicosis. Some veterinarians recommend repeating diagnostic testing periodically in case demodicosis is an early symptom of yet-undiagnosed disease.

Treatment
Localized demodicosis
Localized demodicosis does not require treatment as its can be expected to resolve spontaneously within 6 to 8 weeks. Topical therapy with an antimicrobial agent, such as mupirocin ointment or benzoyl peroxide gel, may be prescribed. Owners should be informed that lesions can be expected to worsen before improving. The general health of the dog should also be assessed at this time, to ensure that any factors capable of suppressing the immune system are controlled.

Generalized demodicosis
Generalized demodicosis is a serious, sometimes life-threatening disease requiring lengthy treatment. Premature discontinuation of therapy is a leading cause of treatment failures. Not all patients with generalized demodicosis require miticidal therapy. Since dogs under 1 year of age can sometimes recover spontaneously, observation with repeated skin scrapings over 4-6 weeks is a reasonable approach to young dogs with mild generalized disease. If mite numbers increase or the condition worsens, miticidal therapy should be initiated.

The general health of the patient should be assessed and addressed when generalized demodicosis is diagnosed. Most dogs with generalized demodicosis suffer from secondary superficial or deep pyoderma. Long courses of oral antibiotics, often 6 weeks or
more, are usually needed. If impression smears from the affected skin show rods rather than the more common cocci, culture and sensitivity testing is indicated to guide antimicrobial therapy.

Therapy for generalized demodicosis must be monitored by both the clinical and the parasitologic responses. Skin scrapings are usually repeated every 4 weeks (sometimes more often). A parasitologic cure means skin scrapings from multiple sites are negative for mites of any stages, including dead mites or mite segments. Treatment is continued for 4 weeks past two consecutive negative skin. A patient can be declared cured of the disease if there has been no relapse within 12 months of cessation of therapy.

Anesthesia and surgery are potent stressors and may exacerbate or precipitate relapses of the demodicosis. Unfortunately, many patients with generalized disease are young dogs in need of surgical sterilization. Elective surgery should be postponed until secondary pyoderma is cleared, but waiting until skin scrapings are negative for mites is not always prudent.

Numerous studies have been published evaluating miticidal treatment protocols for generalized demodicosis. A recent evidence-based review of these studies has been published. (Mueller, 2004) The efficacy of various treatments varies tremendously among studies, and this variability is increased by the inclusion of both juvenile and adult-onset cases, and inadequate follow-up in some studies. The evidence-based review found good evidence for recommending the following treatments for generalized demodicosis: amitraz (0.025-0.05% dips, every 7-14 days), ivermectin (300-600 µg/kg orally daily), milbemycin oxime (2 mg/kg orally daily), and moxidectin (400 µg/kg orally daily).

Amitraz dip (Mitaban; Pfizer) is licensed in Canada for the treatment of generalized demodicosis. Amitraz is an acaricide and insecticide that inhibits monoamine oxidase and prostaglandin synthesis and is also an alpha-2 adrenergic agonist.(Gursoy et al, 2006) It is licensed for use as a 0.025% dip every 14 days in dogs over 4 months of age.(Plumb, 2005) Some dogs cannot be cured with this protocol so a number of variations have been studied.

Amitraz is applied as a dip to both normal and affected skin. For best results, the hair should be clipped in medium- and long-haired dogs. Skin contact is improved by shampooing immediately prior to the dip or the day before administration. Benzoyl peroxide shampoo is used for its follicular-flushing activity. Crusts should be removed whenever possible. The patient must not get wet between applications of the dip. In patients with severe deep pyoderma, treatment of the bacterial infection using antibiotics and shampoos is advisable prior to the use of the dip to reduce toxicity.

Since amitraz is both a monoamine oxidase inhibitor and an alpha-2 adrenergic agonist (like the sedative medetomidine), there is concern for toxicity in both the patient and the handler. Owners commonly report depression and sleepiness in their pet for 24 to 48 hours after a dip. Close observation of the dog for several hours after a dip is recommended to observe for signs of toxicity, which are usually due to its alpha-2 adrenergic activity and include marked sedation, bradycardia, decreased temperature, and hyperglycemia. (Hugnet et al, 1996) Toxicity can be counteracted by the use of the alpha-2 antagonists atipamezole or yohimbine, and patients with a history of adverse reactions may be premicated with these drugs. Low doses of atipamezole (50 µg/kg, IM) were found to reverse symptoms of toxicity within 10 minutes of intramuscular injection. (Hugnet et al, 1996) Small dogs are thought to be at greater risk for development of side-effects, and it has been recommended that they be dipped with a half-strength solution. Diabetic dogs are also a concern due to the potential for hyperglycemia.

Studies reporting the efficacy of amitraz treatment vary widely in the reported success rate. An evidence-based review of a number of studies of the use of amitraz for generalized demodicosis showed treatment success ranging from 0 and 100%, but overall found good evidence for recommending amitraz (0.025 to 0.05% every 7 to 14 days). Increasing the concentration and frequency is associated with a higher success rate, and many be effective in patients who fail conventional therapy. These off-label protocols include the use of amitraz at 0.05 to 0.1% once weekly, 0.125% amitraz on alternating halves of the body, and even 1.25% amitraz weekly with premedication. (Mueller, 2004) Amitraz has also been mixed in mineral oil (1:9) for treatment of podode-modicosis and demodicotic otitis. (Scott, Miller, & Griffin, 2001)

Amitraz may interact with other drugs including other monoamine oxidase inhibitors and sedatives. The safety of not only the patient, but also the handler should be considered. Drugs capable of monoamine oxidase inhibition including some antidepressants and deprenyl/selegiline should be avoided in these dogs. Handlers applying the dips should also be made aware of these potential interactions, although the likelihood of toxicity is not known. People using monoamine oxidase inhibitors, or those with diabetes, Parkinson’s disease, or respiratory diseases probably should not handle amitraz. The dip must be applied in a well-ventilated space using protective clothing and gloves. Since in humans, monoamine oxidase inhibitors have traditionally been stopped 2 weeks prior to anesthesia, it may be prudent not to administer anesthesia shortly following amitraz administration. (Hill et al, 1992)

A novel formulation of amitraz, in a spot-on combination with the insecticide metaflumizone, is labeled for flea and tick control in the U.S. (ProMeris®, Fort Dodge). A study using this product every 14 days or monthly showed rapidly reduced Demodex mite infestations and marked clinical improvement. Success rates (no mites) at 84 days were 42.9 (monthly) to 62.5% (every 14 day application). (Fourie et al, 2007)
Macrocyclic lactones

A common alternative to the use of amitraz in patients with generalized demodicosis is the use of the macrocyclic lactones. This group includes the avermectins (ivermectin, doramectin) and the milbemycins (milbemycin oxime and moxidectin). Macrocyclic lactones potentiate glutamate-gated chloride channels and/or gamma amino butyric acid (GABA)-gated chloride channels of the mite’s nervous system, resulting in neuromuscular blocking and death. (Mealey et al, 2001) These drugs normally do not cross the blood-brain barrier thus do not cause toxicity to most dogs.

Macrocyclic lactones are now the first choice for treating generalized demodicosis by many veterinarians because of several advantages over amitraz. The oral route of administration is preferable to dipping, treatment can be initiated even if severe secondary pyoderma is present, and frequent bathing does not interfere with therapy. There is less potential for sedation and less risk for the person administering the treatment. One disadvantage of using the oral macrocyclic lactones for the treatment of this disease is the off-label use of the drugs when a licensed treatment is available. Another is that their side-effects, though rare, can be extremely serious. Treatment of toxicity has generally involved only supportive care but recently, the use of intravenous lipids has been reported for moxidectin toxicosis (Crandell & Weinberg, 2009), and anecdotally, used for ivermectin toxicosis.

Ivermectin

Ivermectin is usually dosed at 300 to 600 µg/kg orally per day for treatment of generalized canine demodicosis. (Fondati, 1996; Medleau et al, 1996; Ristic et al, 1995) Most often a large animal injectable (e.g. Ivmec), or oral form of the medication is administered orally. An evidence-based review of studies showed good evidence of recommending ivermectin at this dose range for the treatment of generalized demodicosis. (Mueller, 2004) Adverse effects are rare, and may include lethargy, mydriasis, and ataxia. The most worrisome aspect of treatment, is the potential for acute, possibly fatal, neurologic toxicity. It can occur in any dog but is common in Collies and less so in other herding breeds. Ivermectin generally should not be used in Collies and other herding breeds at the doses needed to treat demodicosis. Ivermectin sensitivity in Collies has been traced to a mutation of the multi-drug-resistance gene ABCB1 (previously called MDR1). This gene encodes for P-glycoprotein, an integral part of the blood-brain barrier responsible for limiting drug intake into the central nervous system. Dogs homozygous for the mutation show the ivermectin-sensitive phenotype, and heterozygous dogs may do so as well. (Mealey et al, 2001) A commercial test is available for detecting the mutation in dogs, and should be considered if ivermectin is to be used in a herding or mixed breed breed dog. Information is available at the Washington State University Veterinary Clinical Pathology Laboratory web site (www.vetmed.wsu.edu/depts-vcp/test.asp). It should be noted that a number of drugs, including cyclosporine, calcium channel antagonists, and various antimicrobial agents (e.g. ketoconazole) are capable of P-glycoprotein inhibition and can precipitate neurotoxicity in patients receiving ivermectin. (Tater & Patterson, 2008) There is also an interaction in dogs receiving oral ivermectin and label doses of spinosad (Comfortis®, Elanco) for flea control. Ivermectin side-effects may sometimes occur after several weeks of therapy; subchronic toxicity is not associated with the ABCB1 mutation and thus can not be predicted by testing. (Bissonnette et al, 2009)

Safety in administering daily oral ivermectin may be improved, but not ensured, by gradually increasing the amount administered over several days until the desired dose is reached. For example, if a daily dose of 400 µg/kg is desired, the patient may be given only 50 µg/kg on the first day, a gradual increase to the full dose over 10-14 days. The owners should be instructed to observe the pet closely over this period and to stop administration and contact the veterinarian if any symptoms such as lethargy, tremors, ataxia, hypersalivation, or mydriasis are noted. The dose of ivermectin required to treat dogs with generalized demodicosis varies among patients. Although some veterinarians prefer to treat all patients with 600 µg/kg per day, another approach to reduce cost and side-effects is to use a lower dose such as 300 µg/kg per day, and increase the dose by 100 µg/kg monthly if needed. The decision to increase the dose can be made on the basis of the clinical and parasitologic response.

Milbemycin oxime

A number of studies have evaluated the use of daily oral milbemycin oxime for treatment of generalized demodicosis. Milbemycin oxime is available as an oral heartworm preventative (Interceptor®; Novartis). The doses used to treat generalized disease have ranged from 0.5 mg/kg to 2 mg/kg per day, up to 2.8 mg/kg/day. (Mueller, 2004; Holm, 2003; Miller et al, 1993) Cure rates are highly variable, but an evidence-based review of the literature found good evidence for recommending treatment with milbemycin oxime at 2 mg/kg/day. Reports of the safety of this drug in Collies are contradictory, but homozygous ABCB1 mutants have recently been reported to exhibit toxicity from this drug and likely it should be avoided in these patients as well. (Barbet et al, 2009)

Moxidectin

Moxidectin is another milbemycin that has been evaluated for treatment of canine generalized demodicosis. There are few studies available but there is good evidence for recommending moxidectin 400 µg/kg orally per day. (Mueller, 2004; Bensignor & Carlotti, 1998) Side-effects such as ataxia and lethargy have been seen, as with the other macrocyclic lactones. Moxidectin for oral use is available in a bovine injectable formulation (Cydectin®, Wyeth).
It is also combined as a topical solution combined with imidacloprid, marketed in Canada (Advantage Multi®, Bayer) for monthly parasite control in dogs and labeled as an aid in the treatment and control of demodicosis in dogs. Despite favourable results in one study, this treatment has not been as effective as other treatment options when applied monthly. Application every 1-2 weeks (extra-label) is more effective, although only weekly application approaches the efficacy of oral ivermectin. This topical product is more effective for milder disease and juvenile-onset demodicosis. (Paterson, et al., 2009; Mueller et al, 2009; Fourie et al, 2009)

**Doramectin**

Doramectin is another macrocyclic lactone that has been investigated for the treatment of generalized demodicosis. It has been used at weekly subcutaneous injections of 600 µg/kg without adverse effects and with apparent efficacy. (Johnstone, 2002) This drug is not safe for use in ivermectin-sensitive patients and thus should be avoided in Collies and other herding breeds. It is available as an injectable solution for cattle and swine. Based on one study, there is fair evidence for the use of doramectin. (Mueller, 2004)

**Summary**

Demodicosis continues to be a common and important skin disease of the dog. Deep skin scrapings should routinely be performed on dogs with dermatologic conditions to ensure the timely diagnosis of this disease. Fortunately, the prognosis for the disease has improved in recent years. In part, this is due to a better understanding of the factors that contribute to generalized demodicosis. The greatest progress, however, has been made in the treatment of this disease.

**Material sourced and updated from:**


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Malassezia Dermatitis in the Dog and Cat

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Malassezia dermatitis (MD) and Malassezia otitis (MO) are superficial fungal (yeast) infections occurring on and within the stratum corneum of the epidermis of many mammalian species. Canine Malassezia hypersensitivity (MH) is a Type-1 (immediate) hypersensitivity reaction to soluble allergens produced by the yeast; these allergens are recognized by the host's immune system in a manner similar to aeroallergens, and contribute to the pathology of atopic dermatitis (AD). In dogs and cats, Malassezia pachydermatis colonizes the skin during the immediate perinatal period and is the primary yeast species associated with skin and ear canal disease.

PATHOGENESIS

Malassezia yeast colonize the skin and external ear canals of animals in very low numbers. Overt “infection”, sometimes referred to as “overgrowth” is defined by increased numbers of the yeast on the skin surface in conjunction with inflammation. In a diseased state, alterations of the skin’s surface microclimate contribute to increased susceptibility to yeast infection. Primary diseases that cause increased moisture, altered surface lipids, and/or disruption of stratum corneum barrier function encourage secondary overgrowth of the organism. Pruritic inflammatory diseases (allergic and parasitic) result in microclimate changes due to scratching (disruption of barrier function), licking (added moisture) and increased production of sebum. Endocrinopathies – especially hyperadrenocorticism – directly cause alterations in sebum characteristics and stratum corneum function. Metabolic diseases that result in hyperkeratosis (such as zinc-responsive dermatosis, hepatocutaneous syndrome/superficial necrolytic dermatitis of dogs, and thymoma-associated dermatosis of cats) also appear to be risk factors. Secondary MD is also associated with primary (idiopathic) seborrhea of dogs and cats and the paraneoplastic alopecia associated with internal carcinomas in cats.

In some dogs with AD, antigens produced by M. pachydermatis may be recognized by the immune system as allergens (i.e., Malassezia hypersensitivity), in which case a highly inflammatory and pruritic response can be mounted to relatively low numbers of yeast organisms, blurring the line between cytological definitions of “colonization” and “infection”. However, many dogs with MH will also have overt infection, as defined cytologically by overgrowth of yeast on the skin surface [see section on cytology (below) for guidelines]. Malassezia hypersensitivity has not yet been studied or defined in cats with allergic skin disease, although MD does appear to contribute to the pruritic threshold of some cats with AD.

CLINICAL PRESENTATION IN DOGS:

Several excellent reviews regarding the clinical presentations of MD and MO in dogs and cats are available (see Suggested Reading list). Some key points are included here:

Although MD is usually intensely pruritic, the only primary lesion produced is erythema. Secondary lesions, including excoriations, seborrheic plaques, lichenification, maceration and intertrigo are common, and cannot be reliably distinguished from staphylococcal pyoderma without cytologic examination. Therefore, look for the yeast on the surface of any pruritic skin lesion.

The clinical appearance of the skin in cases of MD is highly variable. It may either be dry and flaky (seborrhea sicca), or tacky/greasy (seborrhea oleosa). In rare cases, M. pachydermatis can cause a folliculitis that mimics staphylococcal folliculitis, dermatophytosis, and demodicosis.

The distribution pattern of canine MD is variable, but most commonly affects some combination of the face (especially periocular and perioral skin), feet (interdigital spaces and claw folds), intertriginous areas (axillae, groin/inguinum, facial folds, vulvar and mammary folds) and perineum. Generalized cases of MD may occur in chronic cases of allergic dermatitis. Similar distributions occur in cats. Malassezia overgrowth can provoke an overwhelming pruritic response in atopic dogs, which can occur acutely and be misconstrued as increased exposure to aeroallergens. Resolution of the yeast infection can reduce the pruritic threshold of an atopic dog by as much as 75 to 100% in some cases, depending on concurrent exposure to other allergens. Therefore, undiagnosed MD is one of the most common reasons for perceived failure in the management of atopic dogs.

Malassezia pododermatitis may occur with or without more widespread MD. The feet are the most common single body area affected in allergic dogs. Patients with interdigital Malassezia pododermatitis will be presented for the complaint of paw licking/chewing. Paronychia (inflammation of the claw beds) may also
occur as the sole presenting sign of MD, and often causes claw biting. Physical examination will usually reveal a reddish-brown staining of the proximal claw or a waxy exudate in the claw fold, with inflammation of the surrounding soft tissue. In any dog with a known endocrine or metabolic disease, MD must be ruled out (by surface cytology) if pruritus, cutaneous inflammation, or even non-inflammatory seborrhea are present. *Malassezia* yeast also play an important role in cases of ceruminous otitis externa, in which they are highly pro-inflammatory. Some cases appear to be primary and associated only with moisture trapping (especially in swimming dogs).

**CLINICAL PRESENTATION IN CATS:**

While a definitive (immunological) relationship between *Malassezia* yeast and atopic dermatitis has not been described in cats, it does appear to be associated with pruritic/inflammatory dermatoses such as AD, adverse food reaction, and ectoparasitism. Cats with increased numbers of *Malassezia spp.* in the external canals often exhibit a highly pruritic ceruminous otitis. In addition to primary pruritic diseases, feline MD appears to occur in conjunction with paraneoplastic skin diseases; in a review of 550 feline skin biopsies, *Malassezia spp.* were most commonly associated with feline paraneoplastic alopecia and thymoma-associated dermatosis/erythema multiforme. All cats with histology consistent with these underlying systemic diseases had died within 8 weeks of skin biopsy sampling.

**Key points:**

- Feline MD is rare in occurrence compared to canine MD.
- It may be associated with any primary pruritic disease, and often causes a diffuse erythematous, scaly to waxy dermatitis. However, not all cases of MD in cats are pruritic.
- MD seems to be especially pruritogenic when associated with facial dermatitis and/or otitis externa in cats. Facial pruritus is one of the most common and frustrating problems encountered in feline dermatology, and the list of primary etiologies that can incite it is extensive.
- Markedly pruritic ceruminous otitis may be associated with *Malassezia spp.*
- As in dogs, *Malassezia* paronychia may be associated with waxy exudates in the claw beds, and rust-colored staining of the proximal claws. The Cornish and Devon Rex breeds appear to be predisposed. *Malassezia spp.* may also be associated with some cases of feline facial acne (affecting any combination of the chin, neck, periloral and periocular regions). Many cases are non-pruritic.
- Cats with thymoma-associated dermatosis (regional dorsal to generalized exfoliative dermatitis), may have MD which may not be pruritic.
- Cats with pancreatic or hepatobiliary carcinomas may develop a paraneoplastic alopecia of the ventrum and legs (skin has a glistening sheen). Secondary *Malassezia* overgrowth may provoke pruritus in this otherwise non-pruritic disease, but even non-pruritic cats should be screened cytologically.

**ZOONOSIS**

It has been documented that the zoophilic species *M. pachydermatis* cause life-threatening fungemia in humans; especially human neonates receiving intravenous lipid-rich infusions, and adults who are immunocompromised. In one report, the source of a yeast infection was shown to be a pet dog owned by a nurse who worked in the neonatal ICU. This observation suggested that *M. pachydermatis* could represent an emerging infectious zoonotic pathogen. An epidemiological survey conducted by the author’s clinical research group has shown that *M. pachydermatis* can be isolated very commonly from the hands of dog owners, regardless of whether the dogs have *Malassezia* dermatitis or healthy skin. However, the public health significance appears to be extremely minor, considering the commonality of mechanical carriage by dog owners and the paucity of fungemia cases reported in the human literature.

**DIAGNOSIS**

Diagnosis of MD and MO are made by microscopic examination of surface cytology specimens. Fungal culture is not generally necessary and may result in a false-positive interpretation since the organism is commensal. A PCR technique is capable of identifying 8 *Malassezia* species simultaneously with an extremely high degree of accuracy, but is primarily used for research purposes or to monitor for point sources in epizootic outbreaks. Diagnosis of MH in dogs is made by intradermal testing with a commercial *M. pachydermatis* extract.

**Cytologic methods:**

- **For dry skin:** Adhesive tape stripping (using clear cellophane tape) allows for quantification of yeast per each microscopic field. Skin scrapings are also effective for dry skin, although it may be necessary to mix the material with saline and heat fix until dry (in order to adhere the material to the slide).
- **For greasy skin:** Direct impression smears with glass slides work best and allow for quantification of organisms.
- **Cotton-tipped swabs are useful for interdigital spaces.**
Interpretation of cytological results: how many is too many?

For skin, 1 yeast per high power (1,000x) oil immersion field (hpoif) is a general guideline used by many dermatologists. For ear canals, a study semi-quantitatively evaluated the expected (commensal) populations of *Malassezia* spp. yeast residing in normal and diseased canals. It showed that normal dogs may routinely exhibit up to 5 organisms per high-dry (400x) field (roughly equivalent to < 2 per hpoif), while cats may harbor up to 12 organisms per high-power dry field (roughly < 5 per hpoif). (Ginel, 2002)

These numbers are guidelines only. Since dogs may mount a hypersensitivity response to *M. pachydermatis*, it is possible that some individuals will suffer a pathological effect from what would otherwise be considered a “normal” population of yeast colonizing the skin or ear canals. In fact, a study examining yeast numbers on healthy and atopic canine skin has suggested that 1 yeast per 27 hpoif may be sufficient to correlate with hypersensitivity. (Morris, 1999) Since a threshold this low is almost impossible to quantify and appreciate clinically during routine practice, the author generally recommends antifungal chemotherapy when > 1 yeast per 5 hpoif is identified on inflamed skin or > 1 yeast per hpoif is identified from swabs of an inflamed ear canal.

Intradermal testing for *Malassezia* hypersensitivity:

A commercial *M. pachydermatis* extract is available for intradermal testing and subcutaneous immunotherapy (Greer Laboratories, Lenior, NC, USA). This allergenic extract is available in 20,000pnu/ml and 40,000pnu/ml concentrations. A study conducted in healthy dogs with normal skin and dogs with AD (both with and without overt MD based upon cytological evaluation), have demonstrated a threshold concentration of 1,000pnu/ml for use in intradermal testing. (Farver 2005) The threshold concentration of an allergen is that to which 90% of the non-allergic population is non-reactive (ie., ceases to develop an irritant reaction). Ideally, the threshold concentration should also correctly identify at least 90% of sensitized individuals, although this is difficult to assess due to lack of a validated gold standard. This extract is now included in the battery of allergens used for intradermal testing in the author’s group practice, for evaluation of dogs with a clinical diagnosis of AD.

To date, a validated *in-vitro* commercial assay for anti-*Malassezia* IgE has not been reported in the scientific literature although at least two companies offer an ELISA. Because of great discrepancies in results reported by research laboratories, any commercial offering of an enzyme-linked immunosorbent assay (ELISA) for detection of anti-*Malassezia* antibodies in canine serum should be scrutinized carefully by sound scientific methods before it can be recommended for routine use.

TREATMENT

The antifungal regimen chosen for therapy of MD or MO should be based upon the distribution of the infection, the general health status of the patient, and expectations of the pet owner in regards to time and effort commitment (relevant to topical therapy) and side-effects (most relevant to systemic therapy). Diagnosing and eliminating (or controlling) underlying diseases are also paramount to long-term prevention of recurrence. Since *M. pachydermatis* is part of the normal cutaneous microflora, complete elimination of the organism is likely to be impossible.

Systemic therapy (see Table 1)

Unless there is a specific contraindication to using an oral antifungal drug, the author prefers to treat all cases of generalized and regional MD (eg., pododermatitis) systemically. Otitis media also requires systemic therapy (assumedly) to achieve therapeutic drug levels within the tympanic cavity. Oral ketoconazole, itraconazole, or fluconazole is most commonly recommended. Griseofulvin is ineffective against *Malassezia spp*. (Negre et al., 2009)

Ketoconazole (Nizoral®, Janssen; and generics) is an imidazole antifungal with proven efficacy for canine MD. It undergoes extensive metabolism by the liver, and its use in patients with hepatic disease is contraindicated. It is also a known teratogen in dogs. Adverse effects include gastrointestinal upset (anorexia, vomiting, diarrhea), thrombocytopenia (rare), and hepatotoxicity, although none of these side-effects are at all common. Hepatotoxicity is a moderate risk in cats however, and its use in feline MD is not recommended. In aged or debilitated dogs, hepatic enzymes should be evaluated prior to use of ketoconazole. The author does not routinely perform screening tests in young/healthy dogs. For long-term or...
Topical therapy

Topical antifungals are most useful for treatment of localized infections, or as adjunctive therapy along with oral drugs. Topicals are also quite valuable in the prophylaxis of chronic or relapsing MD. For regional or generalised disease, shampoos containing miconazole, ketoconazole, chlorhexidine, or selenium sulfide are available, and have met with variable success depending upon client compliance, frequency and technique of application, and severity of disease. For frequently relapsing cases, shampoo therapy (once to twice weekly, 10 minutes minimum contact time) may be adequate for prophylaxis. Leave-on conditioners containing miconazole or chlorhexidine are also available and provide for more residual action that shampoos. Rinses such as lyme sulfur dip and enilconazole can be quite effective, however lyne sulfur dip is not available in the UK, and while enilconazole is available in Canada, it is unavailable in the USA. Miconazole, clotrimazole, or ketoconazole sprays, lotions, wipes, or creams may be used for “spot” therapy of the skin. Ointments and lotions containing nystatin, thiabendazole, miconazole or clotrimazole are commonly used for otitis externa. Some products also contain glucocorticoids and antibacterials. An in-vitro study comparing the efficacy of the azoles against Malassezia spp. yeast indicated that thiabendazole is the least effective, followed by clotrimazole (with efficacy comparable to nystatin), miconazole (with 10x the potency of nystatin), ketoconazole and itraconazole. (Lorenzini, 1985) The author’s clinical bias is that miconazole is the most effective topical therapy on a per-case basis, and poor clinical responses to nystatin, thiabendazole and clotrimazole have been common in the author’s practice population. A systematic review of clinical trials revealed that sufficient experimental evidence exists only for miconazole in combination with chlorhexidine (Malaseb, DVM Pharm/IVX). (Negre et al, 2009)

Table 1: Systemic Drugs for Treatment of Malassezia Dermatitis and Otitis Media

<table>
<thead>
<tr>
<th>Drug</th>
<th>Supplied as:</th>
<th>Dose/Frequency/Duration</th>
<th>Species</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ketoconazole</td>
<td>200mg tablets</td>
<td>5-10mg/kg q daily x 21-28d, or (low dose regimen) 5mg/kg qd x 10d, then Eod x 10, Pulse dose regimen for prophylaxis: 5-10mg/kg 2 consecutive days/week</td>
<td>D</td>
</tr>
<tr>
<td>Itraconzole</td>
<td>100mg capsules or 10mg/ml elixir</td>
<td>5mg/kg q daily x 21-28d, or, 5 mg/kg 2 days/week x 3 weeks</td>
<td>D &amp; C</td>
</tr>
<tr>
<td>Fluconazole</td>
<td>50, 100, 150, 200mg tablets; oral powder for 10mg/ml suspension</td>
<td>2.5-5mg/kg q daily x 21-28d</td>
<td>D &amp; C</td>
</tr>
<tr>
<td>Terbinafine</td>
<td>200mg tablets</td>
<td>30mg/kg q daily x 21-28d</td>
<td>D</td>
</tr>
<tr>
<td></td>
<td></td>
<td>30-40mg/kg q daily x 21-28d</td>
<td>C</td>
</tr>
</tbody>
</table>
Immunotherapy for MH:

The *M. pachydermatis* extract produced by Greer Laboratories has been evaluated in a multicenter study to determine its utility as an immunotherapeutic agent. Atopic dogs that had been on allergen-specific immunotherapy for a minimum of 12 months, but which continued to have chronic/recurrent MD and required antifungal prophylaxis, were enrolled. A total dose of 2,000pnu was administered weekly by subcutaneous injection, and cases were followed for 12 months. Although the data from all member study sites has not been published, it is clear from 4 cases enrolled at the author’s practice that it can be effective. Two of 4 dogs had an excellent response with resolution of pruritus, discontinuation of maintenance antifungal therapy, and negative cytology, while the other 2 improved enough that the owners elected to continue with immunotherapy. Personal communication with clinicians at other study sites (A. Yu, Guelph, Ontario, Canada, L. Henshaw, Tulsa, OK) suggests that this has also been the case elsewhere.

References and Suggested Reading:


Defining pathogenic staphylococci:

Bacteria of the genus *Staphylococcus* are Gram-positive, facultatively anaerobic cocci that exist as part of the normal cutaneous and mucosal microflora of mammals and birds. Many staphylococcal species are also opportunistic pathogens capable of causing serious infections of the skin and other body tissues and cavities. When cutaneous or systemic disease disrupts the skin’s surface defense mechanisms, skin infection (pyoderma) or otitis externa may result from these same staphylococcal species. Invasive infections involving the genitourinary tract, respiratory tract, joints, bone, and body cavities may also result either from ascension along epithelial tracts, introduction via penetrating wounds, or hematogenous spread. Although the coagulase-negative staphylococci are receiving renewed attention with regard to their medical significance, the coagulase-positive species which normally colonize the skin of the domestic dogs and cats have been well characterized, and include *Staphylococcus intermedius*, *Staphylococcus schleiferi*, and *Staphylococcus aureus*. The coagulase-positive species which normally colonize the skin of the domestic dogs and cats have been well characterized, and include *Staphylococcus intermedius*, *Staphylococcus schleiferi*, and *Staphylococcus aureus*. The coagulase-positive species which normally colonize the skin of the domestic dogs and cats have been well characterized, and include *Staphylococcus intermedius*, *Staphylococcus schleiferi*, and *Staphylococcus aureus*. The coagulase-positive species which normally colonize the skin of the domestic dogs and cats have been well characterized, and include *Staphylococcus intermedius*, *Staphylococcus schleiferi*, and *Staphylococcus aureus*. Therefore, the primary canine pathogen is now known to be *S. pseudintermedius* and this appears to be the case for cats as well. The author will use the *S. pseudintermedius* nomenclature from the point forward, although references published prior to 2007 will refer to this organism as *S. intermedius*. Finally, *S. hyicus* has also been isolated from the skin of healthy dogs and cats and from those with skin lesions, albeit rarely.

Etiology of methicillin resistance

Since the inception of antibiotic use in the practice of modern medicine, staphylococci have evolved in response to the presence of antimicrobial drugs in their environments. Currently, all staphylococcal species that infect humans and domestic animals exhibit some degree of antimicrobial resistance. Even non-pathogenic staphylococci harbor drug resistance factors which can be transferred to pathogenic species. In human medicine, methicillin resistance in *S. aureus* has contributed to the scope of multiple drug resistance since the early 1960’s, while it has been realized as a serious and widespread problem in companion animals only within the past two decades.

Methicillin and oxacillin are members of a class of antibacterials known as the semi-synthetic penicillinase-resistant penicillins (SSPRP). Due to its superior stability in-vitro, oxacillin is now used by most microbiology laboratories as the surrogate for testing the susceptibility of bacteria to this entire class of antibiotics. Even so, the term “methicillin resistant” (MR) has persisted in the common vernacular and in most scientific publications. The SSPRP class was developed to circumvent staphylococcal resistance to the first generation penicillins, which is mediated by bacterial production of penicillinase enzymes. Although the SSPRP class is unaffected by penicillinases, it is susceptible to an acquired penicillin-binding protein (PBP), known as PBP2a. This staphylococcal PBP is encoded by the meCA gene, which confers an intrinsic resistance to all beta-lactam antibiotics and their derivatives (including all classes and generations of penicillins and cephalosporins). Methicillin-resistant staphylococcal strains may express co-resistance to any combination of other drug classes, including aminoglycosides, fluoroquinolones, macrolides, tetracyclines, potentiated sulfas, chloramphenicol, fucidic acid, and mupirocin, in which case epidemic
they are referred to as MDR (multiply drug resistant). However, the mechanism of resistance is distinct for each of these antimicrobial classes (i.e., due to mechanisms other than PBP).

**Methicillin-resistant Staphylococcus aureus (MRSA):**

Since the early 1960’s, the incidence of MR has escalated within human hospital strains of *S. aureus*, and hospital-acquired MR *S. aureus* (HA-MRSA) has now become the most prevalent pathogen causing nosocomial infections of people throughout the world.15 During the mid-1990’s, MRSA strains which cause skin and soft tissue infections in people with no known nosocomial risk factors arose *de novo* within the community.15,16 Risk factors associated with transmission of CA-MRSA include crowded living conditions and shared bathing facilities (e.g., military ships, prisons, day cares, sports teams, residential facilities). The proliferation of these “community-acquired” (CA-MRSA) strains has been global, and while variations exist according to geographic area and ethnic populations, at least half of the persons colonized with MRSA in the United States now carry CA-MRSA strains.17 Over the past several years, niche drift has occurred, with the archetypal HA-MRSA strains “escaping” from the hospital and circulating in some communities, while CA-MRSA have become endemic in some hospitals as nosocomial pathogens.15,18 This epidemiological classification is therefore somewhat anachronistic. Yet, phenotypic differences between these strain types do exist: HA-MRSA strains commonly express resistance to several classes of antimicrobials (i.e., in addition to beta-lactams), which has likely resulted from selective pressure exerted by antimicrobial use in health care facilities. By contrast, most CA-MRSA strains have maintained antimicrobial susceptibility profiles comparable to methicillin-sensitive *S. aureus* (MSSA), with the obvious exception of resistance to beta-lactams.19 It is the HA-MRSA strains which are most commonly isolated from dogs and cats, although it remains unknown if this represents simply the odds of exposure in the “community”, or some difference in host tissue tropism.

The prevalence of human nasal colonization by MRSA in the U.S. population has most recently been estimated to be 1.5%, while the proportion of cultured *S. aureus* infections that are methicillin resistant has been reported to be as high as 72% amongst local community-onset cases.17 The prevalence of MRSA infections in domestic animals is difficult to estimate, as national population-based surveillance is not performed. A retrospective analysis of pets presented to the author’s institution during the 24-month period of January 1, 2003 through December 30, 2004, showed that MRSA infection occurred with equal prevalence (1 case per 1,000 admissions) in dogs and cats.20 Overall, the proportion of canine and feline *S. aureus* infection isolates exhibiting MR was 28%. However it should be noted that the prevalence of resistance reported in this study is likely to be higher than in more general patient populations, due to referral bias and case selection bias (for bacterial culture) by specialist clinicians.

**Methicillin-resistant *S. pseudintermedius* (MRSP):**

In recent years, MRSP has emerged as a clinically important pathogen that causes treatment-resistant infections of dogs and cats.20,21 Like MRSA strains, phenotypic resistance of *S. pseudintermedius* isolates to methicillin has been shown to be mediated by penicillin-binding protein 2a, which is encoded by a mecA gene.22 Also, as observed with HA-MRSA strains (see below), most MRSP isolates co-express resistance to several other classes of antimicrobials, such as the fluoroquinolones, macrolides, tetracyclines, and aminoglycosides.20,21 For the first-generation fluoroquinolones in particular, the disparity in resistance between MRSP and methicillin-susceptible *S. pseudintermedius* (MSSP) is striking. Whereas only 56% of MRSP isolates were susceptible to enrofloxacin and marbofloxacin, 98.5% of MSSP isolates maintained susceptibility to both.20 Since human MRSA isolates have a clonal population structure and global dissemination has occurred, it has been hypothesized that MRSP isolates would also be highly clonal. A recent study of the population genetic structure of *S. pseudintermedius* isolates by multi-locus sequence typing showed them to be highly clonal.10 These isolates had been obtained from several countries, including the USA, Canada, Japan, UK and several other EU nations. Additionally, sequencing of the mecA gene revealed a high degree of homology (95-100%) with the mecA gene of *S. aureus*, suggesting horizontal transfer of the gene. The structure of the MRSP phylogenetic tree suggests that the mecA gene has been received by this staphylococcal species on multiple occasions and on several different continents.10

**Methicillin-resistant *Staphylococcus schleiferi* (MRSS):**

*S. schleiferi* is a coagulase-variable species, with both coagulase-positive and negative subspecies. *S. schleiferi* subsp. *coagulans* is tube coagulase positive. While it has been reported by some authors to be the primary subspecies occurring in dogs,20,24 it is the coagulase-negative variant (*S. schleiferi* subsp. *schleiferi*) which is isolated most commonly from dogs presented to the author’s institution. In humans, the coagulase-negative subspecies is most commonly pathogenic, causing post-surgical skin and soft-tissue infections.25 Isolation of either subspecies of *S. schleiferi* from cats remains exceedingly rare.5,20 Infections caused by both subspecies
are commonly associated with prior antibiotic use, suggesting that 
*S. schleiferi* is primarily an opportunistic pathogen.\textsuperscript{24} While both 
subspecies have been isolated from the healthy skin and ear canals 
of dogs, this remains a rare finding, and the true natural reservoir for 
*S. schleiferi* remains in question (although it is likely the dog).

**Pathogenesis of MR-
staphylococcal infections:**

The broad antimicrobial resistance patterns inherent to HA-MRSA 
contribute significantly to the morbidity and mortality associated 
with human nosocomial MRSA infection. Since pets predominantly acquire the HA-MRSA strains, retrospective studies have 
tried to test the hypothesis that outcomes for MRSA-infected pets are worse than for non-resistant *S. aureus* infections.\textsuperscript{20,27} 
Possibly due to low case numbers, these studies have failed to prove an association. Similar studies to examine outcomes 
of MRSP and MRSS infections have not been reported.

The clinical signs produced by MR staphylococci are not generally different from more “routine” staphylococcal infections. As these infections may occur in any organ or tissue, clinical signs vary greatly. In the author’s dermatology practice, the most common presentations of feline staphylococcal infection are otitis externa, facial acne, and surface pyoderma superimposed upon eosinophilic plaques and indolent lip ulcers. For dogs, bacterial folliculitis and otitis externa dominate, followed by furunculosis (deep pyoderma) – especially of the feet. In a retrospective study conducted at the author’s institution, the majority of MRSA infections were of deep soft tissues, fluids, and body cavities, while MRSP and MRSS infections were predominantly of the skin and external ear canals. The overwhelming majority of MRSA and MRSP were MDR, exhibiting resistance to at least 3 classes of antibiotics in addition to oxacillin.\textsuperscript{20} However MRSS strains have maintained more favorable antimicrobial susceptibility profiles, similar to community-acquired MRSA. In a study of infection isolates identified by the author’s laboratory, susceptibility rates greater than 50% were maintained for trimethoprim-sulfamethoxazole, rifampin, chloramphenicol, tetracycline, clindamycin, and erythromycin, although MR isolates did show poor susceptibility (<50%) to fluoroquinolones.\textsuperscript{20}

**Implications for primary-care practice:**

Despite the reported escalation of methicillin resistance (MR) in staphylococci of veterinary origin, the majority of staphylococcal strains residing on dogs and cats continue to be susceptible to most classes of antibiotics, including the beta-lactams.\textsuperscript{4,5} Therefore, empirical therapy of first-time skin and soft tissue infections with “pet friendly” drugs -- such as amoxicillin-clavulanic acid, cephalosporins, and clindamycin -- continues to constitute acceptable practice. However when clinical suspicion of antimicrobial resistance arises due to initial treatment failure, samples for culture and susceptibility testing should be collected as early in the therapeutic process as possible. This is especially important because even clinically ineffective antimicrobial therapy may continue to select for multi-resistant strains. In particular, sequential empirical therapies should no longer be considered the standard for recurrent urinary tract infections, recurrent pyodermia, and non-healing wounds.

Antimicrobial susceptibility to other (non-beta-lactam) drugs is nearly impossible to predict in MR staphylococcal isolates. Clindamycin and the fluoroquinolones are rarely wise choices due to the high prevalence of resistance in MR-S. aureus (MRSA) and MR-S. pseudintermedius (MRSP) strains.\textsuperscript{20,21} In fact, there is evidence that the use of fluoroquinolones may provoke acquisition of MRSA strains in human-source *S. aureus* isolates, suggesting that early diagnosis can be essential to a positive chemo-therapeutic outcome.\textsuperscript{20,24} Conversely, potentiated sulfa drugs continue to be effective against the majority of MRSA, MRSP, and MR-S. schleiferi (MRSS) strains.\textsuperscript{20,21} Therefore, the author will often initiate therapy with potentiated sulfa antibiotics while culture results are pending (if the case cannot wait for final culture results).

The most problematic therapeutic decisions come when an isolate is resistant to all drug classes discussed above. Fortunately, even MDR strains of MRSA and MRSP have maintained a high prevalence of susceptibility to chloramphenicol, rifampin, and amikacin, although each of these drugs presents its own challenges due to cumbersome dosing regimens and/or concerning toxicity profiles. The author strongly recommends that clinicians consult a veterinary pharmacology resource for dosing protocols, pharmac-toxicologic information, and potential interactions with co-administered drugs before prescribing oral chloramphenicol, rifampin, or amikacin (subcutaneously) for dogs or cats.

**Topical therapies:**

**Shampoos and cream rinses:**\textsuperscript{20} In the cases of broadly drug-resistant pyoderma, intensive topical therapy is often beneficial – as either the sole treatment for localized pyoderma, or as an adjunct to systemic treatment of more generalized disease. For shampoo therapy, the author prefers benzoyl peroxide preparations for better penetration of the hair follicles. Ethyl lactate 10% (Etiderm, Virbac Animal Health) is a less drying option, and maintains some follicular activity. Chlorhexidine – especially at concentrations greater than 2% – is an effective agent for therapy of surface pyoderma, but has no known follicular penetration. The author prefers its use as a leave-on cream rinse.

Several antibiotic preparations are available in cream or ointment formats:
2% mupirocin ointment is available under a canine label (Muricin®, Dechra Veterinary Products, Overland Park, KS) and under human labels (Bactroban, Glaxo-Smith-Kline, and generics). Mupirocin is a highly effective anti-Staphylococcal, with no activity against Gram negative bacteria. It acts by a mechanism unique among antibiotics. A nasal preparation is used for MRSA decolonization purposes in people, and mupirocin-resistant MRSA strains have arisen and are of great concern. Resistance in MRSP was not detected in a study which included 12 MRSP isolates. As mupirocin resistance can be plasmid-mediated in staphylococci, there has been a call for prudent use practices. 2% fusidic acid is available in many countries (not in the USA) in ophthalmic and otic formats (which are often used on the skin). It is a narrow-spectrum antibiotic with excellent efficacy against staphylococci. Resistance amongst MRSA strains remains rare. 1% silver sulfadiazine cream (SSD) has excellent activity against staphylococci in general, and appears to also be effective against mupirocin-resistant MRSA strains.

References


Multi-Drug Resistant Bacterial Infections:  
Zoonosis, Client Education & Hospital Infection Control Strategies

Daniel O. Morris, DVM, MPH, DACVD

Human-animal cross-transmission of methicillin-resistant staphylococci:

The current veterinary literature suggests that pets are capable of being infected or colonized by strains of MRSA that are known to circulate commonly in the community or within human healthcare facilities, and which cause serious skin and soft tissue infections (SSTI) of people. In recent years, individual case reports and small case series have been published which suggest that transmission of MRSA from humans to pet animals (zooanthroponosis) has occurred (reviewed by Weese). More rarely, reports have suggested that MRSA-infected or colonized animals have passed the organism back to people (reciprocal zooanthroponosis). For example, a cluster outbreak of MRSA cases in a nursing home was linked to a colonized cat which served as the facility mascot. Other than non-targeted and limited targeted surveillance of veterinary hospital personnel and animals, no published data regarding the prevalence of MRSA colonization within the general population of dogs and cats are available. Weese and colleagues recently reported that in households in which a MRSA-infected or colonized pet resided, at least one person in each household was positive for subclinical colonization. All MRSA isolated were Canadian epidemic MRSA strain 2 -- a strain type that has “escaped” from hospital settings and is now the predominant community-onset strain in Canada (known in the United States as strain USA 100). Amongst the household contacts of human patients with MRSA infection, a frequency of colonization of 14.5% has been demonstrated. Close contacts, defined as a spouse, parent, child, or caregiver, were at a 7.5-fold greater risk of carriage versus casual contacts (other individuals such as roommates, siblings, and friends). The person-pet relationship will often meet the criteria of “close contact”. Still, a study designed to assess the role of sub-clinical MRSA carriage in pets belonging to people with active infection has not been published, making it impossible to know how often colonized pets might be a “weak link” in control of recurrent/cyclical MRSA infections within human household members.

In regards to veterinary health care workers, prevalence studies have been conducted in small regional surveys (reviewed by Hanselman). Isolates of MRSA strains from pets and veterinary personnel have often been indistinguishable, suggesting some mode of cross-transmission (although directionality of transmission is entirely speculative). This has been true for both horses and small animals. In a veterinary teaching hospital, the prevalence of MR staphylococcal colonization of feline and canine outpatients recently ranged from 13 to 19%. At the 2005 American College of Veterinary Internal Medicine Forum in Baltimore, 417 attendees consented to nasal swabbing for targeted MRSA surveillance. Of these, 27 (6.5%) were MRSA-positive including 15 of 96 (15.6%) of persons in large animal practice; 12 of 271 (4.4%) of persons in small animal practice; and 0 of 50 in industry or research. The strains isolated were dominated by two distinct clones. Large animal practitioners harbored a strain (known as USA 500) which is uncommon in people other than horse owners and people who work with horses. However small animal practitioners harbored strain USA 100. In this study, there was lower over-all risk for small animal practitioners, although they were still colonized at a rate exceeding that estimated for the general U.S. population.

Recent evidence also suggests that human colonization by S. intermedius/S. pseudintermedius occurs in dog owners and veterinarians. While the zoonotic potential of MR S. pseudintermedius (MRSP) is not completely understood, it is not generally considered to be a human pathogen. Historical evidence has suggested that S. intermedius strains isolated from humans may be identical to those that infect their pets. Additionally, strains of S. intermedius resistant to multiple antibiotic classes have been isolated from owners of dogs that presented with deep pyoderma.

The potential for cross-transmission of S. schleiferi between humans and animals has not been systematically evaluated. S. schleiferi subsp. coagulans is thought to be a canine-adapted subspecies that causes infections of dogs, while it is the coagulase-negative variant, S. schleiferi subsp. schleiferi, that is most commonly pathogenic to people. The latter is thought to be part of the normal axillary flora of humans. By contrast, only two reports of human infection by S. schleiferi subsp. coagulans have been published. One of these studies suggested that the patient’s pet dog could have been the source, although the dog was not screened at that time by bacterial culture, despite having active otitis externa.
Hospital and community infection control/management:

In the case of MDR bacteria, management refers not only to treatment of the infected patient, but also to reduction of risk for transmission within hospital/clinic settings and the community (including the home environment). Risk reduction, in the ideal scenario, would involve identification and isolation of all infected and colonized individuals (via surveillance cultures), and use of “contact” or “barrier nursing” precautions in handling both patient groups. In regards to nosocomial transmission, the subclinical colonization of health care workers can be a weak link, due to the obvious risk of transmission to immunocompromised patients. However, contamination of clothing (including gloves and gowns), medical equipment, and the environment have all been clearly implicated in transmission between patients as well. This appears to be true also in veterinary hospitals. A report of nosocomial transmission within in a small animal intensive care unit has illustrated the utility of patient screening and barrier precautions in small animal care settings, once an index case has been identified. These measures were successful in arresting the outbreak, but might be impractical in many general practice settings.

Decolonization of nasal carriers has been a major challenge in human medicine for several decades. Regimens generally involve the use of nasal mupirocin coupled with antiseptic baths, since other body sites may also be colonized. Attempts at decolonization are generally performed in health care workers only in the context of nosocomial outbreaks. Family members of persons (especially pediatric patients) with recurrent MRSA infection occurring within the home may also undergo decolonization therapy. In health care workers, re-colonization occurs in 17 to 48% with 4 weeks to 6 months. The rise in mupirocin resistance among MRSA strains and the need for environmental decontamination within the home are also factors that may confound success. Regardless, veterinary health care workers who are concerned about their personal status and potential nosocomial transmission to their animal patients should seek advice from a physician specializing in infectious disease medicine.

Potential regimens for decolonization/suppression of MRSA in pets have not been explored in an organized manner, and before such attempts can be advocated, longitudinal studies of the duration of pet colonization are warranted. Since dogs are not ideal hosts for S. aureus colonization, it could be that longitudinal persistence in them will routinely be short-term, and this seems to be supported by anecdotal reports. The potential situation for cats is even less clear as up to 50% of cats may naturally harbor S. aureus (within some geographic areas), and MRSA nasal colonization in a kitten was documented to persist for 9 months. In extreme circumstances, systemic therapy or topical (nasal) therapy of colonized pets have been declared successful in breaking cyclical recurrence in human patients, although these pets were not cultured at sites other than the nares to prove global bodily clearance. Studies of MR staphylococcal carriage sites in dogs and cats suggest that nasal therapy alone is likely to be futile, as there were no differences in carriage between nares, oral cavity, anus, groin, and hair/skin of the cranium. Regardless, it is the author’s strongly held opinion that systemic antibiotics for colonized pets should be discouraged in favor of barrier precautions or isolation from other susceptible pets and people in the household. Barrier precautions should include covering of wounds and avoidance of contact with exudates; use of protective disposable gloves; scrupulous hand hygiene procedures after each patient contact (including glove changes); and daily washing of food and water dishes and laundering of patient bedding. Immunocompromised persons and those with a personal history of MRSA infection should not be involved in wound care or grooming of a pet harboring MRSA infection. The British Small Animal Veterinary Association has posted very helpful guidelines on the world wide web.

In human hospitals where MRSA prevalence exceeds 5-10%, surveillance cultures and patient isolation/cohort nursing can be expected to be cost-effective. Such measures are actually highly cost-effective compared to universal gowning/gloving as the primary control mechanism. Due to the comparative rarity of nosocomial MRSA, MRSS and MRSP transmission in veterinary hospitals, such continuous surveillance measures are very unlikely to be cost effective, and will remain research tools until more concrete data can be collected. In the meantime, practices should be targeted toward reducing transmission of MDR bacteria between veterinary staff and animals by excellent hand hygiene, barrier precautions, and (when possible) cohort nursing practices within veterinary care facilities.

Client Education:

As veterinarians, we should be comfortable in our role as “ex officio” public health professionals when it comes to educating the public about potential zoonoses. Clearly, when educating our clients about the risks associated with staphylococcal infection transmission and colonization, we walk a fine line between offering prevention guidelines and making medical recommendations. It is well known that colonization of a person by a pathogenic strain of Staphylococcus is a risk factor for infection, when host factors are present that encourage subsequent infection. However, all people are colonized by S. aureus at least intermittently, with only a minority ever becoming infected.

The three most common scenarios that embroil veterinarians in a discussion about cross-species transmission of staphylococci are:
1) The pet is diagnosed with MRSP or MRSS infection.

2) The pet is diagnosed with a MRSA infection.

3) A normal/healthy pet is presented because a human family member has been diagnosed with MRSA infection. Each of these scenarios involves a somewhat different conversation, which may also vary according to the individual circumstances.

1. The pet has been diagnosed with an MRSP or MRSS infection:
   At this time, we believe that the potential for cross-transmission to and subsequent infection of humans is quite remote. Only a few individual case reports have been published describing infections of people by S. intermedius and S. schleiferi. This does not mean that the risk is zero; especially for immunocompromised people. It is clear that pet owners can carry these organisms in the nares, but it is not known if carriage is intermittent, or how long it will persist without regular pet contact. The risk to other pets is also unclear. Over the past 15 years, the author has personally documented only two households in which pets appeared to be “passing” infection to one another repeatedly. In both cases, only 1 pet had clear risk factors for pyoderma (allergic dermatitis); the other pet involved was healthy in all respects. At this time, companion animal pyoderma should still be considered a “non-contagious” disease to pets that are otherwise healthy and have no epithelial or mucosal barrier defects.

2. The pet has been diagnosed with a MRSA infection. The risk for cross-transmission to human family members is probably greater, as S. aureus is a pathogen highly adapted to human tissues. In fact, we usually assume that someone in the household was the original source of the organism. The risk to other pets (anecdotally) seems to be no greater than with other species of staphylococci. Referring healthy family members to their physicians is often not productive, as infection of a pet is not considered a trigger for nasal screening/decolonization of people.

3. When a human family member has a history of recurrent staphylococcal infections, and especially MRSA, physicians will sometimes suggest that the pets be screened for carriage. However screening is fraught with potential pitfalls, and should never be offered without an a priori plan that has been agreed upon between the veterinarian and the client (i.e., what will be the response if positive?). A negative result could promote a false sense of security that discourages owners from practicing excellent hygiene practices. A positive result may be largely meaningless, if carriage is short/transient. Screening on a single day likely has very low sensitivity in pets, although longitudinal studies to prove this are lacking. Also, which site(s) to screen is controversial. For research purposes, the author screens oral cavity, both nares, the groin, and the anus (in order) with the same swab.

In short, the author feels there to be no substitute for excellent hygiene practices including:

- Stellar hand hygiene practices
- Covering any wounds or abrasions
- Avoidance of contact with any exudates or excretions
- No licking (by the pet) of the person’s face or hands
- Daily washing of pet food dishes
- Litter boxes scooped daily/proper disposal of pet fecal matter
- Weekly (or more often if possible) laundering of bedding in hot water w/detergent
- No pets on the bed until the infected individual has been cleared and remained infection-free for at least 1 month or longer
- Separation of the infected individual (whether human or animal) from any immuno-compromised household members (including those with skin diseases)

Common sense will win the day! Don’t panic! And never give in to the temptation to treat an asymptomatic pet with systemic antimicrobials. However topical therapy with medicated shampoos (such as benzoyl peroxide, chlorhexidine, or ethyl lactate) may be recommended for prophylaxis.

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Dermatology on Demand
Kinga Gortel, DVM, MS, Diplomate ACVD / Daniel O. Morris, DVM, MPH, DACVD
"Just Throw On the Probe!": Utilizing Ultrasonography in the Small Animal Emergency

Tim Spotswood, BVSc, MmedVet, Diplomate of the European College of Veterinary Diagnostic Imaging

Objectives of the presentation
Highlight the benefits and clinical uses of ultrasound in the emergency and critical care patient.

Key clinical diagnostic points
Abdominal and thoracic ultrasonography is a well established diagnostic imaging modality in small animal clinical practice. The main advantages of ultrasound over radiography include: improved contrast resolution that allows evaluation of the internal architecture of organs not normally visualized with radiographs; cross sectional imaging eliminates superimposition and summation artefacts; real-time image acquisition; and a high safety index with no radiation hazard. A big advantage of ultrasonography in the emergency setting is that it can be performed during initial triage within minutes of arrival while the patient is being stabilized, and provides a non-invasive, rapid (often less than 5 minute), repeatable, and portable diagnostic modality. A body cavity can be evaluated in spite of being distended with effusion, and focal disease in abdominal parenchymal organs can be accurately visualized and sampled (fine needle aspirate, needle core biopsy) even if situated deep within the parenchyma.

Ultrasound has important limitations including: the inability to scan though a gas or bone interface; numerous potentially confusing artefacts; and a narrow field of view that necessitates systematic and meticulous scanning technique. Ultrasonography is highly operator dependent, and requires considerable training, time and experience to become proficient in technique and interpretation. However in an emergency situation, referring the case to a radiologist may not be an option, and in many cases the delay in performing the study would be detrimental for the animal. It is therefore desirable for the practitioner to have a basic working knowledge of ultrasound in these cases.

Ultrasound is frequently very useful in emergency situations, and is indicated in cases of acute non-responsive vomiting; acute/painful abdomen; suspect intestinal obstruction; acute abdominal effusion (possibly due to blood, urine or peritonitis); and in trauma cases. Ultrasonography should not be seen as a substitute for radiography but rather as a complementary imaging modality, and almost all these patients should also be radiographed as part of the work up. Diagnostic imaging does not replace a thorough, systematic clinical examination.

Ultrasonographic technique in the emergent case
For the majority of clinical cases, a complete and systematic abdominal and/or thoracic ultrasound is desirable for thorough evaluation of the body cavity and organs; these studies generally take no less than 25-30 minutes each to perform, even in the hands of a skilled sonographer. In an emergency setting, there may be no such luxury of time, and an abbreviated and targeted scan may be all that the situation will allow without further compromising the patient. Various emergency scanning protocols have been devised in human medicine for rapid patient appraisal. In veterinary medicine, a focused assessment with sonography for trauma (FAST) protocol for the evaluation of dogs presenting to an emergency clinic following motor vehicle trauma has been described and evaluated (see below). This protocol utilised a 4 point abdominal technique (subxiphoid, prepubic, right and left lateral flank regions) for the sole purpose of detecting presence of free fluid in the shortest time possible while patients are undergoing resuscitative therapy. The FAST examination in dogs is typically performed with the animal in left lateral recumbency, unless an underlying injury precludes this position (flail chest, fractures, or injury to the vertebral column), in which case the animal is examined in right...
lateral recumbency. The subxiphoid region allows detection of fluid between the liver and diaphragm and between liver lobes as well as the pericardial sac and pleural spaces. Emergency clinicians are able to be trained in the FAST technique in short amount of time and on less expensive ultrasound machines, even if they have minimal ultrasound background. In our referral and emergency hospital, the emergency clinicians have 24 hour access to ultrasound, and utilise the FAST technique or variations thereof.

It must be stressed than an abbreviated scan does not replace a full abdominal or thoracic scan, and whenever possible, a complete scan should be performed at a later stage once the patient has been stabilized.

I tend to use a modified abbreviated abdominal and thoracic technique with the animal placed in lateral recumbency on an “echocardiography” table, where sections of the table surface can be removed. This allows access to the dependent side of the animal, which is where free fluid or fluid within a distended viscus will gravitate; scanning from the dependent side allows for smaller volumes of fluid to be detected, as well as allowing any gas to rise away from the the region, thereby minimizing any gas artefact and maximizing the sonographic window.

The lateral scanning technique is utilised for both the thorax and abdomen, although some animals with thoracic disease may be too dyspnoeic to tolerate lateral recumbency and need to be scanned in sternal recumbency or even standing. In general, placing a dyspnoeic animal in dorsal recumbency should be avoided, especially in the presence of pleural effusion.

Key etiologic and pathophysiologic points

Abdominal effusion

Abdominal effusion of significant volumes can often be diagnosed on physical exam or with the use of radiographs. In general, more than 8 ml/kg of fluid must be present in the abdominal cavity before it is consistently detectable as a loss of serosal detail on radiographs, and it may take several hours for this volume of fluid to accumulate. Abdminocentesis should determine the nature of the effusion in these cases. Abdominal blood may come from different sources, such as secondary to trauma or rupture of a mass lesion within the abdomen. Ultrasound is far more sensitive than radiography for detecting free fluid, and can reliably detect 4-6 ml/kg of peritoneal fluid in the dog. Ultrasound is often helpful in determining the origin of haemoadomen (ruptured spleen or liver or mass lesion), and allows for guided aspiration of smaller fluid volumes.

Blind abdominocentesis can be useful in suspected intra-abdominal injury, however its sensitivity is moderate, only detecting 47-62% of intraabdominal injuries. At least 5-7 ml/kg of fluid in the abdomen is required before a consistent positive result can be obtained. Fluid may also accumulate in pockets in the abdomen, especially exudates, and a 4-quadrant tap has a greater sensitivity than a single quadrant tap. Diagnostic peritoneal lavage (DPL) can be a sensitive and specific test for the detection of intra-abdominal injury. In people, ultrasonography has been found to have a similar sensitivity and specificity to DPL for detecting free abdominal fluid and has essentially replaced DPL as the diagnostic test of choice for early evaluation of humans with blunt abdominal trauma.

Following motor vehicle trauma, the incidence of haemoadomen is reported to be as high as 45% in dogs while the incidence of uroabdomen is much lower (2-3%). Both may result in significant morbidity and mortality and can be difficult to detect on physical examination or radiographs alone; physical examination may only detect up to 35% of cases of intra-abdominal injuries. Neither a palpable bladder on physical examination, a visible bladder on radiographs, nor the ability of the animal to urinate rules out the potential for urinary tract rupture. There is typically a delay in clinical signs associated with uroabdomen which may not be present until 24-48 hours following trauma.

A prospective study looked at the use of focused assessment with sonography for trauma (FAST) in the evaluation of 100 dogs presenting to an emergency clinic following motor vehicle trauma; this rapid technique was useful at diagnosing the presence of intra-abdominal injury as determined by the presence of free fluid. The identification of free fluid helped determine the cause of shock in most of the patients, and in conjunction with ultrasound guided abdominocentesis, allowed the early detection of hemoabdomen and uroabdomen. In that study the FAST exams were generally performed in less than 10 minutes. The FAST examination was found to be a rapid, non-invasive, easily performed procedure for the assessment of intra-abdominal injury that could be done during the initial stabilization of injured dogs with a history or suspicion of significant blunt trauma.

Another important cause for abdominal effusion is peritonitis, and often the source can be determined with the use of ultrasound. Ultrasound may also be useful in detecting free abdominal gas.

Ultrasound can give clues to the nature of the effusions. The sonographic appearance of peritoneal and pleural effusion may be sub-classified into: 1) anechoic, 2) homogeneously echogenic or 3) echogenic and septated. Care is needed when setting the machine’s gain levels in order to avoid under- or overestimation of the degree of fluid echogenicity. Low cellularity of the fluid results in an anechoic or mildly echogenic fluid. As the cellularity increases, the fluid generally contains a greater number and larger reflectors and thus becomes more echogenic (fresh blood, pus, or bacteria etc.). Transudates are typically anechoic. Modified transudates frequently contain more echoes than a transudate but fewer than a typical exudate and typically do not have fibrous
Intestinal obstruction
Radiography is often helpful in the diagnosis of obstructive bowel disease and is usually the first-line diagnostic imaging modality. Ultrasound has been shown to be very useful in detecting as well as identifying the nature of the obstruction (foreign body, linear foreign body, mass or intussusception). Ultrasound often provides more precise location, as well as information regarding the integrity of the intestinal wall and the possibility of perforation. Ultrasound has also been shown to be useful in other causes of mechanical obstruction including intestinal loop entrapment and intestinal volvulus. Ultrasound may also be more sensitive than radiography in detecting foreign bodies: A small retrospective study of 16 animals (11 dogs and 5 cats) that underwent survey abdominal radiography and ultrasonography with clinical signs of an intestinal obstruction showed that radiographically identifiable foreign bodies were evident in only 9 animals, while ultrasound detected a foreign body in all 16 animals.

Intestinal perforation
Ultrasound is very useful in cases of suspect intestinal perforation. In a retrospective study performed to evaluate the sonographic features of gastrointestinal perforation in dogs and cats, sonographic findings in 19 animals (14 dogs and 5 cats) included: regional bright mesenteric fat (19); peritoneal effusion (16); fluid-filled stomach or intestines (12); GI wall thickening (11); presence of free air (9); loss of GI wall layering (9); regional lymphadenopathy (8); reduced GI motility (7); pancreatic changes (4); corrugated intestines (4); presence of a mass (3); presence of a foreign body (3), and mineralization of the gastric wall (1). The site of perforation can also be identified in most cases.

Icterus
Ultrasound can evaluate the biliary tract for integrity and diagnose gall bladder rupture or hepatic diffuse disease or obstruction of the extrahepatic bile ducts, including biliary mucocoele. Ultrasound is therefore very useful in identifying post hepatic icterus. The commonest presenting biliary tract emergency that we encounter in our hospital is bile-induced peritonitis from gall bladder rupture, typically from necrosis of the gall bladder wall caused by a biliary mucocoele. In a retrospective study of 14 dogs, loss of gallbladder wall integrity and/or gallbladder rupture were present in 50% of the dogs, all located in the fundus. Gallbladder wall discontinuity on ultrasound indicated rupture, and pericholecystic hyperechoic fat or fluid were suggestive of but not diagnostic for a gallbladder rupture. Positive aerobic bacterial culture was obtained from bile in 6 of the 9 dogs in the study. Note that occasionally, dogs presenting with disease related to biliary mucocoele may not be icteric.

Vomiting
There are numerous causes for vomiting including gastrointestinal tract as well as other portions of the abdomen. Ultrasound is helpful because of its ability to evaluate the gastrointestinal tract and the pancreas as well as the other organs in the abdomen. Ultrasound is considered a sensitive technique for the evaluation of pancreatitis. Pancreatic diseases and abnormalities frequently investigated by means of ultrasonography include pancreatitis, pseudocysts, abscesses, neoplastic lesions, and nodular hyperplasia. Unfortunately, ultrasonographic findings may lack specificity: the appearance of various pancreatic disorders overlap, and incidental findings or age-related changes may mimic pancreatic disease. Also, some pancreatic disorders may not alter ultrasonographic appearance sufficiently for detection. Ultrasonographic findings should be evaluated in light of the full clinical picture, including signalment, history, and laboratory data. Cytology or histopathology may be needed to establish a definite diagnosis.

Thoracic emergency ultrasound
Thoracic ultrasonography is a powerful tool in the emergent dyspneic or collapsed patient, particularly for identifying pleural effusion, pericardial effusion, and large thoracic space occupying lesions. Thoracic ultrasonography also allows for accurate and efficient therapeutic drainage of effusion. In most cases, correlating the radiographic and ultrasonographic changes provides the best clinical information. The basic principles of identifying effusions are detailed above. It is preferable to image as much of the thorax and heart with ultrasound as is allowable before draining any effusions, as the presence of fluid greatly increases the acoustic window. Post-drainage thoracic radiographs may reveal pathology previously effaced by the presence of effusion.

A full discussion of the applications of thoracic ultrasonography is beyond the scope of this presentation.
Common Sonographic Pitfalls and How to Avoid Them

Tim Spotswood, BVSc, MmedVet, Diplomate of the European College of Veterinary Diagnostic Imaging

Objectives of the presentation

Highlight some of the more common sonographic pitfalls and potential errors that are encountered in a small animal clinical setting.

Key clinical diagnostic points

As with any imaging modality, ultrasonography has many artefacts inherent to that modality that could be misinterpreted by the unwary operator. A basic working knowledge of ultrasound physics and origin of these artefacts is essential in avoiding misinterpretation, as well as providing solutions in avoiding them. Ultrasonography is highly operator dependent, and approaching each case in a thorough and systematic manner is necessary to avoid errors of omission, where lesions are simply not imaged. Interpretation of ultrasonographic changes requires a sound knowledge of what constitutes normal, as well as an appreciation of the range of possible pathologies and conditions and their expected sonographic appearance. The ability to integrate clinical, laboratory and diagnostic imaging findings is essential for meaningful interpretation. Interpretation of subtle ultrasonographic changes can be very subjective, many of which are nonspecific, and there is a tendency to over-interpret these changes in pursuit of a diagnosis. Many ultrasonographic findings may be incidental and unrelated to the the animals original presenting complaint. Many disease conditions may not be detectable on ultrasonography, and the absence of conspicuous sonographic abnormalities does not exclude the presence of disease.

The intent of this presentation is to highlight some of the more basic pitfalls as an introduction to the complexities of the modality.

Artefacts

Ultrasound artefacts create echo signals that do not represent true structures or lesions. Successful ultrasound interpretation depends on the ability to recognize the difference between real tissue signals and imaging artifacts. Artefacts are ubiquitous and are present in almost every sonographic image. They may be generated by improper equipment operation, or may be inherent in ultrasound imaging techniques, tissues and certain pathological processes. When recognised and correctly identified, artefacts can provide very useful information as to the physical nature of the insonated tissues. Sometimes they can severely degrade image detail, or pose an acoustic barrier.

The following are some of the more common ultrasonographic artefacts:

**Acoustic shadowing**

Shadowing is an absence of echo signal which occurs deep to a strongly reflective (gas), or absorptive (bone/mineral) interface. Even though no useful echo signal is evident deep to the shadowing interface, shadowing can be useful in detecting mineralized structures in the body, such as renal and cystic calculi or dystrophic mineralization. A “clean” or black shadow is often seen distal to a mineralized structure, while gas tends to produce a “dirty” or white shadow. Gas shadows associated with bowel may move with peristalsis. Shadowing is most pronounced when the shadowing structure is located within the focal zone of the transducer, and a high frequency transducer is used.

**Reverberation artefact**

Reverberation occurs when the ultrasound beam bounces back and forth between two highly reflective interfaces. Reverberations can occur within the tissue or between the tissue-transducer interface and another structure within the tissues. Reverberation artefact is most commonly encountered at a gas/soft tissue interface, such as the pleural surface of the lung. Reverberation artifacts appear as regularly spaced parallel echo signals that do not represent true anatomic structures. The distance between each signal is equal to the distance between the two interfaces, with each reverberation signal appearing weaker than the one preceding it.
Acoustic enhancement

Enhancement occurs when the sound beam travels through tissue with minimal attenuation and no interfaces (such as a fluid-filled structure or cyst-like structure). The beam is attenuated to a lesser extent than the surrounding tissue as it passes through this structure. This results in a beam of greater echoes (hyperchoic) in tissue interfaces deep to the structure through which the enhanced beam subsequently passes.

Refraction

Refraction occurs when the sound wave traverses from one medium to another at an oblique angle. The ultrasonographic beam bends, resulting in improper location of an imaged structure.

Mirror image

Mirror image artifacts occur in the region of highly reflective curved acoustic interfaces such as the diaphragm-lung interface. Sound reflected from this curved interface may not return directly to the transducer: only after being reflected by another interface, does it return to the transducer via the original highly reflective interface. This artefact creates the illusion of anatomic structures in areas where they are not actually located. In the case of the diaphragm-lung interface, the image gets mapped to the far side of the diaphragm where the lungs are situated. It can look like there is a diaphragmatic hernia, with liver present in the thorax, or like there is consolidation of a lung lobe. In this example, the liver parenchyma and gallbladder are mirrored into the thorax with the sharp hyperechoic line of the diaphragm separating them.

Slice thickness

This artefact is also known as “partial averaging” artefact. This artefact is responsible for simulating debris in normally anechoic structures (“pseudosludge”), or sometimes representing cystic structures as solid. When the scan plane is near the edge of a cystic structure, such as the gall bladder or urinary bladder, echoes are also received from outside the structure because of the beam’s thickness. The artefact results from all the echoes from the finite width of the imaging pulse being compressed into an infinitely thin slice for graphic presentation. The artefact is differentiated from actual particulate material in that slice thickness artifacts have a meniscus, the margins are not horizontal, similar echoes may occur on the nondependant surface and margin inclination is independent of patient position. The effects of slice thickness artefact may be minimized by using higher frequency and by scanning in more than one plane.

Ring-down and comet-tail artefacts

These artefacts contain a series of closely spaced echoes or a solid echogenic streak behind a pocket of gas and fluid (ring down), and a metallic foreign body (comet tail). They are often confused with each other in the literature and incorrectly used interchangeably. Although they appear ultrasonographically similar, they are produced by 2 completely different mechanisms. Comet tail artefacts typically occur with metallic objects and arise when there is a large acoustic impedance mismatch between the reflector and the surrounding tissues: intense reverberation echoes resulting in a virtually solid echogenic streak behind the object are generated.

Ring-down artefacts originate from a bugle shaped fluid collection trapped between at least 2 layers of gas bubbles. When the fluid is struck by an ultrasound pulse, it emits a continuous sound wave back to the transducer which may be seen as a solid echogenic streak or a number of closely spaced echogenic lines. Ring down artefacts are especially useful in identifying diseased pulmonary tissue adjacent to the pleura.

Side lobe artefacts

These result from minor or secondary lobes of the ultrasound beam that spread in directions that are different than the primary beam. Curved structures such as the urinary bladder and highly reflective interfaces cause these side lobes to be interpreted as ultrasound waves within the primary beam, there by resulting in lateral displacement secondary structures not within the primary incident beam into the image of the primary incident beam. They are often seen as extraneous echogenic lines within hypoechoic or anechoic structures, such as the urinary bladder.

Machine operation (“Buttonology”)

Of all the imaging modalities available to the clinician, ultrasonography is by far the most operator dependent. Learning how to drive an ultrasound machine requires familiarity with the different knobs and buttons and how to fine-tune and optimize the image. Optimizing a sonographic image is what takes time and patience, a steady hand, a good technical assistant and a cooperative patient. There are a number of basic controls that one needs to familiarize themselves with and their location on the ultrasound machine. Each of these controls typically are displayed somewhere on the image and must be mastered in order to create high quality images.

Poorly acquired images from incorrect machine settings are at best non-diagnostic, and at worst, misleading. Poor image quality is one of the most common reasons for misinterpretation and incorrect diagnosis.

The basic functions for 2D grey scale imaging that need to be correctly set in order to achieve a high quality image are transducer selection, transducer frequency selection, depth and field...
of view, overall gain and selective gain, and focus. The dynamic range contrast scale), frame rate and frame averaging can also be fine tuned if necessary. More advanced functions include tissue harmonics, compound imaging, and various reconstruction techniques. Many of these functions need to be tweaked and reset continuously during the exam in order to optimize various tissues of different depths, echogenicity and sizes. That's a lot of process during a 20-40 minute scan, and takes concentration, hand eye coordination and fine motor skill! Appropriate skin contact is essential. Re-application of ultrasound gel and/or alcohol will happen multiple times during an exam. Start with your highest frequency transducer setting and maximize the penetration capabilities prior to shifting to a lower frequency setting.

This all takes a lot of practice. It is exceedingly optimistic to expect to become a proficient ultrasonographer after a few weekend ultrasound CE courses, even with a hands-on wet lab and good instruction. Remember that it generally takes 4 years to train a full time radiology resident to become proficient in ultrasound, with good reason\(^\text{10}\). The following a suggested guideline to becoming a proficient ultrasonographer\(^\text{11}\).

- 3 years minimal commitment.
- A minimum of two short courses per year (bring specific questions/problem areas to each new short course).
- Practice, practice, practice.
- Pick on organ or area/vessels/small part and ultrasound all spays/neuters for 5 minutes for each area for a minimum of 3 to 4 weeks.
- Create a checklist to ensure the entire abdomen is covered to build a normative data base.
- Consistent examinations no matter what. Create an abdominal road map and stick with it!
- Shared experience. Find another veterinarian that shares the same passion for learning ultrasound. Journal Club, Book reading, VIN reviews, etc.

It is important that the sonographer knows their own limitations and also the limitations of their modality, and has a sense of when to seek a second opinion.

**Scanning technique**

Most ultrasonographic examinations are elective procedures, and patients should be adequately prepared for the scan. The patient should be fasted, preferably for up to 8 hours prior to the scan to avoid excessive gas and ingesta artefact. Water can be given \textit{ad lib.} Preventing the animal from urinating 2-4 hours before the scan is very helpful for a complete evaluation of the urinary bladder.

It is essential when performing ultrasound to establishing a set pattern of exploration. With echocardiography well established imaging planes have been described, but this is not as well defined for the abdomen where conventional windows and scanning sequences are not clearly established. Each structure or organ has a characteristic echogenicity, shape, pattern, location, margin and number. Each organ should be evaluated using multiple imaging planes. The “satisfaction of search error” is particularly prevalent in sonography, where once the operator has identified the expected lesion, the examination is effectively ended (see proceedings “Why do I miss radiographic lesions?” CVMA 2010).

Incomplete evaluation of the abdomen (or body part) is a very important reason for missing a lesion. A hurried scan in a non-compliant, ill-prepared patient is a recipe for misdiagnosis.

Any abnormalities when identified should also be evaluated in multiple orthogonal imaging planes. The two most common methods used for evaluating the abdomen include dorsal and/or lateral recumbency. There are advantages to each, and operator preference plays a large role here. (I use a combination of both techniques, depending on the size and conformation of the dog, and type of pathology I am expecting based on the history and clinical information). If a particular structure or a possible abnormality requires a more complete evaluation, then rolling the patient into dorsal, lateral or an oblique position may facilitate the examination.

Aspirates and biopsies are done with the animal in the appropriate recumbency that puts the area of interest the closest to the surface with the least amount of possible structures between the area of interest and the skin.

**Image interpretation**

Once the veterinarian is satisfied that a thorough, high quality study has been performed, the images and findings need to be interpreted. This is by far the most challenging part of the process: the spectrum of sonographic lesions is almost infinite, and there is also a large overlap between the appearance of pathological and non-pathological lesions. Trying to match a sonographic image to something similar illustrated in a textbook (the so called “Aunt-Minnie” approach\(^\text{12}\)) is usually inadequate. The veterinarian should also be acutely aware that many important disease processes are
The process of interpretation can be a virtual minefield: If the veterinarian was not aware of or disregarded a differential prematurely, and the list is not inclusive, the diagnosis may be missed. If the differential diagnosis could not be prioritized, then other tests could be ordered, following the so-called “shotgun approach”: the work-up could include an assessment of serum globulins (monoclonal or polyclonal gammopathy which might occur in dogs with lymphadenopathy due to lymphoma or infections such as leishmaniasis or monocytic ehrlichiosis); or various cultures, serology or PCR testing and pursuit of possible infectious aetiologies. These additional tests increase cost and time, and may be subject to false positive and false negative results that muddy the waters further.

As highlighted by the above example, many ultrasonographic disease patterns are highly nonspecific, and require ancillary tests for definitive diagnosis. The expectation that the scan will often yield a definitive or pathognomonic result is overambitious. Ultrasound guided fine needle aspiration or needle core biopsy is often performed during the exam in order to obtain a more definitive answer. These procedures require training, experience, planning and skill to be performed safely and effectively.

One of the most common errors of the novice sonographer is to over-interpret sonographic findings: this is understandable in a clinical situation where the veterinarian is under pressure to “solve the case” and provide the owner with an answer. It is tempting sometimes to “retrofit” the imaging findings to the clinical data. Incidental findings are very common in sonography, especially in older animals, that may have little or nothing to do with the presenting problem. The inexperienced or unwary veterinarian may pounce upon the incidental lesion as the cause of disease while ignoring or dismissing the more important pathology. The scan may also uncover a whole new set of unexpected lesions and potential disease conditions that need to be assessed and worked up. This is sometimes referred to as “opening Pandora’s Box”, and the veterinarian should be prepared for dealing with more than that for which they perhaps had bargained.

Conclusions

Ultrasonography is technically demanding and requires considerable training, time and experience to become proficient in technique and interpretation.

Sonographers beware!
Radiographic Artefacts: From the Analogue Pot Into the Digital Fire?

Tim Spotswood, BVSc, MmedVet, Diplomate of the European College of Veterinary Diagnostic Imaging

Overview

Digital radiography (DR) is rapidly replacing film/screen radiographic systems in veterinary medicine. The digital radiography revolution may have eliminated many of the potential artefacts inherent in traditional film/screen radiography, but has created a whole new set of possible digital artefacts. Digital radiography is a fast changing field, and there are excellent texts and veterinary websites dedicated to this technology and its applications.

Objectives of the presentation

An artefact is a spurious structure or an appearance that is not normally present on the radiograph and is produced by artificial means. This talk will discuss some of the artefacts and their solutions that are common to both digital and film/screen radiographic technologies, as well as highlighting some of those that are unique to digital systems.

An introduction to image acquisition hardware

Understanding image artefacts requires a basic understanding of radiographic physics and technology. There are two main categories of digital equipment: Computed radiography (CR) and digital radiography. Digital radiographic systems utilise two main technologies: direct digital radiography (DDR) and charge coupling device (CCD) radiography. Because DDR and CCD do not require a cassette, for simplicity these technologies can be termed “cassetteless” DR.

CR is an indirect digital system and utilizes a cassette containing a CR image plate. The CR image plate functions similarly to a conventional film/screen system, storing the latent image in a photo-stimulable phosphor plate. The CR plate is processed by an image reader device and the resultant image is displayed on a computer monitor. CR imaging plates will degrade overtime but if well maintained, may last for up to 10,000 exposures. Because CR is a cassette system, the main difference of CR vs. DR is that plate processing is necessary, the duration of which is similar to filmed radiography systems with automatic processors. CR is older but well established technology, typically with spatial resolution at least equivalent to that of DR, and often better. CR units are generally cheaper, and the best CR units are often less expensive than the low range DR units.

With DR images are directly sent to and displayed on a computer monitor immediately after exposure i.e there is no plate processing involved. This system is more immediate and allows increased patient throughput, and is therefore preferred in larger busy hospitals. There are several types of DR detector systems that convert x-ray photons into a digital image.

Flat panel detectors (FPD) are plate-like panels that can be fitted into a bucky-tray of a stationary x-ray unit or used table-top, and some units allow for horizontal beam radiology. Some FPD systems are portable and can be used for mobile work (especially equine radiography). Flat panel systems are sometimes classified as direct digital radiography (DDR), because the x-ray energy is converted directly by the photoconductive layer (usually amorphous selenium) into an electric signal.

Charged coupled detectors (CCD) utilize similar light-capturing technology that is universally found in digital cameras, video cameras and other such devices. With CCD devices, the x-ray photons are first converted into light by a scintillator (usually cesium iodide), and then focused by a lens onto the CCD device which converts the light into an electrical signal. CCD is therefore classified as indirect DR. CCD systems are built into an x-ray unit, thus portability and cross-table work (horizontal beam radiography) is not possible.

An important difference between CCD and Flat panel technology is that the CCD detector does not have any electronics directly in the radiation field. Flat panel systems use a thin film transistor (TFT), layers that capture the X-ray generated electrons.

An important difference between CCD and Flat Panel technology pertains to fill factor. High quality CCD systems have 100% fill factor which means 100% of the pixel area is used. Flat panel systems have lower fill factors because they have electronics running through each pixel reducing the X-ray capture surface area which may reduce the spatial resolution of the system.

In practice, the spatial resolution of high quality “cassetteless” systems is often similar, and even experienced radiologists cannot tell the difference between the images acquired by these different technologies.
Generally CCD detectors tend to be less expensive than flat panel detectors, and easier to maintain and service.

**Key differences between DR and film/screen systems**

In film/screen systems radiographic artefacts are very common, particularly with hand processing. Artefacts may originate between the X-ray tube and the cassette as extraneous material on the patient or contamination of positioning aids, or result from debris within the cassette, or damage to, or staining of the screens. These artefacts are white to grey, may have a constant or different position on follow-up radiographs, and their size and shape are reflective of the inciting cause.

A large number of artefacts may occur in the darkroom during handling, developing, fixing and drying of the film. White to shiny artefacts are caused by the contamination of films with fixer, inability of developer to reach parts of the film or loss of emulsion from the developed film. Black artefacts result from improper handling or storage of films, resulting in exposure to light, or from pressure marks or static electricity discharges. Dropped levels of hand-processing chemicals may result in a variety of tide-marks on films.

There are also several artefacts that are uniquely associated with automatic processing, including roller marks, guide-shoe scratches, chatter marks from worn or poorly fitted roller gears, squeegee marks, faulty temperature settings, and various other electrical faults. Most film/screen radiographic artefacts can be prevented by proper storage and handling of films and by optimal darkroom technique.

With DR systems, x-rays are generated with standard radiographic equipment. However, instead of a film/screen detector system, a photon sensitive detector sends x-ray images directly (or indirectly as in computed radiography) to a computer for display and review. DR systems therefore completely eliminates the need for x-ray film processing, thus eliminating many of the above-mentioned artefacts. DR also saves space as there is no longer the need to maintain a dark room, store x-ray film, chemicals, cassettes, or an automatic processor. There is no longer a need for light boxes, x-ray envelopes, or a filmed radiographic study archive. Digital images may be stored on hard drives or designated servers (with backup of course!); images are never lost or misplaced as long as they are appropriately identified/labeled with patient information at the time of exposure, and can be duplicated an infinite number of times without any loss of quality or detail. Should hardcopy images be needed, digital images may be printed to transparent film or even paper or written to CD/DVD.

The spatial resolution of digital radiographs is inherently less than that of radiographic film (at this point anyway); digital images are able to resolve between 2.5 to 5 line pairs per mm, while film/screen systems are able to resolve up to 15 line pairs per mm. This becomes especially apparent when evaluating small parts such as extremities in small animals. There is also an initial learning curve when manipulating, evaluating and interpreting digital images as the appearance of structures can be dramatically altered by the evaluator.

**Monitors and viewing software**

Image quality affects lesion conspicuity. Since digital images are displayed on a computer monitor, serious consideration needs to be given to how images are to be viewed. Monitor selection is based on the specified use of the viewing workstation. For instance, a lower resolution monitor may be adequate to review images with owners in an examination room, whereas a high resolution or medical grade monitor should be used for diagnostic purposes. Standard commercial grade color monitors have less brightness, smaller pixel matrices, and less sophisticated graphics cards. All these factors reduce the diagnostic accuracy and increase the time it takes to read images. Dedicated medical grade black and white monitors are ideal but are expensive ($4000 and up). High quality colour LCD (liquid crystal display) and LED (light emitting diode) monitors have come down in price over the last few years, and are much more affordable. If of sufficient quality and resolution, these monitors may be acceptable for viewing medical images. Although not considered a true artefact, attempting to make a diagnosis off low resolution screens in bright ambient light leads to missed lesion.

To enhance viewing and reduce eye-strain, ambient lighting should be low, adjustable and indirect. When possible, one image at a time should be viewed. Reducing image size, or cramming more than one image on a single monitor, will result in a loss of diagnostic information. For this reason, two or more monitors are required for a dedicated viewing station. In order to view and manipulate digital images effectively, image viewing software is necessary.

**Digital image processing**

The ability to manipulate a digital image after radiographic exposure is one of the greatest advantages of digital over filmed radiography. Digital image processing can be broken down into three main steps: 1) **Pre-processing**—corrects for system irregularities, 2) **Processing**—performed immediately after image acquisition and involves manipulation of image raw data prior to sending images to a workstation for viewing/interpretation, 3) **Post-processing**—image manipulation using image software (viewing/interpretation workstation) but which does not alter the image raw data. The second step is critical in generating diagnostic images, and
has the greatest effect on the final image quality. Depending on the body part selected, the imaging program algorithm will manipulate and assign a certain brightness, contrast and detail/edge enhancement to each pixel to enhance the diagnostic quality of the overall image. These algorithms have usually been preset by the manufacturer, and are often based on algorithms developed for humans. For instance, a processing algorithm for a thoracic image will create sharp edges and a wide dynamic range, while an abdominal algorithm will create smooth edges and a narrower dynamic range. Applying an inappropriate processing algorithm can greatly reduce the images diagnostic quality and usefulness.

Common digital image artifacts

As with any imaging artefact, a working knowledge of the basic physics and peculiarities of the imaging system is required in order to recognize the origin and apply a solution in order to avoid that artefact in future. As DR becomes more commonplace in practice, more artefacts with different variations are discovered, and there have been several excellent recent reviews in the veterinary literature. Digital artefacts are classified according to the step during which it was created. The major categories are pre-exposure, exposure, post-exposure, reading and workstation artefacts. The artefacts differ between cassette and cassette-less systems during the acquisition phase, but are common to both systems in the processing and post-processing phases.

Table 1.

<table>
<thead>
<tr>
<th>Artefacts with cassette based technology (CR)*</th>
<th>Artefact</th>
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<tbody>
<tr>
<td>Pre-exposure</td>
<td>Storage scatter</td>
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<td></td>
<td>Cracks</td>
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<td></td>
<td>Partial erasure</td>
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<td></td>
<td>Phantom image</td>
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<tr>
<td>Exposure</td>
<td>Upside down cassette</td>
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<td></td>
<td>Backscatter</td>
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<td>Grid cutoff</td>
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<td></td>
<td>Double exposure</td>
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<td></td>
<td>Quantum mottle</td>
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<td></td>
<td>Saturation</td>
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<td>Postexposure</td>
<td>Light leak</td>
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<td>Fading</td>
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<td>Reading</td>
<td>Debris</td>
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<td>Dirty light guide</td>
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<td></td>
<td>Skipped scan lines</td>
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<td></td>
<td>Moire*</td>
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<tr>
<td>Workstation</td>
<td>Faulty transfer</td>
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<td>Border detection</td>
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<td></td>
<td>Diagnostic specifier</td>
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<td>Clipping</td>
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<td></td>
<td>Density threshold</td>
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<td>Ubershwinger</td>
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Table 2.

<table>
<thead>
<tr>
<th>Artefacts with cassette-less technology (DDR and CCD)*</th>
<th>Artefact</th>
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<tbody>
<tr>
<td>Pre-exposure</td>
<td>Memory</td>
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<tr>
<td></td>
<td>Dead pixels</td>
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<tr>
<td></td>
<td>Calibration mask</td>
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<tr>
<td></td>
<td>Upside down cassette</td>
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<tr>
<td>Exposure</td>
<td>Grid cutoff</td>
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<td>Double exposure</td>
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<td>Quantum mottle</td>
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<td></td>
<td>Saturation</td>
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<tr>
<td></td>
<td>Paradoxical overexposure effect</td>
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<tr>
<td></td>
<td>Planking</td>
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<tr>
<td></td>
<td>Radiofrequency interference</td>
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<tr>
<td>Reading</td>
<td>Moire*</td>
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<tr>
<td>Workstation</td>
<td>Faulty transfer</td>
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<td>Border detection</td>
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<td>Density threshold</td>
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<td>Ubershwinger</td>
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</table>

* Adapted from Jimenez DA & Ambrust LJ in Moon Larson M, Danial GB. New concepts in Diagnostic Imaging 2009: Veterinary Clinics of North America, Small Animal Practice (39) 4: 667-718

The following artefacts will be discussed in more detail:

Specific CR Artifacts

Double exposures or phantom images may occur if the previously acquired image was not erased from the plate after processing. This is sometimes termed “partial erasure” artefact.

Not unlike intensifying screens, screen CR plates are soft and quite fragile, and may crack or bend causing linear, white artifacts.

Similar to the film/screen artefact, foreign material (hair, dust etc) may lodge within the imaging plate (or cassette housing the
plate), blocking the x-ray or more typically, the light transmission, resulting in a white artifact. Liquid materials on the plate may be transferred to the plate reader resulting in a repeating artifact too.

Light-bulb artifact is secondary to backscatter radiation entering the peripheral portions of the detector screen, resulting in a dark rim around the image. This artifact is more commonly seen in obese patients or when imaging through thick tissues and can be reduced by decreasing kVp, collimating to the patient, and applying lead backing to the cassette used to house the plate.

Reading artefacts can occur in the post-processing phase where the laser light becomes dirty or blocked, creating a white line across the image.

Skipped scan lines may be seen when the CR plate is not fed smoothly through the reader, creating breaks in the image. This may be from mechanical failure or electrical disruption.

CR is not immune from some of the the typical artefacts seen with film/screen systems, including light leaks, upside down cassette, upside down grid and grid cut-off. Note that light leaks in CR result in part of the image being erased rather than a black artifact as with radiographic film.

**Diagnostic specifier artefact**

A described above, this is when the incorrect body part is assigned to the image, resulting in the computer applying an inappropriate processing algorithm to the image. This artefact is very common, especially when radiographing several sites where the radiographer neglects to inform the computer that the anatomical site has changed. Sometimes the manufacturers preset algorithms are not appropriate for veterinary anatomy, and need “tweaking”.

**Uberschwinger (Rebound) artifact**

This is an image processing artifact where there is an apparent lucent halo surrounding metallic structures or areas where there is a marked difference in tissue density between neighboring objects. This is seen most commonly when using edge-enhancement processing filters to increase image contrast. Uberschwinger artifact may mimic loosening of orthopedic implants or pneumothorax.

**Look-Up table errors**

The LUT determines how bright individual pixel values will be displayed. A digital radiograph that is too light or too dark needs an LUT adjustment, and not a modified radiographic exposure. Some loss of information also occurs in the post-processing phase to reduce the sheer size of the raw image data, termed “clipping” (see table). If an LUT is applied in the preprocessing phase, important information may be lost. An example of this is loss of soft tissue resolution in distal extremity examinations. No matter how one attempts to brighten the image using viewing software, the soft tissues cannot be resolved. This artefact may appear identical to over-saturation artefact (see below) but its origin is not exposure but rather, faulty data processing.

**Exposure artifacts**

In general, digital systems have a much wider dynamic range than film-screen systems. Practically speaking, this means that digital systems are far more forgiving of improper technique settings meaning that under- and overexposures can be compensated for, to a limit. Because the LUT can be adjusted to compensate for under- or overexposures, the radiograph will otherwise appear properly exposed.

If the detector plate does not receive a sufficient amount of x-ray input (severe underexposure), the image will appear grainy or pixelated as a result of low signal-to-noise ratio (SNR). This artefact is termed quantum mottle, and can be rectified by increasing the exposure.

If the detector plate receives too much x-ray input (severe overexposure), it becomes saturated and will no longer respond to additional dose, and is termed over-saturation. The margins of structures, particularly thin ones, are lost and no longer viewable, even when post-processing.

A unique artefact seen with over-saturation of a direct digital plate (flat panel) is an artifact called “planking” due to non-uniformities in the detector plate. To correct this artefact, the patient needs to be re-radiographed at a lower exposure setting.

**Calibration mask errors**

If a uniform x-ray source is used to expose a digital detector plate, the response of each pixel should be equal. However, due to inherent inhomogeneities in the x-ray beam, the inverse square law and the heel effect, this is not the case. Therefore most direct digital systems must be calibrated prior to use (usually on a weekly to monthly basis) so that the sensitivity of each detector pixel will be identical, thus creating a homogenous flood field. If anything is present in the x-ray beam during the calibration process or the plate is incompletely flooded, associated artefacts will be present on subsequent radiographic images. The source of these artifacts may be difficult to determine. The key to recognizing this artifact is in identifying a repeatable, paired white and black structure in the image.

**Zipper artifact**

Direct digital systems are sensitive to radiofrequency (RF) interference. These “zipper” artefacts are often intermittent and may occur only in certain locations (in the case of portable units) or with the detector plate held in certain positions. The stray RF source may not be readily identifiable.
Moiré artefact

This artefact is a sampling artefact; digital imaging information is read line by line, one at a time at a certain spatial frequency. Some objects in the image may also have regular spatial frequencies (particularly grids). When the reading frequency and the object frequency intersect at various points, then spurious straight or curved bright lines may occur across the image. The artefact may also occasionally be seen with other medical devices with repeating spatial frequencies, such as in wiring. Repositioning the grid perpendicular to the detector imaging direction can eliminate this artefact.

Ghost images

If an image is rapidly made before the detector plate has lost all charge from a previous exposure, a “ghost” from the previous exposure will be present on the more recent exposure. In most cases, the time required to prepare for making subsequent images is enough to allow for detector plate quiescence.
Does This Case Need CT?

Tim Spotswood, BVSc, MmedVet, Diplomate of the European College of Veterinary Diagnostic Imaging

Introduction

Computed tomography (CT) is an advanced diagnostic imaging modality that utilizes x-rays and high-powered computers to construct tomographic (cross-sectional) images of the patient. Computed tomography (CT) was initially introduced into human medical practice in the 1970s as a brain scanner, but has rapidly developed to include a broad range of imaging applications. The exponential advancement of computing power and development of improved hardware including slip ring technology, helical CT capability, and more recently, multidetector technology (MDCT), have taken CT imaging to previously unimaginable heights. In humans, MDCT is now an established imaging modality that has almost completely replaced single slice helical CT technology. Computed Tomography is now a well established imaging modality in veterinary medicine, and with more practices acquiring machines, CT is increasingly available to the clinician as a diagnostic imaging option.

Advantages of CT

Conventional radiographs depict a three dimensional object as a two dimensional image, and is therefore a complex superimposition of tissue shadows. CT has two main advantages vs. conventional radiography: the ability to obtain cross-sectional images (or tomograms) eliminates superimposition of complex anatomical structures thereby allowing evaluation of internal structures and anatomy; and exquisite contrast resolution, allowing detection and differentiation of tissue densities over a very wide range. Other features of CT include the capability of imaging all regions of the body. Common veterinary applications include the musculoskeletal system, head (including the nasal cavity, skull, brain), spine, thorax and extra-thoracic structures and abdomen. CT studies are rapidly acquired, far faster than other advanced imaging modalities (particularly magnetic resonance imaging). CT utilizes x-rays for raw data acquisition, which means for the clinician familiar with conventional radiography, the images are inherently intuitive to interpret.

Image acquisition

CT studies are acquired under sedation or anesthesia to prevent patient motion during image acquisition. CT images are obtained by a rotating the x-ray tube located within the gantry (“doughnut”). The patient is positioned inside the gantry on the CT table. Axial (cross-sectional or transverse) imaging slices are acquired as the tube makes one complete revolution (360°) to obtain one axial image (slice) while the CT table is stationary. It takes about 1 second to complete one full revolution. The CT table then advances the patient the predetermined slice interval and the next acquisition takes place. Newer spiral CT scanners (helical CT) have the capacity to move the patient through the gantry at a continuous rate while the x-ray tube head rotates simultaneously and continuously around the patient. Helical CT reduces image acquisition time. Both techniques allow reconstruction of high quality images in body planes other than axial, and allows 3-dimensional reformating. Multidetector CT machines allow greater volumes of tissue to be scanned at one time, this exponentially reducing scanning time and increasing the number of data points acquired, allowing for more detailed and powerful reformating techniques.

The information acquired by CT is stored on a computer as digital raw data. Using powerful mathematical algorithms to analyze the data, the computer displays the images on a video monitor or printed on to x ray film. The image is made up of a matrix of thousands of tiny squares or pixels (65000 pixels in a conventional image). Each pixel has a CT number (measured in Hounsfield units) attributed to it. The CT number is a measure of how much of the initial x-ray beam is attenuated by the tissues at each small volume of tissue (voxel) in the body represented by that pixel. This varies according to the density of the tissues. The denser the tissue is, the higher the CT number, ranging from -1000 HU (air) to 1000 HU (bone) and beyond (up to 4000).

The user can select the range and median of the Hounsfield units to be displayed as a gray scale on the image. This is called “windowing” and is used to optimize the contrast resolution of the image in order to evaluate different structures and increase conspicuity of lesions. These can be spread out over the available gray scale so that two tissues with only a little difference in density will be ascribed separate shades and can therefore be differentiated. For example, a range (window width) of 500 is often used for imaging...
the mediastinum, with a window median (level) of 40. The level refers to the CT number at the centre of the selected window. Thus, in this case, all pixels within the range - 210 to 290 will be displayed. Most of the lungs (largely air) will have CT numbers below -210 and will therefore appear completely black on the final image. The bone with density above 290 will appear completely white.

Axial images through the mid nasal cavity at the same level in a dog, displayed in soft tissue (left) and bone windows (right). Bone images are displayed at WW 3600 WL 550. Soft tissue images are displayed at WW 400 WL 87. Note the lack of conspicuity of the turbinates in the soft tissue window vs. the bone window, while the fatty detail tissue of the tongue becomes inconspicuous in the latter.

Standard intravenous iodinated contrast media is often utilized in CT following the acquisition of standard sequences. Contrast CT allows visualization of specific anatomic structures, detection of some lesions otherwise not seen, can yield useful information regarding tissue perfusions, and often assists in typing of pathology.

### Indications for computed tomography

#### Masses

In general, CT is very useful in providing useful information about masses, including: origin of the mass; establishing margins; detecting invasion of important surrounding structures; detecting bone destruction; and determining the feasibility of surgical resection. Gross margins of tumors often extends beyond palpable limits, and the increased sensitivity of CT in determining margins is useful for surgical planning. CT is also used for planning radiation therapy, allowing precise locations for therapy portals. MRI is also very accurate at depicting mass margins.

#### Nasal and sinus

Skull and nasal radiographs are challenging to evaluate due to the complex bony anatomy and superimposition of structures. CT allows precise evaluation of the fine anatomical bone and soft tissue detail of the skull. CT allows precise determination of the location and extent of traumatic, inflammatory and neoplastic conditions of the nasal cavity and facial bone structures, including diseases of the tympanic bulla and temporomandibular joints. Image acquisition is far more rapid and simple than with conventional skull radiography, and patient positioning is straightforward vs. the multiple and rather challenging skull views needed to obtain a full series of nasal or skull radiographs. A nasal CT series typically only takes several minutes to acquire. With these advantages, CT has largely replaced skull and nasal, TMJ and bulla radiography in our practice. One of the more common CT procedures in our clinic is the assessment of retrobulbar masses.

CT can often discriminate between most types of nasal disease. Bacterial rhinitis will generally have diffuse exudate between turbinates and a small amount of sinus fluid. Fungal rhinitis will have the same findings as rhinitis, but also shows regions of turbinate destruction and sequestration. Neoplasia will usually present as a focal or regional destructive mass often involving the facial bones. CT allows accurate planning for biopsy procedures. Because of its greater bone imaging capabilities, CT is preferred over MRI for nasal disease.

#### Brain and skull

Note that CT is less useful in detecting diffuse parenchymal diseases, and MRI is vastly superior for detailed evaluation of brain parenchymal disorders and has become the gold standard for neuroimaging in human and veterinary medicine. For this reason,
we sometimes refer to CT as the “poor man’s MRI” in neuroimaging cases. In general, contrast enhanced CT can detect lesions of greater than 1 cm diameter, but smaller lesion will have a good chance of remaining inconspicuous. Larger mass lesions such as neoplasms (e.g., pituitary tumors, meningiomas, etc.), abscesses and granulomas, and hematomas may be detectable, but are less easily characterized than with MRI. CT is very sensitive at detecting pituitary macroadenomas since the pituitary gland does not have a blood-brain barrier and will readily contrast enhance.

CT is useful in acute trauma cases where rapid acquisition and ultra-short anesthesia or sedation is mandated. Small fractures can be difficult to see on radiographs, but are easy to visualize with CT. This is especially true for fractures involving the TM joints or calvarium. In the first 24 hours after skull trauma, CT can detect subarachnoid and brain hemorrhage. (After this time, MRI is more useful for this purpose).

Importantly, CT and MRI do not replace a microscope, and cannot reliably determine the type of histopathology present.

**Spine**

As with brain imaging, MRI should be considered the standard imaging modality for spinal cord disease, particularly intramedullary disease (neoplasia, hemorrhage, edema) and nerve root tumors. CT is an excellent imaging modality for evaluation of spinal diseases involving the vertebrae (vertebral bony lysis and fractures), and can be very useful extradural (particularly disc herniation) and intradural/extradural spinal cord lesions. CT can also be useful in evaluating the paraspinal soft tissues. CT is commonly used following myelography to further assess the extent of intervertebral disc disease or other mass lesions within the spinal canal, and bony lesions resulting from infectious and neoplastic processes. CT is exquisitely sensitive at detection of iodinated contrast material within the subarachnoid space, requiring lower volumes of contrast media required for conventional myelography (at least a third of the usual dose). As with any spinal imaging, lesion localization by means of a meticulous neurological exam is important to allow a detailed evaluation (small slices) of as small a spinal region as possible. CT is an excellent modality for evaluating the lumbosacral region. The epidural fat around the nerve roots in this region facilitates visualization of lesions at this site without contrast.

**Thorax**

CT is the gold standard for pulmonary and pleural disease imaging in human medicine. Thoracic CT is also now commonplace in veterinary medicine. High resolution CT is more sensitive than conventional radiography for detection of pulmonary nodules and subtle pulmonary infiltration. Pulmonary and pleural masses can be precisely located, and tissue sampling by CT guided aspiration or biopsy may be applied. For these reasons, CT is very useful for surgical planning of thoracic disease. CT is also helpful in further differentiating types of pulmonary infiltration and consolidation, particularly in concurrent pleural effusion is present. Pulmonary perfusion can also be assessed (with non-selective CT angiography) and potentially challenging conditions such as lung lobe torsion and pulmonary thromboembolism can be diagnosed. CT is an excellent imaging modality for evaluation of mediastinal disease, including cranial mediastinal masses, differentiating esophageal pathology, and assessment of hilar lymph nodes. Thoracic CT requires meticulous anesthetic technique for best image quality: respiratory movement must be managed; and the lungs must be properly inflated to prevent atelectasis that may mimic pathology.

**Abdomen**

Although ultrasound is the “work horse” of abdominal imaging in veterinary medicine, CT is can provide invaluable additional information in certain cases. The field of view is limited in abdominal ultrasound, necessitating that the sonographer compiles a virtual 3-D picture from a composite set of multiplanar images. This can be very challenging in some cases, such as where a large abdominal mass is present and the extent and sometimes origin is not clear; if the pathological process is complex and anatomical structures are distorted; and if ultrasound artifact is excessive (particularly gassy abdomens). CT allows a more global assessment of the abdomen without sacrificing contrast resolution and anatomical detail that limit conventional abdominal radiography. The images can also be reformatted in any imaging plane. CT may be able to detect metastatic disease (particularly in the liver and spleen when intravenous iodinated contrast material is used) not seen during an ultrasound examination. Non-selective positive contrast CT has been shown to be an excellent imaging modality for detection of portosystemic shunts, and is considered the optimal imaging technique over sonography and portography for PSS in some centres. One of the commonest indications of abdominal CT in our clinic is the assessment of caudal vena cava invasion/thrombus formation by adrenal gland masses for surgical planning. Conventional excretory urography is often a finicky and time-consuming procedure, particularly for the assessment of ectopic ureters extending into the pelvic cavity; we have largely replaced this procedure with CT for evaluating ectopic ureters in our clinic. CT is also an excellent choice for detecting ureteroliths and other cases of ureteral obstruction.

CT also allows for detailed evaluation of the pelvic cavity, a region not generally accessible by transabdominal ultrasound.
Musculoskeletal System

CT provides exquisite bony detail, and bony lesions are often identified on CT examinations that were simply not detectable on conventional radiographs. CT is capable of detecting early discospondylitis well before radiographic signs are present. The extent of bony neoplasms is better assessed with CT than conventional radiographs, which assists with treatment planning. CT is a very sensitive image modality for assessment of canine elbow dysplasia, far superior to radiography particularly for assessing fragmented medial coronoid process disease, and is routinely utilized in our clinic for this purpose. The canine shoulder, carpus, stifte, and tarsus have all been studied using CT. Other clinical applications include complex fractures, suspected osteochondral lesions, various forms of arthritis, joint neoplasia, bicipital tenosynovitis, etc.

CT is very useful in evaluation of soft tissue masses of the extremities. A good example is the assessment of infiltrative lipomas, which may be clearly differentiated from the surrounding muscles; liposarcomas can also be assessed, which have a wispy appearance and densities intermediate between lipomas and soft tissues masses. CT has been shown to be superior in most cases for detection of wooden or plant matter (grass awn) foreign body migration over ultrasonography and MRI. MRI’s superior contrast resolution is generally considered better for evaluating soft tissues, especially joints (menisci, cruciate and collateral ligaments, and articular cartilage).

Further reading


Why Do We Miss Radiographic Lesions?

Tim Spotswood, BVSc, MmedVet, Diplomate of the European College of Veterinary Diagnostic Imaging

Introduction:

Accurate interpretation of diagnostic images, whatever the modality, requires a combination of skill and experience. The skill component can be obtained and honed by training and study while experience can only be obtained over time. Regardless of the diagnostic imaging modality a consistent systematic approach to interpretation is essential to accurate analysis and reliable diagnoses.

Objectives of the presentation

Review common sources of error in diagnostic imaging.

Key points

Radiological interpretation is based upon detecting changes or alternations from normal.

The principles in film reading technique can be deconstructed into three phases: the recognition phase, the descriptive phase and the analysis phase.

Changes in size, shape, margin, position, number, symmetry and opacity are evaluated. Although this talk will focus on radiography, the same process may be applied to any imaging modality by substituting opacity with echogenicity in ultrasound; attenuation in computed tomography; signal intensity in magnetic resonance imaging and radiopharmaceutical uptake in nuclear medicine.

A thorough analysis of the images presented is essential: all the body parts or components in the image should be diligently evaluated in sequence. There are differences of opinion between radiologists about how to search films for abnormalities. Two methods are described:

1) Directed search pattern, i.e., look at the various structures on the radiographs according to a preconceived sequence in an attempt to avoid concentrating on a central or obvious abnormality at the expense of peripheral or unexpected lesions e.g. working from the periphery to the centre of the image is a common search pattern. Developing checklists of various anatomical regions, particularly for complex images such as thoracic and abdominal radiographs, and abdominal ultrasound and echocardiography may be very helpful.

2) Hypothesis-driven search, i.e., form a hypothesis about possible diagnosis from the history or from the initial observation of a suspected abnormality, then use this to guide further examination of the radiograph. It is important to take into account what disease conditions are likely to involve a particular body part, and then to carefully evaluate the image for evidence of such lesions.

In practice, a combination of both strategies is typically applied. One approach used for training radiologists is to evaluate the images without prior knowledge of the clinical information to avoid idiosyncrasy (tunnel vision) and bias; once the study has been systematically evaluated, a more targeted review can be performed taking into account the clinical, historical and laboratory data. Obviously, this “clinically blinded” approach is not always possible in the clinical setting, especially if the evaluator is also the primary clinician.

Lesion perception

The process of lesion detection can be divided into three phases; fixation, recognition and diagnosis. Radiographs are intricate jumbles of superimposed shadowgrams, where the complex structures of a three dimensional organism have been smeared onto a two dimensional plane. As the eye scans an image, the brain is bombarded by a vast amount of information. Fixation occurs when the eye focuses on portion of the image. Factors that influence how this data is perceived by the brain, include lighting conditions, and image quality (sharpness and contrast). Optical illusions are based on the tendency of the brain to distort data presented to it by the eyes. What the eye sees is not always what the brain sees, and this occurs frequently when viewing radiographs. Once an image has been presented to the brain the second phase, lesion recognition, depends upon a process of comparing this image with the expected normal appearance. This phase is affected by the knowledge and experience of the observer (the quality and quantity of stored normal and abnormal images in the brain). The mindset of the observer may heavily influence this process, including bias or prejudice on the part of the observer and also the clinical data.

Once the brain recognizes a lesion, the third phase of interpretation occurs. In this phase, the brain determines whether the lesion is real or not and attributes a degree of significance to the lesion. The interpretation phase may be affected by factors such as clinical suspicions or prejudices and past observer experience.
The vast majority of radiographic diagnostic errors are from missing or not recognizing a lesion (perceptual errors) rather incorrectly interpreting a lesion. Most missed radiographic lesions (false negatives) are usually obvious once pointed out.

In human radiology, studies using eye position recording techniques have shown that false negatives can be classified into three categories based on how long they are fixated or dwelled on by the radiologist.

1. Some lesions are missed because they are never looked at with high-resolution foveal vision (fixation or search errors).

2. Some lesions are looked at, but not long enough to detect or recognize any suspicious lesion features (recognition errors).

3. Finally, there are those lesions that are looked at for longer periods of time, often as long as lesions that are looked at and reported, but are still missed (decision or interpretation errors).

Common sources of radiology errors in veterinary medicine

**Technique**

Regrettably, this a very common and mostly avoidable source of error in veterinary imaging. Errors arise from substandard or non-diagnostic studies, or studies that have been incompletely performed. Poor radiographic techniques may lead to both the failure to detect lesions (false negatives) or overinterpretation (false positive) diagnoses.

**Under-reading**

This occurs when a lesion is not detected (missed) or not ascribed appropriate significance (under-interpreted). There are many reasons why this could occur. The “satisfaction of search” error occurs when subtle and often important radiographic changes are overlooked when a more obvious lesion(s) is/are present. The clinician directly involved in managing case is particularly vulnerable to this error. Influenced by his/her clinical findings, the clinician may be tempted simply to stop reading the radiograph once the expected or obvious lesion is found.

Under-confidence and lack of conviction of the observer’s own interpretation can also lead to under-reading, particularly if the diagnosis may have severe consequences for the patient. Search errors may occur when assessing complex body parts, such as the thorax. Lesions may be also overlooked in the relatively overexposed parts of a film, unless these parts are examined using a hot light (analogue films), or inappropriately windowed (digital images).

Under-reading errors are frequently repeated when a patient has serial imaging studies. If a lesion is overlooked on the first study, later studies are often evaluated for progression or resolution of the already reported abnormalities, and the undetected abnormalities remain unreported or are simply ignored. Sometimes, the earlier films may not be evaluated at all, particularly when a large number of images is presented, or the previous radiology report(s) are not read, and the lesion(s) continue to be missed.

In a busy clinical practice setting, insufficient time allocated to reading radiographic studies and/or other ubiquitous workplace distractions may lead to incomplete reading or snap judgements.

Sometimes, for whatever reason, the observer is overhasty in reading the study, too casual, or simply complacent. Sometimes, for reasons that can defy explanation, an obvious lesion is overlooked or dismissed on initial review of radiographs. Possible causes include dismissing the lesion because the diagnosis “could not be that simple”, or concentrating on searching for small lesions and simply missing the big one i.e. “could not see the wood for the trees”.

A common error in veterinary imaging is the observer failing to detect the absence of a normal structure. In general, we expect disease to produce lesions on diagnostic images; when the pathological process removes something rather than creating something new, the lesion may be overlooked.

**Over-reading**

Over-interpretation of normal anatomical structures or incidental changes (false positives) is also problematic. This often stems from a lack of appreciation of what constitutes normal. Variations in breed, body condition (particularly intrathoracic fat), age, and technique (exposure factors and phase of inspiration) all play a major role here. An appreciation of the range of variation of expected age associated or incidental findings in also important: a real abnormality may be determined to be the cause of the patient’s signs even though it is unrelated or incidental i.e. normal anatomic or physiologic variations may be interpreted as pathological.

Sometimes, the observer may “reach” for a diagnosis, particularly when under pressure from a clinician or client to resolve the case, or to try and explain a confusing clinical finding and make the radiograph “fit” the clinical picture. Sometimes, it’s just wishful thinking and overuse of the “imagine-o-scope”.

As mentioned above, bias or prejudice is a common cause of misdiagnosis. This is difficult to avoid for the general practitioner who will already have formed an opinion of a case based on history and physical examination before reviewing radiographs. An erroneous diagnosis may also occur if one has seen several similar cases recently and is expecting to see more. Perhaps the clinician has previously missed the lesion on a similar case, and is afraid of making the same error.
Many diseases have specific geographic distributions, which may lead to over-diagnosis because the disease is perceived to be common. Conversely, a diagnosis may also be missed because a disease is considered exotic and unlikely to occur in a geographical region.

Flawed reasoning

It is important to integrate the radiographic, clinical and laboratory data during the interpretation phase in order to reach meaningful conclusions. Errors in evaluating a lesion can occur because of incomplete, erroneous or misleading ancillary information. Over-interpretation or excessive significance can be attached to historical or clinical data resulting in over-reading of imaging lesions (bias). Errors can occur when the clinical data is cherry-picked to support the diagnosis being considered, while other important data is ignored. A previous diagnosis may be erroneously assumed to be the cause of current clinical signs, while a new diagnosis is overlooked.

Image diagnosis by the use or rules, such as numerical formulas to determine normalcy, may also lead to errors. These formulas should only be seen as diagnostic aids, and dogmatic interpretation or over-reliance should be avoided, especially if the findings do not make sense clinically. For each radiographic change, the observer should have a sense of the sensitivity and specificity of that finding for a particular disease condition.

Deficits of knowledge

Limited knowledge may result in failure to recognize a lesion as such or over-interpretation of incidental pathology or normal structures. This may be the result of lack of experience or inadequate training. This can also occur with uncommon or unusual lesions, which are not known to the observer or do not resemble those illustrated in textbooks.

Ignorance is bliss, and sometimes it’s a case of “not knowing what one does not know”. Emerging from this state can be a very humbling experience!

In veterinary imaging this type of error is especially common when one is learning a new imaging modality (such as ultrasound), the training in which is often inadequately at undergraduate level. With the exponential explosion of knowledge in veterinary medicine, with diagnostic imaging often leading the way, it is simply impossible to be proficient in everything.

Poor communication

It takes time, effort, training and practice to produce a complete, well-worded radiology report. Many radiographic studies are interpreted in clinical practice without ever receiving a formal written report. At best, a brief note of the clinician’s impressions will make it into the medical record, and at worst, no written interpretation is available whatsoever.

Communication errors are commonly encountered in reports prepared by veterinary radiologists for other specialists or general practitioners. Imprecise terms such “consistent with”, “most likely” “possible”, “probable”, “suspect” have quite different meanings and convey quite different levels of confidence for different individuals. Incorrect or vague anatomical descriptions are a common source of error and confusion.

Reports containing long lists of possible diseases or conditions with no attempt to whittle these down to a workable differential list, or omitting to offer rational diagnostic solutions or clinical plans, renders many radiology reports practically useless to the referring clinician. It is desirable for the referring clinician and radiologist to develop an open working relationship to foster mutual respect, confidence and trust.

Transcribed reports may get ‘lost in translation” and contain typing errors, nonsensical or misheard phrases, especially if the transcriber lacks experience or adequate medical training. Similar errors are encountered with radiologists utilising voice-recognition software, the technology of which is still far from perfect.

Even when carefully written, reports can contain errors such as incorrect counts (especially ribs or vertebrae) or body parts (right and left, transposing front and hind limb bones).

Summary

Being aware that errors in radiology are inevitable is an important step in learning to avoid making them. Recognizing and understanding the causes of errors should help reduce their frequency and avoid complacency.

Good radiographic technique is non-negotiable.

A systematic and diligent film reading approach, preferably with checklists, is essential to minimize errors. Investing in good radiology textbooks and atlases helps, but you still have to take the time to read and refer to them!

A “second read” by a radiologist or colleague has been shown to significantly reduce the rate of errors.

References


Sunbursts and Onion Skins: Interpreting Radiographic Bone Changes

Tim Spotswood, BVSc, MMedVet, Diplomate of the European College of Veterinary Diagnostic Imaging

Overview

Bone has a limited response to injury. The radiographic features of many diseases affecting bone are therefore often similar, with a large overlap in appearance between benign and more aggressive processes. This confounds interpretation, and it is unusual that a diagnosis can be made from radiographic appearance alone.

Objective

The purpose of this presentation is to review the radiographic features of bone lesions, their interpretation, and provide guidelines for determination of the aggressiveness of the disease process.

Evaluating the orthopaedic radiograph

The radiographs should be thoroughly evaluated in a consistent manner. The following system serves as an example of how to review an orthopaedic radiograph, starting from the outside of the limb/body part and working inwards:

- Check the label to identify the animal and radiographic date.
- Evaluate the radiographic quality, including whether the number of projections and positioning is appropriate.
- Evaluate the soft tissues for evidence or gas, swelling or foreign material.
- Evaluate periosteal margins for new bone formation.
- Evaluate bony cortices and subchondral bone for opacity changes.
- Evaluate the medullary cavities for changes in opacification.
- Evaluate the joint spaces and articular margins.
- Evaluate the joint capsule attachments for bone production or lysis.
- Evaluate the physes as they relate to the age of the animal.
- Evaluate the alignment of the bones.

Evaluation of soft tissues

Soft tissue changes are important in identifying the focus of disease, and often precede bone changes (the footprint of disease). Usually the nature of the swelling gives little clues as to the underlying aetiology. The severity of the swelling is also not necessarily prognostic.

Roentgen signs of soft tissue changes

1. Changes in soft tissue opacity

Fascial planes are normally visible as they contain fat, and often the margins of individual muscles can be discerned. Edema, hemorrhage or inflammation in the area of the fascial planes will obscure their margins. Joint effusion may displace a normally visualized fascial plane (particularly in the stifle and tarsus).

Gas in the soft tissues is seen as focal or regional areas of lucency. Gas in the tissues can occur with a break in the integumental surface (such as a compound fracture), iatrogenic (post operative or following perineural anesthesia), or gas producing organisms (rare).

Mineralization of soft tissues may be seen as an increased opacity in soft tissues. This should be differentiated from artefact or material on/in the integument. Wet fur, dirt, or dried blood on the skin may have a mineral opaque appearance on radiographs.

There are three mechanisms of soft tissue mineralization:

- Dystrophic mineralization is mineralization of dead, devitalized or degenerative soft tissues in the face of normal serum calcium and phosphorus levels.
- Metastatic mineralization is mineralization of normal soft tissue that occurs in the face of elevated serum calcium and/or phosphorus levels. This tends to occur when the product of the serum calcium and phosphorus in mg/dl is greater than 70 for a sustained period.
- Neoplastic mineralization is caused by bone-producing neoplasia in soft tissue. Types of soft tissue neoplasia that can cause this include osteosarcoma (extraskeletal osteosarcoma) and some mammary gland tumours. Some tumours originating from the bone itself can produce tumour new bone that extends into the soft tissues.
2. Changes in soft tissue size (soft tissue swelling)

Soft tissue swelling may originate from within a joint (intra-capsular or synovial swelling), around the joint (juxta-articular) or from outside a joint (extracapsular swelling). Sometimes this differentiation is not possible radiographically, and other imaging modalities (ultrasound, CT or MRI) may be needed. Swelling may be diffuse or focal, and can be caused by edema, hemorrhage, inflammation (cellulitis) or neoplastic tissue.

- Intracapsular soft tissue swelling is typically centred over the affected joint. Joint effusion can be recognised in certain situations: a good example is the infrapatellar fat pad attenuation in the femorotibial joint of the stifle, and displacement of the fascial planes of the gastrocnemius muscle. Synovial masses from synovial proliferation or synovial tumours may also be identified.

- Juxta- and extracapsular soft tissue swelling occurs outside the joint. Diffuse extracapsular swelling may obscure the radiographic signs of intracapsular swelling.

Roentgen signs of bone abnormalities

Bone consists of inorganic (mineral) and organic (protein matrix and cellular) components. The ratio of these components varies with age, with an approximate matrix/cellular : mineral ratio of 65 : 35 in immature animals, and 35 : 65 in mature animals. The mineral component of bone comprises 35% Calcium, 17% Phosphorus, 12% Copper and other. These minerals are incorporated into the bone in a crystalline form (hydroxyapatite).

Bony abnormalities can be focal, regional or generalized. Generalized bony lesions may indicate metabolic or nutritional diseases. Radiographs are relatively insensitive in detecting mineral loss: 30-60% alteration in mineral content of the bone is necessary before radiographic changes are seen. There is a therefore some delay between the onset of clinical disease and the observation of radiographic changes.

Focal lesions are easier to detect than generalized bone loss because of the contrast between the abnormal and surrounding bone. The location and number of lesions can help differentiate the aetiology of the lesion.

Location of lesions:

- Primary tumours are usually metaphyseal in location. Common locations for primary bone tumours include distal radius, proximal humerus (away from elbow), distal femur and proximal tibia (toward the stifle), and distal tibia.

- Fungal osteomyelitis is often metaphyseal in location, often is the same sites that primary bone tumours are seen.

- Metastatic bone tumours may be either diaphyseal or metaphyseal in location, sometimes associated with the nutrient foramen.

- Adult onset bacterial osteomyelitis can be localized to a site of injury or surgery, or can extend the entire diaphysis of the infected bone(s) and involve all cortices.

- Juvenile onset bacterial osteomyelitis often affects the epiphyseal or metaphyseal regions of bone.

Number of lesions:

- Primary tumours are usually solitary lesions that are confined to one bone (mono-ostotic) i.e. they usually will not cross an articular joint or extend into an adjacent bone.

- Metastatic bone tumours are typically polyostotic lesions (often more than one) and are often seen in the flat bones of the axial skeleton (ribs, skull, pelvis, vertebrae). They may also be seen in the diaphysis or metaphysis of the appendicular skeleton. They are not usually seen in two adjacent bones since each bone has a separate bone supply.

- Multiple myeloma is typical polyostotic.

- Fungal osteomyelitis is often polyostotic, although multiple bone sites may not be seen unless a bone scan (nuclear scintigraphy) is performed. Fungal osteomyelitis may involve adjacent bone due to soft tissue extension of infection.

Opacity changes in bone:

An opacity change is either the result of lysis (decreased opacity) or production (increase opacity). Both maybe present in a lesion. Lytic changes can be detected radiographically 5-7 days after initial injury. Productive changes require at least 10-14 days to be seen radiographically.

Patterns of osteolysis (bone loss)

Bone is living tissue, in constant flux with osteoclastic resorption and osteoblastic replacement. Normally, these two processes are in balance. Diseases that are characterized radiographically by decreased bone opacity have excessive osteoclastic resorption, or lack of osteoblastic production. Bone lysis can also occur through direct destruction of trabeculae by tumour cells, or proteolytic enzymes from inflammation or bacteria.
Generalized bone loss

Osteopenia is a term a generalized reduction in bone opacity on radiographs. Signs of osteopenia include:

» reduced bone opacity.
» cortical thinning.
» double cortical line (intracortical resorption of bone).
» endosteal reabsorption (coarse trabeculation).
» relative/apparent increase in opacity of cortical bone and vertebral endplates, resulting in prominent metaphyses or vertebral endplates (“picture frame” vertebral bodies.
» compression or folding fractures.
» loss of lamina dura around teeth (particularly seen with hyperparathyroidism).

Causes of Osteopenia

» Osteoporosis (loss of bone mass) occurs when the quantity of bone per unit volume is decreased, but bone is normal in composition. Osteoporosis involving a single limb is often from disuse, such as seen with a longstanding non-weightbearing lameness.

» Osteomalacia (loss of mineralization of the bone matrix) is characterized by an increased percentage of non-calcified osteoid by insufficient mineralization of the osteoid matrix. The bone then becomes soft.

Localized Bone Lysis

There is a latent period of 5-10 days between histologic destruction and radiographic demonstration of bone loss. Early bone loss may be recognized radiographically as a subtle alteration of bone texture. Bone loss is often easier to detect in the cortical (denser) than in cancellous bone, and more conspicuous in bone of normal opacity than in osteoporotic bone. Lysis that is better marginated is also more easily seen.

Bone lysis can be classified as benign (non-aggressive), semi-aggressive or aggressive depending on the distinctness of the margins. Lesions with poorly demarcated margins between abnormal and normal bone tend to be aggressive processes (tumour, active infection). A non-aggressive lesions tend to have sharp margins, often with a sclerotic rim surrounding the lesion (bone sequestrum, bone cyst), as the body has time to respond to the injury.

Specific types of bone lysis

» Geographic lysis is characterized as one large (>10 mm) area of lysis, typically with clear margins which may be sclerotic. Geographic lysis normally affects the medullary cavity. The overlying cortex may be interrupted, or thinned and displaced outwards (expansile). Geographic lysis is usually associated with more benign lesions. Examples include bone cysts, pressure atrophy or benign dental tumours. However if the overlying cortex is destroyed, this indicates that a more aggressive process may be present.

» Moth-eaten lysis is characterized by multiple foci of osteolysis, (3-10 mm) appearing as radiolucent defects. These may coalesce and may form areas of geographic osteolysis. Moth eaten lesions tend to have less well defined margins and broader zones of transition between normal and abnormal bone. The cortex is often irregularly eroded or disrupted. Moth eaten lysis is seen with more aggressive processes, such as malignant bone neoplasia, osteomyelitis and multiple myeloma.

» Permeative lysis is characterized by numerous small pinpoint areas of lysis (1-2 mm) which are indistinct and fade into the normal bone with a broad or inconspicuous transition zone. Permeative lysis is often better visualised in the cortices, and may cause irregular erosion of the cortices. Permeative lysis is a highly aggressive process usually due to a malignant tumour or fulminant osteomyelitis.

» Mixed bone patterns of osteolysis: many pathological processes involve a combination of patterns of lysis, in which case the lesion is classified by the most aggressive process present.

Patterns of osteogenesis

Periosteal reactions

A periosteal reaction is a reactive or healing response which occurs when the periosteum is irritated or elevated from the bony cortex by inflammation, blood, pus, neoplastic tissue, granulomatous tissue, edema or simply stripped from the cortex during trauma or surgery. The periosteum is composed of two layers. An outer fibrous layer and an inter cambian layer. The cambian layer retains its osteogenic properties and if not destroyed, will form new bone along small fibers and blood vessels (Sharpey’s fibers) back to the cortex directly perpendicular to the periosteum. Periosteal new bone is usually seen 10-14 days after insult, and sometimes earlier in young animals. Periosteal reactions can be characterized as nonaggressive, semi-aggressive, or aggressive, depending on the degree of periosteal elevation. Note that aggressive is not necessarily synonymous with malignancy,
but rather indicates that the disease is progressing faster than the bone can respond. Depending on the aggressiveness of the lesion, the area below the periosteum and between the Sharpey's fibers may or may not fill in with bone: a more aggressive lesion will lift the periosteum away from the cortex faster and farther leaving a larger space for the bone to fill in, producing a more spiculated (interrupted) appearance to the periosteal reaction. Slower processes have time to fill in with new bone, and appear more solid. Young periosteal reaction will be less opaque. As the reaction ages, it will become more opaque or bone like.

Periosteal reactions are classified morphologically as continuous or interrupted.

**Continuous periosteal reactions** are features of a slow disease process, where new bone formation is allowed to be laid down orderly. They are typically uniform in opacity, and fairly constant in depth. They are associated with benign or low grade processes, or healing aggressive processes. They are sometimes found at the edges of aggressive lesions.

Subtypes of continuous periosteal reactions include:

- **smooth-solid**: seen in mild trauma, tumour haematoma and remodeled new bone
- **Codman’s triangle**: a solid triangle of new bone at the edge of a more aggressive lesion. Sometimes seen at the diaphyseal edge of a malignant process
- **rough-solid**: associated with trauma and soft tissue inflammation
- **brush-border**: sometimes seen with hypertrophic osteopathy and soft tissue inflammation
- **palisading**: solid chunks of new bone formed perpendicularly to the cortex, often a feature of hypertrophic osteopathy.
- **lamellar (onion skin)**: forms with recurrent or cyclical episodes of periosteal elevation, such as metaphyseal osteopathy. Also seen with some forms of osteomyelitis and neoplasia, and is considered semi-aggressive.

**Interrupted periosteal reactions** are associated with rapidly changing lesions that breach the periosteum and cortex with little time for orderly repair. New bone will form along the Sharpey’s fibers giving a speculated appearance. The presence of pus, granulomatous tissue or tumour cells may fill in between the small spicules bridging the periosteum to the cortex, and is often accompanied by underlying cortical lysis. Interrupted patterns are usually associated with semi-aggressive to highly aggressive lesion. In general, short thick spicules (“thick brushlike”) indicate a less aggressive process than long thin spicules (thin brushlike to sunburst). If the periosteum is being elevated from a focal point by a rapidly enlarging lesion, such as a tumour, the new bone is laid in a radiating pattern, resulting in a sunburst appearance.

Amorphous periosteal reaction is typified by new bone of variable shape, size and orientation (clumps, clouds or wispy strands), and orientation. This periosteal reaction represents a disease process that is expanding very rapidly, breaking through the periosteum, and may contain spicules and remnants of the original bone cortex. The margins are usually poorly defined. This pattern is the least organized and most aggressive of all periosteal reactions, usually associated with a bone tumour.

**Interpretation and integration**

Radiographic features can be common to many diseases processes, especially in the early stages. This means that a diagnosis based solely on the radiographic appearance will often be erroneous. The most frequent dilemma is the differentiation between infection (fungal or bacterial osteomyelitis) and neoplasia (primary or metastatic bone cancer). The definitive diagnosis depends on bone biopsy. If the cortex is destroyed, then fine needle aspiration through the cortical defect (with ultrasonic or computed tomographic guidance if necessary) has also been shown to be useful in making a definitive diagnosis. It is essential to weigh all supporting clinical data (history, signalment, clinical signs and physical examination), additional laboratory data, response to previous treatment and other imaging findings.

Solitary metaphyseal aggressive lesions should be considered neoplastic until proved otherwise, especially in large breed dogs. Primary bone tumors of the appendicular skeleton other than osteosarcoma include chondrosarcoma, fibrosarcoma and hemaniosarcoma, and are uncommon. The other major differential diagnosis for a monostotic aggressive lesion is mycotic osteomyelitis. A juvenile form of osteosarcoma is seen in the dog (1-2 years).

The major differential diagnoses for polyostotic aggressive bone lesions are metastatic solid tumors and mycotic osteomyelitis. Dogs with mycotic osteomyelitis often present with localized soft tissue swelling in the metaphyseal region of long bones; often the lesion will have a draining lesion. Lameness will usually precede the development of a draining lesion. The origin is haematogenous spread fungal following entry into the body by the respiratory system. Patients with mycotic osteomyelitis tend to be younger than patients with metastatic solid tumors. In dogs, mammary, liver, thyroid and prostatic cancer may cause bone metastasis, and often produce polyostotic lesions on ribs, pelvis and vertebrae.

Bacterial haematogenous osteomyelitis may also cause polyostotic aggressive lesions in young animals but is rare in dogs and cats. The major differential for haematogenous osteomyelitis in the juvenile dog is metaphyseal osteopathy. Aggressive
monostotic lesion is the most common finding associated to bacterial osteomyelitis, usually a consequence of open fractures, surgical contamination or perforating injury (bite wound).

Radiographically, it is difficult to differentiate between aggressive infectious and neoplastic lesions of the digit. Neoplasia of the nail fold is common in the dog. Squamous cell carcinoma (which clinically often resembles a non-healing wound), melanoma, and mast cell tumour are relatively frequent. Less common neoplasia includes nailbed epithelial inclusion cyst, keratoacanthoma, inverted papilloma, and eccrine adenocarcinoma. These tumours usually affect one digit and necessitate aggressive excision therapy. Melanoma and mast cell tumour may metastasize, although squamous cell carcinoma may have a better prognosis provided that excision is carried out at an early stage. Multiple squamous cell carcinomas are seen in black dogs, affecting several digits. Nail bed tumours are rarer in old cats. Those that do occur are squamous cell carcinoma, hemangiosarcomas, and metastasis of primary lung carcinomas (lung-digit syndrome). Digital osteomyelitis can usually not be radiographically differentiated from malignant tumors. Diagnosis is made by histopathology of the removed tumour or infected digit.

References


Fluid resuscitation is indicated in most patients in shock. Two major exceptions include the heart failure patient, who may actually be hypervolemic, and the cardiac arrest patient that was known to have been euvoletic prior to the arrest. It should be kept in mind that congestive heart failure patients that have been receiving diuretics or have been vomiting may present in a hypovolemic state.

There is a wide range of fluids available for treating small animal patients. Each fluid was designed with a specific purpose in mind and understanding the composition of the fluid is essential to understanding the indications and both potential and real advantages and disadvantages of each. The choice of a fluid depends to a large extent on the underlying disease process that is being treated and the abnormalities in the fluid dynamics in the disease process. A patient that is dehydrated has a loss of interstitial volume, which will need to be treated very differently than the patient that is hypovolemic and is not perfusing its tissues adequately. It should be kept in mind that almost all fluids were designed for use in human beings and the effects in small animal patients may be different than what was intended when the fluid was developed. The label on the fluid should always be read to ensure the veterinarian and the nursing staff understand fully what is actually being infused into a patient.

Vascular Access

All patients in shock should have fluids administered into a vascular space. Typically this involves placement of a peripheral catheter; however central catheters may be indicated in some patients and, intraosseous access may be the only accessible route in very small patients, reptiles, birds and rodents. Peripheral vascular access can be achieved by placing a catheter in the cephalic or lateral saphenous vein in the dog and the cephalic and medial saphenous veins in the cat. Rapid fluid administration is indicated in most patients in shock and since flow is directly proportional to the radius to the fourth power and indirectly proportional to length, a large gauge, short catheter will allow the most rapid administration rates. General guidelines are as follows: 18 ga in cats and smaller dogs, 16 ga in medium-sized dogs (10-30 kg), and 14 ga in larger dogs (greater than 30 kg). One large bore short catheter may be sufficient for patients in mild shock; however, those patients in severe shock should have two large bore catheters placed. Central catheters are rarely placed as part of resuscitation unless a cutdown is indicated; however, they are indicated frequently once resuscitation has been completed.

Since these patients may be hypovolemic the veins may not stand up well. Lowering the vein below the level of the heart may help improve filling. Penetrating the skin can be the most painful part of the procedure. In addition burring usually occurs during penetration of the skin. A small cutdown (1-3 mm skin incision) using the bevel of a hypodermic needle decreases pain and the likelihood of burring and permits a larger catheter to be placed than might otherwise be possible. A larger cutdown can be made using the same hypodermic needle if necessary.

Intraosseous catheters can be placed in the humerus, the trochanteric fossa, or tibial plateau. Either an 18-22 ga hypodermic needle or commercial needle is placed in the bone marrow cavity. Because these are “central” catheters, any medications or fluids that would be infused via a central line can be infused via an intraosseous catheter.

What Kind Of Fluid Should Be Used For Resuscitation?

The choice of fluids therapy is dictated by the cause of the shock. Often some combination of crystalloid and colloid will be required. Ideally the fluids used to treat patients should be identical to the fluids lost by the patient. If the patient lost 40% of its blood volume out on the pavement then whole blood is an essential part of the fluid resuscitation. If the patient has been vomiting
for 2 days and has lost electrolytes then electrolyte-replacing fluids are essential. If the patient has a protein-losing enteropathy or a severe vasculitis, then fluids that provide colloid support to counteract the effects of the lost proteins will be needed.

Ideally blood should be used for resuscitation if the patient has lost whole blood. The lower the hematocrit becomes the more important this is to ensure adequate oxygen delivery to the cells. If the patient has lost clotting factors then clotting factors should be replaced. This means administering fresh whole blood or fresh frozen plasma. Platelets are only found in fresh whole blood (administered within 6-8 hours) and in platelet-rich plasma. If the patient has a low albumin or a loss of clotting factors then fresh frozen plasma should be used. If only albumin is low frozen plasma or albumin can be given.

Often the ideal fluid is not feasible due to lack of hospital resources or lack of client resources (finances). In these situations the next best fluid is chosen but the clinician should be aware that there might be negative side effects of a treatment that is less than ideal. The side effects should be monitored for in an attempt to minimize their negative impact on the patient. For instance the patient that is severely hypoproteinemic that is resuscitated

**Crystalloids**

Crystalloids are aqueous solutions of mineral salts (usually predominantly sodium and chloride) or other water-soluble molecules that are capable of distributing to all fluid compartments. They have no oncotic pressure. Since approximately 80% of extracellular fluid is in the interstitial space crystalloids will rapidly redistribute and after as short a period of time as 20 minutes there will be only 20% to 30% of the administered volume remaining in the circulation. On a short-term basis crystalloids certainly will expand the intravascular space, but this effect will be short-lived. Thus, crystalloids should be thought of as interstitial dehydrators, not intravascular volume expanders. An increase in interstitial fluid can lead to tissue edema (thus decreasing the ability of oxygen to diffuse to the cells). Interstitial edema may be extremely detrimental in cases of cerebral edema and pulmonary edema. Buffered solutions are usually indicated for resuscitating patients in shock since administration of a highly acidic solution may worsen a preexisting metabolic acidosis. Fluids such as 0.9% saline and Ringer’s solution do not contain buffers. Most buffered solutions, unless they indicate they have been prebuffered to a pH of 7.4, have a pH of about 6.8; the metabolism of the buffer leads to increase in the pH to normal. Buffered solutions usually contain lactate, gluconate or acetate. The liver must metabolize lactate whereas many cells in the body metabolize acetate and gluconate; however, end-stage liver disease must be present before the patient will have problems metabolizing the lactate. Solutions containing lactate are no longer recommended by some due to the adverse effects of the lactate. These include neutrophil priming and worsening of cellular apoptosis.

Some replacement fluids contain calcium and others contain magnesium. Given that hypomagnesemia is considered to be a concern in critical patients it may be advantageous to use a replacement fluid containing magnesium rather than calcium, unless the patient is predisposed to hypocalcemia. Calcium-containing fluids as well as magnesium-containing fluids should not be administered concurrently through the same line as blood products anticoagulated with citrate since the resultant precipitate may be detrimental to the patient. Crystalloids are generally isoosmolar; however, they become hyperosmolar once other medications or supplements are added to the fluids. This may be important to patient therapy.

Replacement fluids contain an electrolyte distribution that approximates that found in the extracellular fluid. The average sodium concentration in the serum of the dog is approximately 146 mmol/L and in the cat is 156 mmol/L. Based on these sodium concentrations most fluids are hypotonic for the dog and even more so for the cat. This can have serious repercussions as infusion of these fluids can lead to significant hyponatremia if the cat truly needs a replacement fluid. Commonly used replacement fluids are 0.9% saline, lactated Ringer’s solution, Normosol-R, and Plasmalyte. Lactated Ringer’s solution has a sodium concentration of 130 mmol/L. Plasmalyte and Normosol-R usually have a sodium concentration of 10 mmol/L.

Normal saline at 0.9% has a sodium concentration of 154 mmol/L which makes it hypertonic for most patients. The chloride is also 154 mmol/L which can lead to the development of a hyperchloremic metabolic acidosis. Due to its acidifying nature (pH of 5.4) it should generally be reserved for patients with gastric outflow obstructions, patients with hypoadrenocorticism, and patients with hypercalcemia. It is important to note that electrolyte abnormalities cannot be corrected in patients with any of these 3 conditions without administration of saline – usually in very large volumes. Extreme caution should be exercised when giving 0.9% saline to hypotremic patients. If these patients have a corrected chloride ([Cl] corrected = [Cl] measured x 146/[Na+] measured in the dog and [Cl] corrected = [Cl] measured x 156/[Na+] measured in the cat) that is elevated then administration of 0.9% saline may cause or worsen a metabolic acidosis.

**Hypertonic saline** is a hyperosmolar crystalloid fluid used for resuscitation of hypovolemia. It is usually given as a 7.5% solution (2600 mOsm/L). The hyperosmolarity leads to rapid intravascular volume expansion by drawing fluids from the interstitial and intracellular space into the intravascular space. Its major benefit is that it can produce an equivalent intravascular volume expansion to colloids but at one-fourth the volume. Caution should be exercised when infusing this fluid in patients with
uncontrolled internal hemorrhage since the rapid rise in volume and blood pressure can worsen the hemorrhage. Because it is a crystalloid it will rapidly redistribute similar to all other sodium chloride-based solutions; however, its effects can be prolonged by concurrent administration of a colloid. It also appears to have an immunomodulatory effect including decreasing mesenteric lymph production and eliminating neutrophil priming, which decreases susceptibility to sepsis following hemorrhagic shock.

Five percent dextrose in water is a very hypotonic fluid. It can be used sometimes to treat hypernatremia but is generally used as a diluent for medications. It is an acidic fluid and any medication that is added to it must not be inactivated by the low pH. Due to its hypotonicity it should never be used for resuscitation, as a replacement fluid, or as a sole maintenance fluid. It only contains 200 kcal/L so the caloric density is insufficient to provide a useful energy intake.

Colloids

Colloids are fluids containing large molecular weight substances that generally are not able to pass through capillary membranes. They exert an oncotic pull which helps maintain fluid in the intravascular space. Colloids can be considered intravascular volume expanders. Since most patients in shock require sustained intravascular volume expansion, colloids are indicated frequently during fluid resuscitation. Examples include synthetic colloids such as the, dextrans, hydroxyethyl starch (hetastarch, pentastarch), and biologic colloids such as whole blood, plasma, and albumin. Colloids are usually isoosmolar. All synthetic colloids have the potential to cause a dilutional coagulopathy.

Almost all colloids have molecules of varying sizes although the average size is the one that is typically listed. The smaller the size of the colloid molecule, which is measured in kilodaltons (kD), the more potent it’s colloid effect but the shorter its duration of effect. As a general rule the kidney filters all molecules 40 kD and smaller.

All synthetic colloids have the potential to cause a dilutional coagulopathy. Since most patients in shock require sustained intravascular volume expansion, colloids are indicated frequently during fluid resuscitation. Examples include synthetic colloids such as the, dextrans, hydroxyethyl starch (hetastarch, pentastarch), and biologic colloids such as whole blood, plasma, and albumin. Colloids are usually isoosmolar. All synthetic colloids have the potential to cause a dilutional coagulopathy. Most patients in shock require sustained intravascular volume expansion, colloids are indicated frequently during fluid resuscitation. Examples include synthetic colloids such as the, dextrans, hydroxyethyl starch (hetastarch, pentastarch), and biologic colloids such as whole blood, plasma, and albumin. Colloids are usually isoosmolar. All synthetic colloids have the potential to cause a dilutional coagulopathy.

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All synthetic colloids have the potential to cause a dilutional coagulopathy and some have a direct effect on the coagulation cascade.

Since most patients in shock require sustained intravascular volume expansion, colloids are indicated frequently during fluid resuscitation. Patients with SIRS (systemic inflammatory response syndrome) or sepsis frequently have increased vascular permeability which leads to leakage of albumin and other small proteins out of the intravascular space (“third-spacing”). Synthetic colloids that have a larger molecular weight than albumin (69,000 Daltons) usually remain in the intravascular space.

Dextrans are polysaccharides produced by the bacterium Leuconostoc in a sucrose media. Dextran 70 has an average molecular weight of 70 kD. Dextran 70 expands the fluid volume by approximately 1.4 times and has a duration of effect of approximately 4 to 8 hours. Since albumin has a molecular weight of 69,000 Daltons, any disease associated with a vascular leakage of albumin is likely to lead to loss of dextran 70 equally quickly. With dextran 70 a coagulopathy may occur secondary to dilution, coating of platelets, change in the function of factor VIII:Ag, and destabilization of the clot through polymerization with fibrin. Dextran 70 can cause red blood cell cross-linking that leads to rouleaux formation, which may interfere with cross matching.

Hydroxyethyl starch is a molecule made from maize or sorghum and is primarily an amylopectin. The average molecular weight varies tremendously from the130 kD and 200kD commonly found in Europe to the 450 kD commonly used in North America. The starch may be in a 0.9% saline solution, a lactated Ringer’s solution or combined with hypertonic saline to create a potent resuscitation fluid. It can interfere with factor VIII and von Willebrand’s factor, although to a lesser extent than dextran, and the 130 kD hydroxyethyl starch (tetraastarch) has been shown to have fewer coagulopathic effects when compared to the 200 kD hydroxyethyl starch. Hetastarch is one of the main types of hydroxyethyl starches available. Research with hetastarch in endotoxic shock has shown that it has a significant antiinflammatory effect with a subsequent improvement in capillary permeability. It inhibits the accumulation of neutrophils and inhibits other proteins induced during sepsis, which may have beneficial effects. Doses greater than 20 ml/kg/day have been associated with an increased incidence of bleeding problems, which may be due to dilution, increased microvascular perfusion, or decreased platelet aggregation in addition to the direct coagulopathic effects.

Pentastarch is a slightly smaller molecular weight hydroxyethyl starch with an average molecular weight of 264,000 Daltons. This makes it a more potent colloid than hetastarch but it also has a shorter half life with about 70% being eliminated within 24 hours. It is typically in a 0.9% saline solution.

How Fast Should Fluids Be Given?

Fluids should be given as fast as necessary to resuscitate the patient. The ideal goal, although often unobtainable, is to resuscitate the patient during the first hour following injury. Crystalloids and colloids (synthetic and biologic) can be bolused to a dog as fast as the catheter diameter will permit. The use of pressure infusor bags ensures rapid administration of fluids. Crystalloids and biologic colloids can be bolused rapidly to cats but synthetic colloids should be administered over a minimum of 10 to 20 minutes. When given faster than this the cat may become hypotensive (histamine-induced vasodilation??). Human serum albumin should never be bolused.
How Much Fluid?

The amount of fluid to be administered during resuscitation must be based on restoring normal oxygen delivery. This means fluid is administered until respiratory rate and effort are normalized, heart rate is normalized, blood pressure and central venous pressure are normalized, mucous membrane colour and capillary refill time are normalized, temperature (toe web and central) is normalized, and urine output is normalized.

While the blood volume of the dog is approximately 80-90 mL/kg the blood volume of the cat is approximately 55-60 mL/kg. These numbers are kept in mind during fluid administration. Fluids should be infused to achieve or maintain a systolic blood pressure of 100-120 mm Hg, a diastolic blood pressure of 60-80 mm Hg and a central venous pressure of 5-8 cm H2O. If blood pressure is being monitored indirectly a Doppler is recommended since flow can be subjectively assessed. In addition, most oscillometric units are inaccurate in hypotensive cats. If patients do not respond to infusion of fluids (i.e. blood pressure remains low) dobutamine or dopamine infusions may be indicated. Urine output should be monitored in patients in shock or with renal dysfunction. An indwelling urinary catheter may be indicated to be able to quantify urine production.

In the author's experience fluid resuscitation is started with a bolus of 20-30 ml/kg of a buffered, balanced electrolyte solution. This volume is reduced by approximately 30% in cats. If the patient is suspected, based on clinical presentation or initial lab work, of having a low colloid osmotic pressure then synthetic colloids are used during initial resuscitation with boluses of 5 ml/kg to a maximum of 20 ml/kg (15 ml/kg in the cat??). After each bolus the patient's physical parameters are reassessed. Once parameters have been normalized fluid resuscitation is slowed to maintenance rates.

If the patient is assessed to be in hypovolemic shock from blood loss, or is thought to have a protein-losing vasculitis, or is a potential candidate for disseminated intravascular coagulation, then an attempt is made to use blood products during resuscitation. This prevents dilution of remaining hemoglobin and clotting factors. Whole blood (or packed red blood cells if available) should be administered to maintain a packed cell volume as close to 27% in the cat and 30% in the dog as possible, assuming the patient does not have a chronic anemia. Autotransfusion may need to be considered if the patient has a significant hemoadbomen and large quantities of blood products are not available. Fresh frozen plasma should be administered to maintain an albumin greater than 20 g/L and to provide clotting factors to any patient with a coagulopathy. Patients with a prolonged prothrombin time, activated partial thromboplastin time (or activated clotting time), or significantly decreased platelets (<75,000 or 5/oil immersion field) may have a clinically significant coagulopathy. If in doubt it is always better to err on the side of providing coagulation factors and red cells. Preventing a problem from occurring is always better than trying to treat a problem once it has occurred.
Shock can be defined as insufficient perfusion or blood flow to the tissues that leads to inadequate delivery of oxygen, and nutrients to the cells. The lack of blood flow also leads to an accumulation of waste products, primarily carbon dioxide. Energy is depleted, lactic acidosis develops and there is a disruption of normal cellular function. In an acidic environment enzyme-driven systems cease to function appropriately. These enzymes control multiple functions from muscle contraction to coagulation. The fundamental goals of resuscitation are to reverse this process.

Shock can occur secondary to hypovolemia, poor cardiac function, or vascular dysfunction. These causes may overlap since instigating causes can affect the system in multiple ways and all systems are ultimately interrelated. Hypovolemia occurs most commonly secondary to hemorrhage but also can occur secondary to third-spacing of fluids or from excessive losses due to vomiting or diarrhea. Cardiogenic shock at the extreme end of the spectrum occurs during cardiac arrest. In the less extreme scenario cardiogenic shock occurs secondary to heart failure. Septic shock is initiated by an infection which leads to SIRS (systemic inflammatory response syndrome), a cascade of events resulting in vascular dilatation (decreased systemic vascular resistance), increased capillary permeability and ultimately organ failure.

Assessing Tissue Perfusion

Appropriate resuscitation is only possible when the abnormal signs are recognized. History often plays a large role in helping to diagnose a patient in shock. This is true especially for hemorrhagic shock related to trauma. Large breed older dogs that present with a history of collapse and a distended abdomen often are in shock secondary to splenic neoplasia and subsequent hemorrhagic shock. Patients that have been running on the beach in hot weather or were locked in a car in the heat are in shock. The unvaccinated puppy with a history of vomiting and diarrhea that presents recumbent has heatstroke and is in shock. The patient with bradycardia and hypotension is in the early stages of decompensatory shock whereas the patient with tachycardia and hypotension is in uncompensatory shock and often dies within minutes without aggressive resuscitation measures. Blood pressure and heart rate play a key role in identifying the patient in shock and must be monitored closely. Blood pressure should be considered the fourth vital sign and should be measured in every ill or injured patient.

Patients with significant anemia (packed cell volume < 20% acutely or 10% in chronic anemia), metabolic acidosis, or increased blood lactate concentrations should be assumed to be in shock until proven otherwise. Patients with significantly altered electrolytes (sodium, potassium, magnesium, calcium) may be in shock due to altered muscle contractility. Electrocardiographic abnormalities including tall T waves, ST segment depression and premature ventricular contractions or ventricular tachycardia may all be indicators of myocardial hypoxia. An end-tidal capnometry measurement less than 15 to 18 mm Hg indicates severely decreased pulmonary blood flow (unless the patient has been deliberately hyperventilated) and is an indication of impending cardiac arrest. A finding of a significantly decreased fractional shortening on cardiac ultrasound is consistent with cardiogenic shock until proven otherwise.
An objective assessment of splanchnic perfusion is difficult. Urine output less than 1 ml/kg/hr may indicate decreased renal perfusion. Gut perfusion can be assessed by checking for hemorrhage on rectal examination or checking for blood in any vomitus; however, this is very subjective.

**Goals of Resuscitation**

The end goals of resuscitation should be to restore tissue perfusion as well as appropriate oxygen delivery. Physical exam parameters including level of consciousness, respiratory rate and effort, heart rate and rhythm, pulse strength and temperature should be normalized. Blood pressure should be normal (approximately 120/80 +/- 10 mm Hg). Central venous pressure should be in the 4 to 8 cm H2O range. Urine output should be at least 1 ml/kg/hr.

It is possible to impact oxygen delivery through medical therapy but it is difficult if not impossible to affect oxygen uptake by the cells. The goal of resuscitation is to restore normal levels of oxygen (and nutrient) delivery to the cells. Adequate oxygen must be delivered to the alveoli where the pulmonary circulation must be able to take up the oxygen (adequate ventilation and perfusion to the lungs). There must be sufficient hemoglobin to carry the oxygen, adequate blood volume to get the oxygen where it needs to go and an effective pump (heart) to deliver the blood. Each of these factors can be affected by therapy provided during resuscitation.

The content of oxygen in the arterial blood is calculated by the following formula:

\[ CaO_2 = (Hb \times 1.34 \times SaO_2) + (PaO_2 \times 0.003) \]

\( CaO_2 \) is the arterial content of \( O_2 \), \( Hb \) is hemoglobin in g/dl, \( SaO_2 \) is the percent saturation of the hemoglobin, and \( PaO_2 \) is the partial pressure of \( O_2 \) in mm Hg.

The amount of hemoglobin in the blood has a more dramatic effect on oxygen content than the amount of oxygen dissolved in the plasma. For this reason it is recommended that the hemoglobin concentration not decrease below 7-10 g/dl (packed cell volume of 21-30%). Ideally the packed cell volume should be kept as close to 30% as possible, perhaps 27% in cats. This number is somewhat arbitrary and depends to a large degree on the patient’s normal hemoglobin. For instance, a patient with a hematocrit of 50% that has a hematocrit of 21% after a traumatic episode has lost over 50% of its blood volume acutely. This patient is at high risk of developing hypoxia, tissue ischemia, and lactic acidosis, and needs a blood transfusion. A patient with a normal hematocrit of 30% that decreases to 21% may be much more tolerant of this change. Patients at higher altitudes are much more likely to be affected by low hemoglobin than those at sea level.

**Cardiovascular Physiology and Hemodynamic Concepts**

An understanding of cardiovascular physiology and hemodynamic concepts is essential in order to be able to provide appropriate fluid therapy during resuscitation of the patient in shock. Again, the goal of resuscitation is to restore normal or provide increased levels of oxygen delivery to the cells. Cardiac output is calculated as a product of stroke volume and heart rate. Stroke volume is dependent upon preload, cardiac contractility, and afterload (resistance to outflow).

Blood volume can be difficult to assess accurately but it can be estimated. If the animal’s blood volume is normal then preload and blood pressure should be normal, assuming the heart is functioning normal. Approximately 10% of the blood is in the arteries, 20% in the capillaries and 70% in the veins. This makes it essential to evaluate the venous side of the circulation. In clinical terms preload is the volume returning to the right side of the heart. It is assessed most effectively by measuring central venous pressure (CVP). If no central catheter is present the jugular veins should be clipped and examined for distention and filling. Patients with hypovolemic shock will have flat jugular veins and poor filling when they are held off at the thoracic inlet. In breeds of dogs with highly visible lateral saphenous vein, distention of this vein also can be used as a subjective assessment of volume status. Patients with right-sided heart failure or diseases interfering with return of blood to the right side of the heart (pericardial tamponade, tension pneumothorax, etc.) may have distended jugular veins in the face of hypovolemia.

Volume is dictated to a large extent by oncotic pressure within the vessels and albumin is the largest contributor to oncotic pressure. Patients with an acute hypoalbuminemia will always be hypovolemic. (Patients with chronic hypoalbuminemia may not.) Ideally oncotic pressure should be maintained within normal limits; however measuring oncotic pressure is problematic. As a general rule patients with serum albumin concentrations less than 20 g/L require albumin replacement as well as synthetic colloids in order to restore oncotic pressure. Those with serum albumin concentrations between 20 and 25 g/L may require synthetic colloid therapy to restore euvolemia.

Gastric distention secondary to gastric dilation or aerophagic can cause significant impairment of diaphragmatic movement as well as venous return to the heart. This can lead to significant hypoxia, hypercarbia and hypotension. Gastric decompression may be indicated during resuscitation. If the animal cannot breathe because of the distention decompression can be performed immediately by transabdominal trocarization. The rapid decompression may cause cardiovascular collapse and should be avoided whenever possible until fluid resuscitation has been initiated.
Blood pressure is a function of cardiac output and systemic vascular resistance. Cardiac output is a function of stroke volume (largely dependent upon preload) and heart rate. Blood pressure can be estimated by palpating pulses, but extreme caution is warranted. The strength of central pulses (femoral pulse) is based on the pulse pressure (the difference between the systolic pressure and the diastolic pressure). Thus, a blood pressure of 120/80 is equal to a pulse pressure of 40, as is a blood pressure of 100/60. However, these 2 values would dictate different therapeutic approaches. Skill plays a large role in the ability to palpate pulses. The femoral pulse becomes nonpalpable between 30 and 60 mm Hg systolic pressure depending on the skill of the clinician, and patient parameters such as obesity, shivering or shaking. Blood pressure should be measured for more objective assessment that that afforded by palpation of pulses alone and should always be assessed in conjunction with the heart rate. Tachycardia in patients in shock usually implies hypo-volemia or pain. Hypovolemia triggers the baroreceptors and the heart rate elevates in an attempt to restore normal blood pressure.

Cardiac contractility is dependent upon heart muscle function, which is dependent upon heart rate, heart rhythm, acid-base status, electrolyte levels, and the intrinsic health of the muscle. The effectiveness of the pump can be assessed most accurately in a clinical setting using echocardiography. The heart muscle is supplied with oxygen via the coronary vessels. Coronary filling occurs during diastole; therefore, if the patient is tachycardic diastolic time is reduced and coronary perfusion is reduced. Irregular rhythms alter cardiac contractility; therefore, a lead II electrocardiogram should be assessed and arrhythmias that may interfere with cardiac function should be treated. Serum electrolytes levels should be assessed and sodium, potassium, and calcium values normalized as rapidly as possible. Muscle function is impaired when severe acidosis is present.

**Acid-Base Status**

Changes in the blood gases and acid base status of the body reflect abnormalities in the metabolic system or the respiratory system. Without being able to characterize these abnormalities it is virtually impossible to provide the best treatment to a patient. The goal should be to return the pH to as normal as possible by correcting the underlying metabolic and respiratory disorders. Respiratory acidosis is treated by improving ventilation. Metabolic acidosis is treated by improving tissue perfusion. Sodium bicarbonate may be needed in cases of severe acidosis, but is rarely required in patients in shock if fluid resuscitation is appropriately administered. A patient in septic shock with a pH of 7.0 after fluid resuscitation has not been adequately resuscitated and may not be able to maintain normal vascular tone and cardiac function, which may lead to a refractory shock state. A patient in hypovolemic shock that has been given fluids but still has a severe metabolic acidosis has not been adequately resuscitated.

Arterial blood gases must be used to determine oxygen tensions since venous oxygen tensions are dependent to a large extent on oxygen uptake in the tissues, which can vary tremendously in critical patients. Assuming relatively normal tissue perfusion, the carbon dioxide tension in a venous blood gas is usually a fairly accurate reflection of pulmonary function, often varying by only 2 to 3 mm Hg from the arterial tension. During resuscitation from significant shock there can be tremendous washout of carbon dioxide from the tissues and the carbon dioxide tension in this situation is a reflection of tissue metabolic status rather than pulmonary function. Using central venous blood samples will minimize the impact of any washout from regional tissue beds. Comparison of central and peripheral venous blood gases may provide information on tissue perfusion abnormalities in the region where the venous sample was obtained.

**Hypotensive Resuscitation**

Hypotensive resuscitation refers to a controversial form of resuscitation provided to trauma patients that may still be actively hemorrhaging. It involves the use of limited fluid resuscitation until the hemorrhage is controlled. The systolic blood pressure is maintained between approximately 80 and 100 mm Hg. The goal is to avoid increased hemorrhage from normotension or hypertension that might cause fragile clots to be disrupted. At the same time dilution of clotting factors from excessive administration of crystalloids or synthetic colloids is avoided. There is danger of inadequate perfusion (especially splanchnic) since the patient is not being adequately resuscitated. The advantage is that severe hemorrhage may be controlled without requiring administration of multiple units of blood products and the patient’s life may be saved. Hypotensive resuscitation can be helpful in patients with significant intraabdominal hemorrhage.

**Coagulation**

The triad of hypothermia, acidosis, and coagulopathy has been called the “trauma triad of death”. It is important not to worsen any preexisting coagulopathy by excessive dilution of clotting factors using crystalloids or synthetic colloids, by improving the acid-base status and by avoiding hypothermia. This means that the appropriate fluids should be administered based on the underlying disease and that fluids should be warmed to body temperature – especially if the fluids are being given very rapidly. Crystalloids play a role in resuscitation; however, caution should be exercised to ensure the triad of death is being avoided through judicious use of warm fluids and blood products. Patients that are likely to have a coagulopathy (evidence of petechiation, hematoma...
formation at sites of venipuncture, severe heatstroke, estimated greater than 50% blood loss, etc.) benefit from a source of clotting factors during resuscitation. Ideally coagulation tests should be checked first but if that is not possible then it is better to err on the side of providing the fresh frozen plasma or fresh whole blood.

**Hypothermia**

Critically ill patients may present with hypothermia. In cats low rectal temperatures often correlate with poor peripheral perfusion and the core temperature actually may be fairly normal. Patients may become hypothermic during resuscitation secondary to intravascular infusion of large volumes of room temperature fluids. Hypothermia interferes with normal metabolic functions leading to vasodilation, cardiac dysfunction, and interference with the coagulation cascade. Core rewarming should be instituted since peripheral rewarming may lead to worsening of the vasodilation and subsequent worsening of the hypothermia. In the author’s opinion core rewarming should be instituted in hypovolemic shock but should be done with caution in cats with poor perfusion from other causes such as cardiac disease and renal failure since they may actually overheat. Artificial warming devices should be insulated from the patient since they can cause burns. Means of rewarming patients includes the use of warm water bottles, warm water circulating blankets, oat bags, warm blankets, and hot air circulating devices. Fluids should be infused at normal body temperature in the hypothermic patient.

**Too Much Fluid**

Geriatric patients or those with significant underlying renal, hepatic or cardiac disease may not tolerate rapid fluid administration. If the intravascular space is expanded too rapidly the patient may develop signs of pulmonary edema. This is a late sign and indicates the earlier signs were overlooked or the patient had unrecognized cardiac disease (most commonly) or oliguric renal failure. Central venous pressure or jugular filling will increase first followed by an increase in respiratory rate and effort. If excessive crystalloids are administered the patient will develop signs of overhydration. If the kidneys are functioning normally increased urination will be noted. Often the first clinical sign of pathologic overload in the cat is chemosis and serous nasal discharge in the dog. If the patient is hypooncotic the first sign may be peripheral edema.

**Refractory Hypotension**

Pharmacologic intervention is indicated when patients remain hypotensive despite appropriate fluid therapy. This means central venous pressure must have been maximized. Hypoglycemia should be treated if present. Patients with sepsis or underlying heart disease may require a positive inotrope. Dobutamine is the drug of choice in this situation. If vasopressors are indicated dopamine or norepinephrine are the drugs of choice. Pressors should only be indicated in patients with severe vasodilation secondary to anaphylactic shock (in which case epinephrine is indicated) or those with severe sepsis. Whenever a vasopressor is used in appropriately it can decrease tissue perfusion which can ultimately worsen outcome.

**Monitoring**

Close monitoring of these patients is essential if morbidity and mortality are to be minimized. Patient parameters should be monitored as frequently as every 5 minutes during resuscitation and recorded on a flow sheet. Recording numbers is vital since patient treatment frequently is guided based on evaluation of trends.
Transfusion Medicine —
Which Product and How Do I Give It

Jennifer J. Devey, DVM, Diplomate ACVECC

Transfusion medicine involves the infusion of blood products, which can include whole blood or blood components. Understanding the properties of each of the blood products as well as indications for the use of each is essential. This lecture will discuss the use of common blood products such as whole blood, packed red blood cells, plasma and human serum albumin.

Blood Products

Blood can be separated into multiple components including packed red blood cells, plasma, cryoprecipitate, cryopoor plasma and platelet-rich plasma. Packed red blood cells contain up to 80% packed red cells although many blood banks now add a diluent to the red cells to provide a product with a hematocrit of about 40-45%. Fresh frozen plasma is considered fresh frozen for 1 year and then frozen for another 4 years. Fresh frozen plasma contains coagulation factors, albumin and immunoglobulins. Frozen plasma contains Factors II, VII, IX, X, albumin and immunoglobulins. Cryoprecipitate contains 50% Factor VIII/vWf from the original unit, 20-40% fibrinogen and some Factor XIII. Cryopoor plasma contains the remaining clotting factors, albumin and immunoglobulins.

Human albumin made from pooled human plasma is a concentrated source of albumin. At a 25% concentration the COP is 100 mm Hg, making it a very potent colloid that is able to expand the intravascular volume by 5 times the volume infused. It is also hyperosmolar at 1500 mOsm/L. For both of these reasons the patient must be monitored closely for signs of fluid overload when 25% human serum albumin is being infused. The half life is approximately 16 hours. Doses of 2.5-5.0 mL/kg have been recommended with a maximum dose of 2 g/kg. Because it is human albumin allergic reactions are possible. This may manifest as facial swelling, vomiting or fever. Delayed reactions several weeks after administration have been documented.

Anemia

Red cells are the primary carriers of oxygen in the blood and the amount of hemoglobin in the blood has a much more dramatic effect on oxygen content than the amount of oxygen dissolved in the plasma as seen by the equation below. The content of oxygen in the arterial blood is calculated by the following formula:

$$\text{Ca } O_2 = (\text{Hb} \times 1.34 \times \text{SaO}_2) + (\text{PaO}_2 \times 0.003),$$

where $\text{Ca } O_2$ is the arterial content of $O_2$, $\text{Hb}$ is hemoglobin in g/dl, $\text{SaO}_2$ is the percent saturation of the hemoglobin, and $\text{PaO}_2$ is the partial pressure of $O_2$ in mm Hg.

Anemia can be caused by blood loss, red blood cell destruction or lack of production (bone marrow disorders). The severity of the impact of the red cell loss will depend on the amount of functional hemoglobin lost as well as how acutely the loss occurred. The body can compensate for anemia over time by off-loading more oxygen from the hemoglobin; however, this compensatory mechanism is not functional in the acutely anemic patient. Transfusion triggers to a large extent are based on these factors. It is generally recommended that the hemoglobin concentration not drop below 7-10 g/dl (packed cell volume of 21-30%). In critical patients and acutely anemic patients the packed cell volume should be kept as close to 30% as possible. This number is somewhat arbitrary and depends to a large degree on the patient's normal hemoglobin. For instance, a patient with a hematocrit of 50% that has a hematocrit of 26% after a traumatic episode has lost over 40% of its blood volume acutely. This patient is at high risk of developing hypoxia, tissue ischemia, and lactic acidosis, and needs hemoglobin supplementation. A patient with a normal hematocrit of 30% that decreases to 21% may be much more tolerant of this change. Patients at higher altitudes are much more likely to be affected by low hemoglobin than those at sea level.

Hypoalbuminemia

Albumin is the largest contributor to oncotic pressure in the body. Acute losses lead to an inability of the body to maintain adequate intravascular volume. Chronic losses do not seem to have the same impact which probably relates to an adjustment over time to the hypoalbuminemia. Albumin also is
important as a free radical scavenger. It may improve micro-circulatory flow and is important as a carrier of drugs.

One third of the body’s albumin is in the plasma and two-thirds of the albumin is in the interstitial space; therefore, replenishing low albumin concentrations often requires significant volumes of plasma, especially in large dogs. For this reason plasma usually is used to replenish clotting factors and restore albumin levels of 20 g/L and synthetic colloids are used to ensure adequate oncotic pressure is maintained. Maintaining a plasma concentration of 20 g/L has been recommended in the human literature for patients with serious illnesses such as sepsis. This number has somewhat arbitrarily been used in veterinary medicine, although as a general rule it appears to be a reasonable target.

Coagulopathies

Coagulation abnormalities are not uncommon in seriously ill or injured patients. On a fundamental level they can present as hypocoagulation and hypercoagulation. Hypocoagulation is more commonly dealt with since clinical evidence of hemorrhage is easily to recognize and specific tests are available to confirm abnormalities. Early stages of bleeding tendencies may be missed since spontaneous hemorrhage does not occur until approximately only 30% of clotting factors remain. Hypercoagulation or the predisposition to thromboembolic complications is more complex to both recognize and diagnose.

Bleeding disorders can develop due to toxicities such as rodenticide toxicities or drug toxicity (heparin, warfarin). Coagulopathies can develop secondary to loss of clotting factors. This occurs in patients who have experienced severe blood loss secondary to trauma or bleeding tumours. This is often compounded by fluid resuscitation using fluids that do not contain clotting factors. The dilutionary effects may lead to overt bleeding tendencies. This can be a serious problem if the patient requires surgery.

The inflammatory cascades that are activated during SIRS (systemic inflammatory response syndrome) also activate the endothelium. This endothelial activation triggers other cascades including the coagulation cascade which if left uncontrolled can lead to disseminated intravascular coagulation (DIC).

Which Product?

A combination of assessment of the patient’s hematocrit, serum albumin concentration, and results of coagulation tests should be used in addition to physical exam parameters when making a decision on which blood product to transfuse.

Patients that are anemic and hypovolemic, such as those that have lost whole blood, should be transfused with whole blood or a combination of packed red cells and plasma. Patients that are anemic and euvoletic should be transfused with packed red blood cells. If packed red blood cells are not available, whole blood should be transfused with caution, since the patient may become hypervolemic secondary to the oncotic effects of the plasma and may develop pulmonary edema.

Plasma should be transfused to patients that are coagulopathic and, in some situations to help prevent the development of a clinical coagulopathy. Plasma also provides a source of α2-macroglobulin, which binds the activated and liberated proteases in patients with pancreatitis and may be useful in the treatment of this disease. Plasma should be transfused in septic and SIRS patients with acute third-spacing of albumin to maintain albumin levels as close to 20 g/L as possible. In large dogs or patients where plasma is not available human serum albumin can be a useful alternative. Plasma or human serum albumin is rarely indicated in conditions leading to chronic hypoalbuminemia such as inflammatory bowel disease or advanced liver disease, since this often leads to fluid overload and rarely has any positive impact on the albumin concentration.

Blood products are rarely given to raise platelet counts since platelets are only viable for 6 to 8 hours after blood is collected and 1 unit will typically raise the platelet count by only about 10,000.

Calculating How Much Volume To Give

The volume of blood required should be calculated based on an end goal of a packed cell volume. The patient weight in (kg) is multiplied by 90 ml/kg for dogs and 50 ml/kg for cats which is multiplied by the (desired PCV-present PCV)/ PCV of the donor blood.

Typically a range of 10-15 ml/kg of plasma is given for plasma transfusions. If a patient has a coagulopathy then ideally fresh frozen plasma is given until the coagulation tests are normal or near normal.

Basic Crossmatching

The following protocol can be used to perform a basic crossmatch which should control for most common reactions. Collect 2 ml EDTA blood from the donor and the recipient and centrifuge at 1000 rpm for 1 minute and remove the plasma. Mix 0.1 ml of the red blood cells with 5 ml 0.9% saline, centrifuge at 1000 rpm for 1 minute, discard the supernatant and repeat twice more. Place 2 drops of the recipient plasma/serum and 2 drops of the donor cell suspension in a 3 ml tube (major crossmatch). Place 2 drops of donor plasma/serum and 2 drops of recipient cell suspension in a 3 ml tube (minor crossmatch). To perform the controls of the donor and recipient mix the red blood cells with their own plasma/serum. Incubate all samples for 30 min at room temperature then centri-
fuge for 1 minute at 1000 rpm. Check macroscopically for agglutination and hemolysis, and microscopically for agglutination at 40X.

Administration of Blood Products

Unless the blood type of the recipient and donor are known all transfusion recipients ideally should have a major and minor crossmatch. Dogs who are in danger of dying and have never received a transfusion usually can be transfused without a crossmatch but the clinician should watch carefully for any reaction including delayed reactions that may not show up for several weeks. Since cats have naturally occurring alloantibodies they must be typed and preferably crossmatched prior to transfusing since even a few drops of type A blood given to a type B cat can cause death.

Transfusions should not be transfused through the same fluid line as hypotonic fluids (i.e., 5% dextrose in water) or fluids that contain calcium (i.e., lactated Ringer’s solution). Packed red blood cells usually need to be diluted in 80-100 ml 0.9% saline (minimum) or infused piggy-backed with a non calcium-containing crystalloid if the hematocrit is greater than 55%. Some blood banks have already added a diluent to bring the hematocrit of the transfusion to approximately 45%, in which case further dilution is unnecessary. A 170 u filter should be used to transfuse blood; an 18 u filter and a regular drip set can be used for plasma. Red cells should only be administered using special pumps since most regular pumps use a roller mechanism that causes red blood cell lysis. Blood products should not hang at room temperature for longer than 4 to 6 hours. Rate guidelines vary with the patient’s underlying disease and volume status but typically 5 ml/kg/hr can be given in the first 15 min followed by 10-20 ml/kg/hr; if treating hemorrhagic shock blood should be given as fast as needed to resuscitate the patient.

Blood products should be warmed in a warm water bath to room temperature before administration and to body temperature in patients that are hypothermic. Plasma can be thawed in a microwave at low power settings (50% or less) for 2-3 seconds at a time. Every 2 to 3 seconds the plasma should be gently rolled back and forth to mix the fluid. If the temperature exceeds 42°C in any part of the fluid the proteins will denature. If plasma has been thawed to refrigerator temperature and is not spiked it can be refrozen but should be labeled as frozen plasma and the expiration date should be halved.

During a red blood cell transfusion the animal’s temperature should be monitored pre transfusion, every 5 minutes for the first 15 minutes then hourly. During a plasma transfusion the temperature should be monitored every 15 minutes for the first hour then hourly. The patient should be monitored for fluid overload if the patient is normovolemic or has underlying cardiac, liver or renal disease and is receiving whole blood or plasma. The patient also should be monitored for transfusion reactions which may manifest as fever, urticaria or a hemolytic reaction, which can be characterized by tachycardia or bradycardia, hypotension, respiratory distress, cyanosis, emesis, defecation, collapse, opisthotonus, cardiac arrest, hemoglobinuria, and hemoglobinemia.

The transfusion should be stopped if a hemolytic reaction is noted and supportive care should be provided along with treatment for an allergic reaction. Blood samples should be drawn from the recipient if hemoglobinemia is a concern and a sample of the donor blood should be saved for cross-match and culture. The transfusion should be slowed to half the rate if a febrile reaction is noted and the temperature should be monitored every 5 minutes. If the temperature continues to rise the transfusion should be stopped.

If massive volumes of blood or blood products are transfused the patient should be monitored for other potential complications such as significant hypothermia, electrolyte and acid-base imbalances, and, citrate toxicity leading to hypocalcemia.
**Blood Gases and Acid Base — Why Do I Care?**

Jennifer J. Devey, DVM, Diplomate ACVECC

**Why Do We Care About Blood Gas and Acid-Base Abnormalities?**

Changes in the blood gases and acid base status of the body reflect abnormalities in the metabolic system or the respiratory system. Without being able to characterize these abnormalities it is virtually impossible to provide the best treatment to a patient. A low pH (less than 7.1 to 7.2) can be associated with enzyme system dysfunction. This can lead to respiratory muscle failure (ventilatory failure), myocardial failure (poor cardiac contractility), cardiac arrhythmias, vasodilation, and coagulation abnormalities – not to mention the detrimental effects on most cellular processes. A patient in septic shock with a pH of 7.0 after fluid resuscitation has, first of all, not been adequately resuscitated, and secondly may not be able to maintain normal vascular tone and cardiac function, which may lead to a refractory shock state. A patient in hypovolemic shock that has been given fluids but still has a severe metabolic acidosis has not been adequately resuscitated. A patient with pneumonia that is breathing faster than normal but has an increased carbon dioxide level probably has significant pulmonary disease and is far more critical than the patient with a normal carbon dioxide level. A patient that is breathing faster than normal and does not have a lower than normal carbon dioxide also probably has abnormal lung function and the clinician should be alerted to investigate for pulmonary parenchyma, pleural space disease or chest wall trauma. A cat with chronic renal failure with a very low bicarbonate concentration will require bicarbonate supplementation in order to recover more completely and more rapidly but supplementation cannot be gauged unless acid-base numbers are available. A patient with a hypochloremic metabolic alkalosis has a gastric outflow disorder until proven otherwise – no matter what all other tests indicate.

**Arterial vs Venous Blood Gases**

Arterial blood gases are considered the gold standard; however, obtaining an arterial sample can be technically difficult in cats, small dogs and hypotensive dogs of all sizes. Additionally positioning an animal so that an arterial sample can be drawn can be disastrous for patients in respiratory distress even though they are the ones that would benefit most from this information. Arterial blood gases must be used to determine oxygen tensions since venous oxygen tensions are dependent to a large extent on oxygen uptake in the tissues, which can vary tremendously in critical patients. Assuming relatively normal tissue perfusion, the carbon dioxide tension in a venous blood gas is usually a fairly accurate reflection of pulmonary function, often varying by only 2 to 3 mm Hg from the arterial tension. During resuscitation from significant shock there can be tremendous washout of carbon dioxide from the tissues and the carbon dioxide tension in this situation is a reflection of tissue metabolic status rather than pulmonary function. Using central venous blood samples will minimize the impact of any washout from regional tissue beds. Comparison of central and peripheral venous blood gases may provide information on tissue perfusion abnormalities in the region where the venous sample was obtained.

**pH**

Normal metabolic processes lead to the production of carbonic acid (H₂CO₃), which breaks down to hydrogen ions (H⁺) and bicarbonate ions (HCO₃⁻) or to water (H₂O) and carbon dioxide (CO₂).

\[ \text{CO}_2 + \text{H}_2\text{O} \rightarrow \text{H}_2\text{CO}_3 \rightarrow \text{H}^+ + \text{HCO}_3^- \]

The hydrogen ion concentration is actually very low (4 x 10⁻⁸ or 40 nmol/L) so a logarithmic scale was developed to allow the hydrogen ion concentration to be expressed as pH. Normal pH is approximately 7.4. The pH is defined as the negative log of the hydrogen ion concentration so as the hydrogen ion increases the pH decreases and vice versa. Because pH is a logarithmic scale, it should be kept in mind that a minor change in the pH can reflect a major change in the hydrogen ion concentration. For instance a change in pH from 7.4 to 7.1 is equivalent to a doubling of the hydrogen ion concentration from 40 nmol/L to 80 nmol/L.

**Buffers**

There are 4 main buffer systems, which help limit the effects of acidosis and alkalosis on the pH. These buffers allow for hydrogen to be buffered to a weak acid or for an acid to dissociate this forming more hydrogen ions. The major extracellular buffer system is the bicarbonate/carbonic acid system which provides over half of the total buffering capacity. The intracellular buffering systems are the disodium/monosodium phosphate...
system (NaH₂PO₄ and Na₂HPO₄), which is important in the kidney and red blood cells, and the hemoglobin/oxyhemoglobin system (HbO₂⁻ and HHbO₂⁻), which is important in red blood cells to buffer the carbon dioxide that is being transported. The protein buffer system is active in tissue cells and in plasma. Most proteins carry a negative charge which attracts positive hydrogen ions.

These buffers act immediately although some have a rapid impact and some are slower processes, which help maintain homeostasis over the longer term. For instance, the lungs fairly rapidly adjust to help retain or blow off more carbon dioxide. The kidney buffering system may take 2-3 days to be effective at altering the pH.

**Sodium Bicarbonate**

Sodium bicarbonate should never be supplemented without evaluation of a blood gas. Side effects of sodium bicarbonate administration include hyperosmolality, hypernatremia, paradoxical intracellular acidosis and alkaline overshoot. A metabolic acidosis is dealt with by hyperventilation, but a metabolic alkalosis would require hypoventilation — something unlikely to happen in most patients. Typically bicarbonate is not supplemented unless the pH is less than 7.2 and the bicarbonate level is less than 11 mm Hg although these numbers are somewhat arbitrary and good clinical judgment based on the underlying disease pathophysiology should be used to determine the need for treatment.

**Acidosis vs Alkalosis**

The terms acidosis and alkalosis refer to processes (metabolic or respiratory), which lead to changes in production, retention or excretion of acids and bases without necessarily resulting in a change in pH. Acidosis refers to a condition of too much acid. Alkalosis refers to a condition of too much base. Acidemia refers to increased hydrogen ions in the blood or a low pH. Alkalemia refers to too much base in the blood or a high pH. A patient can be acidic without being acidemic. More than one process can occur at one time. For instance, a patient can have a metabolic acidosis and a respiratory alkalosis simultaneously. Acid base abnormalities are classified into four different types — metabolic acidosis, metabolic alkalosis, respiratory acidosis and respiratory alkalosis.

**Metabolic Acidosis and Alkalosis**

A metabolic acidosis results when metabolic processes are producing too much acid. Sulfuric acid, phosphoric acid, lactic acid and ketoacids are the main acids produced. The excess hydrogen ions are buffered by the bicarbonate to shift the above equation to the left. This results in a decrease in HCO₃⁻. A decrease in the bicarbonate concentration indicates a metabolic acidosis. Insufficient hydrogen ions in the blood stream (increased loss, decreased production) will lead to a shift of the equation to the right and an increase in the concentration of HCO₃⁻. An increase in the bicarbonate concentration indicates a metabolic alkalosis.

Base excess is another term used to help identify the metabolic process. It is the amount of hydrogen ions required to return the pH to 7.35 if the carbon dioxide were adjusted to normal. It is a calculated number. A base excess less than –4 (i.e. –8) indicates a metabolic acidosis. A base excess greater than 2 to 4 (i.e. 5) indicates a metabolic alkalosis. (These numbers will vary somewhat depending on the machine being used to analyze the blood gas.)

An abnormal bicarbonate concentration allows the clinician to determine that there is a metabolic component to the acid-base system but does not indicate if the problem is primary or compensatory.

**Respiratory Acidosis and Alkalosis**

A respiratory acidosis results when too much carbon dioxide is being produced or retained (hyperventilation). An increase in the carbon dioxide level indicates a respiratory acidosis. Increased carbon dioxide exhalation (hyperventilation) leads to a decrease in the carbon dioxide concentration and a respiratory alkalosis.

An abnormal carbon dioxide level allows the clinician to determine that there is a respiratory component to the acid-base system but does not indicate if the problem is primary or compensatory.

**Compensation**

The body attempts to rapidly compensate for any acid-base abnormality by returning the pH to normal, initially using the bicarbonate/carbonic acid buffering system. Whenever too much acid is being produced the body will attempt to shift the equation to the left and the excess carbon dioxide will be exhaled. Thus a low carbon dioxide in the face of a metabolic acidosis is an expected compensation. Pulmonary disease leading to carbon dioxide retention will lead to activation of the non-respiratory buffering systems and an increase in the bicarbonate concentration.
Interpreting Acid-Base Numbers

The pH is the first number that should be evaluated and the number classified as normal, increased (alkalemia) or decreased (acidemia). The bicarbonate and carbon dioxide numbers should then be evaluated and classified as an acidosis or alkalosis or normal. The primary event is the process (metabolic or respiratory) that is shifted in the same direction as the pH. If the pH is low and there is a metabolic acidosis then the metabolic acidosis is the primary event. If there is a compensatory response then the other process should be shifted in the other direction. A respiratory acidosis should be accompanied by a metabolic acidosis and a metabolic acidosis should be accompanied by a respiratory alkalosis.

Rule of Fours

While the following numbers are not exact they are suitable for all clinical intents and purposes and allow the clinician to rapidly assess a blood gas.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Normal Value</th>
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</thead>
<tbody>
<tr>
<td>pH</td>
<td>7.4 +/- 0.04 (7.36-7.44)</td>
</tr>
<tr>
<td>PCO₂</td>
<td>40 +/- 4 (36-44)</td>
</tr>
<tr>
<td>HCO₃⁻</td>
<td>24 +/- 4 (20-28)</td>
</tr>
<tr>
<td>BE</td>
<td>0 +/- 4</td>
</tr>
</tbody>
</table>

These numbers will also vary to some extent based on the machine being used to analyze the blood. In addition cats tend to be more acidic than dogs and are less likely to end up on the +4 side of the numbers quoted above.

How Do I Determine If The Compensation Is Appropriate?

Changes should occur to the acid-base buffering system based on the length of time the abnormality has been present. Typically these are classified into acute and chronic changes in compensation.

**Metabolic acidosis**

Decrease in HCO₃⁻ of 1 mEq/L results in decrease in PCO₂ of 0.8 mm Hg

**Metabolic alkalosis**

Increase in HCO₃⁻ of 1 mEq/L results in increase in PCO₂ of 0.7 mm Hg

**Respiratory acidosis**

- **Acute**: increase in PCO₂ of 1 mm Hg results in increase in HCO₃⁻ of 0.15 mEq/L
- **Chronic**: increase in PCO₂ of 1 mm Hg results in increase in HCO₃⁻ of 0.35 mEq/L

**Respiratory alkalosis**

- **Acute**: decrease in PCO₂ of 1 mm Hg results in decrease in HCO₃⁻ of 0.2 mEq/L
- **Chronic**: decrease in PCO₂ of 1 mm Hg results in decrease in HCO₃⁻ of 0.55 mEq/L

**Rule of Fours**

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Blood Gas Problems

Acid-Base Problem #1

5 year old castrated male cat with urethral obstruction
pH = 7.30 HCO₃⁻ = 8.2 CO₂ = 20 BE = -12

Acidemia? Alkalemia?
Metabolic acidosis or alkalosis?
Respiratory acidosis or alkalosis?
Primary problem?
Clinical significance?

Acid-Base Problem #2

2 year old German Shepherd HBC, no head trauma
pH = 7.26 HCO₃⁻ = 13 CO₂ = 40 BE = -9

Acidemia? Alkalemia?
Metabolic acidosis or alkalosis?
Respiratory acidosis or alkalosis?
Primary problem?
Clinical significance?

Acid-Base Problem #3

17 year old cat with chronic renal failure
pH = 7.22 HCO₃⁻ = 6 CO₂ = 34 BE = -17

Acidemia? Alkalemia?
Metabolic acidosis or alkalosis?
Respiratory acidosis or alkalosis?
Primary problem?
Clinical significance?

Acid-Base Problem #4

1 year old dog with 2 day history of vomiting
pH 7.49 HCO₃⁻ = 30 CO₂ = 30 BE = +6

Acidemia? Alkalemia?
Metabolic acidosis or alkalosis?
Respiratory acidosis or alkalosis?
Primary problem?
Clinical significance?
Acid-Base Problem #6
2 yr old FS Lab HBC, post fluid resuscitation
pH = 7.34 HCO₃ = 15 CO₂ = 26 BE = -8
Acidemia? Alkalemia?
Metabolic acidosis or alkalosis?
Respiratory acidosis or alkalosis?
Primary problem?
Clinical significance?

Acid-Base Problem #7 - Arterial
2 yr old FS Lab cardiac arrest
pH = 6.88 HCO₃ = 8 CO₂ = 50 BE = -18
Acidemia? Alkalemia?
Metabolic acidosis or alkalosis?
Respiratory acidosis or alkalosis?
Primary problem?
Clinical significance?

Acid-Base Problem #7 - Venous
2 yr old FS Lab cardiac arrest
pH = 6.88 HCO₃ = 8 CO₂ = 50 BE = -18
Acidemia? Alkalemia?
Metabolic acidosis or alkalosis?
Respiratory acidosis or alkalosis?
Primary problem?
Clinical significance?

Acid-Base Problem #8
5 yr old MN Greyhound fever,
brick red mm, crt < 1 s HR 150/min, BP 130/65
pH = 7.35 HCO₃ = 19 CO₂ = 28 BE = -2
Acidemia? Alkalemia?
Metabolic acidosis or alkalosis?
Respiratory acidosis or alkalosis?
Primary problem?
Clinical significance?
Acid-Base Problem #9
5 yr old MN Greyhound, following fluid bolus painful abdomen
pH = 7.30 HCO₃⁻ = 15 CO₂ = 30 BE = -8
Acidemia? Alkalemia?
Metabolic acidosis or alkalosis?
Respiratory acidosis or alkalosis?
Primary problem?
Clinical significance?

Acid-Base Problem #10
10 yr old MN DSH, hx diabetes
pH = 7.15 HCO₃⁻ = 16 CO₂ = 40 BE = -26
Acidemia? Alkalemia?
Metabolic acidosis or alkalosis?
Respiratory acidosis or alkalosis?
Primary problem?
Clinical significance?

Acid-Base Problem #11
12 yr old FS obese golden retriever, vomited 2x,
sl weak, sl weak pulses, HR 130/min, PCV/TS 34/4.7
pH = 7.28 HCO₃⁻ = 13 CO₂ = 22 BE = -11
Acidemia? Alkalemia?
Metabolic acidosis or alkalosis?
Respiratory acidosis or alkalosis?
Primary problem?
Clinical significance?

Acid-Base Problem #12
8 yr old MN DSH, 3 d hx vomiting (now resolved) & anorexia
pH = 7.48 HCO₃⁻ = 29 CO₂ = 36 BE = 0
Acidemia? Alkalemia?
Metabolic acidosis or alkalosis?
Respiratory acidosis or alkalosis?
Primary problem?
Clinical significance?
Acid-Base Problem #13
1 yr old FS DSH, 3 d hx vomiting (now resolved) & anorexia
pH = 7.45 HCO₃ = 31.5 CO₂ = 45 BE = 7
Acidemia? Alkalemia?
Metabolic acidosis or alkalosis?
Respiratory acidosis or alkalosis?
Primary problem?
Clinical significance?

Acid-Base Problem #14
1 yr old FS DSH, 3 d hx vomiting (now resolved) & anorexia
pH = 7.586 HCO₃ = 22 CO₂ = 23.4 BE = 0
Acidemia? Alkalemia?
Metabolic acidosis or alkalosis?
Respiratory acidosis or alkalosis?
Primary problem?
Clinical significance?

Acid-Base Problem #15
1 yr old FS DSH, 3 d anorexia, vomiting recurred post metoclopramide
pH = 7.508 HCO₃ = 19.9 CO₂ = 25.1 BE = -3
Acidemia? Alkalemia?
Metabolic acidosis or alkalosis?
Respiratory acidosis or alkalosis?
Primary problem?
Clinical significance?
Controlling Hemorrhage — Should I Go to Surgery?

Jennifer J. Devey, DVM, Diplomate ACVECC

Trauma, neoplasia and coagulopathies can all cause hemorrhage. The cause of the hemorrhage should be determined as definitively as possible since knowledge of the underlying pathophysiology will impact patient management. Emergent or urgent surgery is almost always indicated for penetrating trauma and actively hemorrhaging abdominal neoplasia; however, surgery is rarely indicated for blunt trauma and is almost always contraindicated if the patient is coagulopathic. This lecture will discuss how to diagnose the presence and cause of the hemorrhage, how to determine if a traumatic hemoabdomen needs surgery as well as options for controlling hemorrhage, both conservative as well as surgical.

Success

Successful management of the actively hemorrhaging trauma patient requires prompt recognition, aggressive treatment, and diligent follow-up. The traumatic hemoabdomen patient, in particular, must be assumed to have serious internal injuries until proven otherwise. Rapid diagnosis of emergency surgical conditions is important; however, diagnostic tests should never take precedence over resuscitation. Radiographs can wait but improving blood pressure with appropriate fluid therapy should not.

On occasion intrathoracic hemorrhage will be severe enough to warrant surgery but patients with severe thoracic trauma usually do not make it to the hospital alive. Abdominal trauma may involve blunt injuries, penetrating injuries or a combination of the two. Surgery is always indicated for hemorrhage associated with penetrating abdominal wounds due to the potential for bowel injury. Some patients with penetrating wounds may require surgery on an emergent basis and some may only require it on an urgent basis. The decision to take patients to surgery for a hemorrhage secondary to blunt abdominal trauma is more complex. Rupture of a discrete abdominal tumour or tumours leading to active hemorrhage ideally should be managed surgically as soon as possible in order to minimize morbidity.

If diagnostic tests confirm the need for surgery then a team must be able to be called into action. These patients are critical and standard anesthetic protocols cannot be used. The anesthetist is the most important member of the surgical team while the patient is unstable. Positive pressure ventilation is essential and blood pressure must be closely monitored and kept as close to normal as possible during surgery. Blood products are often vital to the short and long term successful outcome of surgery for serious intraabdominal hemorrhage. The surgeon must have an extremely thorough knowledge of anatomy since the source of the bleeding can be coming from many sources. In addition these patients require intensive 24 hour care in order to ensure a consistent positive outcome.

Coagulation Disorders

Hemorrhage can occur secondary to an underlying coagulopathy. This may be due to an inherited coagulation disorder, or secondary to another underlying problem such as an anticoagulant rodenticide toxicity or a neoplastic condition. It can also occur secondary to loss of coagulation factors from blood loss, dilution with crystalloids and synthetic colloid fluids, hypothermia and acidosis. Any time a patient presents with a bleeding condition it is vital that coagulation tests be assessed. Ideally a prothrombin time, an activated partial thromboplastin time (aPTT) and a platelet count should be performed. An activated clotting time can substitute for an aPTT. Platelet counts performed by automated machines are notoriously inaccurate in ill or injured patients and a manual estimate should always be performed. Patients who are high risk breeds for von Willebrand’s disease or a suspected underlying platelet disorder also should have a buccal mucosal bleeding time assessed. If surgery is indicated and a minor coagulopathy is evident it may be able to be corrected or controlled using transfusions of blood products while surgery is being performed. If a serious coagulation disorder is present surgery may need to be postponed until the coagulopathy has been corrected or the hemorrhage may need to be managed medically.

Diagnostic Peritoneal Lavage

Diagnostic peritoneal lavage performed with a multi-holed catheter can be a valuable diagnostic tool for intraabdominal injury. Results of lavage fluid examination are extremely useful in not only diagnosing a condition but also in determining the need to exploratory surgery in cases where the diagnosis is uncertain. Approximately 20-30 ml/kg of blood is required in the abdomen to detect abdominal hemorrhage on palpation or radiographically. Whereas 4
quadrant abdominocentesis may be negative even with up to 50% of the blood volume in the abdomen, DPL is accurate over 95% of the time. False negative results for diagnosing septic peritonitis also exist with abdominocentesis but once again a diagnostic peritoneal lavage has an extremely low incidence of false negative results.

To perform a diagnostic peritoneal lavage the animal is placed in left lateral recumbency. This keeps the spleen away from the midline. Ideally the urinary bladder is emptied. A clip and surgical prep is performed of a 4 cm square area 2 cm distal to the umbilicus on the midline. A local block is placed in the skin and peritoneum 2 cm caudal to the umbilicus either on the midline or just lateral to the midline. Sedation is used if necessary. Surgical gloves are worn and ideally a drape is placed. A stab incision is made in the skin and a multi-holed catheter is inserted into the abdomen. In cats and small dogs an 18g 2 inch (5 cm) catheter is ideal. In medium and larger sized dogs a 16g or 14g 5.25 inch (13 cm) catheter is inserted. Side-holes should be added using a #15 scalpel blade. Alternatively a commercial diagnostic peritoneal lavage catheter can be used. The catheter is inserted in a caudal direction. If fluid is retrieved a sample is collected aseptically for analysis. To complete the lavage 20 ml/kg of warm (body temperature) isotonic crystalloid fluid is infused. Since this will increase pressure on the diaphragm, the respiratory rate and effort should be watched closely and fluid infusion stopped if the animal starts to show signs of respiratory distress. Once the fluid has been infused the animal is gently rotated to mix the fluid around and then fluid samples are collected for analysis. A packed cell volume, protein level, white blood cell count, and microscopic examination of the fluid to evaluate white blood cell morphology as well as the presence of bacteria should be performed. The presence of even a single intracellular bacterium confirms a diagnosis of septic peritonitis. The fluid should be cultured if bacteria are present. Blood chemistries such as amylase, lipase, alkaline phosphatase and bilirubin can be analyzed. Levels that are higher than serum suggest pancreatic or intestinal injury, and a ruptured biliary tract respectively. High potassium levels are consistent with urinary tract rupture. Urea nitrogen levels will equilibrate rapidly between the serum and peritoneum but in an acute bladder rupture the peritoneal level will be higher than serum.

If the catheter is being used to monitor intraabdominal hemorrhage it can be sutured in place and serial samples can be taken at 5 to 10 minute intervals or serial lavages can be performed. An initial hematocrit of greater than 20%, a rising hematocrit by more than 5% between samples which indicates ongoing hemorrhage are strong indicators for surgery. If urine is present the urine should be allowed to drain from the abdomen. The catheter can be left in place and used as a peritoneal dialysis catheter in this situation. Once the catheter is removed a dressing is placed over the incision. A suture or staple can be placed if desired.

Ultrasonography

Ultrasonography is a valuable tool in diagnosing intraabdominal conditions especially because it can be performed rapidly at the bedside. Ultrasound-guided abdominocentesis provides a marked advantage over blind abdominocentesis for procuring samples of fluid. The use of FAST (focused abdominal sonography for trauma) has been validated in the dog as an effective means of determining the presence of free fluid in the abdomen. The scan is ideally performed with the dog in left lateral recumbency. Both transverse and longitudinal views are assessed of 4 areas of the abdomen. The probe is placed just caudal to the xiphoid to check for fluid between the diaphragm and the liver and between liver lobes. The probe is then placed just over the urinary bladder to evaluate for fluid at the apex of the bladder. Following this the probe is placed over the left flank to check for fluid between the spleen and abdominal wall, between the spleen and the liver and between the spleen and the left kidney. Lastly the probe is placed over the right flank to evaluate for fluid between loops of intestine, between the intestine and the right kidney and between the intestine and the right body wall. Although ultrasonography can be used to detecting the presence of intraabdominal fluid it should be kept in mind that it is rare that it indicates whether or not hemorrhage is ongoing although subjective evaluation of volumes of fluid visualized during the scan may help with this assessment.

“Bleeding somewhere, bleeding nowhere, check the retroperitoneum.” Retroperitoneal hemorrhage may be detectable by observing an expanded or expanding retroperitoneal space on ultrasound. Most retroperitoneal hemorrhage can be managed medically; however, continued expansion of the retroperitoneal space despite the use of counterpressure might indicate the need for exploratory surgery.

In addition to evaluating the patient for free fluid, ultrasound can be used for evaluating for specific intraabdominal injuries such as fractures of solid organs such as the liver, spleen or kidney or rupture of hollow organs such as the urinary bladder or gall bladder.

Hemostasis

Accurate hemostasis is key in all patients but even more so in critically ill or injured patients since even minor oozing can lead to significant blood loss if the patient is coagulopathic. Prior to taking a patient to surgery it should be determined that the appropriate equipment is available to deal with any hemorrhage that might be encountered. If equipment is not available or the patient is deemed to be too unstable to take to surgery then use of pressure bandages may be more appropriate.

Minor hemorrhage such as capillary oozing and most venous hemorrhage can be controlled with pressure. Controlling more
significant hemorrhage or hemorrhage from complex trauma can be challenging. External counterpressure to the hindlimbs, pelvis and abdomen can be an effective method of raising blood pressure and controlling hemorrhage from vessels under the wrap. It can be provided using commercial antishock garments, rolls of cotton and bandage material, or towels and duct tape. The intraabdominal pressure should be kept below 25 mm Hg in order to prevent organ damage especially renal failure. A hand should be able to be easily passed under the wrap once it is in place. Respiration must be closely monitored if the wrap encompasses the cranial abdomen as ventilation can be compromised especially if there is a diaphragmatic injury. The pelvic limbs must be included in the wrap if the abdomen is to be wrapped to prevent vascular occlusion of the caudal abdominal vena cava. Counterpressure should remain in place for a minimum of several hours and then be slowly removed and pressures closely monitored. If pressures drop by more than 5 mm Hg, removal is stopped, volume is infused and then removal restarted once the pressure has stabilized. If blood pressure cannot be maintained surgery is indicated.

Blood loss from subcutaneous vessels, omental vessels, and mesenteric vessels can be significant in patients who cannot clot normally. Both monopolar and bipolar electrosurgery devices are indispensable in controlling hemorrhage. Laser-assisted surgery also can be useful in vascular tissues or when very precise dissection and simultaneous hemostasis is desired.

Topical hemostatic agents also can be used to control hemorrhage in certain situations. Fibrin glues, collagen, gelatin sponges and oxidized cellulose are available. Rapid ligation of blood vessels can be performed using vascular clips. Hand ties can be performed more quickly than instrument ties in certain situations. Temporary control of hemorrhage into parenchymal organs can be achieved by placing atraumatic vascular clamps (Satinsky clamp, bulldog clamp) or a Rumel tourniquet. A modified Rumel tourniquet can be formed by passing a small bore red rubber tube around the vascular pedicle and then bringing the tube ends together. A pair of hemostatic forceps are slid down both tube ends until the vessel is approximated at which point they are clamped.

The Final Decision To Cut

Various diagnostic methods can be used to determine whether or not surgery is required and how urgently surgery is required but often the decision to go to surgery is based on how the patient is doing clinically as well as test results. Close monitoring of the 5 vital signs - temperature, pulse rate (and quality), respiratory rate (and effort), blood pressure and pain score - of these patients is essential to minimizing patient morbidity.

Emergency surgery is always indicated if vital signs are not stabilizing, the patient is showing signs of relapsing into shock after initial resuscitation, or if a hemoabdomen is continuing to expand. In the case of abdominal hemorrhage a continually decreasing hematocrit (especially to 20% or below that is not responding to initial resuscitation and hemorrhage control methods) usually indicates that emergency surgery is required. Additional findings that indicate the need for emergency surgery include radiographic evidence of pneumoperitoneum or diaphragmatic hernia, or diagnostic peritoneal lavage findings consistent with septic peritonitis.

Surgery for more minor penetrating wounds such as body wall herniation without internal abdominal organ injury may not be an extremely difficult undertaking but surgery for major abdominal hemorrhage requires an equipped surgical suite including appropriate instrumentation, electrosurgery and positive pressure ventilatory support capability, good lighting, blood products and a trained team of a minimum of 3 people - surgeon, assistant surgeon and anesthetist.

Severe hemoabdomen patients can be extremely challenging to manage surgically – often due to difficulties in controlling the hemorrhage. In a severely hemorrhaging patient a thoracotomy may be required in order to crossclamp the descending thoracic aorta prior to entering the abdomen. In the serious but less severely bleeding abdomen the aorta may need to be digitally compressed cranial to the cranial mesenteric artery, the abdomen packed with laparotomy pads or towels to help control bleeding and improve visualization, and pressure maintained until the source of the hemorrhage can be controlled. If hemorrhage persists despite this maneuver, a Pringle maneuver (occlusion of the portal vein, hepatic artery, and common bile duct) may be indicated.

If the appropriate resources do not exist to take the patient to surgery it may be more appropriate to try and manage the patient conservatively until the resources do exist or until the patient can be referred. For example, taking a hemodynamically unstable, actively hemorrhaging patient to surgery without the right resources has a high likelihood of ending in the death of the patient. Managing this patient with appropriate fluid therapy and blood products, hypotensive resuscitation and placement of external counterpressure may give the patient enough time to clot and thus avoid the need to go to surgery.
Analgesia In The Emergency Patient — What Really Works?

Jennifer J. Devey, DVM, Diplomate ACVECC

Analgesia is an essential component of treatment for many patients presenting for an illness and for all patients presenting with injuries. An injury always causes pain although the degree of pain that is associated with the injury will vary depending on the location and severity of the trauma. The clinician should always make a conscious decision not to give analgesia rather than the other way around.

Pain has many detrimental physiologic effects. Pain can negatively impact cardiopulmonary function, metabolism, endocrine status and immune function. Premature ventricular complexes, ventricular tachycardia, tissue hypoxia, atelectasis, hypoventilation (leading to significant acidosis), anorexia, muscle weakness, and delayed tissue healing are all potential sequelae of pain. In addition pain KILLS. NO patient is so critical that pain relief cannot be provided. In more seriously ill or injured patients the dose of the drug or drugs being used may need to be decreased, sometimes to 10 to 25% of the normal dose; however, analgesics should never be withheld.

Opioids

Opioids are the class of drug most commonly given to animals in pain. Pure agonists include meperidine, morphine, hydromorphone, and fentanyl. Meperidine is a mild analgesic and short acting. Morphine can be used effectively for short-term analgesia (sometimes less than 20 minutes) and is an excellent choice for constant rate infusions for controlling significant pain. It may last as long as 4 hours in animals that are not very painful. Morphine can cause vasodilation and emesis. Hydromorphone is an intermediate acting opioid that clinically is effective at controlling most pain. Common side effects include panting (actually hypoventilation) and noise sensitivity. It may last as long as 6 hours in animals that are not very painful. Fentanyl is an inexpensive, short acting opioid that must be delivered frequently (every 10 to 20 minutes) or via a constant rate infusion. It is an extremely effective analgesic that is 100 times more potent than morphine.

Transdermal fentanyl patches are useful adjuncts in controlling pain; however, since they take as long as 12 to 24 hours to reach peak effect they are rarely useful during the emergency situation. In addition they are rarely effective at completely controlling pain unless the pain is mild. The use of patches avoids the episodes of moderate to severe pain that can break through between intermittent injections or oral pain medication administration. Patches should be avoided in very critical patients since the amount of drug the animal can be exposed to can cause severe depression and sedation. If half patches are being used the patch is not cut in half but rather the adhesive is removed from only half of the patch. When placing a patch, care should be taken to ensure the animal (or other animals in the household or small children if the patient is being discharged with a patch) cannot eat it.

Butorphanol is an agonist/antagonist. It is a short acting analgesic that clinically is most effective for treating soft tissue pain. It has minimal sedative and respiratory depressant effects. Because of these characteristics it is very useful in very critical patients or in patients who have not been fully cardiovascularly resuscitated. It has a short duration of action (sometimes as little as 20 minutes) and clinically it is not as effective as other opioids so it is less useful for treating significant soft tissue pain and musculoskeletal pain. Given as a constant rate infusion it appears to be effective at controlling soft tissue pain in cats without the dysphoric effects.
that may be seen with the extended use of pure mu agonists in this species. It may last as long as 4 hours in animals that are not very painful. Buprenorphine is a partial agonist that is effective for mild soft tissue pain. Clinically it also seems to be more effective in cats than in dogs. Because it is a partial agonist it has fewer side effects than the pure agonists. It may take 1 to 3 hours to reach peak effect which makes it an inappropriate drug to use in the acutely painful animal. The duration of effect may be as long as 6 to 12 hours. Because of its high affinity to receptors it blocks other pure agonists from binding to the mu receptors.

Side effects of all opioids can include bradycardia and respiratory depression. These are both uncommon except in critical patients unless high doses were administered. If opioids are required to provide analgesia but respiratory depression becomes evident then positive pressure ventilation may be required. When bradycardia is noted a blood pressure should be checked and anticholinergics (atropine, glycopyrrolate) should be administered only if the animal is concurrently hypotensive or if the bradycardia is associated with a heart block. Routinely treating patients with anticholinergics should be avoided since tachycardia increases myocardial oxygen demand and may worsen arrhythmias. Opioids are metabolized by the liver and effects may be prolonged in patients with liver disease.

**Local Anesthetics**

Local anesthetic drugs can be injected into wound edges, onto tissue beds, regionally, intraarticularly, intrapleurally, or intercostally. They are extremely effective at controlling pain. Lidocaine or bupivacaine or both mixed together in 50:50 volumes can be used. Pain related to the acidic nature can be modified by warming the drug(s) to body temperature or by adding 10% of the volume as sodium bicarbonate. Dilution of lidocaine to a 1% solution can be useful in minimizing total drug dose while provided adequate volume needed for the block.

Local anesthetic agents also can be used intravenously at low dose constant rate infusions to provide additional analgesia. Lidocaine has been used at 1 mg/kg/hr in the awake patient. Patients receiving intravenous doses or higher doses of local anesthetic agents should be monitored closely for hypotension.

**Nonsteroidal Antiinflammatory Drugs**

Nonsteroidal antiinflammatory drugs are extremely effective in managing pain in small animals especially when used in conjunction with opioids. They generally should not be used in patients with underlying renal, hepatic or gastrointestinal disease or those with poor tissue perfusion. They should be avoided in critically ill or injured patients due to their negative gastrointestinal and renal effects. In addition some have negative effects on coagulation. Some of the newer generation COX-2 specific drugs may prove to be safe to use in more critical patients; however, even this class of drug is not recommended in patients with hypovolemia, compromised gastrointestinal perfusion (related to circulatory disturbances or underlying disease processes), and renal disease.

**Ketamine**

Constant rate infusions of low dose ketamine given to effect also have been used in painful patients and may be a helpful adjunct. Ketamine can be used in conjunction with morphine or fentanyl and lidocaine and given as a constant rate infusion. When using this combination the patient should be assessed regularly and the dose titrated to the minimum possible to avoid sedation and anorexia – both not uncommon complications.

**Painful Conditions and Clinical Signs**

The following is a list of conditions that can be painful and clinical signs that may be seen in association with the condition that indicates that the animal probably is painful. The list is not exhaustive but is designed to provide an overview.

**Historical Conditions Leading to Pain**

- trauma
- surgery (deliberate trauma)
- cancer
- arthritis, intervertebral disc disease, muscle abnormalities
- infection/inflammation
- abdominal conditions – pancreatitis, gastroenteritis/cramping
- poor circulation

**General Signs Consistent with Pain**

- panting
- vocalizing
- biting
- depression, hesitancy to move, hesitancy to lie down
- lameness, abnormal gait
- not eating, drinking
- flinching or biting when touched
» anxiety
» increased heart rate
» increased respiratory rate
» dilated pupils
» purring in some cats

Physical Exam Findings

Head – conditions potentially leading to pain
» abscesses
» dental especially extractions
» ear infections, tympanic membrane problems
» lacerations, wounds
» blunt trauma
» fractures
» eye problems – proptosis, enucleation, uveitis, corneal lacerations/ulcers
» hot spot

Clinical signs
» rubbing face, scratching, pawing (distinguish from pruritus)
» guarding, moving away
» head pressing
» shaking
» mouth open
» squinting, weeping, red eye
» drooling (distinguish from nausea)
» pupil constriction
» not eating/drinking

Neck/Back – conditions potentially leading to pain
» arthritis
» intervertebral disc disease
» trauma – fractures, luxation, blunt
» wounds to muscles
» meningitis
» certain breeds (Beagles, Doberman Pinschers, Dachshund, Corgi)

Clinical signs
» lack of mobility
» hunched gait
» not able or not willing to walk
» abnormal posture when sitting or standing, reluctance to sit or stand
» abnormal posture when urinating/defecating
» lameness
» vocalizing with movement
» licking at distal limb
» tail down
» guarding
» laying down to eat and/or drink
» spasms
Limbs – conditions potentially leading to pain

» fractures
» arthritis
» torn toenails
» lacerations, penetrating trauma
» bone inflammation
» dislocation
» ligament/tendon injury
» thromboembolus

Clinical signs
» lameness
» warm to touch

Skin – conditions potentially leading to pain

» laceration
» bruising, blunt trauma
» abrasions
» inflammation (including dermatitis), hot spots
» burns
» parasites
» torn toenails
» insect bites, allergies

Clinical signs
» redness, scratching, licking
» swelling
» licking, chewing
» missing hair, wet areas
» skin twitching
» warm to touch
» hives
» abnormal gait
Chest – conditions potentially leading to pain

» bruising
» fractured ribs
» diaphragmatic hernia
» infection – lung, pyothorax
» muscle injury
» chest tubes
» cancer

**Clinical signs**

» shallow +/- rapid breathing
» reluctance to sit, lie down, walk
» coughing
» bruising
» air under skin

Abdomen – conditions potentially leading to pain

» trauma
» hernias
» gastrointestinal obstruction
» stretching of hollow organs: gall bladder, gastrointestinal tract, urinary bladder, uterus
» inflammation (stretching) of capsule of solid organs: spleen, liver, kidney

» pancreatitis
» bladder calculi

**Clinical signs**

» tense, guarding
» hunched
» enlargement/distention
» vomiting
» straining – lack of urination, defecation
» reluctance to lie down
» “praying” position
» splinting
» gait abnormalities
» decreased appetite
» groaning
Clinical presentation

Middle aged to older dogs (>5yrs years old) who are overweight or obese appear at higher risk for developing acute pancreatitis. Miniature Schnauzers, Yorkshire and Silky Terriers, non-sporting breeds and perhaps miniature poodles may be at increased risk. There is no clear sex predisposition. Hypertriglyceridemia is a risk factor and may be why Miniature Schnauzers are overrepresented. Hypothyroidism, diabetes mellitus and hyperadrenocorticism may also be risk factors. The history may reveal a recent episode of dietary indiscretion or drug administration. Common clinical signs include lethargy, anorexia, hunched stance, vomiting (± blood), diarrhea (± blood), increased respiratory rate and painful abdomen.

Physical examination findings in dogs with acute pancreatitis are highly variable and include depression, dehydration, apparent abdominal pain, shock (tachycardia, prolonged capillary refill time, tacky mucous membranes, hypothermia), petechiation, icterus and ascites. An abdominal mass is palpated in some dogs. Some dogs with pancreatitis exhibit few localizing clinical signs. Especially in such cases, diagnosis requires a high index of suspicion and use of additional diagnostic tests, particularly evaluation of sonographic findings, serum enzyme activities and cPL results, and careful exclusion of other diseases that may cause similar clinical signs.

Laboratory findings

Findings on the CBC are highly variable, ranging from mild neutrophilia and slightly increased hematocrit, through marked leukocytosis with a left shift, to thrombocytopenia, anemia and leukopenia with a degenerative left shift. If thrombocytopenia is detected, blood clotting tests (OSPT, APTT, FDP) are performed to determine if the patient has disseminated intravascular coagulopathy (DIC). Serum biochemical abnormalities are also variable and include azotemia (pre-renal and renal), increased liver enzymes (ALT, AST, ALP), hyperbilirubinemia, lipemia, hyperglycemia, hypoproteinemia, hypocalcemia, metabolic acidosis and variable alterations (usually decreases) in sodium, potassium and chloride. Obtaining a urinalysis enables azotemia to be better characterized as renal or pre-renal. Transient proteinuria occurs in some dogs with acute pancreatitis. In dogs, the presence of glucosuria and/or ketonuria should prompt consideration of diabetes mellitus, which may be transient while the pancreatic inflammation is active or permanent, presumably due to destruction of a critical number of pancreatic beta cells.

Classically, increases in serum amylase and lipase activity have been used as indicators of pancreatic inflammation in dogs. These tests are not very accurate because dogs with non-pancreatic disorders may have elevated enzyme activities. Many different cell types in the body synthesize and secrete lipases. Serum activities of some lipases may increase with non-pancreatic disorders including intestinal obstruction (amylase), corticosteroid administration (lipase) and azotemia (both enzymes). Dogs with confirmed pancreatitis may also have normal amylase and lipase activities. For example, in two case series of dogs with histologically confirmed pancreatitis, lipase was normal in 28 and 61% of dogs, and amylase was normal in 31 and 47% of dogs, respectively. These limitations led to development of canine pancreatic lipase immunoreactivity (cPLI) with the goal of developing a more sensitive and specific blood test for pancreatitis. In comparisons of the different diagnostic tests in dogs with biopsy-proven pancreatitis, the sensitivity of serum TLI concentration was below 40% and that of serum lipase activity was about 60% (see table, below). (Note that serum cTLI concentration remains the diagnostic test of choice for exocrine pancreatic insufficiency). The sensitivity for serum cPLI for pancreatitis was above 80%, using a positive at >250 µg/L. The effect of azotemia on this test was investigated and it was found that serum cPLI was significantly higher in dogs with experimentally induced chronic renal failure than in clinically healthy dogs, but most renal failure dogs had serum cPLI concentrations within the reference range and none of the dogs had serum cPLI concentrations that were above 200 µg/L.
The cPLI test, developed at the Gastroenterology Laboratory at Texas A&M University, has now been made commercially available through Idexx Laboratories as the cPL test and as a semi-quantitative in-house SNAP test. Based on limited data, serum cPL appears to be quite sensitive for the diagnosis of canine pancreatitis (93% using a cutoff of >400 ug/L). Test specificity of 78% using <200 ug/L indicates that, even in a dog with a positive cPL test, attention must still be paid to ruling out other diseases that may produce similar clinical signs.

Imaging results

Radiographic signs of acute pancreatitis are nonspecific and don’t often contribute to diagnosis except by eliminating the presence of intestinal obstruction. Radiographic findings in dogs with acute pancreatitis may include loss of serosal detail, increased opacity in the right cranial quadrant of the abdomen, displacement of the duodenum ventrally and/or to the right, dilated hypomotile duodenum and caudal displacement of the transverse large intestine. Punctate calcification is occasionally identified in dogs with pancreatitis due to saponification of mesenteric fat in the region of the pancreas. Thoracic radiographs may enable the detection of pleural fluid or pulmonary edema, both of which have been associated with acute pancreatitis, and pneumonia occasionally develops in dogs that are ill with pancreatitis.

Ultrasonographic evaluation of the abdomen may identify a pancreatic mass or an enlarged hypoechoic pancreas that may surrounded by a hyperechoic rim, representing an increase in echogenicity of the peripancreatic fat. Pancreatic changes may be diffuse or involve one limb or region of the pancreas. Pancreatic cysts can also be identified. Ultrasound-guided fine needle aspiration of the pancreas for cytologic evaluation is being performed more commonly and can help confirm the diagnosis. Pancreatic neoplasia can also be detected by pancreatic cytology. Examination of peritoneal fluid (spontaneous or obtained by peritoneal lavage) may aid the differential diagnosis of acute abdominal signs including pancreatitis, gastrointestinal perforation or ruptured bile duct.

Sensitivity of available diagnostic tests for pancreatitis in dogs

<table>
<thead>
<tr>
<th>Test</th>
<th>Sensitivity</th>
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<tbody>
<tr>
<td>Lipase</td>
<td>54.5–73% using &gt;3x upper limit of range</td>
</tr>
<tr>
<td>TLI</td>
<td>36.4% using &gt;50ug/L</td>
</tr>
<tr>
<td>cPL</td>
<td>93% using &gt;400 ug/L</td>
</tr>
<tr>
<td>Radiology</td>
<td>24%</td>
</tr>
<tr>
<td>Ultrasound</td>
<td>68%</td>
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In dogs suspected of having acute pancreatitis, oral intake has traditionally been withheld for the initial 48h or longer and then gradually re-introduced as tolerated. The rationale for giving nothing by mouth was to “rest the pancreas” by decreasing pancreatic stimulation. This rationale is coming under close scrutiny in human medicine and requires re-examination in dogs. Currently, antiemetics are used immediately and as required to get vomiting controlled, and enteral feeding by nasoenterostomy tube is begun as soon as possible (or gradual oral alimentation, if possible). This approach attempts to maintain enterocyte integrity and reduce the risk of bacterial translocation. Recent studies in people indicate that enteral nutrition, administered via a naso-jejunostomy tube, can attenuate the systemic inflammatory response and may decrease complications. As the dog’s appetite returns, small amounts of a bland diet can be frequently offered. This diet should be highly digestible, and relatively low in fat. Boiled rice, rice with chicken, low fat cottage cheese, or prescription diets such as i/d® (Hills Pet Nutrition), or Low Residue (Iams) are effective. The size of the meals should be slowly increased and the frequency of feeding decreased if vomiting does not recur. After about 3 days, the normal diet can be slowly added. Continued fat restriction is usually recommended for dogs that have had pancreatitis.

Based on experimental evidence and experience in human patients with pancreatitis, some referral centers are using hyperbaric oxygen treatments in dogs with severe pancreatitis. No outcome data is available at present.

Prophylactic broad-spectrum antibiotics (e.g., amoxicillin ± enrofloxacin depending on severity) may be warranted in patients with shock, fever, or evidence of break down of the GI barrier. Surgery is occasionally needed to remove devitalized tissue in the unusual case with infected pancreatic necrosis or pancreatic abscess. Serum bilirubin may remain increased for weeks during apparent recovery from a bout of pancreatitis, but only rarely is surgery required to relieve an obstruction of the common bile duct. Resection or surgical drainage of pancreatic pseudocysts is not always necessary as these can resolve spontaneously or following ultrasound-guided percutaneous drainage.

**Prognosis**

The majority of dogs with acute pancreatitis respond to symptomatic therapy, as outlined above. The prognosis for dogs with mild acute pancreatitis is good. Severe or recurrent pancreatitis is associated with a more guarded prognosis. The presence of shock or abnormalities such as oliguria, azotemia, icterus, markedly elevated transaminases, hypocalcemia, hypoglycemia, hypoproteinemina, acidosis, marked leukocytosis, marked decrease in hematocrit, thrombocytopenia and DIC should be considered likely indicators of severe pancreatitis. Permanent diabetes mellitus occasionally follows a severe and/or recurrent pancreatitis. Because the etiology is unclear, recurrent bouts of pancreatitis can occur. Prevention strategies are currently not evidence-based, but may include continuing a well-controlled, fat restricted diet, increasing the omega-3 content of the diet, and supplementation with antioxidants.

**Summary**

Acute pancreatitis is a relatively common cause of vomiting in dogs but the severity of signs is highly variable. Some cases present with mild self-limiting vomiting that is similar to cases of dietary indiscretion. Other cases have life-threatening vomiting and require intensive therapy. Death occurs in some cases despite rigorous therapy. Acute pancreatitis is difficult to definitively diagnose. As the therapy for many causes of acute vomiting is similar to pancreatitis, misdiagnosing a case as having pancreatitis often does not have adverse consequences. However failure to perform additional diagnostic tests in cases with gastric or duodenal ulcer disease, foreign body intestinal obstruction, intussusception, or acute renal or liver failure, can have dramatic consequences. There is currently no single specific test for pancreatitis in dogs and diagnosis is based on a combination of compatible clinical, clinicopathological and imaging findings. Classic findings of include: 1) acute vomiting, 2) cranial abdominal pain, 3) pyrexia, 4) leukocytosis with a left shift, 5) elevated serum amylase and lipase, and 6) ultrasonographic findings of an enlarged hypoechoic pancreas. Supportive findings include: 1) signalment 2) recent fatty meal or dietary indiscretion, 3) lipemia, 4) hypocalcemia, 5) elevated ALT, ALP, and bilirubin 6) hypercholesterolemia and 7) increased cPL. Amylase and lipase can be useful as long as the clinician is aware of the other causes of increased amylase and lipase and does not rule out pancreatitis based on normal enzyme concentrations. Abdominal ultrasound has assumed a major role in the diagnosis of pancreatitis and the differentiation of pancreatitis from other pancreatic disorders, but a normal appearing pancreas (or inability to image the pancreas) does not rule out pancreatitis. Therapy is supportive with fluid and colloidal support, antiemetics, analgesics, and enteral nutritional support (as soon as vomiting is controlled) providing the mainstays of therapy.
Selected References


Feline Pancreatitis: Diagnosis and Management

Jane Armstrong, DVM, MS, MBA, DACVIM

Clinical presentation

Middle aged to older dogs (>5 yrs years old) who are overweight or obese appear at higher risk for developing acute pancreatitis. Miniature Schnauzers, Yorkshire and Silky Terriers, non-sporting breeds and perhaps miniature poodles may be at increased risk. There is no clear sex predisposition. Hypertriglyceridemia is a risk factor and may be why Miniature Schnauzers are overrepresented. Hypothyroidism, diabetes mellitus and hyperadrenocorticism may also be risk factors. The history may reveal a recent episode of dietary indiscretion or drug administration. Common clinical signs include lethargy, anorexia, hunched stance, vomiting (± blood), diarrhea (± blood), increased respiratory rate and painful abdomen.

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Laboratory findings

Findings on the CBC are highly variable, ranging from mild neutrophilia and slightly increased hematocrit, through marked leukocytosis with a left shift, to thrombocytopenia, anemia and leukopenia with a degenerative left shift. If thrombocytopenia is detected, blood clotting tests (OSPT, APTT, FDP) are performed to determine if the patient has disseminated intravascular coagulopathy (DIC). Serum biochemical abnormalities are also variable and include azotemia (pre-renal and renal), increased liver enzymes (ALT, AST, ALP), hyperbilirubinemia, lipemia, hyperglycemia, hyperproteinemia, hypocalcemia, metabolic acidosis and variable alterations (usually decreases) in sodium, potassium and chloride. Obtaining a urinalysis enables azotemia to be better characterized as renal or pre-renal. Transient proteinuria occurs in some dogs with acute pancreatitis. In dogs, the presence of glucosuria and/or ketonuria should prompt consideration of diabetes mellitus, which may be transient while the pancreatic inflammation is active or permanent, presumably due to destruction of a critical number of pancreatic beta cells.

Classically, increases in serum amylase and lipase activity have been used as indicators of pancreatic inflammation in dogs. These tests are not very accurate because dogs with non-pancreatic disorders may have elevated enzyme activities. Many different cell types in the body synthesize and secrete lipases. Serum activities of some lipases may increase with non-pancreatic disorders including intestinal obstruction (amylase), corticosteroid administration (lipase) and azotemia (both enzymes). Dogs with confirmed pancreatitis may also have normal amylase and lipase activities. For example, in two case series of dogs with histologically confirmed pancreatitis, lipase was normal in 28 and 61% of dogs, and amylase was normal in 31 and 47% of dogs, respectively. These limitations led to development of canine pancreatic lipase immunoreactivity (cPLI) with the goal of developing a more sensitive and specific blood test for pancreatitis. In comparisons of the different diagnostic tests in dogs with biopsy-proven pancreatitis, the sensitivity of serum TLI concentration was below 40% and that of serum lipase activity was about 60% (see table, below). (Note that serum cTLI concentration remains the diagnostic test of choice for exocrine pancreatic insufficiency). The sensitivity for serum cPLI for pancreatitis was above 80%, using a positive at >250 ug/L. The effect of azotemia on this test was investigated and it was found that serum cPLI was significantly higher in dogs with experimentally induced chronic renal failure than in clinically healthy dogs, but most renal failure dogs had serum cPLI concentrations within the reference range and none of the dogs had serum cPLI concentrations that were above 200 µg/L.

The cPLI test, developed at the Gastroenterology Laboratory at Texas A&M University, has now been made commercially available through Idexx Laboratories as the cPL test and as a semi-quantitative in-house SNAP test. Based on limited data, serum cPL appears to be quite sensitive for the diagnosis of canine pancreatitis (93% using a cutoff of >400 ug/L). Test specificity of 78% using <200 ug/L indicates that, even in a dog with a positive cPL test, attention must still be paid to ruling out other diseases that may produce similar clinical signs.
Imaging results

Radiographic signs of acute pancreatitis are nonspecific and don’t often contribute to diagnosis except by eliminating the presence of intestinal obstruction. Radiographic findings in dogs with acute pancreatitis may include loss of serosal detail, increased opacity in the right cranial quadrant of the abdomen, displacement of the duodenum ventrally and/or to the right, dilated hypomotile duodenum and caudal displacement of the transverse large intestine. Punctate calcification is occasionally identified in dogs with pancreatitis due to saponification of mesenteric fat in the region of the pancreas. Thoracic radiographs may enable the detection of pleural fluid or pulmonary edema, both of which have been associated with acute pancreatitis, and pneumonia occasionally develops in dogs that are ill with pancreatitis.

Ultrasonographic evaluation of the abdomen may identify a pancreatic mass or an enlarged hypoechogenic pancreas that may surrounded by a hyperechoic rim, representing an increase in echogenicity of the peripancreatic fat. Pancreatic changes may be diffuse or involve one limb or region of the pancreas. Pancreatic cysts can also be identified. Ultrasound-guided fine needle aspiration of the pancreas for cytologic evaluation is being performed more commonly and can help confirm the diagnosis. Pancreatic neoplasia can also be detected by pancreatic cytology. Examination of peritoneal fluid (spontaneous or obtained by peritoneal lavage) may aid the differential diagnosis of acute abdominal signs including pancreatitis, gastrointestinal perforation or ruptured bile duct.

Therapy

The initial medical management of dogs with acute pancreatitis must not be delayed until a diagnosis is confirmed. Intravenous fluid therapy with Lactated Ringers solution or 0.9% NaCl, supplemented with potassium and glucose as necessary is usually required. Potassium supplementation is necessary to replace losses in diarrhea, vomiting, and urine and supplement the lack of food intake. Potassium supplementation (20-30 mEq/l KCl to start), should be based on measurement of serum potassium levels. Symptomatic hypocalcemia (tremors, seizure activity) is a possible complication of acute pancreatitis and requires that calcium gluconate be given at doses of 50-150 mg/kg intravenously over 12-24 hours and serum total or ionized calcium concentrations monitored during therapy.

Plasma transfusion (12-20 ml/kg) has been recommended to provide a fresh source of protease inhibitors and may be indicated in the presence of hypoproteinemia or shock and to treat DIC. Colloids such as hetastarch are also useful for hypoproteinemia and may also have antithrombotic effects that help maintain the microcirculation. Dextran 40 may increase micro-perfusion. Insulin therapy is initiated in diabetic patients.

Nausea and vomiting may be severe in dogs with pancreatitis. The potent antiemetic, maropitant (Cerenia®, Pfizer), an NK1 receptor antagonist, administered at 1 mg/kg subcutaneously once a day is very useful in controlling emesis associated with pancreatitis. Alternatives are one of the 5-HT3 antagonists (ondansetron 0.1-1.0 mg/kg or dolasetron 0.5-1.0 mg/kg, orally or intravenously every 12-24 hours). The dopaminergic antagonist, metoclopramide, is a relatively weak antiemetic, but may be a useful adjunct to therapy to enhance motility in the upper gastrointestinal tract. An H2 antagonist, such as famotidine, is recommended to protect the esophagus from exposure to gastric acid during episodes of vomiting and may have other benefits.

Analgesia is an important aspect of treatment of pancreatitis. It can be provided using injectable opioids such as buprenorphine (0.005-0.01mg/kg SC q6-12hrs) or oxymorphone (0.1-0.2mg/kg IM, SC q 1-3hrs). A transdermal fentanyl patch is a good way of providing a longer duration of analgesia (10-20kg dogs, 50µg/hr patch q 72hrs) but adequate fentanyl blood levels are not attained for about 24 hrs after application in dogs, so another analgesic

In dogs suspected of having acute pancreatitis, oral intake has traditionally been withheld for the initial 48h or longer and then gradually re-introduced as tolerated. The rationale for giving nothing by mouth was to “rest the pancreas” by decreasing pancreatic stimulation. This rationale is coming under close scrutiny in human medicine and requires re-examination in dogs. Currently, antiemetics are used immediately and as required to get vomiting controlled, and nasoesophageal feeding by slow infusion is begun as soon as possible (or gradual oral alimentation, if possible). This approach attempts to maintain enterocyte integrity and reduce the risk of bacterial translocation. Recent studies in people indicate that enteral nutrition, administered via a naso-jejunostomy tube, can attenuate the systemic inflammatory response and may decrease complications. As the dog’s appetite returns, small amounts of a bland diet can be frequently offered. This diet should be highly digestible, and relatively low

<table>
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<th>Lipase</th>
<th>TLI</th>
<th>cPL</th>
<th>Radiology</th>
<th>Ultrasound</th>
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<td>54.5-73% using &gt;3x upper limit of range</td>
<td>36.4% using &gt;50ug/L</td>
<td>93% using &gt;400 ug/L</td>
<td>24%</td>
<td>68%</td>
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Sensitivity of available diagnostic tests for pancreatitis in dogs
Boiled rice, rice with chicken, low fat cottage cheese, or prescription diets such as i/d® (Hills Pet Products), EN® (Ralston Purina), or Low Residue (Iams) are effective. The size of the meals should be slowly increased and the frequency of feeding decreased if vomiting does not recur. After about 3 days, the normal diet can be slowly added. Continued fat restriction is usually recommended for dogs that have had pancreatitis.

Based on experimental evidence and experience in human patients with pancreatitis, some referral centers are using hyperbaric oxygen treatments in dogs with severe pancreatitis. No outcome data is available at present.

Prophylactic broad-spectrum antibiotics (e.g., amoxicillin ± enrofloxacin depending on severity) may be warranted in patients with shock, fever, or evidence of break down of the GI barrier. Surgery is occasionally needed to remove devitalized tissue in the unusual case with infected pancreatic necrosis or pancreatic abscess. Serum bilirubin may remain increased for weeks during apparent recovery from a bout of pancreatitis, but only rarely is surgery required to relieve an obstruction of the common bile duct. Resection or surgical drainage of pancreatic pseudocysts is not always necessary as these can resolve spontaneously or following ultrasound-guided percutaneous drainage.

**Prognosis**

The majority of dogs with acute pancreatitis respond to symptomatic therapy, as outlined above. The prognosis for dogs with mild acute pancreatitis is good. Severe or recurrent pancreatitis is associated with a more guarded prognosis. The presence of shock or abnormalities such as oliguria, azotemia, icterus, markedly elevated transaminases, hypocalcemia, hypoglycemia, hypoproteinemia, acidosis, marked leukocytosis, marked decrease in hematocrit, thrombocytopenia and DIC should be considered likely indicators of severe pancreatitis. Permanent diabetes mellitus occasionally follows a severe and/or recurrent pancreatitis. Because the etiology is unclear, recurrent bouts of pancreatitis can occur. Prevention strategies are currently not evidence-based, but may include continuing a well-controlled, fat restricted diet, increasing the omega-3 content of the diet, and supplementation with antioxidants.

**Summary**

Acute pancreatitis is a relatively common cause of vomiting in dogs but the severity of signs is highly variable. Some cases present with mild self-limiting vomiting that is similar to cases of dietary indiscretion. Other cases have life-threatening vomiting and require intensive therapy. Death occurs in some cases despite rigorous therapy. Acute pancreatitis is difficult to definitively diagnose. As the therapy for many causes of acute vomiting is similar to pancreatitis, misdiagnosing a case as having pancreatitis often does not have adverse consequences. However failure to perform additional diagnostic tests in cases with gastric or duodenal ulcer disease, foreign body intestinal obstruction, intussusception, or acute renal or liver failure, can have dramatic consequences. There is currently no single specific test for pancreatitis in dogs and diagnosis is based on a combination of compatible clinical, clinicopathological and imaging findings. Classic findings of include: 1) acute vomiting, 2) cranial abdominal pain, 3) pyrexia, 4) leukocytosis with a left shift, 5) elevated serum amylase and lipase, and 6) ultrasonographic findings of an enlarged hypoechoic pancreas. Supportive findings include: 1) signalment 2) recent fatty meal or dietary indiscretion, 3) lipemia, 4) hypocalcemia, 5) elevated ALT, ALP, and bilirubin 6) hypercholesterolemia and 7) increased cPL. Amylase and lipase can be useful as long as the clinician is aware of the other causes of increased amylase and lipase and does not rule out pancreatitis based on normal enzyme concentrations. Abdominal ultrasound has assumed a major role in the diagnosis of pancreatitis and the differentiation of pancreatitis from other pancreatic disorders, but a normal appearing pancreas (or inability to image the pancreas) does not rule out pancreatitis. Therapy is supportive with fluid and colloidal support, antiemetics, analgesics, and enteral nutritional support (as soon as vomiting is controlled) providing the mainstays of therapy.

**Selected References:**

Hepatic Lipidosis in Cats

Jane Armstrong, DVM, MS, MBA, DACVIM

PREVALENCE AND PATHOGENESIS

Feline hepatic lipidosis is now a well-recognized syndrome characterized by intracellular accumulation of lipid with clinico-pathologic findings consistent with intrahepatic cholestasis. In a 10-year retrospective study of all feline liver biopsies at the University of Minnesota College of Veterinary Medicine, hepatic lipidosis was the most common form of liver disease diagnosed in cats, accounting for 50% of all cases. This figure may even under-represent the true prevalence in this hospital population as the study did not include cats undergoing fine needle aspiration, but not biopsy. The clinical prevalence of the syndrome remains unknown but pathology surveys have revealed 5% of cats affected with this lesion. The triglyceride content in the liver of cats with lipidosis averages 43% compared to 1% in the liver of healthy cats. While some cases of hepatic lipidosis result from diabetes mellitus, the majority of cases are attributed to the nutritional and biochemical peculiarities of the cat. The cat does not appear very capable of regulating intermediary metabolism during starvation. Histologic evidence of hepatic lipidosis was found to develop within two weeks of the onset of fasting in a feline experimental model. While many cats develop lipidosis during periods of anorexia related to another underlying disease, otherwise healthy cats can also develop the syndrome due to inadequate intake during periods of enforced weight loss, unintentional food deprivation, or stress (e.g. boarding). This understanding has emphasized the importance of maintaining food intake in cats that become hyporexic/anorexic for any reason for periods of longer than a few days.

CLINICAL FEATURES

Most studies suggest that there are no breed, sex, or age predilections for the development of hepatic lipidosis. One retrospective study suggests that female and middle-age cats are at greater risk. Obese or overweight cats that undergo a period of anorexia and weight loss may be particularly at risk. Anorexia, often of several weeks duration, is the primary presenting complaint in cats with this syndrome. Observant owners may report jaundice. Other historical findings are vomiting, weakness, weight loss, and diarrhea. Physical examination commonly reveals dehydra- tion, cachexia, jaundice, and hepatomegaly. All of these findings are also reported in cats with other hepatobiliary diseases and acute pancreatitis. Careful physical examination and laboratory/imaging evaluation is crucial in cats with signs of lipidosis in order not to overlook concurrent disorders. Concurrent diseases were recognized in > 90% of lipidosis cases in one study and included inflammatory bowel disease, inflammatory liver disease and cholecystitis, pancreatitis, septic peritonitis, and toxicosis.

LABORATORY FINDINGS

Clinicopathologic findings in hepatic lipidosis are typical of an intrahepatic cholestatic disorder. The most consistent laboratory findings are increases in serum bilirubin, and serum activities of alkaline phosphatase (ALP), alanine aminotransferase (ALT), and aspartate aminotransferase (AST). Serum ALT and AST are less consistently increased than ALP. Increases in serum gamma-glutamyl transpeptidase (GGT) are inconsistently seen. When cats with cholangitis/cholangiohepatitis are compared to cats with hepatic lipidosis, hepatic lipidosis cases tend to have higher total bilirubin concentrations, and higher ALT and SAP activities. Unlike in hepatic lipidosis, increases in GGT tend to parallel increases in SAP in other forms of liver disease in cats. More severe and chronic lesions may result in hypoglycemia, hypoalbuminemia, hyperammonemia, low BUN, and coagulation abnormalities. A normocytic, normochromic non-regenerative anemia, poikilocytosis, and mild leukocytosis are frequently observed.
IMAGING FINDINGS

Hepatomegaly is a frequent finding in chronic cases; it may not be observed in early cases. Hyperechoic changes in the hepatic parenchyma on ultrasonographic examination are expected. This change has been cited as a pathognomonic finding, but may be seen in other feline hepatic disorders. Diagnosis should be substantiated by aspiration cytology, or better still, tissue biopsy (percutaneous, ultrasound-guided, laparoscopy, or laparotomy). Reliance on aspiration cytology alone may confirm fatty change in the liver but miss other, potentially more important, diagnoses. Fine needle hepatic aspiration cytology typically shows vacuolation of > 80% of sampled hepatocytes. The liver parenchyma is typically very friable in cats with hepatic lipidosis, requiring pre-biopsy screening for a coagulation disorder, atraumatic biopsy technique, and careful post-biopsy monitoring.

THERAPY

In many cases of suspected hepatic lipidosis, it may be prudent to postpone a tissue biopsy (especially if general anesthesia is required) until 2-3 days of fluid therapy, nutritional support and other therapy can be given. If coagulation abnormalities are present, administer vitamin K, 0.5-1.5 mg SQ or IM using a 25 g needle; give 2 doses at a 12 hr interval. Fluid therapy should be accompanied by regular monitoring of serum electrolytes, especially potassium and phosphate concentrations. One prospective evaluation of plasma B₁₂ (cobalamin) concentrations in cats with lipidosis revealed that 40% had subnormal values. All cats with a concurrent diagnosis of inflammatory bowel disease tested had subnormal concentrations. All cats with a concurrent diagnosis of inflammatory bowel disease tested had subnormal concentrations. The route of choice for cobalamin supplementation is parenteral injection (250 µg/injection once weekly for six weeks, once every two weeks for six weeks, then monthly as assessed by regular measurement of serum cobalamin concentration).

Nutritional support is the cornerstone of successful therapy for hepatic lipidosis. Most studies suggest that recovery rates of 90-95% can be expected if enteral feeding is initiated as early as possible in the course of the disease and sustained until voluntary intake resumes. Useful methods for enteral support include feeding via nasoesophageal, esophagostomy or gastrostomy tube. The benzodiazepine agonists (diazepam, oxazepam) and 5-HT₂ agonists (cyproheptadine) should be avoided in cats with hepatic lipidosis as they may exacerbate pre-existing hepatic encephalopathy. Appetite stimulants, especially cyproheptadine, may increase appetite momentarily, but are unreliable for ensuring adequate caloric intake. Oral force-feeding is of limited benefit in cats that have been anorectic for prolonged periods (> 1 week) and can be stressful when performed. It should only be attempted for a short time and abandoned in favor of a tube-feeding technique if unsuccessful in inducing significant voluntary food intake in 1-2 days. Concern also exists about inducing learned food aversion by using force-feeding.

Nutritional support should initially deliver sufficient amounts of a nutritionally balanced food meet the cat’s resting energy requirement (RER) at its current weight when the body condition score (BCS) is 3/5 or less. 60 kcal/kg of body weight/day approximates RER in cats. Cats with a BCS of 4/5 or 5/5 generally have the same muscle mass as those with a BCS of 3/5, therefore RER is calculated based on estimated optimal weight to prevent overfeeding. This is an important consideration as many cats with hepatic lipidosis are still obese at presentation.

Some of the metabolic changes that take place during fasting in obese cats have been studied. Small amounts of protein administered to obese cats during fasting significantly reduce accumulation of lipids in the liver, prevent increases in alkaline phosphatase activity, eliminate negative nitrogen balance, and appear to minimize muscle catabolism. These findings emphasize the importance of providing cats with hepatic lipidosis adequate protein during recovery. In these studies, carbohydrate supplementation reduced hepatic lipid accumulation, but metabolic abnormalities still developed.

Many cats with lipidosis are initially volume sensitive. The feeding schedule is determined by the patient’s ability to tolerate food and the logistics of feeding. Feeding one-third of RER over day 1 (divided into 4-6 feedings) and then increasing the amount by one-third every 24 hours may be better tolerated than schedules that reach RER faster. Foods should be warmed to room temperature, but not higher than body temperature, before feeding. Food boluses must be infused slowly (approximately one minute) to allow gastric expansion. Daily food dosage should be divided into several meals, usually 3-4 per day. Feeding should be stopped at the first sign of gulping, retching or salivating, the meal size reduced by 50% for 24 hours and then increased gradually. Each meal must be followed by a water flush to clear the feeding tube of food residue. When the patient is volume sensitive, it is important to know the minimum volume required to flush the tube. The patient’s daily fluid requirement must also be met and additional water may be administered through the feeding tube to meet that requirement. Liquid oral medications may also be administered through feeding tubes.

Some authors advocate routinely providing L-Carnitine to hepatic lipidosis cats (250 mg PO/day) for the theoretical reason of promoting fatty acid oxidation and retention of lean body mass. This metabolic response to L-carnitine has recently been proven in obese healthy cats undergoing weight loss, but evidence is lacking that it provides any benefit in recovery from hepatic lipidosis. Cats showing heinz body hemolytic anemia may benefit from administration of a thiol substrate donor such as n-acetylcysteine followed by supplemental s-adenosylmethionine for several weeks.
Cats making a successful clinical recovery from hepatic lipidosis demonstrate a gradual reduction in serum enzyme activities and total bilirubin concentrations over time. Expect the total bilirubin concentration to decline by ≥ 50% within 10 days, even though serum enzyme activities may remain close to values documented at the time of case admission. Cats may need tube feeding for several (3-6) weeks, requiring that the owner is an active participant in their cat’s recovery.

Successful recovery of cats with hepatic lipidosis requires correction of fluid and electrolyte abnormalities (especially hypokalemia and hypophosphatemia) and nutritional support. Other therapies, such as supplementation with vitamin K₁ and cobalamin, may be indicated. The cornerstone of therapy, however, is nutritional support concentrating on meeting protein and caloric needs. This is best achieved by early use of a nasoesophageal tube followed by placement of either an esophagostomy or gastrostomy feeding tube. An important component of recovery involves recognition and elimination or therapeutic management of any underlying process initially promoting the onset of hepatic lipidosis.

References available on request.
Inflammatory liver disease is a histopathologic diagnosis; it must be made by tissue examination. The WSAVA International Liver Standardization Group Classification has proposed the following classification scheme:

a) Neutrophilic cholangitis – acute and chronic forms
b) Lymphocytic cholangitis
c) Cholangitis associated with liver flukes
d) Lymphocytic portal hepatitis.

Cholangitis (inflammation of bile ducts and ductules) is a much more important entity than hepatitis in the domestic cat (unlike the dog). Thus, the first two syndromes are better referred to as cholangitis rather than cholangiohepatitis, even though the latter term has been commonly used in the past. There appears to be a separate entity of portal hepatitis in cats, but it remains an open question as to whether it is a disease or a histologic lesion associated with aging (non-specific reactivity).

Inflammatory liver disease is the second most common category of feline liver disease after hepatic lipidosis. The true prevalence of inflammatory liver disease in the overall cat population is unknown. In our retrospective study, 15% of feline liver biopsies had lymphocytic infiltrates confined to the portal areas or lymphocytic portal hepatitis (LPH). The acute form of neutrophilic cholangitis accounted for about 6% of feline biopsies and the chronic form of neutrophilic cholangitis for about 4% of feline liver biopsy diagnoses. Lymphocytic cholangitis is a very chronic disease that has been described almost exclusively in cats from Europe, with Persian cats being over-represented. Liver fluke infestation is associated with chronic cholangitis in cats. The trematode Platynosomum sp is the most common genus identified and is limited to subtropical and tropical climatic zones.

**NEUTROPHILIC CHOLANGITIS**

**Acute neutrophilic form (ANF)**

This disorder has been seen primarily in young to middle-aged male cats with clinical signs of acute vomiting, diarrhea, anorexia, and lethargy. Physical examination findings often reveal fever, dehydration, icterus, abdominal pain, and hepatomegaly (<50% of cases). Laboratory findings frequently reveal mild to moderate leukocytosis with mild to moderate elevations in ALT, AST, GGT, and ALP. Cats affected with this form of cholangitis often have related disease, e.g., pancreatitis and inflammatory bowel disease. Acute cholangitis may begin as an ascending bacterial infection within the biliary tract; however, bacteria have been isolated from the liver or gall bladder in only a few cases. Diagnostic evaluation includes culture and Gram staining of bile, gallbladder wall, liver and cholelith (if found) and biopsy of the liver and/or extrahepatic biliary system. Whenever possible, a 22 g ultrasound-guided gallbladder aspirate for bile cytology and culture should also performed. Bile aspirates are relative safe if the needle is directed through the right medial liver lobe and into the gall bladder lumen. Using this approach, any bile leakage will drain back into the liver and not into the peritoneal cavity. Suppurative inflammation and bacteria may be observed in the bile cytology and is diagnostic for the ANF of cholangitis. Bacterial isolates in affected cases are usually enteric organisms such as E. coli, Enterococcus spp, Bacteroides spp, Clostridia spp, Staphylococcus, and β-hemolytic Streptococcus. Preliminary investigations using culture-independent methods have resulted in identification of DNA sequences of Helicobacter spp from a few cats with inflammatory liver disease (both acute and chronic forms) and these DNA sequences were distinct from gastric species that are found in cats. Congenital or acquired abnormalities of the biliary system, including anatomic abnormalities of the gallbladder or common bile duct and choleliths may predispose cats to cholangitis. Inspissation of bile, which may cause partial or complete obstruction of the common bile duct or intrahepatic bile ducts, appears to frequently accompany acute cholangitis and may require treatment before the disease can be controlled or resolved. The treatment of this syndrome has included appropriate antibiotic based on culture and sensitivity (or an empirical choice if cultures are negative or not obtained), cholelith removal where appropriate, bile duct decompression if necessary, fluid and electrolyte maintenance, and ursodeoxycholate therapy.
Chronic neutrophilic form (CNF)

The chronic form of neutrophilic cholangitis (CNF) is speculated to be a later stage of the acute form of neutrophilic cholangitis. It is characterized by a mixed inflammatory response (equal numbers of lymphocytes or plasma cells and neutrophils) within portal areas and bile ducts. Other features of chronicity include marked bile duct proliferation, bridging fibrosis, and pseudolobule formation. CNF may rarely progress to progressive biliary cirrhosis and the death of the patient. CNF may represent a persistent bacterial infection or an immune-mediated response may result in a chronic self-perpetuating disorder. Affected cats had a mean age of 9.0 years. Clinical signs are usually of a chronic, intermittent or persistent nature. Vomiting, icterus, and hepatomegaly are common findings, and ascites may be present. Hepatic encephalopathy and excessive bleeding are uncommon unless severe end-stage liver disease is present. The best treatments for this syndrome have not been investigated. Suggested treatment plans include glucocorticoids, metronidazole, Ursodeoxycholate, vitamin K₁, and dietary manipulation for presumed IBD.

Lymphocytic Cholangitis

This syndrome is an important cause of hepatic disease in cats in the United Kingdom. It seems to represent a distinct form of inflammatory liver disease from those reported in the USA. It has previously been called progressive lymphocytic cholangitis or cholangiohepatitis. It occurs predominantly in cats 4 years and under. The most common clinical features are ascites, jaundice, and hypergammaglobulinemia. This is one of the only forms of liver disease in cats in which ascites is noted. Fever is not a feature of the disease. Persian cats appear to be over-represented, suggesting a genetic predisposition. The rate of clinical progression is variable. There are two distinct stages of hepatic histopathology. In the active stage, there is marked lymphocytic inflammation of portal tracts, particularly surrounding and infiltrating bile ducts, with occasional extension to periportal hepatic parenchyma. The chronic stage is characterized by predominant monolobular fibrosis but also by a reduction in the intensity of lymphocytic infiltration. This stage results in considerable distortion of liver architecture. Concurrent pancreatic inflammation is not a feature of the disease. Evidence for an immune-mediated pathogenesis has been provided by immunohistochemical characterization of the lesions but DNA evidence of H. pylori have recently been recently identified in the bile of some affected cats, raising more questions about etiology. Helicobacter spp. have also been recently implicated in human primary sclerosing cholangitis and primary biliary cirrhosis.

Cholangitis Associated with Liver Flukes

Liver fluke infestation is associated with chronic cholangitis in cats. The trematode Platynosomum sp is the most common genus identified and is found in subtropical and tropical climates of the world. The life cycle of Platynosomum sp includes two intermediate hosts, the first being a land snail and the second a lizard, gecko or toad. Affected cats acquire the parasite by ingesting the second intermediate host, so affected clinical cases are generally adult outdoor cats. Immature liver flukes migrate to the liver from the intestine via the bile ducts, causing marked thickening and cystic dilatation of the bile ducts. Bile duct enlargement and tortuosity can be observed ultrasonographically but it is important to note that other causes of cholangitis can result in these same changes. Adult flukes or oocerculated eggs may be observed within the bile ducts on histopathological examination or in feces. Episodes of vomiting, anorexia and fever are common and substantial increases in liver enzymes are observed. Icterus from severe cholestatic liver disease or bile duct obstruction can occur. Clinical signs range from asymptomatic cases to death from hepatic failure. The recommended treatment is praziquantel (20-30 mg/kg PO q24h for 3 days).

Lymphocytic Portal Hepatitis

A subset of cats of liver biopsies of cats with inflammatory liver disease have lymphocytic infiltrates confined to the portal areas. As opposed to cats with cholangitis, there is a lack of neutrophilic inflammation, bile duct involvement, infiltration of inflammatory cells into hepatic parenchyma, or periportal necrosis. Lymphocytic portal hepatitis is not associated with inflammatory bowel disease or pancreatitis. Lymphocytic portal hepatitis is a common finding in liver biopsies of older cats. In a retrospective study, 82% of cats greater than 10 years old had histopathologic changes consistent with lymphocytic portal hepatitis, whereas only 10% of cats younger than 10 had these histopathologic changes. This suggests that it is a common aging change or that a subclinical form of disease is prevalent. Lymphocytic portal hepatitis appears to progress slowly with varying degrees of portal fibrosis and bile duct proliferation but no pseudolobule formation. Concurrent hepatic lipidosis is less likely than with cholangitis. Because of the presence of lymphocytes and plasma cells in portal areas and the lack of concurrent diseases, several authors have speculated that lymphocytic portal hepatitis is an immune-mediated disease. This speculation provides the rationale for immunosuppressive therapy in symptomatic cases. Data on the role of immune mechanisms in the initiation and/or perpetuation of lymphocytic portal hepatitis, however, have not been reported, and controlled studies on therapy are also lacking.
TREATMENT

Successful recovery of cats with any form of liver disease requires correction of fluid and electrolyte abnormalities (especially hypokalemia and hypophosphatemia) and attention to nutritional support. Other therapies, such as supplementation with vitamin K, and cobalamin, may be indicated. Nutritional support, concentrating on meeting protein and caloric needs, is important to aid in recovery, treat any concurrent hepatic lipidosis, and prevent fatty change from occurring.

Cats with the acute form of neutrophilic cholangitis require aggressive supportive care. These cats are frequently acutely ill and have fluid and electrolyte derangements which should be corrected. If coagulation abnormalities are present, administer vitamin K, 0.5–1.5 mg SQ or IM using a 25 g needle; give 2 doses at a 12 hr interval. Hepatic encephalopathy appears to be relatively uncommon in cats with acquired liver disease and is manifest most frequently by excessive salivation. Hepatic encephalopathy can be managed by giving lactulose orally (0.5–1.0 ml/kg q8h PO) with or without addition of enteric antibiotics (neomycin 20 mg/kg q8-12h PO). Anorexia must be managed promptly to prevent further deterioration in clinical condition and possible development of concurrent hepatic lipidosis. Assisted oral feeding should be tried for no longer than 12–24 hours, after which a nasoenteral tube should be placed if voluntary intake is inadequate. Ensuring that RER (resting energy requirements) are met through intake of a high energy, high protein diet is a high priority throughout treatment. Protein is an important nutrient for liver repair and regeneration and should not be restricted unless hepatic encephalopathy occurs.

Hepatobiliary disease can be painful and pain management is indicated in those cases. Most acute pain control can be accomplished through the use of injections of hydromorphone or buprenorphine (the latter drug can also be administered sublingually and is absorbed through the buccal mucous membranes). Injections of meperidine or butorphanol can also be used. For longer duration pain control, a fentanyl patch is very effective.

The major specific therapy for neutrophilic cholangitis is antimicrobials. Surgical intervention has been recommended if discrete choleliths or complete biliary obstruction is identified, but biliary-to-intestinal diversion (i.e. cholecystoduodenostomy or cholecystojejunostomy) sold be avoided if possible. Bacterial culture and sensitivity testing of bile, liver aspirate or biopsy specimens, choleliths, or gallbladder specimens, should be used to select appropriate antimicrobial agents whenever possible. Antibiotics chosen for treatment of cholangitis should be excreted in the bile in active form, and should be active against aerobic and anaerobic intestinal coliforms. Ampicillin or amoxicillin combined with clavulanic acid is frequently used. These drugs may be combined with metronidazole (7.5 mg/kg PO BID) to extend the spectrum to anaerobes and more coliforms. Treatment with antibiotics for 4 weeks or longer is recommended.

If the biopsy sections in cats with the CNF of cholangitis contain relatively few neutrophils, concurrent antimicrobial and corticosteroid therapy is often used. Alternatively, prednisolone may be added if cats with chronic cholangitis fail to respond to antibiotic therapy alone within 2 to 3 weeks. A dose of 1–2 mg/kg of prednisolone (or higher) is used initially. If successful, the dosage is slowly tapered to an alternate day dose for long-term maintenance. A schedule commonly used is to start therapy at 1 mg/kg BID x 2 weeks, then progressively reduce the dose as follows: 1 mg/kg SID x 2 weeks; 0.5 mg/kg SID x 2 weeks; 0.5 mg/kg q 48 hours x 4 weeks. Biochemical values should be monitored prior to each reduction in dosage. If the clinical and biochemical response is satisfactory, doses as low as 0.5 mg/kg q 48 hours may be sufficient for long-term maintenance. Long-term corticosteroid treatment is well tolerated by most cats and side effects are usually minimal. An additional therapeutic strategy includes dietary manipulation for documented or presumed IBD.

Lymphocytic portal hepatitis may not require therapy; if treatment is begun it is based on the hypothesis that LPH represents a disease syndrome and that liver injury is immune-mediated. In symptomatic cases, Immunosuppressive doses of corticosteroids are empirically used. Lymphocytic portal hepatitis appears to be a very slowly progressive condition, with mean survival reported to be 3 years.

Ursodeoxycholic acid (Actigall) is recommended for cats with all types of inflammatory liver disease. It has anti-inflammatory, immunomodulatory, and antibacterial properties as well as increasing fluidity of biliary secretions. Ursodeoxycholic acid has safely been administered to cats at a dose of 10 to 15 mg/kg q24 h PO. Efficacy has not been established in any type of feline liver disease, but clinical trials in human patients with hepatitis support improved quality of life. Adverse effects in cats are uncommon and usually limited to mild diarrhea.

There is increasing interest in the use of nutraceuticals in the treatment of feline liver disease. At this time, controlled clinical trials are lacking, but their widespread use likely reflects clinician frustration in treating many forms of liver disease and the belief that they are relatively safe. Oxidative damage from free radical formation is one potential mechanism of cellular damage in liver disease. High concentrations of bile acids, accumulation of heavy metals, and inflammation cause free radical generation in the liver. Vitamin E and SAMe are commonly used; Vitamin C and phosphatidylcholine may also be considered. The herb, milk thistle, has also recently attracted veterinary attention. Clinical trials on its use in veterinary patients have not yet been conducted.

References available on request.
Hepatitis in Dogs: The Two Main Types

Jane Armstrong, DVM, MS, MBA, DACVIM

No Paper Available
Introduction

Cholestasis is defined as impairment of normal bile flow accompanied by the accumulation in the blood of components normally secreted in the bile (a.o. bile acids, conjugated bilirubin, cholesterol). Morphologically it is characterized by the presence of bile in the hepatic parenchyma and can be recognized as bile thrombi in the canaliculi, phagocytosed bile (thrombi) in Kupffer cells / macrophages and as bile granules in the cytoplasm of hepatocytes. Intrahepatic cholestasis is associated with a wide spectrum of liver diseases. In general, microscopic lesions apart from the cholestasis are related to the primary hepatic disease. Extrahepatic cholestasis can be associated with intraluminal obstruction (mucocele, mucinous cystic hyperplasia, gall stones) or luminal constriction (neoplasia or inflammatory processes) of the extrahepatic biliary tract, and occasionally a large intrahepatic duct, and results in stasis of bile and dilatation of the bile ducts proximal to the obstruction. The characteristic microscopic lesions are related to the leakage of bile from the bile ducts into the stromal tissue of the portal areas. In the acute stage this results in enlarged edematous portal tracts with a neutrophilic portal infiltrate and often, a degenerative and proliferative reaction of the bile duct epithelium. The chronic stage of extrahepatic cholestasis is characterized by enlarged portal areas with fibrosis, bile duct proliferation and a mixed inflammatory infiltrate. These histopathological changes may be recognized when liver biopsies are performed in conjunction with working up a dog for biliary tract disease, such as gall bladder (GB) mucoceles.

Mucoceles: What are they?

A mucocele is a dilatation or distention of a cavity with accumulated mucus. Gall bladder mucoceles had not been described in dogs prior to the early 1990s but now appear to be an increasingly frequent diagnosis. Generalized GB wall thickening is a relatively common sonographic finding in dogs. This may be a local manifestation of a systemic cause of edema, such as hypoalbuminemia, right-sided heart failure, pancreatitis, or of localized disease, such as cholangitis. Cystic mucinous hyperplasia of the GB is recognized with some frequency at post-mortem examination of dogs with or without signs of biliary tract disease. It is characterized by hyperplasia of the epithelium with papillary projections and an increased mucin production. Whether cystic mucinous hypertrophy (also called biliary mucosal hyperplasia) is one disease with a range of clinical manifestations (from mild GB wall thickening to wall necrosis +/- gelatinous inspissated bile or “mucocele” formation, with or without post-hepatic biliary obstruction, or separate conditions remains to be determined. Regardless, histologic examination of a resected GB in cases with mucocele often reveal cystic mucosal hyperplasia +/-cholecystitis. Liver biopsies taken concurrently may show periportal hepatitis and/or vascular hepatopathy.

Although the definitions of mucoceles vary, the basic characteristics are an inappropriate intraluminal accumulation of inspissated bile and/or mucus. The GB is distended with either green-black gelatinous material or a mass. The inspissated bile and mucus may extend throughout the biliary tree causing extrahepatic biliary obstruction. Mucoceles are sometimes associated with neutrophilic cholecystitis (characterized by the presence of neutrophils in the lumen and/or the wall of the GB, sometimes with erosion and ulceration) and GB rupture.

Signalment

Gall bladder mucoceles affect dogs of either gender with approximately equal frequency. The median age at diagnosis is was10 years (range of 3.5-15.0 years), and dogs <20 kg accounted for >70% of cases in one study. Three breeds are over-represented: Shetland Sheepdogs, Miniature Schnauzers, and Cocker spaniels. Shetland sheepdogs have been best studied: the breed appears predisposed to several gallbladder disorders, with mucoceles and concurrent dyslipidemia or dysmotility in many affected dogs (Ale et al, 2007).

Clinical signs

Many (probably most) dogs with GB mucoceles are asymptomatic. In the case series of Shetland sheepdogs described above, gallbladder disease was serendipitously discovered in 11 of 38 dogs when they were undergoing abdominal ultrasound for an unrelated reason. Affected dogs can show chronic signs such as anorexia and abdominal discomfort. Acute signs may also occur, especially
if the mucocele leads to GB rupture or if it is associated with acute cholecystitis. Median duration of signs was 3 days in one case series (Crews et al, 2009) and frequency of signs was vomiting (77%), lethargy (73%), anorexia (71%), jaundice (47%), pain on abdominal palpation (44%), diarrhea (29%) and fever (20%). In these acute cases, a mucocele was present in 21 of the 45 (47%), and 9 of those (43%) had GB rupture resulting in bile peritonitis.

**Laboratory findings**

CBC: A stress leukogram is common in dogs that present with clinical signs attributable to GB disease. Leukocytosis due to neutrophilia is seen in some dogs and, in one study (Ale et al, 2007), white blood cell and neutrophil counts were significantly higher among nonsurvivors.

Serum chemistry findings: hyperbilirubinemia and increased activities of alkaline phosphatase, alanine transaminase, and -glutamyltransferase may be present. Hypercholesterolemia and fasting hypertriglyceridemia may be seen, especially in breeds predisposed to idiopathic hyperlipidemia. Serum potassium concentration has also found to be significantly lower among nonsurvivors.

In the above case series of dogs with GB disease undergoing surgery (Crews et al 2009), bile was culture positive in 11 of 40 dogs tested (9 had gallbladder wall necrosis and 5 had a rupture). Only 2 dogs had concurrent positive results of bacterial bile culture and GB mucocele. Organisms isolated were: nonhemolytic Escherichia coli (6 cases), Streptococcus spp and Enterococcus spp (each 2 cases) and Clostridium perfringens (the only anaerobe isolated).

**Sonographic Appearance of a Mucocele**

The classic description of the ultrasonographic appearance of a GB mucocele is “kiwi fruit-like” or “stellate patterned”. Alternatively, a mixed echogenic, mosaic-like appearance may be seen. The key points are that the contents do not move in a real-time study and the GB appears rounded or distended, as if under pressure. (Besso 2000).

In one study of 45 dogs with histologically confirmed GB disease that had ultrasonographic evaluation, the most common sonographic findings were echogenic peritoneal fluid, thickened or laminated GB wall, and echogenic reaction in the GB fossa (Crews et al, 2009). Eighteen of 45 (40%) dogs had GB rupture. Rupture was associated with histologic evidence of GB necrosis, decreased serosal detail radiographically, and pericholecystic echogenic reaction, pericholecystic echogenic fluid, and generalized echogenic abdominal effusion ultrasonographically.

**Concurrent Diseases**

It has been suggested by multiple authors that endocrinopathies such as hyperadrenocorticism and hypothyroidism may play a role in GB disease. A recent retrospective case-control study investigated whether there is a significant association between the presence of endocrinopathies and the development of mucoceles in dogs (Mesich et al, 2009). 78 dogs with a surgical or ultrasonographic diagnosis of GB mucocele and 2 age- and breed-matched control dogs were evaluated for the presence of hyperadrenocorticism, diabetes mellitus and hypothyroidism. The odds of mucocele in dogs with hyperadrenocorticism were 29 times that of dogs without hyperadrenocorticism. No differences in risk were found for diabetes, and the results for hypothyroidism were equivocal (increased risk identified but observation bias may have affected results). The results of this study suggest that hyperadrenocorticoid dogs presented for acute illness with laboratory evidence of hepatobiliary disease should undergo evaluation for the presence of a biliary mucocele. Dogs diagnosed with a GB mucocele should be screened for concurrent hyperadrenocorticism if clinical suspicion exists.

**Treatment**

The management of GB mucoceles has not been investigated in any controlled trials. Most authors recommend cholecystectomy if there are clinical signs associated with the presence of a mucocele, especially signs such as jaundice, fever, marked leukocytosis with or without a left shift, sonographic evidence of pericholecystic echogenic reaction or echogenic fluid, or if generalized (echogenic) peritoneal effusion is detected by any imaging modality (Bowlus 2005). The concern in such cases is the presence of necrotizing cholecystitis, with possible GB rupture and resultant bile peritonitis. Less frequently, cholecystoenterostomy is performed. Whether surgery needs to be performed on an emergency basis in cases with an intact GB is unknown, but may be a prudent choice if the dog is ill. What is even less clear is the optimal approach in dogs with milder clinical signs. Surgery is advocated by some, but cholecystectomy has a mortality of 15-30%. In one study of dogs with sonographic evidence of GB disease, post-operative survival (86% overall) was not significantly associated with presurgical bile leakage, a positive bacterial culture, or the presence of a mucocele. Mucocele or bacterial GB infection is the most common concurrent finding in dogs with GB rupture. The relatively low survival rate after cholecystectomy in clinically affected dogs has lead some to suggest that preemptive surgical interventions (in mildly symptomatic and even in asymptomatic dogs) may be a more appropriate treatment strategy.

Resolution of GB mucoceles with medical treatment alone has been reported (Walter et al, 2008) and one such case was noted.
in the 2007 manuscript of Ale et al. An asymptomatic Shetland sheepdog with hyperlipidemia was treated with a fat-restricted diet and ursodeoxycholic acid (Ursodiol, 10-15 mg/kg PO divided into 2 doses/day). The GB mucocele resolved within 6 months. In the more recent report, resolution with medical therapy was described in two mildly symptomatic dogs. A 12-year-old spayed female Miniature Schnauzer and a 6-year-old neutered mixed-breed dog were each diagnosed with GB mucocele and hypothyroidism. The first dog was treated with S-adenosyl-methionine (SAMe; 20 mg/kg PO on an empty stomach, omega-3 fatty acids, famotidine, ursodiol, and levothyroxine. Substantial improvement in the gastrointestinal signs and complete resolution of the GB mucocele were evident within 3 months, but the dog was not available for further follow-up monitoring. The second dog was treated with fenbendazole, ursodiol, and levothyroxine and fed a hypoallergenic diet. One month after evaluation, abdominal ultrasonography revealed that the mucocele was resolving, and treatment was continued. Ultrasonographic evaluation 2 and 4 months later revealed complete resolution of the mucocele. These cases indicate that surgery is not necessary in all dogs with GB mucocele. Despite this information, until further prospective trials with a control group and standardized treatments and follow-up monitoring can be performed, surgical intervention for treatment of dogs with symptomatic GB mucocele is generally recommended. Medical therapy appears to be appropriate for asymptomatic dogs found to have a mucocele as an incidental finding. Such dogs should be tested and managed for concurrent hyperadrenocorticism and/or hypothyroidism, as indicated. Guidelines for medical therapy remain empirical, and may include a low fat diet, ursodiol, SAMe, and fish oils.285

Selected References


What’s New in Canine Epilepsy

Joane Parent, DMV, MVetSc, ACVIM Neurology

INTRODUCTION

It is important to define the terminology used in the context of this presentation as it greatly affects this speaker’s diagnostic and therapeutic approach to recurrent seizures in dogs. In the context herein, the epilepsy is idiopathic when there “is only epilepsy, with no underlying structural brain lesion or other neurological signs or symptoms; idiopathic epilepsy is presumed genetic and usually age dependent”.1,2 This definition implies that the patient has had a thorough diagnostic work up that includes magnetic resonance imaging (MRI) of the brain and cerebrospinal fluid (CSF) analysis. The epilepsy is symptomatic when “the epileptic seizures are the result of one or more identifiable structural lesions of the brain”.1,2 The epilepsy is probably symptomatic when “the epilepsy is believed to likely be symptomatic but no etiology has been identified”.1,2 The following presentation is the summation of over 25 years of medical neurology referral practice. The objective is to increase the knowledge of the practicing veterinarian as it relates to seizure activity in the hope to improve seizure control and ultimately increase the lifespan of epileptic dogs.

SEIZURE PATTERN AND DIFFERENTIAL DIAGNOSIS

An epileptic seizure is a diagnostic entity with etiologic, therapeutic and prognostic implications.1,2 The cause of seizure in a given patient is intimately linked to the seizure pattern. The seizure pattern includes the breed of the animal, its age at onset of seizures, the seizure type (focal, focal onset with generalization, or generalized) and, the frequency of seizures. A Doberman with seizures, regardless of the age, is suspicious for symptomatic epilepsy because seizures are so unusual in this breed. The same is not true for the Labrador retriever or the German shepherd dog. However, when one of these dogs is presented at 7 years of age with a first seizure, a diagnosis of idiopathic epilepsy cannot be given without serious consideration to structural causes of seizures. Idiopathic epilepsy should never be diagnosed in a dog older than 5 years, or younger than 6 months without a thorough diagnostic work up. Genetic/idiopathic epilepsy has been reported in Finnish Spitz dogs in which all seizures were focal seizures.3 This may occur with idiopathic epilepsy but is far from being the rule. In most cases, the seizures have a focal onset with rapid secondary generalization or, remain focal.4,5,6 First onset seizure presented as a cluster of seizures or status epilepticus, is usually not observed with idiopathic epilepsy. Symptomatic epilepsy is more commonly observed with clusters of focal seizures that progress over a few days to generalized seizures and/or status epilepticus. In dogs showing status epilepticus as the first manifestation of a seizure disorder, intoxication should always be considered.7

At our institution, the most frequent causes of seizures are, by order of frequency, ‘probably symptomatic epilepsies’, encephalitides and cerebral tumors. The most frequent tumor is the meningioma of the large breed dog.

DIAGNOSTIC WORK UP

For many veterinarians, most dogs presented with recurrence of seizures end up with a diagnosis of idiopathic epilepsy if the blood work and the physical and neurological examinations are normal. A thorough diagnostic work up with MRI of the brain and CSF analysis is frequently perceived as not necessary by the veterinarian. This is unfortunately also the case for many of the retrospective studies published on the subject of idiopathic epilepsy despite the fact that focal seizures and seizures with a focal onset are the most common seizures in dogs. In all instances of focal seizures (with or without generalization), the seizure originates from a focus of abnormal cerebral parenchyma. Moreover, of the neurological tests targeting the cerebral hemispheres, the mental status is the most difficult to evaluate because it necessitates the owner’s ability to recognize mild to moderate but consistent behavioral
changes in their animal. This is particularly true with the epileptic dog. Clinical experience has shown that owners of dogs, with significant MRI cerebral lesions involving the frontal and temporal lobes, are poor at evaluating their pet’s behavior. Regardless of the dog’s age, it is wise to assume that the presence of focal seizures or seizures with focal onset harbors a focal cerebral lesion. Low field MRI is not ideal in the diagnosis of canine epilepsy, especially when the dog is described as mentally normal by the owner. Magnet strength is important as low field MRI may not succeed at demonstrating smaller lesions. It has been demonstrated that age at onset of seizures is not a good predictor of MRI results. 

Ideally, a diagnostic work up that includes physical, neurological and ophthalmological examinations, CBC, biochemical profile and urology, MRI of the brain and CSF analysis should be performed in any dog presented with recurrence of focal seizure or seizure with focal onset. If the preliminary results are suggestive of metabolic or neoplastic disease, thoracic radiographs (3 views) and abdominal ultrasound are added prior to anesthesia for neuroimaging. In inflammatory diseases, titers or PCR of infectious diseases encountered in the area are pursued as well. In most ‘probably symptomatic epileptic’ dogs, an endocrine panel including thyroid profile (T4 and TSH) and baseline cortisol is also requested.

When the criteria for the diagnosis of idiopathic epilepsy are applied stringently, one realizes that the syndrome is not as frequent as previously reported. Most dogs with recurrent seizures are affected with symptomatic or probably symptomatic epilepsy. There is benefit for the patient in categorizing its epilepsy as “probably symptomatic” when the seizure pattern detracts from idiopathic epilepsy because it forces the clinician to diagnostically reevaluate the patient every time he is presented to the office.

**THERAPEUTIC PLAN**

Treatment failure in a great majority of cases, results when the wrong diagnosis is posed, the wrong treatment given, the wrong antiepileptic drug (AED) chosen or, when the AED is used inadequately. The seizure pattern has a determining role not only in the establishment of the differential diagnosis but also in the therapeutic plan. In symptomatic epilepsy, the successful control of seizures is intimately associated to the successful treatment of the primary cause of seizures. As an example, the cerebral meningioma of a dog with seizures must be addressed to improve seizure control. In cases where surgery is not an option, an anti-inflammatory treatment such as low dose dexamethasone will be more successful at improving seizure frequency than antiepileptic treatment alone.

In all patients with idiopathic or probably symptomatic epilepsy, the primary treatment is the antiepileptic treatment. The choice of an antiepileptic drug (AED) is based on the type of seizures present, the general health and age of the animal, the family lifestyle and the cost of the drug. Focal seizures or seizures with focal onset are more likely to respond to treatment when the AED targets this type of seizures. It is also important to use the AED adequately measuring serum levels whenever available. By order of frequency, the AEDs used in the maintenance treatment of canine epilepsy include phenobarbital, potassium bromide, zonisamide (5mg/kg q12h when it is used as sole agent and 10/kg q12h if the dog receives simultaneously phenobarbital), levetiracetam (10mg/kg q8h), gabapentin (10mg/kg q8h) and clonazepam or clorazepate.

Veterinarians have somewhat neglected the quality of life of the epileptic patient. For years, the only antiepileptic drugs (AED)s available and used to treat canine epilepsy were phenobarbital (1912) and potassium bromide (1857). Despite the side effects of these drugs, in a recent survey, 82% of the respondents used a combination of phenobarbital and potassium bromide to manage refractory seizure disorders in dogs. However, today, there are other options and owners are willing to pay extra cost to avoid the side effects of the older agents. Although phenobarbital remains one of the most potent and most used AED in the treatment of canine epilepsy, as experience is gained with the newer AEDs, its use will gradually diminished. In this author opinion, potassium bromide should be restricted to dogs with intractable generalized epilepsy when everything else has failed. Its long half-life, the polyuria and polydipsia, and the polyphagia with weight gain that ensue render its use problematic. Levetiracetam is reported as mainly efficacious against focal seizures but its use for focal seizures with secondary generalization is increasing, and, in selected cases, has been successful. The only side effect reported is an occasional mild sedation at onset of treatment. To avoid the sedation often present with AEDs at onset of treatment, it is advised to initiate treatment gradually, treating once per day or giving half of the dose for a few days, if the seizure frequency allows it. Zonisamide has proven effective for generalized and focal seizures with occasional mild sedation. Gabapentin in the author’s experience has not been as effective in the treatment of refractory epilepsy as compared to the AEDs mentioned above. It is safe, has no know drug interaction and only mild to moderate sedation at onset of treatment. The benzodiazepines have been supplanted with newer more efficacious AEDs for maintenance therapy. Epileptic dogs frequently become refractory to this group of AEDs.

**COMPLICATIONS OF SEIZURES**

The most common deleterious complication in epilepsy is the development of status epilepticus (SE). A substantial percentage of idiopathic epilepsy (IE) dogs have episodes of SE. Dogs with greater body weights are more likely to have episodes of SE. Early appropriate seizure treatment does not appear to decrease the risk for dogs to have episodes of SE. Premature death is also reported in dogs with epilepsy with survival time
being shorter for dogs that experience episodes of SE.¹⁴

The most frequent complications encountered in epileptic dogs relate to the antiepileptic treatment. The phenobarbital-induced hepatotoxicity, once quite common, is now rarely observed. Veterinarians and owners are well aware of the necessity to follow the AED levels and liver biochemical profile.¹³ The use of phenobarbital and/or KBr increases the probability of pancreatitis.

The most deleterious and insidious effect of the use of phenobarbital and/or potassium bromide is the tremendous weight gain that occurs in the first few months of treatment in most dogs. The weight gain has detrimental effect on the joints with secondary cruciate rupture, ataxia, somnolence, lethargy, and fatigue.

CONCLUSION

When the criteria are used stringently, idiopathic epilepsy is not as common as previously observed at least in this author’s opinion. It is important to encourage the diagnostic work up of dogs with focal seizures or seizures with focal onset. Many of the newer AEDs are efficacious in the treatment of canine epilepsy without the deleterious effects associated with the use of the older agents, phenobarbital and KBr.

REFERENCES

What’s New in Feline Epilepsy

Joane Parent, DMV, MVetSc, ACVIM Neurology

INTRODUCTION

The symptoms and causes of seizures in the cat have little comparable to that of the dog. Most cats with seizure activity have symptomatic or probably symptomatic epilepsy. To ease understanding of this presentation, some important definitions are given. In the context herein, the epilepsy is **idiopathic** when “is only epilepsy, with no underlying structural brain lesion or other neurological signs or symptoms; idiopathic epilepsy is presumed genetic and usually age dependant”. The epilepsy is **symptomatic** when “the epileptic seizures are the result of one or more identifiable structural lesions of the brain”. The epilepsy is **probably symptomatic** when “the epilepsy is believed to likely be symptomatic but no etiology has been identified”. Keeping in mind these definitions, most cats with recurrent seizures have symptomatic or probably symptomatic epilepsy.

CAUSES OF SEIZURES

The history, signalment and seizure pattern are the most important elements toward establishment of the differential diagnosis. Most seizures in the cat are intracranial in origin. Rare are the seizures resulting from toxicities or metabolic diseases. In a descriptive study of cats with seizures, polycythemia was the only metabolic disease reported as a cause of seizures. In these cats, the cause of seizures was not the metabolic effect of the disease per se, but the vascular events that resulted from the blood hyperviscosity. In the same study, the most common causes of seizures were the viral encephalitides.

The viral non-feline infectious peritonitis (FIP) encephalitides are common causes of seizures in the young to middle-aged cat. The seizures may be preceded by mild unspecific transient systemic signs such as fever, anorexia, cough, vomiting, and diarrhea up to three weeks prior to the onset of the seizures with the animal being clinically normal by the time the first seizure is observed. The seizure onset is acute and frequently progresses rapidly over a few days. The disease is often self-limited but the resulting lesion may be highly epileptogenic in reason of its location, with frequent focal and/or generalized tonic-clonic seizures occurring as clusters and status epilepticus. On the biochemical profile, there is often a marked increase in creatine kinase (CK) due to the constant muscle tremors/shaking these cats experience.

Feline infectious peritonitis is likely if the cat is less than three years of age, has a protracted history, systemic signs and neurological disease. In a study of cats with confirmed CNS FIP, 14/55 cats had seizures. The cats with seizures were more likely to have extensive forebrain inflammation suggesting that the presence of seizures in FIP may indicate an unfavorable prognosis. Central nervous system infection with Cryptococcus has similar presentation but is observed in a wider range of age. These infectious diseases are rare compared to the viral non-FIP encephalitides. Feline leukemia virus (FeLV), feline immunodeficiency virus (FIV) and toxoplasma organism are rare causes of neurological disease in the cat.

Ischemic encephalopathy may also lead to epilepsy in the cat but the seizures may be infrequent and may not necessitate antiepileptic treatment. The outcome is usually good if the animal survives the initial cerebral ischemia. Strangely, a few cats never

SEIZURE PATTERN

The most frequent types of seizures in the cat are the focal seizures, with or without secondary generalization. The seizure is classified as focal when there is no loss of consciousness even though the mental status may be altered. Focal seizures may be violent, the animal propelling itself in the air, running and colliding with objects, biting its tongue, avulsing its claws, etc. The focal seizures may also be subtle and not recognized by the owners as seizure activity. In these cases, there is frequently repetitive ear, eyelid or whisker fluttering and twitching. Occasionally, cats admitted to the intensive care unit with on-going seizure activity continue to seize even though they seem deeply sedated by the antiepileptic treatment; when touched, their muscle mass is vibrating with activity. The muscular necrosis can be marked in these patients (creatine kinase > 50,000U/L). With generalized seizures, there is loss of consciousness; in most cases the seizures are convulsive, with tonic-clonic movements, salivation and urination.

The seizure pattern plays a crucial role in establishing the differential diagnosis and in orienting the antiepileptic treatment. The seizure pattern includes (1) the age of the patient at onset of seizures, (2) the seizure type (focal, generalized, or focal with secondary generalization) and (3) the seizure frequency.
display signs characteristic of feline cerebral ischemia but are presented later on with recurrent seizure activity. On these cats, the presumptive diagnosis is made on magnetic resonance imaging (MRI) of the brain demonstrating bilateral but asymmetrical atrophy of the cerebrum. Many metabolic/endocrine diseases in cats (renal failure, diabetes mellitus, and hyperthyroidism) cause hypertension; however, for the frequency of these diseases in the aged cat, ischemic events are comparatively rare findings.

Brain tumors, especially meningiomas, are frequent causes of seizures in the cat > 10 years of age. In all, there is a behavioral component that often, unfortunately, is unnoticed by veterinarians and misinterpreted by owners as “old age”. Meningiomas are relatively easy to remove surgically. Recurrences are frequent especially if the removal is not complete.

Young cats (6–12 months) that develop recurrent seizures and in which the neurological examination and ancillary tests (MRI, cerebrospinal fluid analysis) are unremarkable often have epilepsy that becomes intractable. A retrospective study has reported however that those young epileptic cats live generally longer.5

**DIAGNOSTIC WORK-UP**

There is benefit to the patient into categorizing its epilepsy as “probably symptomatic” when the seizure pattern detracts from idiopathic epilepsy and, when a thorough diagnostic work up has failed to identify a cause for the seizures, because it forces the clinician to diagnostically reevaluate the patient every single time the patient is presented to the office. The diagnostic work up advocated in all cats with seizures includes: physical, neurological and funduscopic examinations, CBC, biochemical profile (with CK and T4 measurements), FeLV, FIV and urinalysis, thoracic radiographs (3 views) and abdominal ultrasound, and, magnetic resonance imaging of the brain and cerebrospinal fluid analysis. The FeLV and FIV tests are done, not to eliminate causes of seizures, but as part of the patient’s general health evaluation. Note that when the neurological examination is done, the mental status, response to menacing gesture, response to nasal septum stimulation and proprioceptive positioning are the tests that target the cerebral hemispheres. They must be done with attention to details especially the questioning of the owners regarding the cat’s mental status.

**TREATMENT**

Treatment failure in a great majority of cases results when the wrong diagnosis is posed, the wrong treatment applied, the wrong antiepileptic drug (AED) chosen or, when the AED is used inadequately. The biggest deterrent to an improved seizure control is the overt diagnosis of idiopathic epilepsy. Treating the seizures alone in a cat with encephalitis or tumour is doomed to fail. As an example, the seizures in a cat with cerebral meningioma are more likely to be controlled if the peri-tumoral oedema is addressed. Moreover, the choice of AED is based on the type of seizures present. Focal seizures or seizures with focal onset are more likely to respond to treatment when an AED that targets this type of seizures is used. Focal seizures can be very difficult to abate. It is also important to use the AED adequately measuring serum levels whenever available. Although guidelines exist, the antiepileptic treatment must be customized to the patient and to the owner’s lifestyle.

There are many AEDs available to treat seizures in cats. It includes phenobarbital, gabapentin, levetiracetam, and zonisamide. As a rule, if the seizure frequency allows it, it is preferable to introduce the AED gradually to avoid overt sedation.

**Phenobarbital** remains the first choice because it is well tolerated in this species and the serum level measurement is readily available in most commercial laboratories. The drug is used in the treatment of focal and generalized seizures. The optimal serum therapeutic concentration is 100-130 µmol/L. The dosage is not calculated by weight but by cat. Some cats require 7.5mg q12h and a few 15mg q12h, but most cats necessitate 22.5mg/day, given in two treatments (15mg and 7.5 mg), to reach optimal serum concentration. The sedation is present in the early stage as observed in the dog but the hepatotoxicity, the most concerning side effect in dogs, is not a problem in the cat. Usually, there is no polyuria, polydipsia or polyphagia, although possible.

**Gabapentin** is used in the treatment of focal seizures and seizures with focal onset. Its use is safe and well tolerated in cats. The dosage used is 10 to 40 mg q12h to q8h per cat. In Canada, the commercial drug is not available in concentrations smaller than 100mg capsule necessitating compounding. There is sedation in the early stage of treatment. This author uses gabapentin as second line AED in the cat.

**Levetiracetam** is used in the treatment of focal seizures and seizures with focal onset. It seems to be well tolerated in cats but our experience with the use of this drug in this species is still limited. The dosage advocated is 20 mg/kg orally every 8 hours to reach plasma concentrations within the therapeutic range established for humans.6

**Zonisamide** is used for focal and generalized seizures. There is still limited experience with this drug in cats. The dosage advocated is 5 to 10mg/kg once daily per os.7 Diazepam, a long time first or second AED of choice has been relegated to the treatment of refractory feline epileptic patients in reason of the 17 cases of acute hepatic necrosis and liver failure reported in the 90s.8, 9 Potassium bromide should not be used in the cat in reason of the life threatening lung disease it may cause.10
PROGNOSIS

Contrary to the dog, the severity of seizures at onset of disease does not seem to have a determining effect on outcome. Indeed, it is not rare that a cat, severely epileptic secondary to encephalitis or ischemic encephalopathy, stops to seize once treatment is applied. This is possibly related to the frequent self-limiting causes of seizures in the cat.

When the seizures are the result of an acquired cause, the antiepileptic treatment is continued for 6 months seizure-free, then very gradually the patient is weaned off treatment.

CONCLUSION

Feline epilepsy is usually intracranial in origin. The epilepsy in most cases is symptomatic or probably symptomatic. For this reason, a thorough diagnostic work up including MRI of the brain and CSF analysis is advocated in all cats with seizures. It is only once a final diagnosis has been posed that the appropriate treatment can be applied. If the epilepsy is symptomatic, it is crucial to treat the primary cause to optimize seizure control. Among the AEDs available, phenobarbital and gabapentin are the treatments of choice. The prognosis for arrest of seizures in the cat is superior to what is observed in dogs.

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The Neurological Examination of the Geriatric Dog

Joane Parent, DMV, MVetSc, ACVIM Neurology

As in humans, dogs have an increased incidence of problems as they age. No system is immune to aging and the neurological system makes no exception. The neurological examination of the older dog is challenging in reason of the concomitant presence of other diseases. When performing the neurological examination of the older dog, there are abnormalities observed that are non-neurological in origin but do have an impact on the test results, others that are neurological but do not relate to the presenting problem, others that relate directly to the presenting neurological problem, and finally abnormalities that are indicators of significant neurological illness but that go unnoticed due to poor technique or interpretation.

Non-neurological abnormalities that impact on the results of the neurological tests

There are multiple non-neurological abnormalities in the older dog that may affect the results of the neurological tests.

- The menace responses may be poor to absent secondary to cataract formation.
- The pupils may be asymmetric and the pupillary light reflexes poor to absent due to iris atrophy or cataract formation.
- The responses to the menace and nasal septum stimulation may be poor to absent bilaterally in the frightened animal. These tests evaluate the cerebrum and consequently are influenced by an altered mental status. The abnormalities should be bilateral and symmetrical in these instances.
- Bilateral and symmetric masticator muscle atrophy in the older dog is frequent and often an indication for presence of an on-going systemic disease or chronic illness, which may or not be associated with the primary neurological problem. The atrophy is secondary to muscle abnormality and not neurological in origin. In this case, there is dropped jaw.
- Musculo-skeletal diseases have an enormous impact on the animal’s gait and posture, postural reactions and spinal reflexes. The older large breed dog is always a challenge because of the orthopaedic and neurological problems that often co-exist. The gait associated with orthopaedic diseases such as hip dysplasia, degenerative joint disease, chronic arthritis secondary to cruciate rupture/repair and fibrotic myopathy must be differentiated from the gait associated with neurological diseases such as lumbosacral stenosis, intervertebral disc disease and degenerative (radiculo)myelopathy.
- Generalized or musculo-skeletal weakness and pain from joint diseases frequently lead to weak withdrawal reflexes making the objective evaluation of the flexor reflexes difficult. The reflex may be weak, but it must be complete, i.e., each articulation must flex.
- While the flexor reflex is the most difficult reflex to interpret, the patellar reflex is the most difficult to elicit. If you cannot obtain a patellar reflex, ensure the position of the limb is appropriate and the technique correct. If the reflex is truly absent, evaluate if the lesion involves the sensory and/or the motor part of the nerve. The motor part of the femoral nerve is responsible for weight bearing of the hind limb. If the lesion is motor in nature, the animal should have difficulty standing since the femoral nerve is responsible for the extension and fixation of the stifle. If the animal is walking, the motor part of the reflex is present. In this case, if the patellar reflex is absent, the lesion is then at the level of the ascending (sensory) pathways, or dorsal roots.
- Back pain may be caused by benign arthritis of the vertebral facets. In these instances, the owner is not aware that the animal has back pain. This is an incidental finding.
- An abnormal tail carriage may be secondary to perianal fistulas.
Neurological abnormalities that do not relate to the presenting problem

Older dogs may have had other neurological illnesses in the past that may, or not, have been recognised by the owners. It stresses the importance of good history taking. The neurological examination must always be done with attention to details. In the old dog, many abnormalities are often disclosed.

- Cognitive impairment secondary to senile degenerative cerebral changes is often present in the older dog.
- The menace responses and the pupillary light reflexes may be absent secondary to progressive retinal atrophy.
- Idiopathic facial paralysis is the most common disease entity affecting the cranial nerves. There may be presence of an old facial nerve paresis/paralysis. Following paralysis, facial muscular fibrosis ensues causing, over months, a progressive natural tuck up and mild deviation of the facial mimic on the affected side. The normal side becomes the droopy side. The affected side is diagnosed by gently passing a finger on the eyelids. On the affected side, the eyelid closure may be incomplete, or the eyelids appear closing because there is a rapid retraction of the eyeball (cranial nerve VI) and subsequent protrusion of the third eyelid.
- Many old dogs have suffered an episode of geriatric vestibular disease which has left them with a permanent ipsilateral head tilt.
- A laryngeal paresis may be present. This is more prevalent in the large breed dog. It is abnormal to hear a dog breath during examination. In cases of laryngeal paresis, the breathing is noisy.
- Some older dogs, especially small breed dogs, have neurological hind limb gait. The hind limbs do not advance at the same pace or speed then the front limbs. There is a superposed automatism. This is characteristic of chronic thoracolumbar spinal cord compression, usually secondary to intervertebral disc disease in the small breeds of dogs. In these instances, the animal has a history of on-going bouts of back pain +/- hind limb incoordination.
- Older Doberman dogs with cervical spondylomyelopathy (CSM) may walk with toed-in front paws. As CSM becomes clinical, the posture changes to assume a more wide-based stance by rotating the elbows outwardly. Hind limb ataxia is often subtle in these cases but nevertheless present.
- Back pain may be secondary to facet arthritis and not necessarily to nerve root compression or bony destruction/infiltration. Lumbosacral pain is common in the older large dog. When the problem is significant, the dog often cannot posture for longer period to defecate and ends up walking while doing so toward the end of the bowel movement. When evaluating for back pain, it is important to evaluate down to the lumbosacral region.

Neurological abnormalities that directly relate to the presenting neurological problem (frequent neurological diseases of the older dog)

- Abnormal behaviour, circling, seizure activity: thalamocortical tumours
- Head tilt, nystagmus, vestibular ataxia: geriatric vestibular disease
- Degenerative (radiculo)myelopathy of the older German Shepherd dog, German shepherd mix, boxer, Welsh corgi
- Acquired lumbosacral syndrome of the middle age to older large breed dog.

Neurological abnormalities that go unnoticed due to incomplete examination or inadequate interpretation

It is important when performing a neurological examination to follow and fill a neurological form. It ensures that every test is done. It forces the examiner to critically evaluate each of the neurological aspect of the animal even if at first sight, the function appeared normal. The examination must be done with attention to details looking for subtle but consistent changes.

- The veterinarian must be proactive in the domain of cognitive dysfunction if he/she is to make progress in its recognition and treatment. Veterinarians frequently assume that the behavior observed in the examination room is the behavior experienced in the home environment. Pets are submitted to an adrenaline surge when presented to veterinary clinics. The behavior displayed is rarely representative of what is present in the home environment. One cannot stress enough the necessity of a thorough history to evaluate the animal’s mental status. This is by far the most time-consuming part of the examination. History taking is difficult because owners
anthropomorphize. The more educated the owner, the more difficult it is sometimes to separate what truly is from what is perceived. The senile dog is interpreted as deaf because not greeting the owner at the door, blind because colliding with objects, anxious and fearful because pacing at night, stubborn because “ignoring” the call, pensive because staring at walls, etc. These behavioral changes are often NOT the reason why the owners present their animal to the veterinarian.

- When presented with a cranial nerve deficit, it is important to question the family for presence of somnolence, or lack of activity when the animal is not stimulated.

- The pupils of a dog are normally dilated and slow to respond to a light source in the clinical setting due to fear and anxiety. Beware of the small pupil size in the older dog presented with a complaint of abnormal behavior of a few weeks in duration. There may be impending brain herniation.

- It is important to perform a neurological examination in the dog diagnosed with pituitary dependant hyperadrenocorticism. Not only must the behavior be critically assessed but the pupillary light reflexes as well. If there is presence of a macroadenoma of the pituitary gland, there may be compression of the optic chiasm which is situated closely to the pituitary gland. The pupils in this case are dilated but a stronger than usual light source is necessary to cause a constriction of the pupils.

- When evaluating the nasal septum responses, a cotton swab is used to stimulate alternatively the septum on each side. Blindfolding the animal, this must be done slowly and gently starting with the stimulation of the whiskers and external nostrils, progressing if need be to the nasal septum. The emphasis is on the presence of a consistent asymmetry between sides. The presence of an asymmetry is indicative of ascending (sensory) pathway involvement, or along the ipsilateral trigeminal nerve, or at the level of the contralateral thalamocortex.

- When evaluating the facial nerve (CN VII), it is important to look for complete lid closure and strength of closure. The palpebral closure should be complete at the first gentle touch of the eyelids. In dogs having facial nerve paresis, a gentle touch on the upper and lower lids causes the third eyelid to rapidly protrude to protect the eye instead of causing a closure of the eyelids. The palpebral fissure is already small from the upper lid droop and when the eyeball retracts, it gives the impression that there is eyelid closure. If this is bilateral, it may be the first and only sign of polyneuropathy. If present, a thorough tumor search should be done as part of the diagnostic work up to eliminate a paraneoplastic polyneuropathy.

- It is abnormal to hear the breathing of a dog in the examining room. If the respiration is noisy, there is likely recurrent laryngeal nerve paresis. This may be idiopathic, the result of a metabolic disease, or a sign of a paraneoplastic polyneuropathy. The onset of laryngeal paresis is frequently insidious. The problem goes unnoticed until a life-threatening emergency arises on a hot and humid summer day.

- The clinician may choose to ignore an absence of patellar reflexes because of a lack of lower motor neuron deficits in an old dog. This however indicates the presence of a sensory polyneuropathy. A paraneoplastic polyneuropathy must be ruled out before concluding at an incidental finding.
Hind Limb Weakness in the Large Dog

The older large dog presented with hind limb weakness is a challenge that deserves more attention. The observation of the gait and posture, the postural reactions and the spinal reflexes must be correctly performed and interpreted, and specific diseases ruled out by an elimination process.

The nervous system is composed of the peripheral and the central nervous system, the latter including the thalamocortex, brainstem and spinal cord. The clinical signs observed with diseases of the peripheral nervous system include sensory (proprioceptive deficits without ataxia, and hyper-, hypo-, or anaesthesia) and motor deficits (paresis or paralysis, hypo- or atonia, hypo- or areflexia and neurogenic atrophy). The clinical signs with diseases of the spinal cord include sensory (ataxia with or without proprioceptive positioning abnormalities and hyper-, hypo-, or anaesthesia) and motor deficits (paresis to paralysis, normal to hyperreflexia, normal to hypertonia and disuse atrophy). In peripheral nervous system diseases, there is no ataxia whereas ataxia is the hallmark of spinal cord disease. The gait must be observed carefully on a non-slippery surface and in a large enough space to allow free movements of the animal.

The proprioceptive pathways include the proprioceptors located in the muscles, tendons and joints, the ascending fibres in the peripheral nerve, the ascending proprioceptive pathways within the spinal cord, and, the reflex proprioceptive fibres (spino-cerebellar) that terminate in the cerebellum and conscious proprioceptive pathways that travel along the brainstem to end in the cerebral cortex. Anywhere along this path, a lesion may potentially lead to a paw that knuckles over. The proprioceptive positioning (knuckling of the paw) must be done carefully. It is important to stimulate as a few proprioceptors as possible to enhance subtle deficits. It is the association of other neurological abnormalities that indicate the location of the lesion and if there is a neurological lesion. If the lesion is in the peripheral nervous system, the reflexes should be decreased and there should be NO ataxia. If the lesion is in the spinal cord, there should be ataxia and weakness. If the lesion is in the brainstem, there is accompanying somnolence and cranial nerve deficits. If the lesion is unilateral in one cerebral hemisphere, there are accompanying behavioural changes while the gait usually remains normal. In cortical diseases, the proprioceptive deficits involved the contralateral limbs and the gait in most cases remains unchanged.

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Equine Cushing’s Disease: Pathogenesis and Diagnosis

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Although the frequency of diagnosis and treatment of pituitary pars intermedia dysfunction (PPID) in horses has clearly increased over the past decade, there is no evidence that the prevalence of PPID is actually increasing. Increased recognition of the disease is likely a consequence of clients maintaining their horses to more advanced ages as well as improved health care (e.g., diet and dentistry) being provided to older horses. A recent survey of horse owners in Queensland, Australia revealed a prevalence of 15-20% of PPID in horses and ponies 15 years of age and older. There is no gender predilection and average age of affected horses is around 20 years. All breeds and types of equids can be affected with PPID but Morgan horses and ponies appear to be at greater risk (Figure 1).

Pathophysiology:

In humans and dogs, Cushing’s disease is most commonly attributed to a corticotroph adenoma in the pars distalis of the pituitary gland. These adenomas are thought to arise spontaneously. In contrast, Cushing’s disease in horses is almost exclusively attributed to hyperplasia or adenoma formation in the pars intermedia that appears to be due to loss of hypothalamic innervation. Abnormal pars intermedia tissue in horses contains markedly reduced amounts of dopamine, about 10% that of normal pars intermedia tissue, consistent with a specific loss of hypothalamic dopaminergic innervation. Recent evidence suggests that this loss of dopaminergic innervation is due to oxidant-induced injury to hypothalamic tissue. Thus, a risk factor for affected horses may be reduced anti-oxidant defense mechanisms in neural tissue. Further, insoluble aggregates of the neural protein α-synuclein have been found in dopaminergic nerve terminals of PPID-affected horses. These protein aggregates are also found in humans with Parkinson’s disease suggesting that the two neurodegenerative disorders may share a similar pathogenesis. However, the population of neurons affected in horses, as compared to humans, appears to be somewhat different leading to the difference in clinical signs observed in each species.

Abnormal pars intermedia cells produce excessive amounts of pro-opiomelanocortin (POMC) and a number of POMC-derived peptides including adrenocorticotropic (ACTH). Also unlike Cushing’s disease in humans and dogs, adrenocortical hyperplasia accompanying equine Cushing’s disease is relatively uncommon, occurring in ~20% of affected horses. These differences in location and pathophysiology between human, canine, and equine pituitary adenomas have lead several authors to suggest that the disease in horses should not be called equine Cushing’s disease; rather, pituitary pars intermedia dysfunction (PPID) has been advanced as a more appropriate descriptor.

Figure 1.

Pituitary pars intermedia dysfunction (PPID) affects equids of all sizes and breeds although it may be more common in ponies (image courtesy of Dr. J. H. van der Kolk, Utrecht, Netherlands).
Clinical signs:

The classic clinical sign of PPID in horses is hirsutism, a long and curly hair coat that fails to shed. In some affected horses, coat color changes have also been observed (Figure 2, left). The pathogenesis of hirsutism, which is characterized by arrest of hair follicles in telogen, remains unclear. Hyperhidrosis is also observed in up to two-thirds of horses with PPID, most commonly over the neck and shoulder areas, and has been attributed to a thermoregulatory response to the long hair coat. Weight loss and lethargy, or poor performance, are also commonly observed in horses with PPID. In addition to true weight loss, protein catabolism due to increased cortisol activity leads to loss of muscle mass. This is most notable in advanced cases as a loss of epaxial and rump musculature. Despite weight loss, appetite in affected horses is normal or even increased (polypagia). However, dental abnormalities, leading to painful mastication and quidding, may compromise feed intake and contribute to weight loss in some horses. Combined with, or often preceding, loss of muscle mass is deposition of fat along the crest of the neck, over the tail head, and in the sheath of male horses. Another area where abnormal fat deposition may occur is above and behind the eyes (supraorbital area, Figure 2, center). Horses with PPID have also been described as overly docile and more tolerant of pain than normal horses. The latter signs have been attributed to increased plasma and cerebrospinal fluid concentrations of β-endorphin that are 60- and more than 100-fold greater, respectively, in horses with PPID than in normal horses.

Chronic, insidious-onset laminitis is perhaps the major clinical complication of PPID with more than 50% of horses affected in most reports. Although the condition is more amenable to management in ponies due to their lower body weight, chronic or recurrent pain with exacerbation of laminitis or associated foot abscesses is often the reason for euthanasia. Polydipsia and polyuria (PU/PD) develops in about one-third of horses with PPID. Equids with PPID tend to have delayed wound healing and are frequently affected with secondary infections. Commonly recognized infections include skin infections (e.g., refractory “scratches” and fistulous tracts), recurrent subcorial abscesses, conjunctivitis, sinusitis (Figure 2, right), gingivitis, alveolar periostitis, and bronchopneumonia.

Other signs that have been reported in horses with PPID include persistent mammary secretions and infertility. Central nervous system (CNS) dysfunction, including ataxia, blindness, and seizure-like activity, are occasionally observed in equids with PPID. A major complication of hypercortisolism in affected human patients is osteoporosis.

Clinicopathologic findings

Abnormal laboratory data in horses with PPID may include mild anemia, an absolute or relative neutrophilia, and an absolute or relative lymphopenia. Although one or more of these abnormalities is usually found in a third or more of equids afflicted with PPID, the true prevalence is not well documented. As well as being increased in number, neutrophils in affected animals may appear hypersegmented. This finding reflects maturity of neutrophils and can be attributed to a longer half-life of circulating neutrophils because cortisol excess limits diapedesis from the vasculature. Eosinopenia is also recognized in human and canine patients with hyperadrenocorticism but is difficult to document in horses because equids typically have a low numbers of circulating eosinophils. The most common abnormality detected on serum biochemical evaluation is mild to moderate hyperglycemia, reported in 25-75% of cases, depending on the upper end of the reference range used. Additional abnormal biochemical findings may include elevations in liver enzyme activities, hypercholesterolemia, and hypertriglyceridemia.

Diagnosis

Practically, the diagnosis of PPID is most commonly made by observation of hirsutism and other clinical signs in older equids. However, establishing a diagnosis of PPID in less severely affected horses can be challenging. As a result, a number of endocrinologic tests have been used to evaluate horses with suspected PPID.

Plasma cortisol concentration and loss of diurnal cortisol rhythm. Resting cortisol concentration does not routinely exceed the upper end of the reference range in horses with PPID. Thus, measurement of plasma cortisol concentration alone is not a valid diagnostic test. Because plasma cortisol concentration has a diurnal rhythm of secretion, with an increase in the morning hours and a nadir around midnight (that should be at least 30% lower than morning values),
loss of the diurnal rhythm has been advanced as a screening tool for evaluation of horses with suspected PPID. However, the effects of external stressors and disease on plasma cortisol concentration, not to mention collection of the evening sample at a late hour, makes loss of cortisol rhythmicity a poor screening tool for PPID.

Dexamethasone suppression test

The overnight dexamethasone suppression test (DST) is considered by many equine clinicians to be the “gold standard” endocrinologic test to support of a diagnosis of PPID. However, this statement is not without controversy and there is concern, although poorly documented, that administration of dexamethasone may induce or exacerbate laminitis in PPID-affected equids. In its most simple form, the overnight DST consists of measuring cortisol in the late afternoon (typically 5 pm) followed by administration of dexamethasone (40 µg/kg, IM = 20 mg to a 500 kg horse) and subsequently measuring plasma cortisol concentration 17 to 19 hours later (between 10 am and noon the following day) (Figure 3). The major limitation of the overnight DST for ambulatory practitioners is that it requires two visits to the horse. However, considering the fact that the most important value is the cortisol concentration following dexamethasone administration, the overnight DST can be simplified by dispensing dexamethasone to the client for administration and limiting the test to one visit the following morning. When using this test, it is probably wise to consider dexamethasone as a “sledgehammer” in terms of feedback to the hypothalamic-pituitary axis. In other words, failure of dexamethasone to induce suppression of circulating endogenous cortisol concentration is strongly supportive of PPID. However, the overnight DST may be less effective in diagnosis of PPID in the earlier stages of the disease process. In this clinician’s opinion, this is not an important limitation of the test because in the earlier stages of PPID, when DST results may be normal (not supportive of PPID), it may be difficult to justify treatments other than body clipping to limit hirsutism (unless laminitis is the primary clinical problem for which treatment with pergolide can be use on a “trial and error” basis).

Figure 3.

Overnight dexamethasone suppression test (DST) results in 43 horses with pituitary pars intermedia dysfunction (PPID) confirmed at necropsy and 18 non-PPID horses. Endogenous cortisol was measured prior to dexamethasone administration (40 µg/kg, IM) and again 15 and 19 hours later. Only 2 of 43 PPID-affected horses had an endogenous cortisol concentration <1.0 µg/dL (≈30 pmol/L, dashed line) at 15 hours and all 43 horses had an endogenous cortisol concentration >1.0 µg/dL at 19 hours. In contrast, all 18 non-PPID horses had suppression of endogenous cortisol concentration to <1.0 µg/dL at both 15 and 19 hours. (adapted from Dybdal NO, Hargreaves KM, Madigan JE, et al. Diagnostic testing for pituitary pars intermedia dysfunction in horses. J Am Vet Med Assoc 1994;204:627).

Another limitation of using the DST is that seasonal variation can affect results. In a recent study of horses and ponies without clinical signs of PPID, abnormal DST results were found in 10 of 39 equids in September. To further examine the effect of season on DST results, the author performed the overnight DST monthly for a year in a group of 18 aged horses (>19 years) without clinical signs of PPID. Seven of 18 horses had normal overnight DST results throughout the year while 11 horses had overnight DST results supportive of PPID from 1 to 9 months of the year. Test results from late July through late October were most commonly affected by seasonal variation (Figure 4). Thus, the overnight DST is best performed from December through June and overnight DST results from July through November, if abnormal, should be interpreted with caution. Although the author prefers not to perform the test during these months, it warrants emphasis that normal overnight DST results during late summer to fall are valid and can be useful in case assessment. A further observation in the author’s study that warrants mention is that no signs of laminitis were induced in this group of older horses during performance of 216 overnight DSTs.
Plasma ACTH concentration. Horses with PPID have excessive amounts of ACTH in abnormal pars intermedia tissue and increased amounts are released into plasma. Thus, plasma ACTH concentration would seem a likely choice for a single sample test to support a diagnosis of PPID. In fact, increased plasma ACTH concentrations, with a maximum reported value exceeding 12,000 pg/ml, have been documented in several reports of PPID in equids. Further, ACTH concentrations exceeding 27 or 50 pg/ml (~6 and ~11 pmol/l) in ponies and horses, respectively, have been reported to have a high sensitivity for diagnosis of PPID. Limitations of using plasma ACTH concentration as the only endocrinologic test to support a diagnosis of PPID are that sample handling can be problematic and that different laboratories may use different assays for measuring ACTH. Because ACTH can be adsorbed onto glass and can be degraded by proteolytic enzymes in both whole blood and plasma, collection of blood into plastic tubes, rapid separation from red cells, and freezing of plasma prior to shipment for analysis has been recommended. Practitioners interested in using ACTH concentration as a diagnostic aid should contact the testing laboratory prior to sample collection for sample handling recommendations and should only send samples to a laboratory using an assay that has been validated as specific for ACTH in equine plasma. Another limitation of using plasma ACTH concentration is seasonal variation in test results. In normal ponies and horses without signs of PPID, plasma ACTH concentrations measured in September were above the threshold for diagnosis of PPID. Finally, ACTH is released in a pulsatile fashion from the pituitary gland. Consequently, plasma ACTH concentration can vary considerably over the day such that the absolute elevation in ACTH may not be all that useful for monitoring disease improvement with treatment. These limitations complicate use of plasma ACTH concentration as the sole endocrinologic test for both diagnosis and monitoring response to treatment of PPID.

Thyrotropin stimulation test and combined dexamethasone suppression/thyrotropin stimulation test. Thyrotropin (TRH) is a releasing hormone for several pituitary hormones that has been shown to increase plasma cortisol concentration when administered to horses and ponies with PPID. Although the TRH stimulation test has not been as well validated as the overnight DST, it has been advocated for use in horses with laminitis because of concerns about exacerbating foot pain following dexamethasone administration. When used, a 50% increase in cortisol concentration between 15 and 90 minutes after administration of TRH can be supportive of a diagnosis of PPID. However, interpretation of the response is complicated by considerable variability of the initial cortisol concentration as well as the problem that up to 50% of normal horses may have a false-positive result with this test. In an attempt to overcome these problems with this test, a combined DST/TRH stimulation test has been developed. Three hours prior to TRH administration, dexamethasone (40 µg/kg) is administered to suppress cortisol concentration to similar values in both PPID-affected and normal horses. Cortisol concentration is subsequently measured before and 30 minutes after TRH administration and equids with PPID show a greater increase in comparison to normal animals. After 24 hours, plasma cortisol concentration remains suppressed in normal horses while it returns to the basal (pre-dexamethasone) concentration in PPID affected horses (Figure 5). Although this combined test improves the accuracy of the TRH stimulation test, it is both more expensive for the client as well as less practical for the ambulatory clinician than the overnight DST. As a consequence, this combined test has not been widely used.

The combined dexamethasone suppression test (DST) / thyrotropin (TRH) stimulation test involves administration of dexamethasone (40 µg/kg, IM) at time 0 followed by administration of TRH (1 mg, IV) 3 hours later. Support for a diagnosis of pituitary pars intermedia dysfunction is provided by detection of either a >66% increase in endogenous cortisol concentration 30 min after TRH administration or an endogenous cortisol concentration >1.0 µg/dL (>10 ng/mL) 24 hours after dexamethasone administration (adapted from Frank N, Andrews FM, Sommardahl CS, et al. Evaluation of the combined dexamethasone suppression/thyrotropin-releasing hormone stimulation test for detection of pars intermedia pituitary adenomas in horses. J Vet Intern Med 2006;20:987).
Domperidone stimulation test. The most recent endocrinologic test developed for diagnosis of PPID is a provocative test utilizing administration of domperidone, a dopamine receptor antagonist. In theory, this drug should exacerbate the loss of dopaminergic inhibition in horses with PPID and thereby increase release of endogenous ACTH by pars intermedia melanotropes. To test this hypothesis, plasma ACTH concentration was determined in 33 horses with or without clinical signs of PPID prior to and 4 and 8 hours after oral administration of domperidone (3.3 mg/kg). After testing horses were euthanized for histopathological examination of the pituitary glands. In this study, plasma ACTH concentration increased modestly (by about 50%) in horses without clinical signs of PPID or significant pars intermedia pathology while plasma ACTH concentration more than doubled in horses with clinical signs of PPID and more advanced pars intermedia histopathologic abnormalities. Unfortunately, the domperidone challenge test was performed in the late summer and fall in some horses leading to seasonal variation as a possible confounding factor. Nevertheless, this novel test may offer promise of detection of PPID in the earlier stages of the disease and further investigation is warranted.

Serum insulin concentration. Some equids with PPID, especially ponies, may have insulin resistance. As a consequence, an elevated fasting serum insulin concentration could support a diagnosis of PPID. However, hyperinsulinemia can also accompany the “equine metabolic syndrome”. Thus, use of serum insulin concentration alone for diagnosis of PPID can be misleading because hyperinsulinemia is not specific to PPID. However, measurement of fasting insulin concentration may be of benefit in the initial evaluation of equids with suspected PPID because one case series found poorer survival in PPID-affected equids with hyperinsulinemia as compared to PPID equids with a normal insulin concentration.

Pathophysiology


Diagnostic testing


Equine Cushing’s Disease: Approach to Management

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Management of pituitary pars intermedia dysfunction (PPID) in equids consists of improved husbandry, including adequate nutrition and limiting competition for feed, body-clipping, dentistry, and appropriate treatment of concurrent medical problems. In addition, specific treatment with the dopamine agonist pergolide can improve quality of life and reverse many clinical signs of the disease in PPID-affected equids. Combination treatment with both pergolide and cyproheptadine, in the author’s experience, may also prove beneficial in more advanced cases. For patients with chronic laminitis, appropriate trimming or shoeing and judicious use of analgesic medications is also necessary. Although many nutritional supplements and nutraceuticals have been advocated for use in equids with PPID, none have established data to support their touted benefits. Finally, due to the expense of lifelong medication, a decision of whether or not to treat affected horses with pergolide should be made on a case-by-case basis in consideration of the client’s goals for the patient.

Husbandry and nutritional considerations: Management of equids with PPID initially involves attention to general health care along with a variety of management changes to improve the condition of older animals. In the earlier stages of PPID, when hirsutism may be the primary complaint, body-clipping to remove the long hair may be the only treatment required. Next, since many affected animals are aged, routine oral care and correction of dental abnormalities cannot be overemphasized. In addition, assessment of diet and incorporation of pelleted feeds designed specifically for older equids (e.g., senior diets) should be pursued. In the author’s experience, aged horses both with and without clinical signs of PPID can easily gain 50 or more pounds within 3-4 weeks of placing them on a Senior feed.

Sweet feed and other concentrates high in soluble carbohydrate are best avoided (unless that is all that they will eat), especially when patients are hyperinsulinemic, hyperglycemic, or both. Also, affected equids may need to be separated from the herd if they are not getting adequate access to feed. Unfortunately, because the abdomen may become somewhat pendulous, weight loss and muscle wasting in more severely affected animals may not be well recognized by owners. In these instances, measurement of body weight, or estimation with a weight tape or body condition score, are important parameters to monitor during treatment.

Whether or not it is “safe” to allow PPID-affected equids to graze pasture as a forage source remains controversial. Pasture, especially lush spring and early summer pasture, should be considered similar to feeding concentrates high in soluble carbohydrates and many veterinarians recommend that PPID-affected equids NOT be turned out on pasture. In my opinion, it is important to assess the overall condition of the patient. If the horse or pony is overweight and has abnormal fat deposits, supportive of insulin resistance, pasture turn out would not be recommended. Instead, feeding grass hay at 1-1.5% of the body weight daily would be the preferred forage diet and animals that are overweight clearly do not need an additional “low starch” concentrate feed. However, if body condition is somewhat poor, strategic grazing for several hours per day can be a useful way to increase caloric intake and produce weight gain. Again, caution is advised and access to lush spring or early summer pasture should be avoided or at least limited to one or more shorter periods per day, preferably during the early morning hours.

Since the major musculoskeletal complication of PPID is chronic laminitis, regular hoof care is essential to lessen the risk of flare-ups. It is important to emphasize to clients that starting medical treatment for PPID (i.e., pergolide) may not lead to complete resolution of the pain and intermittent hoof abscesses that can accompany chronic laminitis, due to the damage to the laminar bed that has previously been sustained. Further, intermittent use of non-steroidal anti-inflammatory drugs, primarily phenylbutazone, may be necessary. Although flare-ups of chronic laminitis remain a leading cause for a decision for euthanasia in PPID-affected equids, it also warrants emphasis that a combination of medical treatment for PPID along with regular hoof care can lead to substantial clinical improvement (Figure 1). Finally, because many PPID affected patients may have secondary infections (e.g., sinusitis, dermatitis, and bronchopneumonia), intermittent or long-term administration of antibiotics, typically a potentiated sulfonamide, may be necessary.
Figure 1.
Photographs of the front feet of a pony with pituitary pars intermedia dysfunction and chronic laminitis: left, initial evaluation (September, 2006); middle, 5-month re-examination (February, 2007); right, 14 month re-examination (November, 2007). Despite a visual appearance to the hoof that may actually seem worse over time (e.g., lower hoof angle after 5 months), a marked improvement in lameness was observed. In addition, hoof conformation was nearly normal after a year of treatment and corrective hoof care.

Medications for treatment of PPID

Medications that have been used to treat equids with PPID include serotonin antagonists (cyproheptadine), dopamine agonists (ergolid mesylate), and, more recently, an inhibitor of adrenal steroidogenesis (trilostane) (Figure 2). Cyproheptadine was one of the initial drugs used because serotonin had been shown to be a secretagogue of ACTH in isolated rat pars intermedia tissue. Early indications that cyproheptadine (0.25-0.5 mg/kg, q 24 h) results in clinical improvement and normalization of laboratory data within 1-2 months have been disputed as a similar response has been obtained with improved nutrition and management alone. The margin of safety of cyproheptadine appears to be high as several horses have received twice the recommended dose twice daily without untoward effects. Mild ataxia has been described in some horses treated with cyproheptadine but this has not been observed by this author.

Figure 2.
Medications used for treatment of pituitary pars intermedia dysfunction (PPID) in horses: the dopaminergic agent pergolide is used to replace dopamine lost as a consequence of hypothalamic dopaminergic denervation; cyproheptadine is an antagonist of serotonin, a neurotransmitter that may potentiate secretion of pro-opiomelanocortin (POMC); and trilostane, a competitive inhibitor of 3β-hydroxysteroid dehydrogenase, that may limit cortisol production by the adrenal gland. \( \beta LPH = \beta \)-lipocortin; \( \beta END = \beta \)-endorphin; \( \alpha MSH = \alpha \)-melanocyte stimulating hormone; CLIP = corticotropin-like intermediate lobe peptide.

Because loss of hypothalamic dopaminergic innervation appears to be an important pathophysiologic mechanism for PPID, treatment with dopaminergic agonists represents a logical approach to therapy. Pergolide administered in both “high dose” (0.006-0.01 mg/kg, PO, q 24 hours [3-5 mg to a 500 kg horse]) and “low dose” (0.002 mg/kg, PO, q 24 hours [1 mg/day for a 500 kg horse]) treatment protocols have been reported to be effective. Adverse effects of pergolide may include anorexia, diarrhea, and colic; however, the latter problems are more often associated with higher doses of the drug. Usually, only transient anorexia is recognized during the initial few weeks of “low dose” pergolide treatment and can be overcome by stopping treatment for a couple of days and starting back at half the dose for 2-4 days, slowly increasing to the desired dose.

Trilostane (0.4-1.0 mg/kg q 24 hours in feed), a competitive inhibitor of 3β-hydroxysteroid dehydrogenase, has been reported to be effective in reversing both clinical signs (primarily laminitis) and abnormal endocrinologic test results in a series of equine PPID cases. However, horses and ponies in that study received additional management for laminitis and the “improvement” in endocrinologic test results was not overly convincing. In contrast, early attempts at treatment with the adrenocorticolytic agent o,p’-DDD were largely unsuccessful. Because adrenocortical hyperplasia has been recognized in, at most, 20% of horses with PPID, drugs targeting adrenal steroidogenesis would intuitively seem less likely to be successful. However, it is possible that concurrent use of pergolide and trilostane (currently not available in the United States) could produce a greater clinical response than use of pergolide alone.
At present, it is the author’s opinion that the initial medical treatment for equids with PPID should be pergolide mesylate at a dose of 0.002 mg/kg, PO, q 24 hours (1 mg/day for a 500 kg horse). If no improvement is noted within 8-12 weeks (depending on season as hair coat changes will vary with the time of year that treatment is initiated), the daily dose can be increased by 0.002 mg/kg (to 2mg/day) with reassessment after 60-90 days. The author typically increases pergolide to a total dose of 0.006 mg/kg (3 mg/day for a 500 kg horse). If only a limited response is observed with 0.006 mg/kg of pergolide and endocrinologic test results remain abnormal, addition of cyproheptadine (0.5 mg/kg, PO, q 24 hours) to pergolide therapy has been effective in a limited number of cases treated by the author. It is important to recognize that the rate of clinical improvement is higher than that for normalization of endocrinologic test results. For example, in a treatment study performed by the author, 13 of 20 pergolide treated horses were reported to have improved clinically while only 7 of 20 had normalization of endocrinologic test results. Thus, it is prudent to regularly measure blood glucose concentration and perform follow-up endocrinologic testing when managing an equid with PPID. The author currently recommends performing an overnight DST (or measurement of plasma ACTH concentration) at least yearly (between December and June in horses in the northern hemisphere) in horses that appear to be stable and 8-12 weeks after a change in medication dose or addition of cyproheptadine). Finally, it is important to remember that, at present, treatment with either pergolide or cyproheptadine remains both empirical and off-label, as pharmacological studies of the drugs have not been performed in equids and no approved drugs are available. Further, although pregnant mares have been treated with the drugs, safety of use during pregnancy has not been studied in equids. Pergolide mesylate is currently only available from a number of compounding pharmacies as the pharmaceutical grade tablets (Permax®) were recently removed from the human market due to development of heart valve problems in a limited number of patients. A major advantage of the compounded products is lower cost; however, pergolide may not remain stable in an aqueous solution (suspension) for longer than 7 days. Thus, it is important to determine how the drug is prepared and dispensed by the compounding pharmacy before specific formulations can be recommended for use.

As with many chronic diseases in the horse, specific nutrient supplementation and complementary or alternative therapies, including acupuncture, homeopathy, and herbal remedies, have been recommended and used in equids with PPID. Both magnesium and chromium supplementation have been advocated for supportive treatment of this condition. Magnesium supplementation (to achieve a dietary calcium:magnesium ratio of 2:1) has been recommended because magnesium deficiency appears to be a risk factor for insulin resistance and type 2 diabetes in humans and anecdotal reports suggest that supplementation may help horses with obesity-associated laminitis. Similarly, chromium supplementation is recommended to improve carbohydrate metabolism (specifically glucose uptake) and improve insulin sensitivity in type 2 diabetes. A herbal product made from chasteberry has also been advocated for treatment of PPID. However, the claim was supported with a series of case testimonials in which the diagnosis of PPID was poorly documented and a recent field study demonstrated that this herbal product was ineffective for treatment of PPID.

**Prognosis**

Once present, PPID is a lifelong condition. Thus, the prognosis for correction of the disorder is poor. However, PPID can be effectively treated with a combination of management changes and medications. Thus, the prognosis for life is guarded to fair. There has been little longitudinal study of equids with PPID but in one report survival time from initial diagnosis to development of complications necessitating euthanasia ranged from 120 to 368 days in four untreated horses. Further, there are numerous anecdotal reports of horses being maintained for several years as long as response to medical treatment was good and close patient monitoring and follow-up was performed. The author has followed a handful of horses treated for PPID with pergolide for nearly a decade and has gained a clinical impression that the drug improves the quality of life but that does not necessarily equate to prolonging life. A recent case series also found that concurrent presence of hyperinsulinemia with PPID was a negative prognostic factor. This finding supports measurement of fasting insulin concentration in the initial evaluation and ongoing management of horses with PPID.
References

**General reviews**


**Treatment**


Equine Thyroid Disease: Fact or Fiction

Harold C. Schott II, DVM, PhD, DACVIM

No Paper Available
Equine Metabolic Syndrome: Pathophysiology and Diagnosis

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Equine Metabolic Syndrome: Can they Ever Eat Grass Again?

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No Paper Available
Endocrine Case Presentations

Harold C. Schott II, DVM, PhD, DACVIM

No Paper Available
Tooth Extraction A to Z

Michael Q. Lowder, DVM, MS

Introduction

Extracting equine teeth has been a job of the veterinarian for years. While the techniques and equipment have changed little over time, the ability to diagnose which teeth need extraction and the level of pain management has greatly improved.

Prior to extracting any tooth from a horse, the practitioner needs to be certain that he has the equipment and experience to perform the extraction with minimal complications. As with any disease process, the history of the patient and the management of the diseased tooth should be obtained in detail. Questions in regards to disease duration, treatment duration and response, attempted extraction, etc. should be asked.

The area for performing the procedure should be secure and away from distracting noise and movement. A complete set of radiographs should be obtained prior to most extractions to augment the clinician’s ability to determine the area of disease involvement, diseased tooth shape, size and location. An exception might be the older horse with diseased teeth that are loosely attached, as these extractions should cause few if any potential complications. Post-extraction radiographs are indicated in most cases to ensure complete extraction of the diseased tooth.

Wolf Teeth Extraction

Equipment:

Extraction of wolf teeth requires a limited number of tools. In most cases a simple elevator and a pair of extraction forceps is all that is needed. However, most practitioners, like myself, have numerous tools for the extraction of wolf teeth. I frequently use a pair of ronguers as my extraction tool of choice after I have elevated around the tooth.

Numerous types of elevators are on the market. The most important factor for an efficient effective tool is sharpness. A sharp elevator will hasten separation of the gingiva from the tooth and the underlying periodontal ligament. The reason to use the curved rongeurs is access to the wolf tooth from the side of the mouth and if broken, the rongeur can be used to extract/cut out the remaining root.

Technique:

Anesthesia is indicated in extracting any wolf tooth except in cases where a fragment or a very small residual tooth is in place. Infiltration anesthesia of the adjacent tissue was described in Chapter 11.

All wolf teeth extraction tools perform the same basic principle of elevating the periodontal ligament away from the tooth and freeing the tooth from any attachment to the gingiva. Once the periodontal ligament is elevated away the tooth, it is grasped with a pair of forceps and oscillated to augment its movement within the alveolar socket. If the practitioner takes the time to loosen the tooth, he will be surprised at the number of wolf teeth with a significant root.

Once the tooth is loosened, slow steady pressure should be applied away from the gingiva and the tooth will come out in whole. If the tooth root fractures, the fragments should be extracted if possible. More elevation of the fragment may be necessary. If the fragment cannot be extracted, the practitioner may leave the fragment alone and recheck the horse at a later date. In some cases, the fragmented root will erupt more, due to an intact periodontal ligament. If the fragmented root is believed to cause discomfort to the horse, a pair of ronguers may be used to ‘chip’ away at the fragment and smooth out any sharp edges. Alternately, the horse can be placed under general field anesthesia for elevation and extraction.

In most cases, the remaining tooth fragments do not cause any mastication or riding problems. The vacant socket needs no special attention post extraction.
The age of the patient will determine the type and arsenal of tools required by the practitioner for extraction of incisors, premolars and molars. If the practitioner is going to limit his extraction cases to older horses, then the number of extraction tools, e.g., forceps, will not have to be as extensive as someone whose cases include younger horses.

Exodontia of equine incisors will require a specific incisor forceps (both the short and long handles varieties), periosteal elevators and maybe a high-speed headpiece or a flex-cable rotary tool, e.g., dremel, to remove the labial surface of the alveoli plate in some cases.

As noted, the horse’s teeth are anisognathous (maxillary teeth are wider than the mandibular teeth), which will influence tool selection for the extraction process. If the practitioner is going to be performing extractions on a routine basis, then I would encourage him to purchase a complete set of upper and lower, i.e., maxillary & mandibular, extraction tools.

Few companies make a complete set of equine dental extraction forceps, and the practitioner should be discriminating in his purchase, as these tools should last a lifetime in most cases. Be mindful of the quality of metal and the coating. Good tools require careful selection. Note the length of the tool, keep in mind that a large warmblood or draft horse may have an oral cavity that is 16-18 inches deep. The handles of the tool must reach past the speculum and give the practitioner enough working room for his hands.

The degree of movement at the forceps’s head does not correlate with the degree of movement of the handles. Depending upon the craftsmanship of the tools, the amount of play at the end of the handles where the practitioner’s hands hold the tool is important. Too much play (movement) will only give a sense of false achievement and cause the practitioner to try premature extraction of a tooth. In addition, tools with lots of play just function to tire out the practitioner.

A prerequisite to extraction of any tooth is a thorough knowledge of the exfoliation/eruption times of each tooth. Variation with breeds should be noted. Large breeds and exotics mature slower and thus may exfoliate their teeth at a later date than normal sized horses.

**Incisors**

Extraction techniques of deciduous incisors will vary with the maturity and length of the tooth. The length is affected by the maturity of the tooth and the attrition of the deciduous tooth via eruption of the permanent tooth directly behind it. Any eruption of the permanent tooth other than the path of the deciduous is abnormal. Eruption of the permanent tooth in an aberrant pattern is a genetic defect. Horses that have one aberrant erupted tooth or a persistent deciduous tooth (i.e., cap) usually will have more than one tooth affected (incisors or premolars).

Infection of deciduous incisors is rare. Most extractions are due to trauma, aberrant eruption of a deciduous tooth or its permanent counterpart, eruption of the permanent distal tooth, delayed exfoliation or in rare cases, a supernumerary incisor tooth.

**Equipment**

Extraction of mature term deciduous incisors requires minimal equipment. A periosteal elevator and a small pair of dental forceps are all that are needed in most cases. Extraction of immature deciduous incisors will require the same tool arsenal as mature permanent incisors due to their length.

**Technique**

The first step in extraction is to determine the age of the tooth. A deciduous tooth that is at term and ready to exfoliate will have a dome shape. A deciduous tooth that does not have a permanent tooth undermining its root structure may have the same shape as the permanent erupting/erupted tooth. In most cases the permanent tooth erupts distal to the deciduous. Radiographs may be indicated to determine the length, size and configuration of the deciduous tooth root.

A periosteal elevator is used to detach the labial gingiva from the deciduous incisor at term (ready to exfoliate) in the sedated horse. Infiltration anesthesia may be used if desired. There is minimal detachment to do and a pair of forceps is placed on the labial and palatal surfaces of the tooth. A sharp twist and pull will extract the tooth. There is no post-extraction care of the extraction site. In most cases the practitioner can either see or palpate the permanent tooth. Some cases may require post-operative pain medication.

The principium for any extraction should be adequate restraint and anesthesia. The extraction procedure is dictated by the clinical examination, radiographic findings and clinician’s experience. Extraction may be done standing or under general anesthesia. Involved (long rooted teeth) extractions should incorporate prior pain medication, systemic and infiltration anesthesia, and facial nerve blocks. In most cases, systemic antibiotics are indicated prior to the extraction procedure(s), due to the depth of the alveolus.
Extraction of deciduous incisors with an intact root involves the same techniques as permanent incisors. Radiographs are indicated in most cases prior to extraction to determine the length, size and shape (direction) of the root. Post-extraction radiographs are indicated anytime pre-extraction radiographs were taken to ensure complete removal of the tooth and any tooth fragments.

First, the mucosa is incised along the lines that the practitioner desires to elevate it (usually down the center or edges of the tooth). Once incised, a sharp periosteal elevator is used to free the mucosa. When elevating the mucosa, keep in mind the type of closure you will desire (primary vs. secondary).

Once incised and elevated, a retaining suture may be placed to hold the flap out of the way. To hold the lip out of the way in the standing horse, a bungee cord can be connected to one side of the halter positioned under the lip, and connected to the other side. Alternatively, a piece of two-inch tape can be used to hold the lip up. It is imperative that a free airway is maintained if the upper lip is retracted.

Next, the labial alveolar plate needs to be removed to free the distal end of the root. This is best done with a No. 8 round burr on a high-speed dental headpiece. A small flex cable grinder with a proper head can be used also if a dental handpiece is not available. Water should be dripped on the grinding burr to minimize heat generation and dust if a flex cable grinder is used.

The tooth should be grasped with a pair of medium sized incisor forceps on the labial and palatinal surfaces. The forceps should be oscillated slowly at first as the horse becomes accustomed to the pressure of the tooth movement. The practitioner should guard against any sudden movement of the head, especially sharp upward movements that might cause the tooth to fracture.

As the tooth is oscillated, the practitioner will note progress as the periodontal ligament is broken down and the socket distorted as foamy blood is noted around the gingiva tooth junction.

As the tooth begins to move freely within the alveolus, do not attempt premature extraction. It is only when the tooth is extremely loose and little blood is seen that it is ready for extraction. Most often the extraction site is allowed to heal via secondary intention.

During the extraction process, the anatomy of the tooth should be kept in mind. Depending upon the tooth and the age of the tooth, its length and shape may vary greatly. The clinical crown will change shape from a rectangular to a triangle with age. Concurrent changes are taking place in the tooth structure below the gingiva margin. In most middle age to older horses, the tooth changes shape from being wider in an abaxial axial direction to a labial distal direction.

### Premolars and Molars: Equipment and Technique

#### Dental Mirror:

No cheek tooth extraction can be done without the aid of a good mirror. I prefer one that has a fixed mirror head and you will need two or three. Most can be bent for various mirror angles. Make sure the mirror is about 18 inches long and has a large diameter head.

Dental picks are used to elevate the gingival away from the diseased tooth. This is important to reduce discomfort to the horse during the extraction process and to allow for proper placement of the forceps on the crown. Prior to elevating the gingiva from the diseased tooth, it is helpful to infiltrate the surrounding gingiva with mepivacaine using a butterfly catheter. This will allow painless elevation of the gingiva. Be careful when placing the forceps that they do not slip as the dental pick could lacerate the palate and/or the palatinal artery.

#### Three-root molar forceps:

Three-root molar forceps, like all forceps, were adapted from human dentistry. The three-root (claw) molar forceps is designed to enclose the crown of the tooth with the roots of the forceps. The upper cheek teeth have two lateral roots and one large medial root. The single claw of the forceps is placed between the two lateral roots of the tooth and the two-clawed side of the forceps is placed on the medial side of the tooth to fit around the single medial root of the tooth.

As you would think, the forceps are functionally best used on teeth that have short reserve crowns, i.e., old horses, but not in the case of a young horse. However, the three-root forceps often offer superior holding ability on some teeth. The practitioner is encouraged to purchase both an upper pair (left & right) and a lower pair (left & right). The upper pair of three rooted forceps is wider between the claws of the forceps than the lower pair.

#### Box-jaw Molar Forceps:

Box-jaw molar forceps are the most commonly used molar forceps. They are called box-jaw because the claws are square to one another. It is important in selecting this pair of forceps to view the serrations of the teeth on the claws. They should not be too short or worn smooth, if so, have the head of the tool re-milled or replace it.

This is the often the tool of choice in most extractions. The box-jaw is placed as far up on the clinical crown (close to the gingiva) of the tooth as possible. Once placed on the tooth, a bicycle tire inner tube should be placed around the handles of the forceps. To do this, make a loop out of one end of the inner tube and slide it over one handle of the forceps. With one end looped around
one handle of the forceps, take the free end of the inner tube and pull it around the other handle of the forceps. Wrap it around the handles a few times and then bring the free end of the inner tube between the handles and wrap it around the section of inner tube between the handles and tuck it between the practitioner’s fingers so that the inner tube may be freed in case of emergency.

Once secured, the handles of the box-jaw forceps should be moved in a lateral-to-lateral oscillating movement. This is done to set the jaws of the forceps into the sides of the tooth. As the jaws work their way into the tooth, the inner tube will keep steady pressure on the tooth. It is only after the forceps are set in the tooth should the handles be rotated side to side. If this is done before the handles are set into the tooth, the forceps will make vertical grooves and slip off the tooth. I usually oscillate the handles of the forceps for about 45-60 minutes before I try rotating them as oscillating takes less effort.

It is very important to get a nice impression into the side of the tooth to keep the forceps from sliding up the side of the tooth when force is applied.

A leverage bar can be used to hasten the loosening of a tooth. The head of the leverage bar is slid over the handles of the forceps as close to the head of the forceps as possible. It is important that the leverage bar be constructed of one piece of solid stainless steel to add weight to the tool to reduce the amount of force needed by the practitioner. The head of the leverage bar must fit securely around the handles of the forceps to prevent any “play” in the bar as it is used to apply leverage to the forceps head. A practitioner may need more than one leverage bar to fit different manufacturer’s forceps.

Only moderate pressure should be applied with this tool. It is very easy to fracture the crown of a tooth if used improperly. The practitioner must allow the weight of the leverage bar to do the work. The leverage bar is only used when rotating the handles of the forceps and not when oscillating the handles.

Molar spreaders (separators) are used to move the diseased tooth in a mesial distal direction. There are several types of spreaders sold with the difference being in the jaw width and the angle of the jaws. The first spreader used in most cases is one with thin straight jaws. Their selection is based on the fact that the diastema (interproximal space) between teeth in a young to middle age and even in an older horse is very tight. Only in aged horses is there a true diastema that one may see.

In application of the molar spreaders, the practitioner has to keep in mind the natural curvature of the upper dental arch. What this implies is that the buccal jaw of the spreaders will be inserted into the interproximal space rostral to the palatal spreader jaw. The long handles of the spreaders will be projecting across the mid-plane of the horse’s head pointing toward the contralateral arch. This is an extremely important thing to note. If the spreaders are applied in a straight line with the dental arcade, the buccal jaw will be closed on the tooth and may fracture the crown.

On the lower arcade, the spreader’s jaws are inserted in alignment with each other as the lower dental arcade is in a straight line. Spreaders are not indicated when trying to use in the caudal part of the lower dental arcade in horses with a spee curve (upward curvature of the lower dental arcade as the last few molars are erupting at an angle at the transition between the vertical and horizontal ramus of the mandible) to the arcade as the long handles of the spreader will prevent them from being inserted into the interproximal space.

An inner tube is applied to the handles as with any other forceps. The spreader is allowed to remain in place for 2-3 minutes then moved to the opposite side of the tooth. Repeated several times, this process aids in the breakdown of the periodontal ligament and distorts the alveolus. Once the jaws of the spreaders are closing within the interproximal space, a larger (thicker jaw) set of spreaders should be selected. The process of slowly closing the thicker spreaders is done as previously. I find that using the spreaders is as much an art as anything. I have broken more crowns and distal roots with spreaders than with any other dental forceps.

Spreaders with an angle jaws should only be used in geriatric cases and with caution. The angle allows for much more pressure to be applied to the tooth, and the potential for tooth fracture is high.

Occasionally, the practitioner will come across two check teeth adjacent (side by side) with one another. Traditional forceps will not allow contact and special forceps that close front to back and not side to side are needed. If the affected tooth is an upper, the jaws of the tool should be angled forward and if a lower tooth, the jaws should be at a right angle to the tool handle.

Simply simply putting a rod inside a pipe and having the end forged to the desired shape can make this tool. The opposite end of the rod should be threaded and a large nut threaded into the rod to adjust the width of the head.

Fulcrums are used to apply leverage to the head of a forceps only when the diseased tooth is ready for extraction. The diseased tooth is only ready for extraction when the practitioner sees foamy blood around the tooth and the movement of the tooth makes a “squishy” sound (like wet tennis shoes). This may not be the case in some older expired teeth.

Most anything can be used as a fulcrum. It is helpful if more than one size of fulcrum is available. I commonly like to use a small piece of square wood. Pine is a nice soft wood that will give with pressure without splintering. In addition, small square pieces of rubber mats can be used and if needed, can be stacked on top of one another to give more leverage.
It is important that the fulcrum be placed close to the head of the forceps when attempting to extract the tooth.

Offset molar forceps are used most often to remove cheek teeth in the caudal aspect of the dental arcade when the diseased tooth is long, as in a young horse, and there is little room for extraction. These forceps may be mistaken for a pair of incisor forceps. Incisor forceps are of the same shape but about a third of the size.

The offset molar forceps are placed upon the crown of the diseased tooth once it is sufficiently loose and the tooth is extracted toward the medial plane of the head. It should be noted that extraction with these offset forceps should only be attempted once the diseased tooth has been partially extracted with a “normal” pair of molar forceps. As the diseased tooth is partially extracted, the length of the tooth prevents it from being brought straight up out of the socket. Thus, the offset forceps are used to remove the tooth towards the medial plane.

The practitioner may try to cut the tooth in half in young horses with long teeth. **WARNING!** If the tooth is cut in half, it must be prevented from falling back into the socket. I would suggest not doing this. If one decides to do this, I would secure the bottom half of the tooth with umbilical tape. If the cut half of the tooth does fall into the socket, the practitioner can use a small Steinmann pin to repulse the tooth.

**Post extraction**

Take the horse's head out of the head support and wash the mouth out with an antiseptic solution. Allow the blood to set in the socket a few minutes. In older horses with shallow alveoli, I usually do nothing to prevent feed materials from entering the space. However, in young to middle age horses, I use some type of material to cover the extraction site. It is very important here NOT to put anything down into the alveolus.

I have tried several types of compounds and still find some type of polymethylmethacrylate material the best to make a patch out of. I will rinse the deep socket, dry it with gauze and then place a patch. Placement of some petroleum jelly on the practitioner's hand will help prevent the patching material from sticking. The patching material should be shaped to the form of an 'H' with the cross-arm of the 'H' over the hole (alveolus) and the legs of the 'H' on the sides of the teeth in front and behind. It is very important to keep the surface of the patch very smooth as it dries. The legs of the patch should be kept on the surface of the supporting teeth and off the gingiva. The ‘patch’ should cover the entire hole of the alveolus and rounded over the edges. The depth of the patch should be no more than ¼ the depth of the socket.

If I am placing a patch over the extracting site of a sinus maxillary tooth, I will place a small sheet of dental wax over the hole and then place the patch. As the patching material cures, it will melt the wax forming a tight seal to prevent migration of feed materials. This is especially important when placing a patch over a maxillary tooth with sinusitis or one that has an oral-nasal fistula.

I like to watch the horse eat later on that day to see if the height or shape of the patch is causing discomfort. I always check on the patch the next day to ensure a tight fit. Depending upon the tooth location and disease state of the surrounding area, e.g., sinusitis, I will usually remove the patch in 2-4 weeks.

**What To Do When Things Go Bad**

Things will go bad every now and then. One way to minimize this is to select your cases very well. As suggested before, I would encourage the inexperienced practitioner to work with an experienced practitioner or attend an extensive “hands-on” short course before attempting an extraction of a tooth in a middle to younger age horse.

The most common complication that I see is the inability to remove the tooth. There is nothing wrong with not being able to remove a tooth. If the practitioner finds himself unable to remove a tooth, he should refer the case. The most common reasons for the inability to remove a tooth is not allowing sufficient time for the procedure, fracturing the crown of the diseased tooth or not having the right equipment. If the crown is fractured, the tooth should be repulsed.

Frequently, the practitioner will fracture a tooth into multiple fragments. If this occurs, the tooth can still be extracted. Select a pair of forceps with small jaws with little or no gap between the jaws and use them to remove the fragments. If a fragment is lodged against the side of the socket, select a dental pick and elevate the fragment away from the socket and extract it. If a fragment is below the depth of the pick, then the fragment will have to be repulsed with a Steinmann pin.

Occasionally, I will see a horse with a fractured alveolus from an attempted extraction. If the alveolus is fractured, the fragment should be left in situ in most cases and allowed to heal.

**Things not to do**

A frequent complication is when a practitioner discovers a fractured tooth and grinds (floats) the fracture fragment(s) down. This does nothing to improve the immediate situation and only makes it harder for the referring practitioner to remove the diseased tooth.

Often, when performing a standing extraction, the horse is aware of his surroundings and quite comfortable if proper sedation and restraint has been administered. However, one must remember that once the initial stimulation of tooth extraction is over and the facial nerve blocks are working well, it will
take less sedation to continue the working procedure of extraction and the level of sedation should be closely monitored.

Be mindful of placing the extraction forceps on the correct tooth or loosening adjacent teeth. Guard against bruising the tongue with the extraction forceps. Do not leave the speculum on a horse for more than about 45 minutes at a time without giving the horse a chance to close its mouth for about 5 minutes. Do not allow the horse’s mouth to dry out during the extraction process.

Horses will urinate as a result of the anesthesia and may become dehydrated. Always put in an intravenous catheter for prolonged extractions. Make sure the horse stays hydrated via intravenous fluids at 1-5 mls/lb/hour in these cases.

Don’t forget to take post-extraction radiographs to indicate all tooth fragments are removed. If a tumor is suspected, submit the extracted tooth for histology.

When doing facial nerve blocks, be sure and let the anesthetic have enough time to work before repeating the nerve block. If you do a maxillary or mandibular nerve block, be sure and watch the horse after the procedure and when they are back in their stalls. A few horses will rub their face or chew on their tongue as the anesthetic is wearing off. If a horse becomes irritated as the nerve block is wearing off, just have someone walk the horse until the irritation is gone.

Geriatric horses may have special considerations. Their liver and/or kidney function may be altered. Either could affect the blood clotting time post extraction, and the type of medication used with the horse. It is encouraged for the practitioner to have blood chemistries done on geriatric horses with suspected disease.

Summary

In conclusion, intraoral extraction of equine teeth can be rewarding but the practitioner should be prepared to handle the potential complications that come with this procedure. Having proper equipment, good patient selection and allowing time for the procedure will increase the chances of positive outcome.

All owners should be informed that if intraoral extraction does not work that they should be prepared to have surgical repulsion of the diseased tooth, and that once a tooth has been disturbed, extraction should be completed.
Misalignment of the incisor arcade occurs commonly in middle age to older horses. The arcade can attain various shapes including a smile, frown, tilt and step. The smile misalignment is corrected by reducing the length of the middle and lateral incisors (teeth 302, 303, 402, 403) of the lower arcade using the length of the central incisors (teeth 301 and 401) as markers for scoring the adjacent teeth (Figure 1).

The teeth should be scored at slightly longer than the desired length, cut and then filed to the desired length. The upper incisors forming the convex part of the smile (teeth 101, 102, 201, 202) may have to be reduced some as well, to allow correct occlusion of the molar arcade. Once corrected, there will be a discontinuity to the table surface of the incisor arcade that will be corrected with frequent floating and time. The frown misalignment is corrected using the same principles as for the smile misalignment, except that the lengths of the upper middle and lateral incisors (teeth 102, 103, 202, 203) are reduced.

The tilted (slant, Figure 2) incisor arcade is reduced in the same manner except that tooth reduction occurs in both arcades. The elongated teeth at opposite ends of each arcade are reduced to correct this misalignment. A stepped incisor arcade is managed using the same techniques used to treat a single elongated incisor.

Canine Teeth

Canine teeth are most common in stallions/geldings older than 3 1/2 years but frequently occur in mares. In young horses the unerupted or erupting canine tooth might be a cause of head shaking or tossing. The gingiva over the tooth should be incised if an eruption cyst is present.

Indications for reducing (floating) canine teeth include growth of a canine tooth into the opposite palate, partial fractures of these teeth, preventing injury to the practitioner’s hands during inspection of the molar arcade, facilitating insertion and removal of the bit, and reducing the chance of an injury to a mare during breeding. In addition, these teeth are frequently the site of large calculi in older horses that sometimes interfere with prehension.

Canine teeth nippers can be used to reduce the length of these teeth. The nippers are positioned over the upper half to two-thirds of the tooth with the handles perpendicular to the horse’s head and the tooth cut off. Care must be taken to avoid placing the nippers near the gingiva as this increases the possibility of fracturing the tooth below the gingiva. Alternatively, the canine tooth may be cut-off with a dremel diamond cut-off wheel. In this case the tooth is cut at the desired length, thereby eliminating the chance of fracturing the tooth.

Regardless of how the length of the tooth is reduced, some floating of the tooth will be needed to leave the tooth at the proper length or smooth the cut edges of the tooth. A carbide chip dremel burr or a rubber mandrel with a sand paper sleeve also can be used to reduce the length of the canine tooth. Less sedation usually is required to reduce the teeth with nippers in contrast to what is needed to complete the process with the dremel burr or wheel. Safety glasses should be worn at all times when cutting off canine teeth as tooth chips frequently will fly through the air, especially when nippers are used. Surrounding personnel also should wear protective glasses. Due to the fine dust particles produced when a dremel tool is used, it is recommended that everyone wear safety masks to prevent inhalation of these particles.
Molar Madness
Michael Q. Lowder, DVM, MS

Supernumerary teeth (polyodontia) occur in the molar or incisor arcades. This congenital defect is due to division of the primary tooth germ. In the incisor arcade, the extra teeth are usually caudal or lateral and may be removed if they cause problems, (e.g., interfere with the bit), or for cosmetic reasons. They are removed using the same technique used to remove a retained deciduous tooth. In the cheek arcade, the extra tooth is usually caudal to the normal teeth, but may be located on the lingual or buccal sides of the normal teeth. If the tooth is causing no problem, it is best left alone.

Dentigerous cysts (periauricular cysts, temporal teratomas, odontomas, heterotopic polyodontia, ear teeth, ear fistulas, conchal fistulae, cysts or sinuses) are due to misplaced germ cells from the branchial cleft and may appear at any age. Most often, dentigerous cysts are located near the ear, but occasionally may be located in the cranial vault or maxillary sinus. The cysts usually have a fistulous tract draining either into the pinna or exiting directly over the cyst. Usually some form or amount of dental tissue is present within the cyst. Dentigerous cysts should be removed via surgical excision with the horse under general anesthesia. Pre-operative radiographs should always be taken to determine the extent of the cyst and bony involvement. Characteristically, the cyst will be attached to the temporal bone and require careful dissection. Care should be taken to prevent fracture of the temporal bone during removal of the cyst. To prevent development of a draining tract after surgery, the entire cyst must be removed and it is recommended that the surgical site be lavaged with sterile fluid and sutured. Prognosis is excellent unless complications arise as the dental remnants are separated from the underlying bone.

Oligodontia (absence of teeth) is a common finding and might be considered normal in some individuals. Mares commonly have fewer teeth than males, with the most commonly missing teeth being the canines and first premolars (wolf teeth).

Overcrowding and shifting of teeth are encountered commonly in miniature horses and small headed horses (primarily Arabians), and these individuals often have a curved mandible (“Spree Curve”). This curvature in the jaw causes the last cheek tooth to erupt at an abnormal angle and this tooth does not meet its counterpart in the opposing arcade. Consequently, both teeth erupt without proper attrition and wear. These teeth are frequently mis-diagnosed as a caudal hook or ramp. Also, these teeth can be managed by frequent filing.

Periapical Infection

Periapical infections are most commonly encountered in young horses when teeth are erupting; they may occur in horses of any age. Infections of this area have also been called root abscesses, periodontal or apical disease, dental caries and eruption pseudo-cyst. Infection of the periapical area appears to be due to either a delay in tooth eruption or the development of a centralized channel of tooth necrosis (infundibular necrosis). Impaction of a permanent tooth (delayed eruption) by a deciduous tooth or trauma may cause vascular lysis in the bone around the tooth. As the permanent tooth continues to grow, it leads to local hyperemia in which periapical disease may occur. It is proposed that opportunistic bacteria invade this hyperemic tissue, thereby initiating the infection. The diseased tooth should be examined radiographically to determine the extent of bone lysis and to evaluate the eruption space. If a retained deciduous tooth (cap) is present, it should be removed and, if needed, the adjacent teeth filed to provide sufficient room for the erupting permanent tooth. If a draining tract is associated with the tooth root, then the tract should be curetted and lavaged with an antiseptic solution. In addition, the horse should be administered antibiotics and a non-steroidal anti-inflammatory agent systemically. Some horses will respond to this form of therapy, while others will have a temporary cessation of the swelling and discharge. If the area becomes inflamed again, the tooth will either require endodontic therapy or will need to be removed.
Endodontic therapy (root canal surgery) is a viable option in these cases and should be considered. Criteria for endodontic therapy include a stable tooth with a healthy periodontal ligament. The basic goal in endodontic therapy is to maintain a functional tooth by removing the diseased pulp.

Periradicular (inflammatory dental) disease refers to inflammatory conditions involving the dental pulp, root and surrounding tissue (pulpitis, apical, periapical, tooth root abscess formation or osteitis). Practitioners frequently encounter horses with periradicular disease that require focused attention to make a diagnosis and decide upon a suitable treatment. Referral hospitals are often presented with horses with suspected periradicular disease in which routine diagnostic procedures, e.g., masticatory exam and radiography, have failed to yield a diagnosis. Clinical signs of periradicular disease include, but are not limited to, abnormal head carriage, abnormal eating and riding habits, ptalism, halitosis, purulent nasal discharge, swellings, draining tracts, and rarely anorexia. The disease may result from direct causes (trauma, impacted deciduous teeth, fracture of the tooth and maxillary or mandibular fractures) or indirect causes (trauma, impacted deciduous teeth, fracture of the tooth and maxillary or mandibular fractures) or indirect causes (trauma, impacted deciduous teeth, fracture of the tooth and maxillary or mandibular fractures) or indirect causes (trauma, impacted deciduous teeth, fracture of the tooth and maxillary or mandibular fractures) or indirect causes (trauma, impacted deciduous teeth, fracture of the tooth and maxillary or mandibular fractures) or indirect causes (trauma, impacted deciduous teeth, fracture of the tooth and maxillary or mandibular fractures) or indirect causes (trauma, impacted deciduous teeth, fracture of the tooth and maxillary or mandibular fractures) or indirect causes (trauma, impacted deciduous teeth, fracture of the tooth and maxillary or mandibular fractures).

**Therapeutic Protocols**

As with all cases, a complete history and physical exam should be obtained. The signalment of the horse will often aid the practitioner in ascertaining if tooth eruption is involved and which tooth may be affected. In addition, a complete masticatory examination using a full mouth speculum is indicated. High quality radiographs of the head should be obtained on horses with clinical signs of dental disease, whether or not abnormal findings are identified on physical examination. Sedation of the horse sufficient for the animal to rest its head upon a stable surface (a stool or table) will reduce motion and enhance radiographic quality. If an external draining tract is present, injection of contrast media into the tract will facilitate identification of the affected tooth. Radiographic evidence of periradicular disease confirms the diagnosis, thereby allowing the practitioner to proceed with the appropriate treatment.

It is important to note that a lack of radiographic changes does not exclude the possibility of dental disease. In such cases, nuclear scintigraphy may identify the affected tooth. While scintigraphy is less specific than radiography, it is an extremely sensitive diagnostic modality, detecting both soft tissue and osseous inflammation. If scintigraphy demonstrates that the tooth is traumatized without significant osseous change, affected horses may respond to short-term non-steroidal anti-inflammatory therapy (phenylbutazone 4.4-8.8 mg/Kg PO q 12 hrs) and 6-8 weeks of antimicrobial therapy (trimethoprin-sulfadiazine 30 mg/Kg PO q 12 hrs and metronidazole 25 mg/Kg PO q 12 hrs).

Response to therapy should be monitored with repeated radiographic and or scintigraphic evaluations. In horses with recurrence of clinical signs after cessation of antimicrobial therapy, and those not responsive to antimicrobial therapy, tooth removal or long term antimicrobial therapy (12-16 weeks) is required.

If the affected tooth must be removed, the method used depends upon the age of the animal, tooth location, amount of exposed crown, integrity of the tooth (intact vs. fractured), and financial considerations. Young horses tend to have retained deciduous teeth (dental caps), which may impede eruption of permanent teeth. Correction is achieved by removal of the retained deciduous tooth. A retained deciduous cheek tooth should only be removed when a permanent tooth can be seen radiographically, and the permanent tooth is at or above the gingival margin. Removal can most often be performed with short forceps but occasionally may require the use of large sixteen-inch forceps. The permanent tooth should erupt uneventfully or if erupted and displaced, should eventually realign with the arcade. Premolars and molars can be removed via oral extraction, by repulsion via trephination, flap sinusotomy or lateral buccotomy approaches. Oral extraction may be performed in the sedated standing horse. Trephination, flap sinusotomy, and lateral buccotomy are best performed in the anesthetized patient.

Oral extraction techniques can be used to remove any of the incisor or cheek teeth. The more cranially located teeth are more easily extracted using this technique than are the caudal molars. With experience, the practitioner can extract caudal teeth, thereby avoiding the complications commonly associated with the repulsion methods. Oral extraction can be performed on the standing sedated horse or with the horse under general anesthesia.

If the affected tooth is the second, third or fourth mandibular premolar or the first or second mandibular molar, the tooth may be removed through a trephine opening made via a ventral approach. The third mandibular molar is best approached via a more lateral approach, with elevation and retraction of the tendinous insertion of the masseter muscle over the ventrolateral aspect of the ramus of the mandible.

In the maxillary arcade, the third and fourth premolars and first molar are the most commonly affected teeth. Maxillary premolars and molars are best removed by oral extraction or a flap sinusotomy, but trephination is an alternative preferred by some clinicians. Trephination produces poorer cosmetic results and appears to cause more anxiety for the owner. Flap sinusotomy allows better surgical exposure to the affected tooth and corresponding sinus. Maxillary molar involvement is usually associated with sinusitis and requires lavage of the sinus with saline during surgery. It is advisable to insert an indwelling lavage system to facilitate postoperative lavage of the affected sinus. The lavage tube can either be made out of tubing from an intravenous drip set or a commercial fenestrated sialastic drain may be used. The sinus should be lavaged one to
two times daily with 1-2 liters of saline. Antimicrobials may be added to the lavage solution. The selection of topical and systemic antimicrobials should be based on the results of bacteriologic culturing and sensitivity testing of the affected tissues at surgery.

Endodonic therapy can be a useful technique for tooth restoration in the horse. Endodonic therapy preserves the affected tooth and eliminates the need for repulsion or extraction. Endodonic therapy does, however, require special equipment and is usually restricted to mature horses with a well-developed tooth root system. The more rostral mandibular cheek teeth have a less complex root system and are more surgically accessible, therefore rendering them more amenable to endodontic therapy. The affected tooth root is identified by survey and/or contrast radiography. If a fistulous tract is present, radiography with a metal probe inserted in the tract may facilitate identification of the affected tooth root. Once the affected tooth root is located, apicoectomy and subsequent filling of the affected root are performed. Teeth in the maxillary arcade are unsuitable for endodontics due to the presence of a complex medial root.

**Prognosis**

Tooth repulsion in the horse has a high complication rate. Horses undergoing maxillary tooth repulsions have more complications (47% vs. 32%) and longer periods of hospitalization (22 vs. 8 days on average) than horses with mandibular tooth repulsions. When applicable, oral extraction eliminates the need for general anesthesia and decreases the incidence of many secondary complications usually associated with tooth repulsion techniques. It is imperative that the horse’s owner understands the potential problems associated with tooth removal and the long-term management required. The owner must make a firm commitment to have regularly scheduled masticatory examinations and dental floating performed for the remainder of the horse’s life.

**Conclusion**

Periradicular disease in horses can be extremely challenging with respect to recognition and diagnosis. Ancillary diagnostic modalities such as survey and contrast radiography, or nuclear scintigraphy may confirm a diagnosis of dental disease. A successful resolution may be achieved with long term antimicrobial therapy, however, in non-responsive cases, tooth extraction or repulsion is necessary for complete resolution of the clinical signs.
Equine Oral Examination
Michael Q. Lowder, DVM, MS

The Modified Triadan System is a three decimal numerical system for labeling teeth (Figure 1). This system assigns a three-digit number to a specific tooth. The horse’s head is divided into four quadrants with the horse’s upper right side labeled “1”. The remaining quadrants are numbered in a counter-clockwise direction. Numbers 1-4 are used to identify the quadrant for permanent teeth, and 5-8 are used for the temporary dentition. The first of the three digits thus describes the quadrant the tooth is located in, and the last two digits identify the specific tooth number ranging from 1-11. For example, the left lower second premolar is tooth “306” and the last molar on the right mandible is “411”.

In contrast, the anatomic system uses tooth function to describe a tooth and a number to depict location within the arcade. Tooth function is denoted via the first letter of the common name of the tooth, with lower case letters being used for temporary teeth and upper case letters for permanent teeth. For example, the second temporary premolar on the upper right side of the horse would be labeled “2p”, whereas the second permanent premolar would be “2P”.

The use of dental charts provides the practitioner with a way of describing findings on dental examinations, anticipating future needs or potential problems, and strengthens the practitioner’s stance against possible litigation. Specific symbols to be used on dental charts have been published and are easily adapted to equine dental charts. Equine dental terminology is very helpful when describing a diseased tooth. This terminology should be used correctly and consistently to communicate with other practitioners and to record aspects of dental disease.

Once the clinician is familiar with the Modified Triadan System and dental terms, the examination process can begin. Before performing a masticatory examination, a general physical exam should be completed and a complete history obtained that describes the horse’s daily activities. The basic masticatory examination should include a visual, auditory, and tactile examination. In some cases radiography, ultrasonography, or scintigraphy are indicated.

Dental Examination and Diagnostic Techniques

The examination process should include a complete history and physical examination with particular attention being paid to questions regarding the horse’s eating and riding habits. If necessary, the practitioner should watch the animal while it eats or while it is being ridden to detect subtle abnormalities. The dental examination process always should include visual and tactile examinations, and in some cases ancillary diagnostic aids are necessary. The visual examination is best done first at a distance, noting the symmetry of the animal’s head and its demeanor. Closer inspection of the head should be performed to identify asymmetry, bumps, swellings, and draining tracts. External palpation of the horse’s head can also aid the practitioner in detecting subtle changes in...
the anatomy and sharp enamel points on the upper cheek teeth by palpating the external cheek. The lips and lip commissures should be inspected for any scars, cuts, or ulcers. Subsequently, the incisor arcade can be inspected for any abnormalities including broken, missing and/or malaligned teeth. Upon completion of the visual examination, the horse’s breath should be smelled for a fetid odor characteristic of tooth infection or impacted feed material.

The auditory part of the examination involves listening to the excursions of the dental arcades. The veterinarian must stand beside the horse’s head with one hand on the maxilla just caudal to the nostrils and the other hand on the mandibular bars. The mandible is moved from side to side while the maxilla is held still. The veterinarian should note sounds made during excursions of the dental arcade and the amount of movement of the mandibles. Normal lateral excursion of the mandibles is 1 1/2 to 2 1/2 times the width of the upper central incisor. To judge the degree of excursion, horses may require sedation. Normal movement of the mandible during mastication occurs from side to side. Because some horses tend to chew in a left to right direction and others chew in a right to left direction, the arcade is usually more difficult to move in one direction. If difficulty is encountered in causing lateral excursion of the mandibles, this may be indicative of enamel points or misalignment of the cheek teeth or incisors.

The tactile examination can be done with or without a mouth speculum, but a full-mouth speculum is required to palpate the caudal molars. In addition to the teeth, the buccal surface of the lips and lip commissures should be inspected for any scars, cuts, or ulcers. Subsequently, a full-mouth speculum is required to palpate the buccal surface of the speculum, but a full-mouth speculum is required to palpate the buccal surface of the lips and lip commissures. The practitioner must be comfortable with the level of restraint required to conduct a complete exam and this will vary with individual practitioners and patients. Proper performance of equine dental procedures requires that the veterinarian develop an efficient and effective examination method. This is best accomplished by working in a consistent and sequential manner.

The most important aspects of the masticatory examination are to have patience with the horse during the examination process, to conduct the exam in a safe environment for the horse and examiner, to use the least amount of restraint that is both necessary and safe, and to slowly introduce the horse to the examination process and application of the equipment. No equipment or procedure should be applied or conducted if the horse becomes restless or agitated. Often a little extra time taken in the first examination will expedite future examinations.

Diagnostic Aids

Radiography can be an essential component of the examination process and satisfactory radiographs can be obtained in the field. A portable x-ray unit capable of producing 80-100 kVp with exposure times up to 0.5s will provide satisfactory x-rays in most cases. Rare-earth 400-speed film provides the best results. The practitioner should always take four views (lateral, dorsoventral, and two obliques) of the skull when evaluating horses with suspected dental disease. The injection of contrast media into draining tracts will often aid in the identification of the affected tooth.

Radiography of the equine head is useful in confirming the presence of dental disease. Indications for radiography include: tooth eruption, impacted teeth, fractures of the teeth or skull, abnormal dental wear patterns, tooth abscesses, foreign objects, and missing, malaligned, sinusitis or supernumerary teeth. It is advisable to sedate the horse to facilitate radiography of the head. The horse’s chin is allowed to rest on a stool or table to stabilize the head, and a thin rope halter should be placed on the horse to minimize interference with the image. Lateral, oblique, and dorsoventral projections can be obtained of the incisors and cheek teeth. A good review of dental radiography and interpretation of the images has been published.

Ultrasonography can be used to detect pockets of fluid, (e.g., abscesses) in the oral cavity and near the base of the tongue or cheek. The ultrasound probe is placed in the intermandibular space or against the cheek if the horse is presented with a history of a localized swelling and/or dysphagia especially if the horse had a history of a previous floating or oral trauma.

Scintigraphy (nuclear imaging) is a diagnostic modality with increasing availability that will allow the practitioner to isolate dental abnormalities that may have been inapparent on radiographs. Scintigraphy of the equine is infrequently done. However, it can be a useful diagnostic technique to determine involvement of teeth in a disease process. While the resolution is less the specificity for disease tissue is better than that of radiology. It provides both anatomic and physiologic information base upon the uptake of the radiopharmaceutical agent used.
A gamma camera is necessary to secure the images produced after the intravenous administration of a radiopharmaceutical agent to the horse. Scintigraphy is divided into three scan phases: Phase 1 (lasting about 30 seconds post-injection) provides an angiogram of major vessels, phase 2 (10-20 minutes post-injection) is used to identify changes in soft tissue structures and phase 3 (minimum of 2 hours post-injection) captures bony changes. The latter two phases can be used in equine dentistry to identify diseases of the soft tissues or bony structures of the head. Scintigraphy is more sensitive but less specific than radiography and should always be used in combination with radiographs to secure a diagnosis in equine dentistry.
Sedation of the equine patient for a complete oral examination is essential. Sedation can vary from mild use of xylazine to a continuous drip of detomidine (Dormosedan®) and butorphanol. The way I approach sedation of a horse is to view the horse coming into my stocks or floating area, i.e., wash rack. Small Quarter Horses I will often use 0.3 ml butorphanol and 1.5 ml xylazine. Whereas, most horses I will use 0.3 ml butorphanol, 0.4 ml detomidine and 0.5 ml xylazine IV.

When I am doing prolong dental procedures I will use continuous drip infusion of 2.0 mls of detomidine and 1.0 ml of butorphanol in 500 mls of saline. I use a 10-drop/ml-drip set and drip as needed after the initial sedation.

Facial nerve blocks can be performed to augment the effects of a tranquilizer or sedative. Indications for facial nerve blocks for equine dentistry include masticatory examination, tooth removal, evaluation of sore gums or cheeks, probing of periodontal pockets, teeth floating, examining sensitive teeth, and repair of lacerations or fractures. The dental nerve blocks include those that desensitize the infraorbital, mandibular, and mental nerves (Figure 1).

The infraorbital nerve block is easily performed in the sedated horse. If the nerve is blocked as it emerges from the canal, the anterior portion of the face ipsilateral to the block is desensitized. Desensitization to the level of the first molar can be obtained by inserting a 5 cm needle about 3.5 cm into the canal, and depositing 3-5 ml of a local anesthetic agent into the canal. The mental nerve can be desensitized in the same manner except that a 7.5 cm needle must be inserted into the canal about 5 cm to desensitize to the level of the first molar.

Although the mandibular nerve block is not performed very often, with some practice this nerve can be blocked just as it enters the mandibular canal with a 20 gauge 30 cm needle; the canal itself is not penetrated. The veterinarian should be aware that there are potential problems associated with desensitization of the facial nerves. These include prolonged anesthesia, and laceration of the cheeks or tongue. In some cases, (e.g. to remove a portion of an incisor) the area surrounding a tooth can be desensitized with local infiltration of an anesthetic agent directly into the gingiva.
How to Dentistry Procedures

Michael Q. Lowder, DVM, MS

Diagnosing equine dental disease often requires radiographs of the head. Recently, a new method of taking radiographs of the head of horse with the dental arcades open has been reported.\(^\text{[1, 2]}\)

The techniques are fairly simple with the mouth of the sedated horse held open via the induction of a short piece of PVC pipe (7.5-11.5 cm in diameter and about 10 cm in length) between the incisor arcades. A slide showing how to make one of the PVC wedges with a guard will be shown. The PVC pipe is inserted between the incisor teeth and the horse’s head is allowed to rest on two bails of hay stack on top of one another or a table or stool.

Most practitioners will use a portable x-ray unit with 80-100 Kvp and 15-20 MA. A focal film distance of 100 cm (40 inches) is most often used. Rare earth cassettes with medium to fast-speed film are used.

Place the cassette on the tabletop adjacent to the affected side of the horse’s head. One advantage of this method is that it allows you to distinguish fluid lines better when the film is placed on a view box as the fluids lines will be parallel with the horizontal of the box. Using 34cm x 43cm (14 x 17 inch) cassette centered at the facial crest the practitioner should be able to obtain a radiograph of the cheek arcade.

To image the clinical crowns of mandibular teeth a 10-15° cassette angle from the ventrolateral-lateral obliques are used. For the maxillary teeth a 10-15° cassette angle from dorsolateral-lateral obliques are used. These views are only for the clinical crown of the cheek arcade. If an image of the roots are desired then a standard 30°dorsolateral-lateral oblique if used for the maxillary cheek teeth and a 35-45° ventrolateral-lateral oblique view is taken for the mandibular roots.

References:


Sepsis, also referred to as septicemia, remains one of the most common diagnoses in equine neonates and accounts for the majority of foal deaths in neonatal intensive care units (NICUs). Foals are inherently susceptible to sepsis due to their unique immunologic system including the requirement for adequate transfer of passive immunity, and are at further increased risk if other predisposing conditions such as prematurity or hypoxic-ischemic encephalopathy are present. Exposure to pathogens is mostly from the environment, other ill foals and the dam. The likelihood of successfully treating septic foals is improved with early recognition, aggressive and appropriate management and careful monitoring, however, the prognosis remains guarded in many cases.

In the human literature, sepsis is defined as the systemic inflammatory response (SIRS) to infection, and parameters used to establish the presence of SIRS include physical abnormalities (fever, tachycardia), signs of inflammation (leucopenia, left shift), hemodynamic abnormalities (hypotension), signs of organ dysfunction (oliguria, coagulopathies) and evidence of abnormal tissue perfusion (hyperlactatemia). Similar parameters are evaluated in equine medicine; however, the diagnosis of SIRS remains subjective. Presence of infection is typically shown through microbiologic testing of blood or other samples such as tracheal aspirates or joint fluid. Importantly, while sepsis is most commonly caused by bacterial infection, viral and fungal sepsis is also recognized in foals. Common bacterial organisms recognized in equine neonatal sepsis include Gram-negative, often coliform, bacteria as well as *streptococci, staphylococci* and anaerobes. Several reports have suggested a rise in the number of Gram-positive organisms causing sepsis in foals, and this trend warrants further monitoring. The most important entry route for infection is the gastrointestinal tract, however, the respiratory tract, umbilicus and skin must also be considered. Following infection in utero, foals may present with signs of sepsis from birth.

Early clinical signs of sepsis in foals are often subtle and may include behavioral changes (increased recumbency, reduced activity), reduced sucking and failure to gain weight as well as hyperemia of mucous membranes and the coronary bands. Fever may be present but is inconsistent, and hypothermia is often noted with advanced disease. Advanced signs of sepsis include petechiation, uveitis and hypopyon as well as signs of localized infection such as umbilical enlargement, joint effusion and lameness, respiratory abnormalities or diarrhea. It appears important to institute diagnostic procedures and treatment based on early clinical signs in order to avoid complications such as joint infection or pneumonia. The prognosis for septic neonates with multiple septic joints or evidence of bacterial meningitis (seizures) is poor.

The diagnostic approach to a septic neonate should include a thorough physical examination along with assessment of the complete blood count (CBC), serum chemistry and adequacy of transfer of passive immunity. Sampling for microbiologic testing should be based on clinical signs and samples should be obtained prior to institution of antimicrobial therapy as long as this does not result in an unreasonable delay of therapy. Calculation of a sepsis score is generally practiced at referral institutions, but is also applicable to field situations. The sepsis score takes into account historical information, physical examination findings and the results of diagnostic testing and provides the clinician with a more objective assessment of the likelihood that an individual patient is suffering from sepsis. Sepsis scoring also improves record keeping for neonatal foals and can be used to evaluate trends during treatment and thereby re-assess prognosis based on response. Evaluation of the dam by physical examination and, if indicated, additional diagnostic testing should be performed as well in order to appreciate potential predisposing factors such as colostrum loss by premature lactation, lack of milk production or presence of placentitis or retained placenta.

Treatment of septic foals depends on severity, organ involvement and complications; however, the following principles apply. Appropriate antimicrobial therapy is crucial and should ideally be based on results of microbiologic testing. Limited evidence suggests that it is not very feasible to predict the presence of specific bacterial...
organisms based on physical examination and routine hematologic testing. Furthermore, reports of unpredictable sensitivity patterns and presence of multidrug resistant organisms underscore the value of microbiologic testing. Until culture results are available, however, or in the absence of microbiologic testing, treatment should be based on the knowledge of common offending organisms as well as the properties (including negative side effects) of available drugs. As mentioned above, Gram-negative organisms are still most common in septic neonatal foals, and empirical treatment should address this fact. Common antibiotics used as first-line drugs in septic foals include combinations of a penicillin and an aminoglycoside, cephalosporins, or combinations of a cephalosporin and an aminoglycoside, and foal-specific dosages have been published. Side effects such as nephrotoxicity should be considered when using aminoglycosides in neonates and assessment of renal function as well as therapeutic drug monitoring during therapy should be considered, especially with longer-term treatment. Other drugs such as chloramphenicol and enrofloxacin may appear attractive from the standpoint of antibiotic sensitivity and tissue penetration; however, side effects including arthropathies (enrofloxacin) and the human risk when handling chloramphenicol must be strongly considered.

In addition to antibiotic therapy, treatment of septic neonates must address failure of transfer of passive immunity, dehydration and acid-base as well as electrolyte abnormalities, especially in foals with diarrhea. Anti-inflammatory therapy is often instituted initially, at least in febrile animals, but should be used cautiously because of potential side effects. Specific treatment of localized sepsis, e.g. removal of infected umbilical remnants or lavage of infected joints, is applicable in some patients and may warrant referral to an equine hospital. Similarly, recumbent foals and those requiring oxygen therapy, ventilation, vasopressors or parenteral nutrition are best cared for in an intensive care unit setting. Supportive care to ensure adequate hydration and nutrition as well as respiratory support, and to avoid further complications such as corneal ulceration or decubital ulceration becomes crucial in severely ill foals and may well determine the outcome of treatment.

The prognosis for septic neonatal foals is overall guarded but probably depends largely on the timeliness of recognition, promptness and appropriateness of therapy, availability of adequate care facilities and owner commitment. Retrospective studies from NICUs at referral teaching hospitals report survival rates between 50 and 80% of admissions; however, inclusion criteria must be considered when evaluating these studies and trying to apply them to another clinical situation. Similarly, little information about the long-term outcome and athletic capacity of septic equine neonates is available; one study reported comparable performance in Thoroughbreds that entered a training program.

In summary, sepsis remains a common and life-threatening condition in equine neonates. Recognition of risk factors and early clinical signs, appropriate diagnostic testing and aggressive therapy are important in managing septic neonates. In the author's experience, treatment of foals with early signs of sepsis is often very rewarding; however, the prognosis for foals that develop complications remains guarded and a realistic approach to therapy should be taken. Having said that, increasing knowledge and application of intensive care strategies to septic foals has resulted in encouraging treatment reports from large-scale neonatal intensive care units, and may ultimately modify our approach to the treatment of septic foals. Given the experiences in human intensive care units, continued monitoring of the organisms responsible for development of sepsis as well as their susceptibility patterns is further indicated.
Non-infectious and Infectious Respiratory Conditions in Foals

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A recent textbook on equine neonatology lists prematurity/dysmaturity, neonatal asphyxia, viral infections (Equine Herpesvirus, Influenza), bacterial infections, aspiration of infected amniotic fluid, milk or meconium, pneumothorax and idiopathic tachypnea as common etiologic categories causing respiratory distress in newborn foals. Cardiovascular diseases, metabolic derangements such as acidosis, hypovolemia and anemia as well as neurologic conditions also need to be taken into account when assessing a foal with respiratory problems. Based on the 2005 NAHMS (National Animal Health Monitoring) study of equine operations in the United States, respiratory conditions - most of which were likely infectious in nature - accounted for 3.6% of foal deaths during the first 30 days following birth. Infections such as those with *Streptococcus equi equi* (strangles) or *Rhodococcus equi* are also common causes for concern in older foals. In the NAHMS study, non-infectious conditions such as surfactant deficiency/lung immaturity or meconium aspiration may have accounted for some of the 10.7% of deaths due to “dystocia, trauma or complications at birth”, while congenital respiratory conditions such as sinus cysts, choanal atresia, guttural pouch tympany and laryngeal malformations may have contributed to the 8.9% of foal deaths from birth defects.

Respiratory distress in foals is a serious condition that requires immediate diagnostic testing in order to identify a cause and institute appropriate therapy. Aside from addressing the cause of disease, clinicians need to be aware that foals tend to fatigue when having respiratory difficulty for a prolonged period of time, and may require respiratory support in the form of oxygen therapy or even ventilation. Clinicians should also recognize that foals may not always show distinct signs of respiratory involvement such as coughing and abnormal lung sounds, which puts greater emphasis on diagnostic tests such as arterial blood gas analysis and thoracic radiography and ultrasonography.

**Surfactant deficiency/lung immaturity**

Surfactant is a complex phospholipid mixture produced by type II pneumocytes in the lung. Surfactant production takes place relatively shortly before birth and, therefore, premature foals – especially those with an exogenously induced premature birth - are at risk of inadequate lung function due to surfactant deficiency. Secondary surfactant deficiency has been identified in children recovering from acute lung injury.

Foals with immature lungs typically exhibit respiratory difficulty shortly following birth. Surfactant deficiency results in decreased lung compliance and reduced functional residual capacity, and favours alveolar collapse which must be overcome at each breath. Potential contributing factors to this increased work of breathing in premature foals are meconium aspiration and resulting lung inflammation, weakness and recumbency (especially lateral recumbency) as well as fractured ribs from birth trauma or vigorous resuscitation efforts.

Blood gas analysis is vital in establishing respiratory failure, which is indicated by hypercapnia and hypoxemia. Hypoxemia can to an extent be overcome by intranasal oxygen insufflations; however, persistent and worsening hypercapnia with PaCO₂ levels above 60 mm Hg indicates a need for positive pressure ventilation.

Treatment of foals with respiratory failure is primarily supportive and may include oxygen insufflation, frequent repositioning and maintenance of a sternal position to prevent atelectasis, antibiotic coverage to prevent pneumonia and other complications, and in some cases ventilator support. Corticosteroids given prior to birth (if an impending premature delivery is suspected) or at birth may help to hasten lung maturation; however, potential side effects must be considered. Surfactant administration (both from BAL obtained from adult horses and by administration of commercially available surfactants) has been reported and may be useful in some cases.
Rhodococcus equi pneumonia

*Rhodococcus equi* (*R. equi*) is a Gram-positive, soil-borne, opportunistic pathogen that causes pyo-granulomatous pneumonia in foals and has rarely been identified as a cause of disease in immune compromised adult horses. *R. equi* pneumonia occurs endemically on many breeding farms, and prevalence of *R. equi* pneumonia has been associated with the mare and foal density on individual farms. Interestingly, generally accepted “good” management practices have not been associated with a reduced prevalence of *R. equi* pneumonia.

Infection with *R. equi* in foals likely occurs early in life; however, the typical age of onset of clinical signs is between 1 and 4 months. Experimental studies suggest that clinical signs of disease may not be obvious until the disease is well advanced and considerable lesions are present in the lungs. Commonly used diagnostic criteria include physical examination findings (fever, tachypnea, increased respiratory effort), leukogram abnormalities and elevated fibrinogen concentration, radiographic and ultrasonographic changes and culture of the organism from trachea-bronchial aspirates. Extra-pulmonary manifestations of *R. equi* infection have been described. Treatment is by prolonged administration of antibiotics such as erythromycin or azithromycin alone or in combination with rifampin; other drugs have also proven effective.

Prevention of *R. equi* pneumonia has been an area of intensive research; however, vaccination efforts have not been very effective to date. Administration of hyperimmune plasma may reduce disease severity and delay onset of disease, and is used routinely on many affected farms. Recent evaluation of metaphylactic antibiotic therapy has shown promising results but needs to be evaluated further before making general treatment recommendations.

Interstitial pneumonia

Interstitial pneumonia has been described in older foals and adult horses; however, the etiology is date ill-defined as no consistent viral or bacterial cause has been identified. Interstitial pneumonia in foals typically presents with an acute onset and rapid progression to respiratory distress; however, the condition has also been reported in foals presenting for chronic respiratory disease. Foals show tachypnea, coughing, nasal discharge, nostril flaring and marked abdominal breathing effort, and typically have adventitial lung sounds (crackles and wheezes). Presence of pyrexia is variable. Routine bloodwork may reveal leukocytosis or hyperfibrinogenemia. Radiographs show a diffusible interstitial pattern, which usually extends throughout the lung field. Cytology of tracheobronchial aspirates is most consistent with a non-septic neutrophilic inflammation, and culture may yield varying bacterial species. As viral infections, specifically those with Equine Influenza Virus and Equine Herpesvirus-2 and -5, have been suggested as a cause, testing may be warranted. Histopathologic evaluation of lung biopsy specimens has also been suggested for diagnostic purposes.

Treatment of interstitial pneumonia combines broad spectrum antimicrobial and anti-inflammatory therapy and typically includes administration of corticosteroids. Supportive care may include oxygen insufflation, administration of bronchodilators or inhalation therapy. Specific antiviral therapy (valacyclovir) has also been suggested.

Prognosis for this condition is guarded, but may depend on the severity and duration of disease as well as intensity of treatment and available resources. All foals in one retrospective study of chronic interstitial pneumonia survived, and their owners reported normal performance capacity. Conversely, poor response to medical therapy and a high case fatality rate have been reported in foals with the acute form of interstitial pneumonia.

References


Colic and Diarrhea in Foals
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Colic and diarrhea are common presenting complaints in foals and can pose a diagnostic challenge to the attending clinician. Based on the 2005 NAHMS (National Animal Health Monitoring) study of equine operations in the United States, colic and diarrhea were responsible for 1.5% and 6.4%, respectively, of foal deaths during the first 30 days of life. Diagnostic evaluation of the colicky foal is hindered by the inability to perform transrectal examination; however, this limitation is compensated for by an increased usefulness of imaging modalities such as radiography, contrast radiography and ultrasonography. Evaluation of colicky foals should focus on differentiation between surgical and nonsurgical conditions in order not to delay surgical exploration in suitable candidates. Congenital conditions such as hernias and atresias must be considered in young foals presenting with colic. When evaluating diarrheic foals, clinicians should keep in mind that diarrhea is a common clinical sign of sepsis, and evaluation of passive transfer as well as completion of a sepsis score or other appropriate testing should always be performed.

Meconium impaction

Meconium impaction is a common cause of colic in neonatal foals and generally responds well to medical therapy. Meconium impactions may occur as secondary problems in septic, dehydrated foals, foals with failure of passive transfer (as colostrums acts as a laxative) and foals with perinatal asphyxia syndrome. Routine administration of enemas post-partum likely prevents many cases of meconium impaction; however, clients must be carefully advised on appropriate administration of enemas in young foals.

Diagnosis of meconium impaction is generally based on clinical signs of colic in a foal of the appropriate age that may not have been observed to pass meconium. Abdominal distension may be present and pain can vary from mild and intermittent to severe. Careful digital rectal examination may reveal firm meconium and may further suggest a narrow pelvis as a potential contributing cause. Retrograde administration of contrast material for radiographic demonstration of meconium impaction has been described but is not routinely required. Important differential diagnoses include atresias and Overo Lethal White Syndrome in paint foals.

Treatment of meconium impaction combines fluid therapy (oral or IV) with pain management. Withholding of milk is often useful in controlling pain and abdominal distension, but should be done carefully to avoid a negative energy balance and weakening of the foal. Enemas are often employed and various options are available. Repeated administration of phosphate-based enemas is generally not recommended due to the potential for hyperphosphatemia. Retention enemas with acetylcysteine can be useful in cases refractory to treatment and if the impaction is thought to be more proximal than the rectum and distal small colon.

Uroabdomen

Uroabdomen typically results from dorsal tears in the urinary bladder, but can also be secondary to disruptions of other parts of the urinary tract. Foals are typically several (2-5) days of age by the time clinical signs occur, which may include lethargy, failure to suck, abdominal distension, dyspnea and colic. Affected foals may or may not have been observed to urinate following birth.

Diagnosis is based on presence of urine in the abdominal cavity, which can be confirmed using ultrasonographic evaluation and sample collection by abdominocentesis. A fluid to serum creatinine ratio greater than 2:1 is generally accepted as confirmation of uroabdomen. In some cases, a collapsed bladder or a bladder defect may be visible. Supportive diagnostic findings include electrolyte abnormalities (hyponatremia, hypochloremia, hypernatremia) in foals that have not undergone fluid therapy prior to sample collection.

While treatment is ultimately surgical in most cases, medical stabilization prior to general anesthesia is imperative, and constitutes an emergency in foals exhibiting neurologic, cardiac or respiratory complications. Fluid therapy along with drainage of the accumulated urine is indicated until electrolyte balance is restored as much as possible. Broad-spectrum antibiotic coverage is also indicated, especially when abdominal drains are left in place for intermittent or continuous drainage. Some clinicians recommend placement of an indwelling bladder catheter for several days following surgical repair in order to reduce pressure on the bladder and prevent suture dehiscence.
Diarrhea

Recognition of diarrhea as a problem is straightforward; however, establishment of an etiologic diagnosis can be quite challenging. Infectious causes of diarrhea must always be considered and appropriate biosecurity measures instituted to prevent transmission of disease to other foals. Infectious causes of diarrhea must also be primarily considered when dealing with an outbreak of diarrhea on a farm, or with repeated cases of diarrhea in a breeding operation. Diagnosis of “foal heat diarrhea” is probably not appropriate in foals that are showing additional clinical signs such as lethargy and dehydration, and until more serious causes of disease have been ruled out.

Important infectious causes of diarrhea include viral (rotavirus, coronavirus), bacterial (Salmonella, E. coli, Clostridia, Aeromonas spp., Lawsonia intracellularis), parasitic (Parascaris equorum, Strongylus vulgaris, Strongyloides westeri) and protozoal (Cryptosporidium spp.) etiologies. Non-infectious causes include dietary disturbances (especially in milk replacer-fed foals), gastric ulceration and lactose intolerance. Antibiotic-associated diarrhea is also considered common in foals, although it is typically not as severe as, and may be more amenable to treatment, than, the same condition in adult horses. Diagnostic testing should be tailored to the suspected cause; however, failure of passive transfer and sepsis should probably always be considered a possibility (especially in younger foals) and appropriate testing must be undertaken.

General principles of fluid therapy apply in diarrheic foals, and specific therapy should be based on evaluation of hydration status, electrolyte and acid-base balance as well as nutritional needs. Plasma administration is indicated in patients with failure of passive transfer, those with hypoproteinemia and if a specific treatment effect of plasma can be expected. Withholding of milk to “rest” the intestines can be helpful, but must be practiced with caution to avoid a negative energy balance. Parenteral nutrition may be considered in cases of prolonged diarrhea in anorexic foals or those that exhibit weight loss.

Additional treatments for diarrheic foals – depending on the suspected diagnosis – include anti-ulcer medication, anti-diarrheals (bismuth subsalicylate, charcoal, smectite), anti-inflammatories, anthelmintics, and antibiotics. Probiotics are often given but have questionable efficacy and, in some cases, have resulted in adverse effects.

Prevention of diarrhea should address passive transfer and general peri- and post-partum hygiene. As the mare must be considered a potential source of infectious organisms, some clinicians advise washing the mare’s hind end as well as changing the bedding (if applicable) prior to allowing the foal to suck. Administration of colostrum by stomach tube prior to allowing the foal to udder seek may also be considered. A rotavirus vaccine is commercially available and may be useful in herd outbreaks of this condition. Metaphylactic antibiotic therapy may also be indicated in certain cases; however, due to economic concerns and potential side effects, this approach should probably be based on a definitive diagnosis and sensitivity testing of the offending organism.
Endometritis: Update on Pathogenesis and Therapy

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Context and Significance
Endometritis affects 15% of mares and is a major cause of lower pregnancy rates. It is the third most common medical condition in horses. Mares that are prone to repeated episodes of endometritis are recognized as having lower pregnancy rates and are called subfertile. Hughes and Loy stated that “Each missed conception and each pregnancy loss becomes a major economic setback for the breeder, who is battling the season, life expectancy of the mare, availability of the stallion, rising cost of management, and interest on their investment.”

In this review the predisposing factors that culminate in endometritis, the new information on the pathogenesis of endometritis and the principles of treatment and therapy for this condition will be discussed.

Predisposing factors
The long term reproductive health of the mare depends on the: mare’s age, breed, perineal (vulvar), and pelvic conformation, immunologic constitution, reproductive history, and internal physical features such as the vestibulovaginal sphincter, cervix, ovaries and uterus. Breeds such as the Thoroughbred, where the focus is more on performance and breeds with a narrow genetic basis such as the: Tennessee Walker, Clydesdales, Shires and Friesians are more prone to subfertility. Older mares are over represented in the population of mares with endometritis presumably from age related deterioration of their anatomic barriers to infection, and the cumulative toll of inflammatory and degenerative changes in the uterus. There is also evidence that the immune system of the mare ages as well “inflammaging.”

Perineal Conformation
Perineal conformation is very important for reproductive health. There is a big focus on the shape of the perineum and tilt of the vulva of brood mares. The vulva should be vertical in its orientation and in line with the anus. The mares should have 1/3rds of the vulva above the pelvic floor and 2/3rds below. In defective conformation the top of the vulva is recessed inward and slopes downward below the anus. Opening the lips of the vulva should not let the air rush in (pneumovagina). Air rushing into the vagina when the vulvar lips are parted is called “wind sucking.” Passage of a speculum should be met with resistance. The physical barriers, such as the vulvar seal and the position of the vulva relative to the pelvic floor, prevent manure from entering the reproductive system. If these are suboptimal the mare will have increased contamination of the reproductive tract with genital flora and are predisposed to infections such as endometritis. For example pneumovagina is a common predisposing factor for endometritis, and is usually due to a conformational defect such as: defective vulvar shape, or abnormal angulation of the vulva, so called tipped perineal conformation.

In addition mares that have cervical injuries, tumours of the reproductive tract, endocrine disturbances, abnormal physiological responses (such as long cervix that does not relax while in estrus), or a dependent uterine location are prone to excess genital contamination due to impaired physical barriers.
Pathogenesis of Endometritis

Endometritis is inflammation of the endometrium (mucosa and submucosa of the uterus). The endometrium contains the luminal epithelium, stratum compactum, and stratum spongiosum of the lamina propria. The endometrium is an essential tissue for the nurturing of the equine embryo, and it contributes to supporting implantation, endocrine / immunologic / nutritional and other functions of fetal and placental development. There is a physiologic and a pathologic form of uterine inflammation.

Mild acute physiologic endometritis is part of a normal mare’s constitution at the time of main reproductive events such as breeding and parturition. The endometrial inflammation is transient and short lived. This process helps a mare to remove bacteria, sperm and debris in preparation for the embryo.

The main means of studying endometritis became the use of an experimental infection model using the most common genital isolate, *Streptococcus equi zooepidemicus* (*Strep*). This Gram positive organism is commonly found on the mare. Mares when challenged were subsequently divided into 3 groups. Mares with injuries to the cervix or other non-uterine based causes of infertility were excluded. Fertile mares were noted to resolve the intrauterine *Strep* challenge quickly, while subfertile mares were noted to develop excess amounts of free intrauterine fluid, and persistent inflammation 72 hours post inoculation. In the older literature mares that could clear low level *Strep* infections were called “Resistant” those that could not clear the *Strep* challenge were called “Susceptible” (Hughes, 1969). Over time studies showed that these mares have an underlying defect in uterine clearance due to poor myometrial contractility (Rigby, 2001). The myometrium is further disadvantaged by having to contract against gravity in mares with a dependent uterine location. The myometrial contractions were reported to be delayed in onset and weaker in susceptible mares (Troadsson, 2008). Weak myometrial contractions were reported to contribute to poor cervical / uterine clearance and lymphatic drainage. The underlying mechanisms for the delay in onset of the uterine contractions and poorer myometrial contractility remain unclear. Rigby et al (2001) reported that intracellular calcium may be involved.

Studies showed that breeding introduces sperm cells, seminal plasma, bacteria and debris into the uterus. Bacteria, sperm and debris were described as the main agents of inflammation with seminal plasma having a modulating role on uterine inflammation (Troadsson M.H.T., 2001; Troadsson et al., 2008).

Bacteria

Post mating endometritis is most commonly used by Gram positive bacteria. Gram negative and mixed infections are also reported. Gram Positive organisms account for 70% or more of all infections, and are due to infection with the first 2 pathogens listed in Table 1. Bacteria may be present in the uterus before breeding, the conditions induced by breeding may facilitate growth in some mares, and bacteria are introduced by breeding or insemination. Breeding may also create the right conditions for bacteria to grow.

<table>
<thead>
<tr>
<th>Gram Positive</th>
<th>Gram Negative</th>
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<tr>
<td><em>Streptococcus equi zooepidemicus</em></td>
<td><em>Escherichia coli</em></td>
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<tr>
<td><em>Staphylococcus aureus</em></td>
<td><em>Klebsiella pneumoniae biotype 1</em></td>
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<tr>
<td><em>Actinobacillus</em></td>
<td><em>Pseudomonas aeruginosa capsule type 5</em></td>
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<tr>
<td>Other <em>Strep</em></td>
<td><em>Enterococcus</em></td>
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<tr>
<td><em>Anaerobes</em></td>
<td><em>Fungal</em></td>
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<td><em>Bacteroides</em></td>
<td><em>Aspergillus (fungus)</em></td>
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<tr>
<td><em>Candida (yeast)</em></td>
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Table 1:
Common Causes of Endometritis in Mares

Immunoglobulin

Historically the amount and type of opsonising antibody (IgG) was evaluated as an underlying cause of excess inflammation in mares (Asbury, 1984). This was the basis for uterine therapy using plasma as a source of IgG in subfertile mares. The recognition of *Strep* as main pathogen led to attempts to make a vaccine against *Strep* infection. These attempts were unsuccessful in remedying the inflammatory response of susceptible mares (Causey et al., 2006; Weiss et al., 2007). Sperm in utero are all coated in IgG. This IgG is present in seminal plasma and the uterine and tubal secretions (Rumke, 1974; Aguilar and Reyley, 2005). The opsonizing IgG is chemoattractive to polymorphonuclear cells (PMNs), which are predominately neutrophils, to phagocytose these sperm cells.
Complement

The IgG-coated sperm activate the complement cascade by cleavage of C5 and release of factors C5a, C5b, and C3b (Troedsson et al., 2001). These factors in turn activate the rest of the complement cascade within the endometrium and attract PMNs. Specifically, C5a is chemotactic for neutrophils and induces their activation leading to degranulation, oxidative bursts, and changes in adhesiveness. The C5a also induces other signs of inflammation such as vasconstriction and increased vascular permeability (Miller and Krangel, 1992). The C3b fragment aids activated PMNs to bind to activated spermatozoa (Troedsson, 2006b). Resistant and susceptible mares have similar levels of IgG and C3 for the first 24 hours, but after 36 hours S mares showed a continual decline whereas R mares showed an increase in levels (Troedsson et al., 1993).

PMNs and Neutrophils

The attracted PMNs have multiple roles including phagocytosis of bacteria, debris, and sperm. No difference in PMN function and ability has been detected between R and S mares (Zerbe et al., 2004). It was reported that sperm induced the breakdown of complement and that the complement fragments C5a and that C3b induced neutrophil chemotaxis (Watson, 1988; Troedsson M.H.T., 2001). The phagocytosis by neutrophils releases prostaglandin F2α (PGF2α) which elicits myometrial contractility and expulsion of debris through the cervix. The release of PGF2α occurs by the metabolism of arachidonic acid via the cyclooxygenase pathway (Troedsson, 2006a). Leukotriene B4 produced from the lipoxygenase pathway is chemotactic to equine neutrophils in vitro and is a potent stimulator of the phagocytic activity. Neutrophil influx was believed to be associated with prostaglandin secretion and production of nitrous oxide (Alghamdi et al., 2005).

Seminal Plasma

The inflammatory response was believed to be modulated by seminal plasma (Troedsson M.H.T., 2002). One author showed that seminal plasma: decreases uterine contractions when compared to sperm alone, and that neutrophil counts are higher in mares bred with sperm plus seminal plasma rather than sperm alone (Portus et al., 2005). Troedsson’s group reported that seminal plasma inhibited the chemotaxis of neutrophils in vitro, was bacteriostatic and decreased the duration of breeding-induced inflammation but not the severity (Troedsson et al., 2001; Troedsson et al., 2002). Seminal plasma may modulate the inflammatory response itself or modify myometrial contractions (Troedsson, 2006a). Seminal plasma protects sperm from opsonization within the uterus thereby allowing them to reach the oviduct (Troedsson et al., 1993; Dahms and Troedsson, 2002; Troedsson et al., 2002). It had been reported that a seminal plasma protein may protect live sperm (Troedsson et al., 2005). Authors suggest that the concentrated nature of frozen semen and lack of seminal plasma may trigger more inflammation.

Immune responses — Female Reproductive Tract (FRT)

Understanding the immunity of the FRT is of worldwide importance due to the high incidence of diseases such as endometritis and placentitis in mares and the presence of sexually transmitted diseases such as contagious equine metritis (CEM), equine viral arteritis (EVA), coital exanthema (EHV III), and dourine (Trypanosoma equiperdum) (Bridges and Edington, 1986). It was recognized that the immune system in the female reproductive tract has evolved under diverse evolutionary pressures to have the ability to: support normal vaginal flora, respond to allogeneic sperm, eliminate sexually transmitted viral, bacterial and protozoal pathogens, and support the development of an immunologically distinct fetus and placenta (Wira, 2002). The components of the inflammatory response that had been identified to date included complement fragments and phagocytic cells such as neutrophils which are part of the innate immune system in the mare.

Mucosal Immune System

The breakdown of complement and the activation of phagocytic cells are now known to be part of the innate immune system. The typical endometrial response of the susceptible mare is an excessive suppurative endometrial inflammation. Researchers began to question what treatments could be used to modulate the inflammation and to evaluate the nature or the mare’s immune response to inflammatory challenges such as sperm and bacteria. It was also known that inflammation usually peaked at 6 – 12 hours in resistant mares (Kotilainen, 1994).

Innate Immune System

The innate immune system is part of a continuum with the adaptive immune system which collectively coordinates and enhances the ability to conceive, support pregnancy, and eliminate challenges that threaten the health of the dam or the success of the pregnancy. In brief, the adaptive immune response is a pathogen specific response that involves the presentation of antigen by antigen presenting cells (APCs). The antigen forms a complex with the Major Histocompatibility Complex (MHC) class II receptor that induces T cell activation (Watson and Dixon, 1993). The activation of T cells leads to cytokine production, cytotoxicity, and eventually to specific antibody synthesis. Antibody production by B cells is termed humoral immunity, and destruction of infected cells by T cells is
termed cell mediated immunity. The mucosal immune system is that portion of the immune system which protects the various mucous membranes of a horse, such as the urogenital, gastrointestinal and respiratory systems, from invasion by potentially pathogenic microbes. It protects mucous membranes against infection, prevents the uptake of unwanted antigens, microorganisms, and other foreign materials, and moderates the horse’s immune response to that material. Mucosal immunity therefore integrates innate immune mechanisms in protecting a horse from pathogens (Watson and Dixon, 1993; Wira, 2002; Zerbe et al., 2003).

The innate immune response therefore is distinct from the adaptive response which involves memory cells and humoral antibodies, in the: immediacy of the response, the cell types involved (phagocytic cells such as macrophages, neutrophils, dendritic cells, natural killer cells, and epithelial cells), and the universality of the receptors. It is only recently that a series of receptors called Toll-like receptors (TLRs) have been recognized as having a role in uterine inflammation.

**Toll-like Receptors and Pathogen — associated molecular patterns**

The innate immune system has evolved to recognize foreign structures called pathogen-associated-molecular patterns (PAMP’s). These conserved molecular patterns of the PAMPs are recognized by receptors called pattern recognition receptors (PRR). Included in the PRR are TLRs. Toll-like receptors were first recognized in drosophilia (fruit flies) and were found to be highly conserved among animals. The TLRs are a family of protein receptors, and at least 15 TLR have been described. The TLRs have been shown to be expressed in macrophages, neutrophils, lymphocytes, endothelial cells and epithelial cells in horses (Suri et al., 2006; Berndt et al., 2009). The downstream signaling that results from the binding of PAMPs to TLRs involves a host of other cytokines and other adaptors that lead to the recruitment of immune cells, and as well as the production of intracellular and secreted anti-microbial factors. Characterization of the signaling pathways activated by specific TLR binding showed that different intracellular messengers are activated following binding of different TLRs. It is now established that Toll-like receptor 4 (TLR4) regulates the response to lipopolysaccharide (LPS) while Toll-like receptor 2 (TLR2) does the same for products of Gram positive bacteria. The significance of these receptors and their activators to equine innate immunity is that they concern the main causes of endometritis (Berndt et al., 2009).

While infections with Gram positive organisms such as Strep and Staph account for 70% of endometritis cases there are a number of cases caused by pure Gram negative infections such as E. coli, Klebsiella pneumoniae, Pseudomonas aeruginosa or mixed infections. The clinical responses of mares to Strep and E. coli were found to be not the same and that different TLRs were likely to be activated by these bacteria (Zerbe et al., 2003).

**Pro-Inflammatory Cytokines**

The new insights into the inflammatory responses of the endometrium lead researchers to investigate the pro-inflammatory cytokine profiles of mares susceptible and resistant to endometritis. Fumuso showed differences between resistant and susceptible mares in the levels of expression of endometrial cytokines including of interleukins (IL) such as IL-1β, IL-6, and Tumour Necrosis Factor alpha (TNF-α) mRNA in non-bred resistant and susceptible mares (Fumuso et al., 2007). This work suggested that mares susceptible and resistant mares might have differential immunologic responses. Interleukin IL-1β, IL-6 and TNF-alpha are recognized as pro-inflammatory, and increases in their expression are associated with increased inflammation. The mediators and pathways that trigger cytokine secretion are presently being elucidated in equine tissues and are known to include activation of TLRs. As our understanding of these process evolves our ability to development immunomodulatory therapies will improve.

**Therapy for endometritis**

Figure 1 shows a schematic of the pathogenesis of endometritis. The principles of therapy involve the elimination predisposing factors, treating infection/inflammation, recognizing that mechanical clearance is a problem, and managing and treating mares to reduce the inflammatory burden (one breeding per heat, lavage and ecbolics such as oxytocin). The therapies for most infectious conditions generally begin with the elimination of the predisposing causes and a focus on prevention. For example a Caslick surgery is performed to prevent pneumovagina in athletic mares and to stop pneumovagina and fecal contamination of the reproductive tract in mares with undesirable perineal conformation. Mares with pre-existing bacterial infections or inflammation should be treated to eliminate inflammatory debris and bacteria. Diagnosis of endometritis is made by finding >10% neutrophils on an endometrial swab cytology in combination with isolation of bacterial pathogen by uterine culture or low volume lavage. Endometrial biopsy or biopsy culture is also a useful tool to diagnose endometritis.
Therapeutic Approach

1. Diagnose, treat and eliminate pre-existing endometrial infection and inflammation. Treat with the appropriate antibiotics identified by culture, systemically if possible for 1 week. Perform the ultrasound and see if there is free intrauterine fluid, perform a vaginal to look for discharge. Obtain an endometrial sample for cytology and culture to obtain a preliminary diagnosis. A Diff Quik and Gram stain of the cytology will allow you to determine if there is evidence of infection (neutrophils and microorganisms). Use Gram stain to determine class of bacteria to determine what type of antibiotic to consider. Submit culture if neutrophils or bacteria are present.

2. The underlying defect is poor myometrial contractility which results in the accumulation of debris and poor contractility, therefore mechanical flushing of the uterus using a bivona type catheter and sterile warm saline or LRS is used (generally 2L in drain 2L out and repeat until fluid coming back is clear if the mare is infected or if she develops fluid accumulation post breeding. Use uterotonics stimulation by teasing the mare, treating with oxytocin (pre and post ov) or prostaglandin (preov). May treat up until 3 days after ovulation (with ovulation as day 0).

3. Breed only once at the right time. Bred once in subsequent heat. This means we usually induce ovulation with hCG when the preovulatory follicle is between 35 - 40 mm).

4. Monitor for ovulation and follow the mare post breeding for fluid accumulation, or poor tone. Post-breeding treatment is usually required. Monitor for post-mating inflammation and treat as needed using lavage, oxytocin and antibiotics.

5. If Gram negative allow a cycle off, or short cycle mare with PG to breed

6. Intensify the management if the mare repeatedly experiences endometritis. Prebreeding saline uterine lavage in estrus a day before breeding, followed by oxytocin 20 - 40 IU. Start antibiotic therapy. Limit the number of breedings (use hCG when the preovulatory follicle is between 35 - 40 mm). Lavage at 4 - 6 hours post breeding as above with oxytocin 20 - 40 IU. Viable sperm are in uterine tube by 4 hours post-mating. Follow post breeding for fluid accumulation, may need to lavage 1-2x daily until the uterus is clean. Perform or Close Caslick after done with post-breeding therapy

7. Other treatments: Chemical curettage (100 mls kerosene) administer to mare in diestrus then give PG, mechanical curettage, DMSO New therapies

8. Other: Expose mare to sexual stimulation while still in heat to cause endogenous oxytocin release, or use low dose prostaglandin pre ov (125 µg cloprostenol), or carbocetin (0.175 mg IV)

9. Treat with Regumate (10cc 0.44 mg/kg) if tone remains poor

10. If mare continues to repeat with the same infection and fails to conceive, consider reculture, biopsy, give a cycle off, clitoral sinus and fossa ablation

11. Immune stimulants, steroids

12. Referral

How do I chose an Antimicrobial? What about the route of delivery?

Microbial therapies are usually initiated by identifying the type of bacteria on a cytology. The majority of the infections are due to Gram positive cocci so the presence of coci in a cytology generally means a Strep or Staph infection, which are usually sensitive to penicillin. Controversy continues over whether to use intrauterine or intramuscular therapy, however usually you achieve better MIC levels over time with IM treatment. Intratu-terine antibiotics result in uterine invasion, they are costly and they may cause super- infection, however in some mares with pathologic biofilms they may be more effective (LeBlanc and Causey, 2009). The expensive antibiotics are sometimes used IU, however they may eliminate the organisms in other reservoirs in the mare such as the clitoral sinus or clitoral fossa. Note mares do not have the temperament for twice daily IM injections.
Match the antibiotics with the culture and sensitivity

**Gram positive**  Procaine pen G 22,000 IU/kg IM BID for 5 – 7 days, Oral TMS 30 mg/ kg SID PO 5 days, cefotiofur IU (not for IM use as it doesn’t penetrate the endometrium)

**Gram Negative**  Gentocin (buffered with bicarb) 1 - 3 g IU or 6.6mg/kg IM, ticarcillin 3 g IU, or 40 – 80 mg/kg TID IM or IV, amikacin 7.5 mg/kg BID IM.

**Anaerobes such as Bacteroides**  Metronidazole 15 mg/kg QID PO

**Yeast fungi**  betadine 0.5%, vinegar 1-2%, clotrimazole 500 – 700 mg IU, amphoteracin B

### Summary - Intramuscular versus Intrauterine antibiotics

Antibiotics are chosen based on clinical signs, culture and sensitivity, and those that achieve good endometrial levels. Generally IM is better, IU route is more expensive, shorter duration, invades the uterus, and may lead to superinfection with yeast or fungi.

### Immunomodulation

There is currently interest in investigating the use of immunomodulation as a therapy for pathologic endometritis. An understanding of which mares to treat, for how long and with what substances remains to be elucidated.

### Glucocorticoids

There are reports of the use of glucocorticoids (dexamethasone 25 mg, or prednisolone 0.2 mg/kg) to modulate the endometrial inflammatory response in mares (Bucca et al., 2008; LeBlanc and Causey, 2009). The exogenous administration of glucocorticoids has been shown to regulate TLR levels in certain animals and tissues. Historically members of the veterinary profession were reluctant to glucocorticoids systemically in bacterial infections because of the suppressive effects on leukocytes. Indeed notable examples where locally high delivery of steroids worsened patient outcomes included ophthalmic application for corneal ulcers, mastitis in dairy cows, and an association the development of laminitis in horses. Glucocorticoid therapy was used primarily for treatment of allergic airway disease, shock, and non-infectious inflammation such as autoimmune disease.

Glucocorticoids decrease inflammation and suppress the immune system by diffusing through the lipid membrane into the cytoplasm and binding to glucocorticoid response elements on DNA which inhibit the production of IL-1 and IL-6 and other cytokines (Almawi et al., 1996) (Brattsand and Linden, 1996). Glucocorticoids also inhibit the downstream effect of interleukins which have a positive feedback loop on further interleukin production and on the recruitment of T-cells (Almawi et al., 1996). Another proposed mechanism of glucocorticoid function is the binding of glucocorticoid-receptor complexes to pro-inflammatory transcription factors which leads to their inhibition and a decrease in the half-life of cytokine mRNAs (Brattsand and Linden, 1996).

Dell’Aqua performed a study evaluating the pregnancy rate of normal and barren mares with and without steroid administration after breeding. Initial results are encouraging, however, the true reproductive status of these mares was not described, neither was there any mention of any adjunctive therapies used on each group of mares so a true evaluation of the efficacy of glucocorticoid treatment is still pending (Dell’ Aqua Jr et al., 2006). In other studies there was no effect of dexamethasone treatment on pregnancy rate (Bucca et al., 2008).

### Immune stimulants

For the past 10 years immunomodulatory therapy has been available for horses and products include mycobacterial cell wall extracts derived from *Mycobacterium phlei* which is available commercially as Equimmune (company and address), killed *Propionibacterium acnes* which is available as Equistim (company and address), and *Parapox ovis* which is available as Zylexis (Pfizer Inc., city). Mycobacterium cell wall extracts and *Parapox ovis* preparations are used prophylactically in cases where horses are at high risk of exposure to respiratory pathogens. Presumably the products are used to non-specifically stimulate immunity in the respiratory tract. Killed preparations of *Propionibacterium acnes* have been associated with higher levels of cell mediated CD-8 T cell activation and interferon-gamma secretion, and has been termed a biologic response modifier when used in respiratory prophylaxis. Both the MCWE and the *Propionibacterium acnes* preparations are labeled for use in cases of endometritis. There are few reports however of these immunomodulatory products being used as a sole therapy in mares with endometritis.

### Mycobacterium cell wall extract

Immunomodulators have been promoted as substances that “normalize” the uterine immune response to breeding Fumuso examined the inflammatory response to *Strep zoo* endometritis in susceptible mares treated intravenously with MCWE. The MCWE administration did not affect cytokine profiles during estrus and diestrus in in R and S mares, however after breeding...
MCWE treated S mares had decreased IL-1β and IL-6 mRNA levels compared to non-MCWE-treated S mares (Fumuso et al., 2007). It was concluded that “resistant” mares had lower levels of mRNA for proinflammatory cytokines (IL-1β, IL-6, and TNF-α) than “susceptible” mares, and that breeding up-regulated mRNA expression in both “resistant” and “susceptible” mares, with this effect persisting into diestrus in “susceptible” mares. The immunomodulator MCWE was reported to decrease IL-1 mRNA expression (IL-1 is believed to be secreted by activated macrophages). The authors suggest that the MCWE restored homeostatic regulation of local inflammation (Fumuso et al., 2005).

**Propionibacterium acnes**

A commercial preparation of *Propionibacterium acnes* is available for the treatment of endometritis with a label claim that treatment with *Propionibacterium acnes* “normalizes” a mare’s inflammatory response to insemination. One field study reported an improvement in pregnancy and live foal rates when breeding was performed from 2 days before to 8 days after systemic administration. However the product was not used as the sole therapy for the treatment of endometritis (Rohrbach et al., 2007).

**Philosophy**

Lastly don’t forget it is okay to advise not breeding some of these mares. We need to cull more animals out of the breeding herd. We geld a large number of stallions, but we spay very few mares. Advice on broodmare selection is an important part of a veterinarian’s role in management.

**References**


Figure 1. Pathogenesis of Endometritis

Opsonization of Sperm and Bacteria
Activation of TLR
Breeding / Insemination
Inflammation
Bacteria, Sperm, Debris
Seminal Plasma
Uterine Secretion: Complement and AB
TLR
C3a C5b
Neutrophil Recruitment

Phagocytosis of Sperm
Activation of Complement
Production of Cytokines Chemokines
Prostaglandin
iNOS Nitrous Oxide
Interkeukins IL-8
IL1-β, IL-6, TNF-α
Myometrial contractions
Mechanical Clearance: Expel debris and bacteria through the cervix
Inflammation controlled = conception
Breeding Strategies for Broodmares
Claire Card, DVM PhD Diplomate ACT

Introduction
Reproductive management of mares primarily follows the natural changes in day length. Pasture breeding, hand breeding using teasing with estrus detection by a stallion, and artificial insemination (AI) of cooled and frozen semen are still commonly used in Canada. In Europe it has been estimated that about 80% of all mares are bred using AI, and in Alberta it was reported that 25% of mare owners now use artificial insemination as a breeding strategy in some, or all of their mares. Due to the large geographic area of Canada the ability of horse owners to access high quality genetics will continue to rely on either movement of mares and or semen. Transportation costs and risks associated with the movement of semen are much lower than the costs of transporting horses. The future of the equine industry will likely continue to include expansion of AI which requires an understanding of the mare’s physiology and options for breeding management.

This paper will briefly review the physiologic (seasonal) changes in the mare and how to advance the breeding season of the anestrus or transitional mare; the basis for hormonal therapy, the hormones used and the success rates obtained; and breeding strategies for use with cooled and frozen semen programs.

The Physiologic Cycle – Seasonal changes and transitions in mares
The mare is a seasonal breeder and she senses changes in day length through retinal receptors and the pineal gland, which influences her secretion of melatonin. Melatonin increases with increasing darkness. Melatonin lowers GnRH secretion and decreases ovarian activity. Additional input from other hypothalamic factors such as Kisspeptins may also be important. When day length shortens, less and less GnRH is secreted, which corresponds to lower amounts of FSH and LH being secreted. The effect of photoperiod on mares is so strong that around 85% of mares will enter a fall transition in October, and cease all ovarian activity becoming anestrus by November. The anestrus period typically lasts from November to February. Spring transition may begin in February or March. Once days lengthen after the spring Equinox in March the increasingly stimulatory photoperiod induces increased GnRH secretion, and follicle wave development. Three follicular waves in what is termed a transitional phase, which occurs in March and April and finally a mature wave will occurs resulting in an ovulation that begins the physiologic breeding season (usually late April or early May)(Ginther, 1992).

If a mare owner wishes to breed outside of the natural season, such as often occurs with Thoroughbred horses, and halter type Quarter horses, one option is to make a plan in the fall forstimulating the mares to cycle outside of the natural breeding season. January 1st is the arbitrary birth date for all foals in these breeds. There is an idea that older yearling and 2 yrs perform better. The photoperiod of mares foaling in winter may also have to be manipulated if the owners wish to breed the mare back right away. Options for advancing the breeding season include a manipulation of the mare’s photoperiod, a use of hormones to stimulate ovarian activity or both.

Manipulation to hasten the onset of the breeding season

Artificial photoperiod
The reason the planning is important is that a ratio of 16 hours of daylight to 8 hours of darkness, or 2-3 additional hours of artificial light beginning at sunset needs to be provided. One 200-watt incandescent bulb or two 40-watt fluorescent bulbs are used in each box stall to give at least 100 lux of light in all corners of the stall. There is a dose response to the power of the light, with less light will have some effect but will be less effective. The mare needs to have the light during her photosensitive window which is metered from the onset of darkness. “Flash Lighting” protocols exploit this fact and a single one-hour exposure of artificial light, given 9-10 hours after sunset, has been shown to stimulate follicular development in anestrous mares. This is because the photosensitive window is about 10 hours after the onset of darkness. Following winter solstice, light increases at about 5 minutes per day, hence using flash lighting the time for lighting needs to be adjusted weekly to achieve the desired effect. Mares need at least 60 days of supplemental light exposure to become stimulated and the additional light must be continued at least until the spring Equinox. The easiest protocols have extra light added at the end of the day consistently until about 11pm.
Hormonal Therapy for Anestrus

Not all farms or management situations are suitable for artificial lighting. The simplest protocols to advance the breeding season hormonally include the use of injectable deslorelin. The physiologic mechanism behind deslorelin (a GnRH agonist) in seasonally anestrous mares is the stimulation of FSH and LH synthesis and release from the anterior pituitary which, in turn, induces follicular development and ovulation. Deslorelin is a potent GnRH agonist and is available as Ovaplon and as a compounded liquid product. Ovaplon pellets may be placed under the skin every 72 hours for up to 10 treatments until ovulation, with hCG being given when a follicle (F) reaches 35 mm. The injectable deslorelin product may be administered at a dose of 62.5 ug per mare BID until ovulation with an additional treatment of 2000 IU hCG administered when a follicle (F) reaches 35 mm. Mares with 20 mm F are more likely to respond than mares with no ovarian activity. Treatment of anestrus mares may result in 30-50% ovulation rates. Mares may not experience a second cycle and may need to be retreated if not in foal (Johnson A, 1987; Raz, 2009).

Anestrus and Transitional Mares

Hormonal and light regimes

New protocols describe the use of increased photoperiod combining 2 months of additional light with pretreatment with estradiol benzoate 11 mg/mare for 10 days before adding a dopamine antagonist, sulpiride 250 mg/mare SQ (Kelley et al., 2006). Others describe using supplemental light beginning in January for 2 weeks and然后 to the increased photoperiod daily administration of dopamine antagonists (sulpiride 1 mg/kg IM BID) or domperidone (1.1 mg/kg PO SID), which is administered for 3 weeks or until ovulation. Sulpiride appears to be more potent than domperidone (Mari et al., 2009). This research points to a potential role that prolactin or dopamine regulation may play in the control of deep anestrus and seasonality in the horse. Dopamine is a prolactin inhibiting substance. The use of a dopamine antagonist such as sulpiride or domperidone will increase prolactin. Prolactin normally increases as daylight increases. It has been suggested that lowering dopamine will increase the number of gonadotropin receptors in the ovary, or dopamine may act directly through ovarian receptors or at the hypothalamic level. The hormones may increase in receptor number or sensitivity may result in heightened responsiveness to the gonadotropins LH and FSH.

Hormonal regimes early transition

We have found the dopamine agonists to be less effective and to have a longer treatment to response time than treatment with either deslorelin, or eFSH in mares in early transition. Some authors say it is due to our low ambient temperature in Canada. Several recent studies have demonstrated that deslorelin (64 micrograms per day BID IM) or eFSH (12.5 mg BID IM) can be used successfully to induce ovulations in early transitional mares (mares that have at least one 20 mm follicle) when administered as twice daily treatments, or serial insertion of slow release deslorelin implants (2.1 mg each) (Raz, 2009). The mares receive a new implant every 3 days for up to 3 weeks or until they ovulate. Using these protocols the hCG is still administered to induce ovulation. Ovulations occur in approximately 78% of early transitional mares treated, and eFSH stimulates more ovulations per mare. The duration from the onset of treatment to ovulation is approximately 7 – 8 days. Hormonal treatment of early transitional mares may result in additional cycles following treatment however a proportion of mares (around 15%) will return to anestrus following the end of treatment, even if ovulation was induced, if they fail to become pregnant. Pregnancy rates following ovulations induced by hormonal means in early transitional mares are not significantly different than pregnancy rates following the first spontaneous ovulation of the year.

Hormonal Manipulation of Mares In Late Transition

The treatment options for mares in late transition (April) usually are progestagen (Regumate) or an ovulation induction agent such as hCG or deslorelin or both. The progestagen (altrenogest 0.044 mg/kg/day PO) is administered orally to the late transitional mare (>20 mm follicles in April) for 10 – 14 days. The treatment is proposed to alter the secretion of gonadotropins or change the sensitivity to them such that withdrawal of treatment results in an LH surge and ovulation, however most transitional mares are treated with hCG. Statistically progestagen treatment only advances the breeding season by 2 weeks. Mares with a >30 mm follicle may simply be treated with deslorelin (1.5 mg injectable, or 2.1 mg implant) or hCG (2000 IU IM) to stimulate ovulation. Transitional mares tend to be more LH rather than FSH deficient. Ovulation rates of around 50% should be expected.

Practice considerations - Early season farm work: Usually veterinarians begin farm calls in February and March to: perform prepartum checks, evaluate foals, examine mares after foaling to determine if they are ready to breed (large size of the uterus, presence of fluid means mare is not ready to breed), and to begin to evaluate the non-pregnant mares (maiden or barren mares) for evidence of transition or cyclicity. Mares that foal in March or later generally have a foal heat and then continue to have regular cycles, and are therefore often cycling ahead of the barren/open mares. The barren or maiden mares will usually exhibit some behavioral signs of heat (transitional estrus behaviour). The behaviour of these transitional mares is variable: some show heat for weeks, others on and off again. These open (non-pregnant) mares are usually in the transition phase where they will have some follicular waves but do not ovulate. The intensity of the transitional mare’s estrous
behaviour has a variable relationship with the size of the follicles on the mare’s ovaries. Owners who want early foals may ask you to try to treat the transitional mares to ovulate. However once a mare has had a spontaneous ovulation she will continue to cycle.

Age and Body Condition

Once a mare has ovulated she enters the physiologic breeding season from late April through October. Age and body condition also seems to play a role in the onset of cyclicity, with mares in poor body condition starting later in the season (July) and some young 2 year old mares or mares over the age of twenty may not start cyclical activity until June or July. Two - year old mares may have only a few heat periods before becoming transitional again (August). The aged mare may only have 2 or 3 heat periods per year. Many mares over the age of 24 do not have cyclic reproductive activity, they enter the equine equivalent of menopause. Mares that lose body condition excessively during lactation may develop an anestrus like state that may be related to negative energy balance or opioid inhibition of ovarian activity.

The Breeding Season

The 21 day estrous cycle of the mare can be divided into 2 phases based on sexual receptivity: estrus and diestrus. Estrus refers to the period during which mares are receptive to the sexual advances of the stallion. Estrus typically lasts 5-7 days and is driven by estrogen produced by developing follicles. Common behavioural responses of mares in estrus when teased with a stallion include raising of the tail, passive urination, repeated eversion and exposure of the clitoris (winking), and assuming a mating posture. The strongest expression of estrus is in mid to late estrus where the mare will posture, and stand to be mounted. Estrus signs and intensity may be very variable mare to mare and therefore are not considered highly reliable. Mares will foals at side are notorious for only showing heat briefly, usually when they are about to ovulate.

Diestrus refers to the period during which mares reject the advances of the stallion. As little as 12 hours post ovulation mares will reject the stallion. Progesterone production by the corpus luteum controls the behavioural responses of the mare during the 14-16 day diestrous period. When teased to a stallion, mares in diestrus will pin their ears back, kick, switch their tail, vocalize and attempt to actively move away from the stallion.

Examination of Mares in the Breeding Season

Veterinarians are asked to get mares ready for breeding, typically on farms where there are no stallions. A few examinations may be required to determine where in her estrus cycle the mare falls. When examining a mare it is best to use both palpation and ultrasound to determine if she is in diestrus or estrus. Data sheets including palpation, ultrasound and teasing information are valuable clinical tools.

During an examination the changes in the reproductive tract are recorded including:

By Palpation

Cervical and Uterine Tone is recorded as tone (typical of pregnancy), mod tone (typical of diestrus), mod soft (early estrus), soft (estrus).

By ultrasound

Edema is scored as 0 (none)(ovulation through diestrus), grade 1 light (first signs of estrus), grade 2 moderate (early estrus), grade 3 heavy (mid estrus), grade 4 extreme (late mid estrus)

Maximal follicular diameters are noted, and follicle consistency (firm smaller non-mature follicles; soft preovulatory follicles)

Presence of a CL is noted and its age (new (large 1/2 – 1/3 of the ovary or more, mid – organized 1/3 - 1/5 of the ovary, old small and echogenic)

Getting Mares ready for breeding

Teasing mares with a stallion is the natural way to determine when a mare is ready to breed. Rectal palpation and ultrasound are also used to confirm or to determine the mare's status. Early in the season (Feb – April) it is important to determine if mares are transitional or if they are cycling. This is done by evaluation of the uterine / cervical tone (which will be atonic or flaccid) and determination of the activity on the ovaries. Active luteal tissue and progressive follicle growth culminating in an ovulation means a mare is cycling. Transitional mares will have no progesterone, some follicle growth but will not ovulate. Therefore monitoring follicle growth on 2 or 3 visits spaced 3 days apart will allow a determination of the mare's status if careful records are kept.
Breeding Strategies

Hormonal Manipulation of mares

Hormonal therapy has become an integral part of routine equine breeding strategies practice. Hormones commonly used to manipulate the equine estrus cycle include: prostaglandin (PG), human chorionic gonadotropin, estradiol 17-beta, deslorelin (gonadotropin releasing hormone analogue), progestagen / progesterone.

Induction of Ovulation

Induction of ovulation is used to time the breeding of a mare with the availability of the semen. Two products are used hCG and deslorelin. Human Chorionic Gonadotropin (hCG) is used for the induction of ovulation in cycling mares (McCue et al., 2007). When administered to a mare showing signs of estrus with a follicle 30 > 35 mm in diameter, hCG will induce ovulation of that follicle 75% of the time within 24-48 hours. The average time to ovulation post injection is 36 hours. The use of hCG improves the efficiency of a breeding program by reducing the duration of estrus, decreasing the number of breedings at each estrus and provides a means of synchronizing ovulation with a stallion’s breeding schedule, or the availability of the semen. Forms, Doses and Routes of Administration of hCG - Chorulon, 1,000 IU per ml dose 750 - 2,000 I.U. I.M. or I.V. Ginther recently suggested that mares that tend not to ovulate in response to hCG may have higher anti-hCG antibody levels (Siddiqui et al., 2009). Deslorelin - is available as a 2.1 mg implant delivered SQ (Ovuplant) and as a liquid injectable compounded product. It is as effective as hCG with the mean time to ovulation is 40 hours, which is longer than hCG at 36 hours. Some mares will have a delayed return to estrus if the implant is not removed in 48 hrs. Some advise insertion of the implant in the vulva after local anesthetic (lidocaine is injected) and then removal at 48 hrs. It was reported to have a tighter bell curve on response of mares. Liquid formulation does not have the delayed return to estrus (1.5 mg IM). Main advantage is the narrower bell curve distribution of ovulations (36-44 hrs).

Prostaglandin and hCG or Deslorelin:

One of the simplest and least expensive means of inducing estrus is to use PG to bring a mare into heat. If the mares are cycling most owners want their mares treated to get them into heat quickly and bred quickly to reduce costs. This is usually done by finding mares not in heat (teasing or palpation and ultrasound) with CL’s (diestrous mares), evaluating them for follicle size, and then treating them with PG. The mean time from PG to the onset of estrus is 3 days, and the mean time to ovulation if using ovulation induction (hCG (Chorulon) or deslorelin (Ovuplant) is 8 days. The status of the follicles on the mare’s ovaries at the time of PG treatment determines how fast the mare will come into heat. Calculate that a follicle grows at around 3 mm a day and determine how many days it will take a mare to reach a follicle size of about 30 - 35 mm. An estrus mare with a follicle size of 30 - 35 mm will show early signs of heat, and typically will respond to ovulation induction. In some cases you may not wish to induce ovulation but plan on breeding the mare the day the follicle is over 40 mm. Note the wide variability in the size of the follicular structures on the mare’s ovary is the reason for the range of PG response from 1 – 7 days. Generally when shipping semen if you are playing the odds PG is given on Thursday to expect the mare in heat by Monday. Therefore you have a Monday and Wednesday semen shipment to access and Friday if your carrier and customs people allow for Saturday delivery. Measurement of the follicle size is essential for you to know when to expect her in heat and on what day to breed her.

Please note a reason the character of the CL (new mid or old) is that a single shot of Prostaglandin is only effective between days 5 – 16. If a young CL is noted, the CL may not be mature enough to respond. The mare may need to be retreated. Mares that have a large follicle (>40mm) at the time of PG treatment pose additional challenges. Most commonly they have a short heat (3 days), less common is a full 6 day heat, and least likely is that they ovulate within 24 hrs after treatment with PG, thus making a new CL with 24 hours. These mares do not show heat but will conceive if bred.

Mares in heat with follicles. Proceed by quantifying palpating and noting uterine and cervical tone, then use ultrasound to measure follicle diameter, and score the uterine edema. Administer an ovulation induction agent at >30-35 mm, or follow to the end of natural estrus (most light mares ovulate a follicle around 42 - 45 mm; draft and warm bloods closer to 55 mm).

Forms, Doses and Routes of Administration of Prostaglandin:

Lutalyse (PGF2-α) 5 mg SQ estrus induction, or 0.5 - 2.5 mg IM given twice 12 hours apart.
Estrumate (clopostenol) 250 µg, i.m. or SQ.

Side effects of PG’s include: often causes profuse sweating, increased heart rate and respiratory rate, abdominal discomfort, locomotor incoordination and lying down. Signs occur within 10 - 20 minutes of injection and typically abate in another 20 - 30 minutes. Caution should be used when handling these products, especially pregnant women and asthmatics. Avoid inhalation and self injection.
Progestosterone and estradiol 17β

This method is highly suitable for rural practitioners, and those with limited experience in ultrasound and palpation. Mare are administered a combination of 150 mg Progesterone and 10 mg Estradiol – 17β also called “P&E” daily for 8 – 14 days. The product is obtained through a compounding pharmacy. This protocol is used to regulate follicular waves for synchronization of estrus. This combination of steroids causes follicular suppression. Prostaglandin is given on the last day of P&E treatment in the breeding season. Stopping the treatment allows a new follicular wave to emerge and generally around 8 days elapses before a mare is showing estrus and is ready to induce ovulation. Breeding is usually at 10 – 12 days post P&E. Advise a check at 6 -8 days after stopping the P&E to monitor follicle growth and plan ovulation induction and ordering / timing of breeding. Ovulation induction is used to further tighten the window when a 30 - 35 mm follicle is present the mare is given hCG 2000 IU IM, or deslorelin. The whole protocol therefore takes around 18 days. The advantage is that mares can be anywhere in their cycle when the protocol starts and the protocol is very, very reliable. The longer the mare is on the P&E the larger the tendency for her follicle wave to emerge more slowly. Temperament of the mare may be a consideration is she is prone to dislike repeated injections.

Breeding strategies for mares bred with Cooled Transported Semen

The considerations for this breeding strategy include: making sure the mare owner importer has a GST number, clearing CFIA paperwork (4 -5 business days) if importing semen from the USA, making sure the shipper is able to obtain a Zoosanitary Certificate from the USDA in the USA with the semen, making sure a commercial invoice accompanies the semen, aligning the mare’s estrus and ovulation with the stallion’s semen collection dates and the courier arrival and delivery schedule! If it is a domestic shipment make sure that Friday overnight shipments may be picked up on the weekend from airlines or couriers, and that the forms are marked for Saturday delivery accordingly.

Cooled semen

Mares should be bred while in heat so that ovulation occurs within the next 48 hours. This is because most inseminations with semen of acceptable quality provide good fertility for at least 48 hours in the mare, and some provide fertility longer than that. Therefore a single timed insemination based on ovulation induction is widely used. Mares may be bred by following their cycle, inducing estrus and then ovulation, or they may be allowed to have a natural heat and then are bred. Due to the expense of shipping and collecting cooled semen, a single insemination is used with this breeding method.

Frozen semen is either used as a fixed time insemination after ovulation induction or it is used as a single insemination (Crowe et al., 2008). Recently deep horn insemination has gained popularity. Fertility is a function of the quality of the mare’s reproductive tract, the timing of the insemination in relationship to ovulation and the dose and quality of the semen. Generally the most limiting factor is the stallion’s fertility of the frozen semen. Frozen semen of acceptable quality (>100 million normal and motile sperm post thaw) will remain fertile for around 12 hours before and at least 8 hours after ovulation. Less than optimal timing for example may still result in pregnancy, however the likelihood of pregnancy decreases. One strategy is to divide the semen dose in half and use fixed time insemination of a half dose of semen at 28 and 40 hours after hCG induction of ovulation (for example treat with hCG at noon (Time 0), breed the following day at 4 pm (28 hrs) and then the day after that at 8 am (44 hours). If using deslorelin administer it a noon, breed at 28 hrs (4pm next day) and 44 hrs (8 am the day after that). One author reported deslorelin induced ovulation between 38 and 42 hours in 94% of mares (Hemberg et al., 2006).

Other Hormones

Progestagen– Regumate – Altrenogest

Progestagen is used most for the Suppression of Estrous Behavior. Administer to mares beginning 2-3 days prior to show or race to suppress estrous behavior that may detract from performance. Reliable and safe for use in mares. The progestagen Regumate (altrenogest) is used to help mares with the Maintenance of Pregnancy. Administered to mares with a history of repeated embryonic loss or low mean plasma progesterone concentrations Synchronization of estrus but not ovulation is achieved by administering progestagen to mares for 10-14 days. Most mares will come into estrus within 2-3 days of drug withdrawal. This and other progestagens do not suppress the follicle wave so there is wide variability in time to estrus and ovulation with progestagen treatment. Progestagen does NOT cause luteolysis, nor does in interfere with luteolysis, it does NOT suppress follicle development, mares will ovulate while on progestagen, and they may undergo luteolysis, but they don’t show heat.

Forms, Doses and Routes of Administration of Progesterone/progestagen:

Progesterone-in-oil (50 mg/ml), may be used to suppress estrous behavior and at 150 mg (3 mls) administered as an intramuscular (IM) injection once per day. Altrenogest (Regumate) (Synthetic progestagen) 0.044 mg/kg (1 ml per 110 lbs) administered orally once/day. Wear gloves.
The CIDR device, for use in cattle, contains native progesterone (1.9g). The device is inserted into the vagina of the mare. Cut the plastic string off the end of the CIDR to avoid irritation of the vulvar mucosa. In transitional mares a 14 day exposure is recommended, in cycling mares a shorter exposure may be effective (8 to 14 days) if combined with prostaglandin at withdrawal of the CIDR. May be used in mares during competitions to prevent estrus behavior but should be inserted at least 3 days prior to the event. Mild vaginal discharge is present in most mares treated with a CIDR. Shorter exposure is associated with less discharge. The use of the CIDR device in subfertile mares has not been investigated.

Other Hormonal protocols for Estrus Synchronization

The main options for estrus synchronization are:

Progestagen (altrenogest = Regumate) for 10 -14 days.
Give PG on the last day. Stop treatment and expect mares to come into heat in 2 – 3 days. Follow into heat and use ovulation induction with hCG or deslorelin. However (not synchronizing the wave), so the time frame to estrus and ovulation is variable.

Prostaglandin – mares receive 2 treatments with prostaglandin (5mg SQ) 14 days apart. Mares will come into heat around 3 days after the last treatment. This is a long protocol and not very reliable. Follicular wave is not synchronized. Mares are followed into heat and usually given an ovulation induction agent to tighten up the synchrony. Must use this long interval due to the long length of estrus.

Mares are given prostaglandin and are on the average coming into heat in 3 days, then they are given an ovulation induction agent. Due to lack of synchronization of the wave there is a variable time to estrus based on the follicle size on the mare’s ovary at the time of treatment with PG.

References


Reproductive Examination of Stallions
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Reproductive examinations are performed in stallions for the purpose of: an insurance examination, a prepurchase examination, following retirement to evaluate status and plan the mare book, assessment for cooled or frozen semen, investigating suspected male factor fertility problems, or following injuries, neoplasia or severe illness. Generally when performing a reproductive examination of a stallion it is a good idea to follow the clinical evaluation format of and use the guidelines of the Society for Theriogenology’s Breeding Soundness Examination (BSE). This is because this information is widely recognized in legal circles as a standardized means of assessing a stallion’s reproductive potential or function. The BSE and the common abnormal findings in reproductive examinations of stallions will be discussed below. Details concerning stallion semen evaluation and the ultrasound examination of the accessory sex organs will be discussed.

The purpose of the BSE is to determine the fertility potential of an individual, it is not a guarantee of fertility because laboratory measurements do not assess all functions of the sperm that are necessary for fertility. For example the semen evaluation determines the percentage of motile sperm, the number of morphologically normal sperm, and sperm with intact membranes, but it does not assess the ability of the sperm to bind to the oocyte and induce the cortical reaction. The vast majority of stallion fertility problems are generally explained by integrating the physical examination findings on testicular size, position, and consistency with the semen parameters such as volume, concentration, motility and morphology. Presently there is alot of interest in developing other tools such as special stains to reveal mitochondrial function, DNA integrity, and genomic analyses as adjuncts to a BSE to identify abnormalities, patterns of expression or mutations that are associated with lower fertility.

Guidelines for the stallion BSE are set by the Society for Theriogenology and are based on a stallion breeding by live cover a book of 20 - 40 mares per season, or by articial insemination (AI) 100 mares in one season. To meet these guidelines a stallion should be capable of producing 2 billion normal and progressively motile spermatozoa in 2 ejaculates collected 1 hour apart. The Classification System uses 3 terms: Satisfactory (meets the standard), Questionable (is close to meeting or may meet the standard in the future), or Unsatisfactory (does not meet the standard). In cases where the stallion is classified as Questionable a recheck examination is usually requested in 2 months time to determine if the stallion has poor intrinsic fertility (i.e. is genetically a poor stallion and is a substandard breeding prospect) or whether he is improving over time as the process of the stallion is still maturing, or a problem with the output or production of sperm is resolved. Theriogenologists estimate that between 8 – 15 % of bulls are not satisfactory breeders. Stallions are not generally selected for fertility so the percentage of BSE unsatisfactory stallions is expected to be higher than bulls. The time frame is based on spermatogenesis in the stallion, where a new population of sperm are ejaculated about every 70 days.

Age at Examination
Generally a BSE of a stallion is not typically performed in stallions that are less than 24 months old, because of the wide range in the onset of puberty. Stallions do not generally experience a decrease in fertility associated with aging that is seen in other species such as stud dogs, and bulls. Stallions may continue productive breeding activity well into their twenties.

A Complete health history
is obtained including any history of past injury or illness. A Complete physical examination with an evaluation of vision, cardiac, pulmonary, neurologic status is performed. A genital exam is performed initially including visual inspection of the scrotum, palpation of the testes/epididymides and measurement of the testes (length width and height). An internal examination may or may not be performed, however some insurance companies require this procedure to be performed. The stallion should be evaluated for the presence of 2 scrotal testes in a normal orientation. Testicular consistency should be noted. Total scrotal width (TSW) should also be measured by pulling the testis down into the scrotum and using a caliper to measure the total width. Generally the total scrotal width in most fertile horses is >10cm, with concern expressed for stallions with a TSW<7cm. By measuring the height, width and length of each testis the volume cm3 of the testis may be calculated. Changes in the testicular volume may be tracked over time. Stallions maintained under artificial photoperiodic conditions may maintain peak sperm production.
Complete reproductive history
This is used to determine if the stallion has ever bred, is actively breeding or is sexually rested. The number of mares bred / foal crops produced, the per cycle and seasonal pregnancy rates are calculated. History included an inquiry about the interbreeding intervals in the herd (short intervals are a concern), or increased early embryonic losses. The fertility of close relatives, grandsires, sires and brothers is also investigated. The stallion owner/handler is asked questions regarding blood, pus or urine in the ejaculate.

Sexual Behaviour
The normal behaviour of a stallion includes vocalization, prancing, arching the neck, sniffing with the neck, striking, pushing into the mare, sniffing noses, rubbing on the mare’s back, nuzzling the mare, scent marking manure and flehman response. When a stallion approaches a mare in heat he should become sexually aroused. He may exhibit the flehman response, particularly in response to mare’s urine.

Observations in the Breeding shed
The observations of a stallion in the breeding shed allow direct observation of a stallion’s temperament, libido, erectile function, neurologic and musculoskeletal function. In the breeding environment exposure to an estrus mare should result in sexual arousal and the penis should drop and become erect. Once aroused the stallion’s penis and urethral fossa is washed with warm water. The prepuce and penis are examined at this time for lesions such as squamous cell carcinoma, herpes III viral lesions, sarcoids, warts, melanoma, lacerations etc. Pre and post ejaculatory samples from the urethra may be obtained. Following the washing procedure the stallion may need to be re-teased (aroused) again by bringing him back to the mare before collection. Pain on mounting is one underlying cause of poor ejaculatory quality, as only a partial ejaculate may be obtained. Degenerative joint disease of the hocks and back of the stallion are usually the problem. These stallions tend to “dance” on the mare/phantom rather than achieving a good position.

Preparation of the artificial vagina
Semen is usually collected using an artificial vagina (AV). The types of artificial vaginas include: Missouri (most popular), Colorado, Nisikawa, and Hannover. Semen may be collected through use of a condom (lubricated inside and out), or by a process of chemically induced ejaculation using imipramine and alpha 2 agonists (xylazine). The internal temperature of the AV is from 43 - 50°C. The AV must be prepared using NON-spermicidal lubricant or vasoline. Place the lube in the top third of the AV to prevent contamination of the sample. Because it is an artificial vagina, a higher temperature than body temperature and more pressure is generally used to stimulate the stallion to ejaculate.

Copulatory Act - Semen Collection
The stallion’s libido and behaviour are rated. The copulatory act includes: mounting and coupling up to the mare / phantom, seeking the vulva/AV, intromission, thrusting, tail flagging, ejaculation, and dismounting. His ability to mount a phantom or a mare and obtain the correct position (called coupling up) for semen collection is observed. Stallions will sore backs or hocks tend to be positioned too far off the phantom as they shift more weight onto their abdomen. More than one mount and intromission may be required before ejaculation. The end of a stallion’s penis, called the glans penis will bell if he is highly aroused or if he has ejaculated. The process of ejaculation is usually indicated by a specific jerky tail flagging motion that occurs in synchrony with urethral pulses. Stallions that have pasture breeding experience are generally more cautious in their mating behaviour, and may initially mount a mare /phantom without an erection. They may only become sexually aroused by mares in the peri oovulatory period. Stallions trained for hand mating rely on the people to do the teasing and may spend little time checking the mare before mounting / breeding her.

Mechanics
The stallion handler and the semen collector are on the same side of the mare or phantom. The semen collector diverts the penis into the AV, feels for urethral pulsations (these correspond to jets of semen), and follows the stallion off the phantom or mare as he dismounts to collect all of the semen. Another sample is collected 1 hour later. Regarding the 2nd sample the concentration is usually half that of the 1st sample, the same or slightly higher motility, and similar morphology is expected. If the 2nd ejaculate does not follow this pattern, as might happen if only a partial ejaculate was obtained, then a 3rd ejaculate is obtained

Preparation of a sample
The stallion’s sperm sample must be warm at 37°C until extended to prevent cold shock. The semen is filtered to remove the gel fraction because gel makes the sample impossible to interpret. Sperm are mixed with Eosin Nigrosin for the morphologic assessment in a 60:40 stain to sperm ratio. The stain is hyposmotic to the sperm so the slide should be dried quickly, such as on a warming tray, to prevent any hyposmotic artefacts from forming (usually curved mid and end pieces). Wet mount preparations viewed under phase contrast do not usually allow the same detailed examination of the defects present. The morphologic examination is performed using 100x objective (oil immersion) which yields 1000x magnification.
Semen Processing

In a complete semen analysis the total sperm number is determined (concentration x volume), the motility, and the morphology of the semen is examined. Morphology is often not routinely evaluated in stallions on farms unless there is a problem with fertility. However, tracking changes in semen quality may provide the clinician with early and timely information to allow for appropriate interventions.

The volume of the raw filtered semen. Check the Motility of the raw semen (average total and progressive individual sperm motility in a high power field (400x)). Check pH, note the colour and gross appearance. Extend the semen 1:1, then record the extended motility. Extending the semen usually increases the motility and % motile sperm by about 10 -15%.

Concentration

Evaluate the concentration (hemocytometer or other means), most methods are accurate from 10 -100 million sperm / mL. Dilute the semen more if needed. Most highly fertile stallions have sperm >100 mill/ mL.

Live Dead Count

Using the supravital stain Eosin Nigrosin, live and dead sperm are evaluated in at least 100 sperm. Sperm which stain pink are considered dead or devitalized, while the cells that stain white are considered alive. The Live / Dead sperm ratios are determined, to indicate the vitality of the sperm.

Sperm Cell Morphology

Differential spermiogram

This is performed using stained dried slides (eosin nigrosin stain). In a Differential Spermiogram at least 100 cells are counted. All cells in a field are counted whether they are live or dead. Detached heads but not detached tails are counted. In the Differential Spermiogram a frequency distribution of all defects is performed on a minimum of 100 sperm cells, using a cell counter. All the sperm in a representative microscopic field are categorized. Therefore if a sperm has a macrocephalic head and a midpiece defect both are enumerated. To enumerate both defects simultaneously one presses both keys down at the same time on a cell counter device. This will only advance the counter by one number in the total column. Mathematically because this is a frequency distribution the percentages for the categories of defects will not add to 100%. The sperm defect categories used in the Differential Spermiogram include: head, midpiece, detached normal head, detached abnormal head, principle piece, proximal droplet, distal droplet, and acrosomes. Germinal epithelial cells are counted if more than just an occasional cell is present. All the sperm in a field are counted irrespective of whether they stain white or pink with the Eosin Nigroin stain. Figure 1 shows a normal sperm. When detached heads are encountered the head but not the corresponding headless midpieces are counted. There are also subcategories of the head and midpiece defects, and the relative frequency of occurrence with the most common subcategory being noted first, followed by the others in a descending fashion. This is done by memory.

Sub-categories

Head defects: Microcephalic (too small), macrocephalic (too large), pyriform (pear shaped), tapered (long and narrow sperm) teratoid (almost non-recognizable) and vacuolated. The range of values for fertile stallions was as follows: length, 4.9-5.7 µm; width, 2.5-3.0 µm; Higher percentages of morphometrically normal sperm heads are found in fertile than in subfertile stallions (52 vs. 19%). Essentially sperm are twice as long as they are wide, and variations in size resulting in sperm heads that are >25% larger or smaller should be considered abnormal. Pyriform sperm (pear shaped with a narrow base, and a proportionately wider apex) may be associated with chromatin instability. Tapered cells are long and narrow and may not be significant if the whole population of sperm are uniformly tapered. Teratoid sperm have the mid and principle coiled around head. Nuclear vacuoles in sperm are visible as dark spots on the head using Eosin Nigrosin. Small single apical vacuoles may not be highly significant in stallions. Larger and more confluent vacuoles are of significance. The overall frequency of the head defects present in both the intact and detached sperm are noted. These are listed from most frequent to least frequent. Regarding acrosomes, knobbed, missing, or abnormal acrosomes are listed. Figures 2 and 4 show sperm with pyriform, vacuolated heads, and a knobbed acrosome. Midpiece defects: The subcategories of the midpiece defects include: segmental aplasia of the mitochondrial sheath, swollen mitochondrial sheath, pseudo-droplet, fractured midpiece, and distal midpiece reflexes. Axial insertion of the midpiece is not considered an abnormality in the stallion or the boar. Figure 3 shows sperm with fractured midpieces. Segmental aplasia of the mitochondrial sheath has the appearance of gaps in the mitochondrial sheath, a swollen sheath involves visible thickening of the sheath, a pseudo-droplet is bunching of the mitochondria on the midpiece with a corresponding region on the midpiece of the boar. Figure 3 shows sperm with fractured midpieces. Segmental aplasia of the mitochondrial sheath has the appearance of gaps in the mitochondrial sheath, a swollen sheath involves visible thickening of the sheath, a pseudo-droplet is bunching of the mitochondria on the midpiece with a corresponding region on the midpiece of the boar.
Other cell types

in the stallion the presence of other cell types in the ejaculate (sperm, neutrophils, lymphocytes, red cells, squames, germinal epithelial cells) is noted. Additional stained slides (Diff Quik) may be used to identify the other cell types.

Laboratory Errors in Preparation

Laboratory errors causing the appearance of sperm defects are sometimes referred to as tertiary defects. There are a few defects that may be produced through errors in sample handling such as failure to filter the semen, excessive stain, cold shock and hyposmotic shock. Gel in the sample makes the background appear white and makes the sperm difficult to see. Excessive stain will create stain cracks along the sperm. Artefacts associated with cold shock of sperm include distal midpiece reflexes, while hyposmotic shock may result in curved midpieces or curled endpieces. In general it is very important to emphasize that the vast majority of defects present in a sample come out of the stallion. Errors or rough handling of the sample will not produce defects such as detached heads, or nuclear vacuoles. Most sperm defects are either developmental or maturational in nature.

Serial evaluation of the Sperm Morphology

Stallions typically have around 50% morphologically normal sperm. Serial examinations of semen using the Differential Spermiogram system, is a powerful clinical tool when it is possible to gain insight into the fertility potential of an individual. The problems that arise as a result of the stallion’s intrinsic fertility (genetic), a point source problem such as a stress (fever), or a long standing alteration (testicular degeneration) may be diagnosed by serial examinations of ejaculates spaced at least 1 month apart. The clinical interpretation of semen samples is challenging because an examiner must determine which parameters and abnormalities are constitutive and reflect an individual stallion’s age and intrinsic genetic ability, and which changes are extrinsic due to a disturbance (nutritional, hormonal, infectious, toxic, degenerative, idiopathic) in spermatogenesis

Semen Quality

Values for semen of average quality semen are: 30 - 70% progressive motility, 30 - 50% morphologically normal sperm, at a concentration of 50 - 100 million sperm per mL. Below average semen usually has a low concentration (<50mill/mL) and a low percentage (<30%) of morphologically normal sperm. Above average semen has a higher motility (>70%) and a higher percentage of morphologically normal sperm (>50%) and a high concentration (>100 mill/mL). The general health of the stallion is considered. There is no minimum total scrotal width, morphology, or motility in the stallion. The sperm parameters are interpreted in light of all other factors (age, history, external examination of the genitalia etc) to determine the classification status of the stallion.

Sperm Math

To determine total sperm number multiple the:

1. Filtered raw semen volume x the concentration of the raw filtered semen = total sperm number (concentration is obtained from using the hemocytometer at 1:100 dilution, loading 10 microliters per chamber on each side, and counting 16 large corner squares on the grid)

2. Multiple total sperm number x progressively motile (PM) spermatozoa = progressively motile sperm (progressive motility is determined by counting forward moving sperm as a percentage of the total number using 100x magnification on the microscope)

3. Multiple the progressively motile spermatozoa number times the % morphologically normal spermatozoa = progressively motile and morphologically normal sperm (PMMN spermatozoa) (where morph normal is done by counting/classifying 100 eosin nigrosin stained cells)

Sum the PMMN spermatozoa numbers from both collections for the BSE. The goal is to achieve 2 billion PMMN spermatozoa in 2 ejaculates, along with suitable soundness for classifying a stallion as satisfactory.
Figures

Figure 1:
Normal sperm

Figure 2:
Sperm with pyriform head/segmental aplasia of the mitochondrial sheath (upper) and vacuole/pyriform head/segmental aplasia/abnormal principle piece (lower)

Figure 3:
Sperm with broken midpieces (all) and proximal droplets (left and uppermost)

Figure 4:
Sperm with a loose head (left), pyriform head/vacuole/distal midpiece reflex (centre) and a knobbled acrosome/pyriform head (right).
Figure 5:
Top panel A shows an Eosin Nigrosin stain of an ejaculate, the dark round cells are germinal epithelial cells. The bottom panel B is a Diff Quik stain of the same sample, demonstrating that the cells are not neutrophils.

Figure 6:
Abnormal sperm in a stallion with Spermiostasis, note the presence of many detached heads.
In an reproductive examination of the the stallion the prepuce, penis, testis, accessory sexglands and semen are evaluated. The penis and prepuce of the stallion should also be examined when the stallion is sexually aroused.

Prepuce
The prepuce of the stallion is evaluated for abnormalities such as excess discharge, and problems such as tumours with squamous cell carcinoma (SCC), and melanoma being most frequent. Sarcoids and papillomas have also been described.

Penis
The penis of the stallion is evaluated for the presence of old injuries such as scars, or incisions. Light skinned horses are more prone to develop SCC. Healed lesions of equine herpes virus III may have the appearance of flat depigmented areas, which may mimic the signs seen with precancerous lesions of Squamous cell carcinoma. The lesions of SCC are however raised, depigmented spots. Scars on the penis that may indicate a past trauma, may result in shunt formation that may cause a failure of a complete erection or deviation of the penis (phallocampsis).

Testis measurements
Following semen collection testicular (LxWxH) and total scrotal width are measured. An ultrasound examination of the scrotal contents and and internal accessory sex organs is optional when performing a BSE of a stallion with no history of fertility problems. Measurement of the total scrotal width usually exceed 10cm in most stallions with values less than 8 cm causing concern. Serial measurements of a stallion’s testicular size may provide information about changes that are occurring. In most stallions testicular mass will increase until the stallion is 4 years of age.

Anatomy of the accessory sex glands
The stallion should be sedated if anxious, sensitive to rectal examination, or hard to handle. Products such as hyoscine bromide (Buscopan) may be used to relax the rectum, however this is seldom required. The tail of the stallion is wrapped, and the rectum emptied of manure. A small linear 7.5 mHz transducer is preferred because it is small enough to be positioned at 90 degrees to the urethra so cross sections of the accessory sex glands may be viewed. The stallion has a complete set of accessssory sex glands which include; the bulbourethral glands, prostate, seminal vesicles, and ampullae (fig 7-9). The stallion may be teased prior to examination for 30 minutes to enhance the ability to visualize the accessory sex glands. Sexual arousal will increase the diameter of the bulbourethral glands and the prostate, but was not reported to change the diameter of the ampulla or seminal vesicles. The bulbourethral glands are large and ovoid in the stallion (mean width 2 cm, and length 3.2 cm) and are located at 5 and 7 o’clock within or near the anal sphincter. The pelvic urethra is present on the midline, and is more echogenic that the surrounding penile tissues. The prostate is non-palpable per rectum, but is bilobed and is often prominent on ultrasound examination because it is filled with small pockets of secretion. The lobes of the prostate are 3.4 cm wide and 2.4 cm high, and are connected by an isthmus that is 0.6 cm wide. A view slightly off midline is the best location to view the lobes of the prostate. The colliculus seminalis (note cystic structures remnants in this area) is where the ejaculatory duct (common duct of the seminal vesicles and ampullae) enters the urethra. In the stallion only the ampullae and seminal vesicles may be palpated. The ampullae are long and tubular in shape, and they are initially located side by side, with the seminal vesicles located lateral to the ampullae. The seminal vesicles have a roughly tear drop shape with dimensions around 2.6 cm wide and 0.9 cm high, the glands are sometimes distended with secretion. By turning the probe so that it is 90 degrees to the urethra (horizontal view) the 2 ampullae will appear in cross section and may be followed to the colliculus. The diameters of the ampullae and seminal vesicles are measured, the presence of the lumen confirmed, and the nature (echotexture) of the secretion noted. Excessive edema in the wall of the ampullae is an abnormality. Returning to the midline each ampulla may be followed to the vas and then to the inguinal ring. The vasa deferens traverse through their respective inguinal rings to the epididymides. The dimensions of the ampullae are 1.6 cm wide and 1.2 cm high.
Abnormalities Detected using Ultrasound

Cysts of the uterus masculinus (Müllerian Duct Remnant / Paramesonephric duct remnant) may be found between the 2 ampulla near the colliculus. Urethral cysts may be found typically near the prostate. These cystic structures have been variably associated with ejaculatory dysfunction in stallions[1, 4-7]. Ampullary obstruction or blockage may be identified by an asymmetry of the 2 ampulla in a cross sectional view in a unilateral obstruction, edema of the wall of the ampulla, a corded feeling on palpation, a loss of the lumen and in some cases dilation of the epididymal ducts. Ejaculates characterized by large numbers of detached heads may be noted. Stallions with this condition may be managed by frequent collection, massage of the ampulla, prostaglandin or oxytocin treatment. Seminal vesiculitis may or may not result in palpable or echographic changes in the glands. In a few cases thickening of the wall of the seminal vesicle and flocculent material in the lumen were noted. Abscessation of the vesicular glands has been reported, and may result in gross enlargement of the gland[6]. Measurement of alkaline phosphatase in the semen indicates complete ejaculation has occurred in the stallion to indicate the ducts are patent in cases where bilateral obstruction is suspected.

![Figure 7: Bulbourethral gland in a stallion](image1)

![Figure 8: Ampulla (solid white arrows) with lumen (star) and seminal vesicle (thin black arrows).](image2)

![Figure 9: Prostate gland (arrows)](image3)
Common reproductive conditions in the stallion include:

- **Poor intrinsic fertility**: genetic – poor semen quality
- **Partial ejaculation**: older stallions with musculoskeletal problems – ejaculates with 3 or 4 urethral pulses rather than 7 – lower than expected ejaculated sperm based on testicular mass
- **Disturbances in spermatogenesis**: injury, systemic illness/fever, anabolic steroid treatment, glucocorticoid treatment, toxin exposure, nutritional – increased prevalence of defective, dead sperm, low motility
- **Testicular degeneration**: (>10% round cells, low percentage of normal sperm)
- **Trauma to penis prepuce**: (breeding accident) symptomatic care NSAIDS, Hydro, intermittent pressure wraps, sling when required or temporary purse string of the prepuce
- **Spermiostasis**: (tends to be permanent in the horse - large number of sperm with dead detached heads)
- **Bacterial** (Klebsiella pneumoniae, Pseudomonas aruginosa - pyospermia) or **viral EHV III** (vesicular lesions or scabs), EVA typically no signs, virus is shed in the semen
- **Hemospermia**: urethral injury or tears
- **Urospermia**: detrusor sphincter dysinergia train horse to urinate before collection
- **Low libido**: differentiate from pasture breeding stallion behaviour
- **Psychogenic ejaculatory failure**: young stallion or after injury, or in poorly socialized stallions
- **Neoplasia**: Penis/prepuce - squamous cell carcinoma (precancerous lesions flat raised spots, neoplasia is characterised by its smell and ulcerative appearance), melanoma (grey horses, raised nodules), papillomas (proliferative lesions). Testis – seminoma (aggressive), teratoma benign usually in a cryptorchid testis, Leydig cell and sertoli cell.
- **Dynamic testicular rotation**: – stallion is asymptomic testis can rotate 90°, extreme rotation causes testicular infarction
- **Habronemiasis**: prepuce and urethra – massive tissue disruption from parasitic larvae of habronema, drasha. Sulphur granules are common. Condition has a rapid onset – treat with ivermectin or topical organophosphates

Other diagnostic tests that may be performed include EVA titers, coggins tests, CEM test matings, pedigree analysis, HYPP / HERDA / CID / overo paint testing, biopsy of lesions, test matings, electronmicroscopy of the semen, biopsy of lesions, test sensitivity of the semen and urethra, urethroscopy, chromosomal analysis, hormonal analysis, and special stains of semen.

References:

Epidemiology in Every Practice

Robert Larson, DVM, PhD, ACT, ACVPM-Epi

Introduction

One component of epidemiology is to understand the interaction between animals, potential pathogens, and the environment in order to implement a herd-specific biosecurity plan. Biosecurity is the attempt to keep infectious agents away from a herd, a state, or a country and to control the spread of infectious agents within a herd. Infectious agents (bacteria, viruses, or parasites) alone are seldom able to cause disease in cattle without contributing factors from other infectious agents and/or the cattle's environment. Therefore to develop biosecurity plans for infectious disease in cattle, veterinarians must consider pathogen, environmental, and animal factors.

Pathogen Factors

Pathogens differ in their virulence, contagiousness, and their modes of transmission (Table 1). These differences exist not only between pathogens, but for virulence and contagiousness, can also differ between strains of the same species of pathogen. A more virulent pathogen causes more severe clinical signs of disease and a greater likelihood of death following infection. A pathogen with greater contagiousness will infect a larger number of animals in a shorter period of time when introduced into a population. These factors are not related, in that a highly virulent pathogen may not be very contagious and a very contagious agent may not be highly virulent. In addition, different pathogens have various methods of transmission that impact how they interact.

Table 1.
Examples of pathogen factors affecting diseases of cattle

<table>
<thead>
<tr>
<th>Pathogen Factors</th>
<th>Examples</th>
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<tbody>
<tr>
<td>Virulence</td>
<td>Infection with some strains of BVD or IBR (and other agents) causes much more severe disease and higher mortality percentages than infection with other strains.</td>
</tr>
<tr>
<td>Contagiousness</td>
<td>IBR and PI3 will infect more animals in a shorter period of time than will BVD following introduction of the virus into a herd.</td>
</tr>
<tr>
<td>Method of transmission</td>
<td></td>
</tr>
<tr>
<td>Inhalation</td>
<td>IBR and BVD</td>
</tr>
<tr>
<td>Ingestion – any age</td>
<td>BVD, Salmonella sp., leptospirosis</td>
</tr>
<tr>
<td>Ingestion – age specific</td>
<td>E. coli K-99 - only for a few hours following birth</td>
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<tr>
<td></td>
<td>Johnne's - most infectious early in life</td>
</tr>
<tr>
<td>Sexual contact</td>
<td>Vibrio, Trichomoniasis</td>
</tr>
<tr>
<td>Intermediate host</td>
<td>Liver flukes (snail), Neospora caninum (canine)</td>
</tr>
<tr>
<td>Fomite</td>
<td>BLV, Anaplasmosis – ticks, horse flies, surgical instruments</td>
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with a host population. Some pathogens are spread via inhalation or ingestion. Infectious agents spread in these ways are further differentiated by the length of time the agent can survive outside the host in the environment, by the distance they can travel and still be infectious, and by the age of host that is susceptible to infection or disease. Other pathogens are spread only through sexual contact and are not contagious outside the act of mating. And still other pathogens require an intermediate host or transmitting fomite such as an insect, snail, or other mammal.

Environmental Factors

A cattle population's environment includes its housing type, animal density, air quality, weather effects, mud, dust, footing, and health antagonists such as internal parasite burden, external parasite burden, and social stress. These environmental factors influence the innate immunity of a herd by their impact on immunosuppression.

In addition to effects on immunosuppression, a herd’s environment also dictates the “animal flow” or contact and mixing patterns of potentially infectious and susceptible animals. Some infectious agents preferentially infect only certain ages of cattle. Mycobacterium avium subspecies paratuberculosis (Johne’s) is primarily infectious to young animals, while Trichomonas fetus (Trichomoniasis) is primarily infectious to older bulls. Some infectious agents will infect all ages of cattle, but are only likely to cause disease in certain ages. Rotavirus and coronavirus infections are likely to cause clinical disease (calf scours) in young calves, but not in adults. In contrast, initial infection with the parasite Anaplasma marginale (anaplasmosis) is not likely to cause clinical disease in young animals but will cause disease in adult animals.

Animal Factors

The two primary animal factors that affect protection of cattle herds from disease are specific and innate immunity. Specific immunity relates to an immune response directed at a specific infectious agent that the animal has been exposed to in the past, either via natural infection or vaccination, for which “memory” remains. Innate immunity is strongly influenced by the overall health of the animal. Nutritional status such as adequate energy, protein, and dietary requirements of vitamins and minerals impacts an animal’s overall health and immune status. Stress due to crowding, inclement weather, unsanitary housing, or concurrent disease can cause varying levels of immune suppression.

In populations of animals, not only do pathogen factors such as virulence, and the length of the latent and infectious periods influence the number of animals infected; but animal factors such as the number of immunologically protected individuals (either due to specific or innate immunity) also determine the number of individuals the pathogen is able to infect and the speed of spread through a population.

Interaction Between Animal and Pathogen Populations

Whenever a veterinarian is looking at a beef herd he/she is observing not only a population of animals, but also an unseen population of present or potential pathogens. Both populations have their own life cycle, immunology, and adaptations. When we investigate a disease, we almost always consider the animal side of the infectious disease interaction because the incubation period (time from infection to onset of clinical signs), the symptomatic period (time from onset to end of clinical signs), and non-diseased state (time following the end of clinical signs) are relatively visible and measurable. At the same time however, the pathogen population and its dynamics are often given little attention. For the pathogen population, its interests are described by the latent period (time from infection of one host until its offspring can infect a new host), the infectious period (time period that the pathogen’s offspring can infect other hosts), and the noninfectious period (time when the pathogen population of one host can no longer infect other hosts). The infectious period can end when the immune system clears the organism from the host’s body, the infectious host animal is removed from the susceptible population (isolation), the animal is sent to slaughter, or the host animal dies. The relationship between the time line of infectiousness (pathogen’s perspective) and the time line of disease (animal’s perspective) differs between pathogens/diseases and is influenced by the virulence of the pathogen and the host response to it.
Understanding the relationship between these two time lines for a particular disease is important when developing a biosecurity plan to deal with that disease. The biosecurity tools available are (Table 2): 1) Test and cull, 2) Test and isolate, 3) Test and treat, 4) Prophylactically treat all, 5) Vaccinate, and 6) Management.

If an animal with a particular infectious disease becomes infectious about the same time as clinical symptoms appear, diagnosis and isolation or culling will help, and may completely stop the spread of the disease (example = rotavirus and coronavirus scour in calves). In contrast, if particular pathogen infects a host before the animal shows symptoms, diagnosis and isolation will not greatly affect the spread of the disease (example = IBR- and BVD-induced respiratory disease). If a disease has a long-term carrier state that accounts for all or most of the source of the infectious agent (anaplasmosis, BLV, BVD, Johne’s, Vibrio, Lepto, etc.) and a testing system has both high sensitivity and specificity, a test and cull strategy may be appropriate (anaplasmosis, BLV, BVD, Vibrio); while a test and isolate strategy could be considered for a disease with a short-term carrier state or a minimally pathogenic disease with a long-term carrier state (BLV, anaplasmosis). A test and treat strategy may be appropriate if the carrier state can be cleared with treatment (anaplasmosis, lepto). If an effective treatment exists to clear a carrier state, but testing or diagnosis lacks sensitivity or exceeds the cost of testing, prophylactic treatment of an entire population may be an appropriate biosecurity strategy (anaplasmosis, BRD). For disease with no easily defined carrier state or for which accurate tests are not available, vaccination should be considered as a primary biosecurity tool if vaccination will result in an increased percentage of animal resistant to infection or a decreased likelihood of transmission of the agent (IBR). Vaccination can be an adjunct biosecurity measure for diseases that have an accurate test used for test and cull as long as vaccination does not interfere with interpretation of the test. For some diseases that lack either a defined carrier or an accurate diagnostic testing strategy, management intervention to decrease the probability of effective contact is the primary biosecurity intervention strategy (Johne’s). Biosecurity for almost all diseases are enhanced by management strategies to reduce the likelihood of transmission and to decrease the events that lead to immunosuppression.

**Conclusion**

The future of food animal veterinary medicine will involve a greater emphasis on biosecurity to implement and monitor systems to prevent the introduction and spread of common livestock diseases. Cattle farms will increase the use of testing and isolation systems as well other biosecurity strategies to minimize the costs of disease to production. With an increased level of sophistication and knowledge necessary to implement these systems, veterinary involvement in food production will continue to increase.
Table 2.
Biosecurity strategies available to veterinarians and their requirements or characteristics

<table>
<thead>
<tr>
<th>Biosecurity Strategy</th>
<th>Requirements / Characteristics</th>
<th>Example Diseases</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Test and cull</strong></td>
<td>Accurate test</td>
<td>Bovine Viral Diarrhea (BVD)</td>
</tr>
<tr>
<td></td>
<td>Carrier animals are only or primary source of infectious agent</td>
<td>Bovine Leukosis virus (BLV)</td>
</tr>
<tr>
<td></td>
<td>Complete strategy that combines testing with movement restriction prior to testing</td>
<td>Brucellosis</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Neospora caninum</td>
</tr>
<tr>
<td></td>
<td></td>
<td><em>Mycobacterium paratuberculosis</em> (Johne’s) – not ideal examples because tests are not highly accurate</td>
</tr>
<tr>
<td><strong>Test and isolate</strong></td>
<td>Accurate test</td>
<td>Calf scours (coronavirus, rotavirus)</td>
</tr>
<tr>
<td></td>
<td>If using clinical signs as test, infectious period must not begin before clinical signs</td>
<td>BLV, Anaplasmosis (life-long isolation)</td>
</tr>
<tr>
<td></td>
<td>If using diagnostic laboratory test, the carrier state must be short lived and self-limiting or isolation must be life-long</td>
<td></td>
</tr>
<tr>
<td><strong>Test and treat</strong></td>
<td>Accurate test</td>
<td>Anaplasmosis</td>
</tr>
<tr>
<td></td>
<td>Treatment must effectively clear carrier</td>
<td>Leptospirosis ?</td>
</tr>
<tr>
<td><strong>Prophylactically treat all</strong></td>
<td>High prevalence or high cost disease</td>
<td>Anaplasmosis</td>
</tr>
<tr>
<td></td>
<td>Prophylactic treatment must effectively clear carrier state or prevent transmission</td>
<td>Bovine Respiratory Disease</td>
</tr>
<tr>
<td><strong>Vaccinate</strong></td>
<td>If combined with testing strategy, must not interfere with test accuracy</td>
<td>Infectious Bovine Rhinotracheitis (IBR)</td>
</tr>
<tr>
<td></td>
<td>Must either prevent infection or decrease transmission</td>
<td></td>
</tr>
<tr>
<td><strong>Management</strong></td>
<td>Even with same animal density, will decrease number of contacts</td>
<td>IBR ?</td>
</tr>
<tr>
<td><strong>Limit population size</strong></td>
<td>Decrease transmission</td>
<td>Calf scours, Johne’s, Trichomoniasis</td>
</tr>
<tr>
<td></td>
<td>Decrease animal density, isolate susceptible age animals from potential carriers</td>
<td>Calf scours, Johne’s, leptospirosis</td>
</tr>
<tr>
<td><strong>Decrease transmission</strong></td>
<td>Sanitation to decrease environmental transfer</td>
<td></td>
</tr>
<tr>
<td><strong>Decrease immunosuppression</strong></td>
<td>Decrease social stressors: commingling, aggressive handling</td>
<td>Almost all diseases</td>
</tr>
<tr>
<td></td>
<td>Decrease environmental stressors: mud, heat, cold, dust</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Adequate nutrition</td>
<td></td>
</tr>
</tbody>
</table>
Testing for Diseases with Chronic Carrier Status

Robert Larson, DVM, PhD, ACT, ACVPM-Epi

Introduction

Many veterinarians express frustration when trying to provide their clients with the best advice on which diagnostic tests to recommend for purchased cattle. The goal is to screen apparently healthy cattle to identify carriers of infectious disease that could cause reproductive losses and other health problems in the herd. To determine the economic return for diagnostic testing strategies, veterinarians need information on the accuracy (sensitivity and specificity) of available tests, commonness of the condition in question in the cattle population at large and specified sub-populations, disease dynamics such as reservoir, transmission pattern, incubation period, immune response, treatment efficacy, and negative or unintended consequences of diagnosis or treatment. In addition to the federal eradication programs for brucellosis and tuberculosis, the diseases with long-term carrier states that are most often considered for screening purchased cattle include: Trichomoniasis, Bovine Viral Diarrhea (persistent infection (PI) status), Bovine Leukosis, Anaplasmosis, and Johnes.

Determining Diagnostic Test Usefulness and Diagnostic Strategy

A valid question confronting veterinary practitioners is whether to use available diagnostic tests to screen a particular herd or purchased replacements (bulls, heifers, and cows) for a particular condition. The input needed to arrive at a logical conclusion includes epidemiologic data about the condition or disease, diagnostic test sensitivity and specificity data, disease or condition dynamics, and economic costs of the condition and its treatment. Literature review and mathematic aids, such as computer spreadsheets and expert systems, are the tools used to create the necessary outputs. These outputs include post-test predictive values of diagnostic tests, economic value of testing, sensitivity of the decision to the individual inputs, and the importance of individual inputs to the decision. These outputs are used to evaluate alternate diagnostic testing strategies in order to indicate the best testing strategy, and to identify the control points to be monitored for change that can trigger a re-evaluation of the decision.

Sensitivity and Specificity of Diagnostic Tests

Sensitivity and specificity are properties of a diagnostic test that are determined by comparing the test to a “gold standard”. The gold standard is considered the true diagnosis, and may be made using a variety of such information as clinical examination, expert opinion, laboratory results, or postmortem results. Sensitivity is the proportion of known positive (gold standard-positive) samples that the test in question identifies as positive. Specificity is the proportion of known negative samples that the test in question identifies as negative. In other words, sensitivity answers the question, “How effective is the test at identifying animals with the condition?” and specificity answers the question, “How effective is the test at identifying animals without the condition?”

Because in almost every situation, there is overlap between the test results of truly negative bulls and truly positive bulls, it is generally impossible to have a test that is 100% accurate. Because diagnostic tests (both laboratory and clinical examination tests) use an arbitrary cut off to separate test-positive and test-negative populations, sensitivity and specificity are inversely related, and placing the cut off is always a trade-off between the impacts of false-negative and false-positive results.
Prevalence (Commonness)

Prevalence is the number of cases of a condition at a given time compared to the population size at that time. Each practitioner’s judgment, based on history and clinical examination of both individuals and the population, aided by available prevalence information, is often all we have to estimate disease probability.

For a test with imperfect specificity, an increasing proportion of the test positives will be false positives as prevalence decreases. At low prevalence, the majority of test positives will be false positives, so that for an uncommon condition, even a highly accurate test will render results that must be interpreted with care when applied to the animal population as a whole. In other words, in the case of very rare conditions, most tests for that condition that appear to be positive are actually false-positive.

Post-test Predictive Value

The post-test predictive values of a test are determined, not in the laboratory, but in the field and they tell if a valid test is useful. The positive predictive value is the proportion of animals with a positive test result that are actually positive, and is influenced by test specificity. The negative predictive value is the proportion of animals with a negative test result that are truly negative, and is influenced by test sensitivity. Both positive and negative predictive values of a test are affected by the commonness (prevalence) of the condition. As the prevalence of the condition rises, more animals in the population have the condition, and we have greater confidence that a positive test result is correct. With increasing prevalence, the positive predictive value of the test increases and the negative predictive value decreases, while the reverse is true as prevalence of the condition is decreasing.

It is often impossible to estimate prevalence with any confidence, but one must still consider predictive value in test interpretation. When screening a herd, one often has no data to suggest that an individual animal is in a particularly high-prevalence group. In such a mode with a test with good sensitivity, a negative test result has a high negative predictive value and is useful in striking a rule-out off the list, but a positive test result (which is most likely a false positive) is useful only in keeping a rule-out on the active list and does not mean a the diagnosis has been confirmed.

Diagnostic testing strategy

To rule-in a potential diagnosis, many times it is necessary to use more than one test, either in series or in parallel. Running tests in series, where a second test is submitted only after the first test returns a positive result, is used to confirm a positive test with a low positive predictive value (low specificity or low prevalence). A two-test series is interpreted as negative if either test results in a negative response (i.e. BVD PI testing). Running two or more tests in parallel, where they are submitted simultaneously or taken sequentially from essentially the same population, is used to confirm a positive test with low negative predictive value (low sensitivity or high prevalence). Parallel tests are interpreted as positive if any test results in a positive response (i.e. Trich testing).
Conclusions

Interpretation of diagnostic tests is an important component of clinical veterinary practice. Veterinarians use a variety of testing methods including physical examination, laboratory tests, diagnostic imaging, and evaluation of performance records. Proper interpretation of test results involves close communication with the diagnostic laboratory and other experts, and an understanding of how commonness (prevalence) of the condition in question affects one's interpretation of the test results. An important component of diagnostic testing is the proper use of more than one test either in series or in parallel. By using properly using one or multiple tests, veterinarian can optimize their diagnostic accuracy to provide the greatest value; and can determine the factors that are producing the greatest impact on the economic return for the testing strategy.

References


Economic Considerations for Disease Testing Strategies

Robert Larson, DVM, PhD, ACT, ACVPM-Epi

Introduction

The most appropriate method to determine the economic value for diagnostic testing will vary depending on: the condition in question, the time frame involved, and how the diagnostic information will be utilized to make decisions. The most straightforward method is by partial budgeting. For rare conditions or events, it may be more appropriate to determine the cost of a negative outcome and the cost of intervention - and working with the client, determine his/her level of risk aversion, and together, determine the value of reducing the risk of a rare event.

The data that we need to make logical conclusions about diagnostic and treatment strategies include: sensitivity and specificity of available diagnostic tests, prevalence of the condition in question in the population at large and specified sub-populations, disease dynamics such as reservoir, transmission pattern, incubation period, immune response, and pathogenicity, treatment efficacy, and negative or unintended consequences of diagnosis or treatment. In addition, food animal veterinarians must also have data on the economic cost of the disease and the economic cost of intervention.

We must make use of tools that are as old as Thomas Bayes’ theorem published in 1764 and as new as computer spreadsheets and probabilistic modeling software (expert systems) to apply numeric probabilities to decision inputs in order to arrive at conclusions with a level of certainty that can be communicated to clients. By evaluating the sensitivity and importance of the inputs of our decisions, we must also determine a rational strategy to re-evaluate our conclusions when inputs change either due to new information removing some of the previous uncertainty or because of the expected variability of biologic events.

Determining Economic Benefit of Diagnostic Strategy

Determining Cost of Disease

Biology and economics intersect when veterinarians determine the cost of a negative condition. A number of tools are available to approximate the cost of a negative condition (disease presence, suboptimal body condition, non-pregnant after bull exposure, subfertile bull, etc.), and the biologic characteristics of the condition determine the proper economic analysis. Partial budgets are appropriate for diseases that are horizontally transmitted and immunity or other responses (death, sterility, removal from population, sale of feedlot pen, etc.) confine the negative effect of the disease to a short period of time; or for conditions whose negative effects are confined to a short period of time (i.e. correction of low body condition score effect on subsequent time period’s feed costs).

Multi-year enterprise analyses are more appropriate to estimate the economic cost of diseases that are vertically transmitted, due to an environmental source, have a chronic production-losing component, or to estimate the cost of conditions that have an impact on costs in subsequent years (open cows, subfertile bulls, etc.).

Determining Cost-Benefit of Diagnostic Testing Strategy

The cost effectiveness of alternate diagnostic testing strategies can be compared with a partial budget (Figure 1). In this partial budget, the post-test predictive values, test cost, cost of the negative condition, treatment cost, and cost of false positives are used to calculate the return for true positives, true negatives, false positives, and false negatives. The economic benefit is simply the costs for true negatives, false positives, and false negatives subtracted from the return for true positives.
Determining Sensitivity of One Variable When All Others Are Held Constant

Spreadsheets are able to do a straightforward calculation to determine the effect on the outcome following a specified change in one variable while all the other variables in the decision are held constant. A variable that is very sensitive will cause the output to be quite different if the variable is changed only a small amount. In contrast, an insensitive variable must be changed a great deal in order to significantly change the output and the resulting decision.

Using Probabilistic Models

Probabilistic decision-making is the everyday challenge of making decisions in situations of uncertainty. Unfortunately, not even highly trained experts are able to model probabilistic decision-making in their minds. Even committees of experts are unable to do probabilistic decision-making. Computers are required to do stochastic simulation when several uncertain inputs and their myriad of interactions are evaluated. Probabilistic models provide the decision maker with the range in outcomes that will occur as long as the assumptions are within the range submitted. RISK™ is one commercially available probabilistic decision tool.
Flaws of Deterministic Models

An analysis is considered deterministic when a variable is treated as having a single value even when it is known to vary, or when its true value is not known with certainty. Variability is the natural phenomena of inherent dispersion. This type of dispersion is not reduced by more research or better knowledge. In contrast, uncertainty is used to describe the fact that we have incomplete knowledge. An uncertain variable has a knowable number or distribution, but we have not done the necessary research to arrive at an accurate estimate. Uncertainty can be reduced with better knowledge. Both variability and uncertainty are ignored in a deterministic model, which promotes a false sense of certainty, and allows one’s biases to influence the outcome of the model.

Monte Carlo Simulation

It is difficult for the human mind or spreadsheets to deal with the amount of variability inherent in biological decisions. The term Monte Carlo simulation refers to the process of using computer simulation to randomly select a value from the distributions of values for each variable in a model and then calculate the net return. The computer program (@RISK™ in this case) repeats the entire procedure many times (>10,000 times) and tracks the results. After many simulations, it is possible to graph the distribution of net returns and to examine the descriptive statistics of the output such as the mean, standard deviation, and the probability of a particularly negative or positive return. If the computer plays the game long enough, we will have a very good idea of the distribution of the possible results.4 @RISK™ can do Monte Carlo simulations easily because it provides a wide array of different random-number generators, and an automatic summary of the simulation results.

Determining Expected Distribution Outcome

The advantage of probabilistic models is that the outcome of the model is not a single value, but an expected range of values with the probability for each level of output. This appropriate uncertainty in the output allows the veterinary decision maker to not only determine the most commonly expected outcome, but also the range of expected outcomes and the likelihood that the outcome will be particularly negative or positive.

Determining Sensitivity (Importance) of Inputs to Outcome

@RISK™ and other probabilistic models are able to calculate the sensitivity of the output in question to input variation based on the mathematical contribution of each input to the output and the input’s dispersion due to variability or uncertainty over all the ranges of all the other inputs. This method of sensitivity analysis is in contrast to spreadsheets that must keep all other variables constant. A tornado diagram is a common graphic used to show the regression sensitivity of the output to the inputs in the model, and is an effective depiction of which inputs have the greatest effect on the decision.

Exceptions (Alternatives) Methods of Cost-Benefit Determination

The value of a testing strategy, whereby all incoming animals are tested and the true-positive animals are isolated or euthanized, is the value of avoiding disease spread in the population. The cost of true negatives is essentially the cost of doing the diagnostic tests, including laboratory costs, veterinary labor and consulting costs for handling the tests, and labor for handling the animals. The cost for false positives is the cost of isolating or euthanizing an animal that was not truly infected. And, the cost of false negatives is the cost of leaving a positive animal in the herd.

For conditions that are rare (low prevalence), even with an accurate test (but less than 100% specific), many of the positive test results will be false-positive, and the costs of finding true negatives (i.e. testing cost) and the cost of false-positives may be greater than the value of finding the few true-positives. In this situation, a partial budget evaluation may indicate little or no economic benefit for a testing strategy unless the cost of disease is substantial.

Because some of the relatively infrequent negative conditions of interest to veterinarians can have significant production and economic costs when present, the cost of an infrequent but significant condition can be better evaluated as an assessment of risk and cost of risk avoidance. Once the cost of the risk is quantified, the producer and veterinarian can determine the effects such an event would have on a confined period’s cash flow, and can evaluate that effect with the cost of risk reduction.
Conclusions

Use of diagnostic tests for biosecurity purposes offers veterinarians a tool to reduce the cost of disease for livestock clients. Veterinarians should use information about test sensitivity and specificity, disease prevalence, test cost, and the cost of disease to calculate the expected value of testing for biosecurity reasons.

References


Feedlot Bovine Respiratory Disease (BRD): Is The Disease Changing?
Amelia R. Woolums, DVM, MVSc, PhD, DACVIM, DACVM

Introduction

The bovine respiratory disease complex (BRD) is the leading cause of morbidity and mortality in feedlot cattle in the U.S. and Canada. The complex occurs when cattle are transported or mixed with cattle from multiple sources; other stressors can also influence development of the disease. Disease is initiated by infection with one or more infectious agents and compounded by inadequate immunity. Information from research in the last 10 years indicates that some aspects of feedlot BRD may be changing, as compared to descriptions of the disease from the 1970's. This presentation will review the risk factors, epidemiology, pathology, and infectious agents of feedlot BRD as described in some foundational papers from the 1970's - early 1980's, and will compare these with findings from studies published recently. New developments will be highlighted, and theories regarding how feedlot management may need to be modified to address these developments will be discussed.

Epidemiology and Risk Factors

While BRD (also known as “shipping fever”) has been recognized since at least the 19th century, our current understanding of the syndrome was well established by research in the 1970's. In a series of papers, Jensen et al described the diseases identified in feedlot cattle that died in a population of 407,000 yearling cattle during 1974 (1). Approximately 5.1% of the total population became sick, and 1.0% died. Of the 3,943 cattle that died, approximately half were subjected to necropsy, which revealed that respiratory disease was the cause of death in 64% of the cases. Shipping fever was the type of respiratory disease present in 75% of the cases of respiratory disease, and 72% of the shipping fever cases that ultimately died occurred within the first 45 days of the feeding period. The report by Jensen et al was the first comprehensive survey of feedlot disease in a western U.S. feedlot, and the pattern of disease they described is still occurring today.

Another detailed report described health outcomes and necropsy findings in cattle from over 60 operations in Bruce County, Ontario over a 3-year period (3). The Bruce County study was noteworthy because it was the first large scale study to prospectively evaluate management practices in order to determine which were associated with adverse health outcomes. Mortality was approximately 1% over the course of the study, and the majority of cattle that died were submitted to the Ontario Veterinary College for necropsy. The most common cause of death was BRD, which caused 50% of the deaths in the first year of the study. Of many management factors included in the analysis, several were more common in groups of cattle with higher mortality. Respiratory vaccines given at arrival, feeding of corn silage in the first weeks after arrival, and mixing cattle from various sources were consistently associated with higher mortality in groups of cattle. The negative effect of corn silage feeding was diminished by feeding of grain in addition to silage. The negative effect of vaccination at arrival was decreased if vaccination was delayed for 2 weeks. The results of this study led many to question the value of vaccination on arrival for control of respiratory disease. A relevant finding was that infectious bovine rhinotracheitis virus (IBRV) was more often associated with respiratory disease in cattle that were not vaccinated for IBRV than in cattle that were vaccinated for IBRV, suggesting that IBRV vaccination protected cattle from disease associated with IBRV.

Two studies occurring approximately 10 years later also evaluated the epidemiology and risk factors associated with BRD (4,5). In the study by Alexander et al (4), data from 17,696 cattle in a single feedlot were evaluated, and factors related to morbidity (treatment) for respiratory disease were evaluated (rather than BRD mortality, as in the Jensen and Martin studies). BRD was the cause of treatment for 50% of the cattle treated, with 58% of BRD cases developing in the first 40 days on feed. If more groups of cattle were arriving at the feedlot on separate days, more BRD occurred; this was considered to be evidence that mixing cattle from multiple sources increased BRD. Pregnancy checking heifers and wide
ranges in environmental temperature over the first 14 days on feed were also associated with increased BRD. Unfortunately the impact of vaccination on arrival was not evaluated; this would have been of interest given the results of the Bruce County study.

Ribble et al evaluated the pattern of fatal BRD in over 58,000 calves entering a large feedlot over 4 years (5). BRD was significantly more common in cattle entering the feedlot in November in each of the 4 years of the study, with cattle entering in November having a 2- to 8-fold greater risk of developing BRD than cattle entering in September or December. A number of reasons for this association were speculated, including factors related to movement of increasing numbers of cattle through livestock auctions and into feedlots, and factors related to increased workload at feedlots receiving large numbers of cattle.

In comparison with these reports, a survey of feedlots published in 2008 evaluated risk factors associated with treatment for respiratory disease (6). The data represented information from over 20,000 cattle entering 102 feedlots for approximately 1 year beginning in 1999. Treatment for BRD was more common in cattle from pens with mixed gender (both steers and heifers), groups containing cattle from multiple sources, and cattle shipped a longer distance.

Because these studies measured different outcomes (mortality vs. morbidity), and did not always evaluate all the same management factors that could put cattle at risk for BRD, it is difficult to make extensive direct comparisons among them. However, it is noteworthy that mixing cattle from multiple sources was associated with either mortality or morbidity by direct or indirect measure in each of these studies. This may have been true at the level of the individual pen, or at the level of the feedlot as measured by increased numbers of cattle from different sources entering the feedlot (5,6). The pattern of BRD appears to be consistent over many decades, in that most cattle developing BRD that is fatal or requires treatment are identified within the first 60 days of the feeding period.

Pathology and Microbiology

In the 1973 report by Jensen et al, 48% of all cattle subjected to necropsy had lesions of bronchopneumonia. The pathology of affected cases was described in detail, with characteristic findings including lobules that were firm, airless, pink to gray in color, and separated from adjacent lobules. Some lobules contained infarcts or areas of coagulation necrosis. Pleural surfaces were covered with variable amounts of fibrin. These findings are typical of acute fibrinous bronchopneumonia. A subset of the cases were subjected to microbiologic testing; of 354 cases tested, Pasteurella (which would have included both Mannheimia haemolytica and Pasteurella multocida) was isolated from 62%, and Mycoplasma (species not identified, so not known if Mycoplasma bovis or other) from 50%. Thirty-five percent had both Pasteurella and Mycoplasma. Pathologic and microbiologic findings in the Bruce County study were similar; 40% of the cases subjected to necropsy had fibrinous pneumonia (3). In the first year of the project, microbiologic assessment indicated that Mannheimia haemolytica was isolated from cattle more frequently than Mycoplasma bovis; however, M. bovis was significantly associated with both fibrinous pneumonia and other types of pneumonia (exact “other types” not specified).

In more recent surveys of causes of feedlot mortality, it appeared that a greater proportion of cases were affected by pneumonia of more chronic nature than in the Jensen and Martin studies. In a 2006 report (7) describing pathologic findings in 99 cattle from 72 Ontario feedlots which died within the first 60 days of the feeding period, the majority of fatalities (68%) were due to pneumonia. Of all cases subjected to necropsy, 40% had lesions of acute fibrinous pneumonia, 18% had lesions of chronic fibrinous pneumonia (characterized by abscesses and mature fibrosis), and 55% had caseonecrotic pneumonia. Caseonecrotic pneumonia, which was not described in either the Jensen or Martin studies, was characterized by the presence of grossly visible raised yellow-white foci of dry, friable, caseous material in 10-90% of the cranioventral lung. Histologically these lesions were identified as foci of amorphous eosinophilic debris surrounded by a region of necrotic leukocytes and then normal leukocytes and fibrosis (8). Mycoplasma bovis was isolated from 98% of cases with caseonecrotic pneumonia, while Mannheimia haemolytica, Pasteurella multocida, and Histophilus somni were isolated from 35%, 20%, and 17% of the cases, respectively. In contrast, M. haemolytica was isolated from 68% of the cases of acute fibrinous pneumonia, whereas Mycoplasma bovis was not isolated from cases of acute fibrinous pneumonia that lacked lesions of chronicity. Many cattle had more than one type of pneumonia simultaneously; for example, both acute fibrinous pneumonia and also caseonecrotic pneumonia. Thirty-one percent of cattle subjected to necropsy had lesions of both fibrinous pneumonia and caseonecrotic pneumonia, indicating that more than one disease process was active in the lung at the time of necropsy.

A 2008 western Canadian study (9) evaluated pathology and microbiology of feedlot BRD cases occurring in the first 60 days of the feeding period in cattle from 17 feedlots. Of 99 animals subjected to necropsy, 90 (91%) had lesions of pneumonia. Thirty-seven percent of the animals subjected to necropsy had peracute to acute fibrinous pneumonia, while 43% of animals necropsied had subacute to chronic pneumonia. In order to be defined as subacute to chronic, lungs had to have abscesses and/or fibrous adhesions. Microbiologic assessment in this study was carried out entirely by the use of immunohistochemistry (IHC), as opposed to bacterial culture, which had been used in previous studies. Mannheimia haemolytica was more commonly found in the peracute, acute, and subacute pneumonia cases, while Mycoplasma bovis was more commonly found in the subacute and chronic groups. Bovine viral diarrhea virus (BVDV) was commonly found in conjunction with M. haemolytica (and thus in peracute to acute pneumonia),
while Histophilus somni was commonly found in conjunction with Mycoplasma bovis (and thus in chronic pneumonia). A 2009 report (10) describing 237 fatal cases of BRD from a single Oklahoma feedlot over 1 year similarly found a relatively high proportion of cattle to have subacute to chronic pneumonia, as compared to acute fibrinous pneumonia. The majority of cases were subjected to necropsy, which revealed that 21% of cases necropsied had lesions of acute pneumonia, while 40% had chronic pneumonia. As in the study by Booker et al, isolation of M. haemolytica was correlated with acute pneumonia, while isolation of M. bovis was correlated with chronic pneumonia; and isolation of M. haemolytica was correlated with identification of certain strains of BVDV, while isolation of M. bovis was correlated with isolation of H. somni. It is noteworthy that, while BVDV has been described to frequently occur in cattle with chronic pneumonia due to M. bovis (11), in the three recent prospective surveys of fatal feedlot BRD (8,9,10), BVDV was not found to be associated with M. bovis infection. This indicates that while BVDV is sometimes associated with chronic pneumonia due to M. bovis, it is not invariably present.

**Summary**

In summary, the importance of BRD as a leading cause of morbidity and mortality in feedlot cattle does not seem to have changed in the past 35 years. Also, the pattern of occurrence of feedlot BRD, wherein the majority of cases occur within the first 60 days of the feeding period, has not changed. However, it appears that BRD cases with lesions indicating a more chronic course of disease make up a greater proportion of cases described in reports from the past 5 years. Mycoplasma bovis is consistently associated with these cases of chronic BRD, but BVDV is not. Histophilus somni has been associated with M. bovis in cases of chronic BRD, but it is isolated from significantly fewer cases than M. bovis. Mannheimia haemolytica is still a consistent isolate from acute fibrinous BRD cases, but these cases are not the most numerous in recent surveys of fatal feedlot BRD. It should be emphasized that the reports by Jensen et al and Martin et al from the 1970’s did not specifically describe the number of BRD cases with lesions indicating chronicity, but the emphasis on discussion of acute fibrinous pneumonia in these reports suggests that chronic BRD did not make up a major portion of the cases seen at that time. If this apparent change in the nature of fatal feedlot BRD in U.S. and Canadian feedlots is real, the reason for the change has not been determined. Many have advanced theories, including the possibility that this apparent shift in the nature of feedlot BRD is related to the widespread use of various antimicrobials that are very effective at controlling M. haemolytica, but much less reliable at controlling M. bovis. The fact that BRD is still the leading cause of fatal disease in feedlots after 35 years indicates that there is still room for improvement in control of BRD, but the approach used may need to be modified to improve prevention of chronic pneumonia often associated with Mycoplasma bovis infection, while not “letting up” on efforts to prevent acute fibrinous pneumonia related to M. haemolytica, which still contributes to feedlot morbidity and mortality to an important degree.

**References**


Vaccination in BRD Control: Do Vaccines Work?

Bovine respiratory disease (BRD) is perennially among the leading causes of loss for cattle producers. For years a variety of vaccines have been available for prevention of bovine respiratory disease, and research is ongoing with the goal of identifying more effective products. The best evidence to support the efficacy of vaccines to control BRD comes from well-designed clinical trials in the field, where meaningful outcomes are measured in animals that have been exposed to the diseases for which they were vaccinated. In recent years a few reports describing studies of this type have been published (for example references 1-3). In some of these reports, vaccines improved resistance to BRD, or the productivity of cattle in production settings where BRD occurs; in other reports, they did not. Of the field trials published, the vaccines most commonly assessed are those for bovine respiratory syncytial virus (BRSV) and Mannheimia haemolytica, with some trials supporting the efficacy of BRSV and M. haemolytica vaccines, and others not supporting their efficacy. In contrast, as yet there has not been a published report of a clinical trial testing the efficacy of vaccines against Mycoplasma bovis or Pasteurella multocida. Clinical trials specifically testing vaccines against infectious bovine rhinotracheitis virus (IBRV), bovine viral diarrhea virus (BVDV), and Histophilus somni are few, and many have included relatively few cattle, or have not tested more recently marketed vaccines (i.e., within the past 10 years). Thus there is a need for more clinical trials testing the efficacy of certain BRD vaccines. When clinical trials are not available, the next level of quality of evidence to evaluate the efficacy of BRD vaccines is that from experimental challenge studies. In such studies, a group of cattle or calves are randomly allocated into groups that are vaccinated or not vaccinated, and then they are exposed to one or more infectious agents that cause respiratory disease. Results from such studies are more widely available than data from clinical trials, because they are less complex and less expensive to run. Moreover, vaccine manufacturers are required to perform challenge studies before vaccines can be licensed for sale. If an experimental challenge leads to disease that is similar to that seen in the field, then data from such studies is useful. However, because an experimental challenge study is by nature artificial (i.e., because cattle are not purposely exposed to infection in the field), such studies do not reflect the “real world” situation like well-designed clinical trials do.

In summary, there is some evidence indicating that some respiratory vaccines can help decrease respiratory disease or improve production measures in some settings; the vaccines most often tested have been those against BRSV and M. haemolytica. However, in well-designed studies vaccines sometimes do not show a clear benefit (for example, reference 2). Moreover, there is insufficient data from clinical trials to determine whether or not some vaccines are efficacious (for example, vaccines against Mycoplasma bovis or Pasteurella multocida). An important point to remember is that when vaccines against BRD are tested in any type of experiment, it is quite rare for them to completely prevent infection in all cattle vaccinated—thus some vaccinated cattle will likely still become infected even if they do not become as sick as unvaccinated cattle. And in some studies testing the efficacy of vaccines, even if vaccinated cattle become less sick than unvaccinated cattle, some of them still become sick. So vaccines should not be expected to completely prevent infection, or even completely prevent disease in all animals exposed; they should instead be viewed as a method to decrease the likelihood of infection or disease, and to decrease the severity of disease if it does occur. Veterinarians generally understand this, but we may not adequately communicate this to clients, leading to unrealistic expectations for vaccination.

I vaccinated cattle, but they still got respiratory disease. Why did this happen?

As noted above, clients should have realistic expectations of what BRD vaccination can accomplish. Given realistic expectations, if vaccinated cattle develop BRD, multiple possible causes should be considered. Possible causes of apparent vaccine failure are related to 1) the administration of the vaccine, 2) the ability of host to respond to vaccination, and 3) the nature of pathogen exposure. In category 1, apparent vaccine failure may be due to mishandling of vaccine (e.g., improper storage of live organism vaccines, use of residual product in a bottle reconstituted days to weeks previously, or chemical disinfection of multi-dose syringes leading to inadvertent inactivation of live vaccines); improper or
careless administration, so that less than an optimum dose is
delivered, or so that vaccine is not administered at recommended
site (e.g., administration into nuchal ligament rather than cervical
muscle, or intradermal rather than subcutaneous administra-
tion); concurrent administration of antibiotics with live bacterial
vaccines; and incorrect timing of vaccination, such as vaccinating
animals already incubating the disease, or failing allow time for an
immune response to vaccination prior to pathogen exposure, or
administration of booster too soon, too late, or not at all. In general,
if only a single vaccination is possible, modified live viral (MLV)
vaccines should be used, as these nearly always give a stronger,
more long-lasting response after only a single dose as compared
to inactivated vaccines. If not given at a 2-4 week interval, even 2
doses of an inactivated product may not elicit protective immunity.

In category 2, factors that may lead to apparent vaccine failure
through inability of the host to respond to vaccination include
the presence of very high levels of maternal antibodies (although
some vaccines can immunize in the presence of moderate
levels of maternal antibody); age of the animal, with very young
calves (<1 month) sometimes manifesting suboptimal immune
responses; immunocompromise due to concurrent disease, poor
nutrition, or high levels of stress; and genetic influences, which
can be expected in a genetically heterogeneous population and
which result in a continuum of responses to a given vaccine,
such that some animals fail to respond as desired even when
the majority of the population does. Poor ventilation in housed
animals may contribute to persistent problems with respiratory
disease even in the face of an appropriate vaccination program.

In category 3, one must consider expected and actual pathogen
exposure. Failure to vaccinate against pathogens to which animals
are actually exposed may result in apparent vaccine failure.

Regular postmortem evaluation with diagnostic microbiology
may help confirm or rule out suspected pathogens and guide
more rational vaccine choices. Strain variation of pathogens as
compared to vaccine strains may impact vaccine success; BVDV
is an example. Most mainstream BVDV vaccines contain both
type 1 and type 2 BVDV to provide optimal immunity against
both viruses. Nature of challenge may affect vaccine efficacy;
even the immune response provided by a reliable vaccine can
be surmounted by an overwhelming number of pathogens, an
unusually virulent pathogen, or by concurrent exposure to ad-
tional pathogens unrelated to the vaccine. While the above
points may appear obvious, it is important to consider and rule
out these possibilities when a vaccine has failed to prevent BRD
before making a quick switch to another brand or type of vaccine.

How can I best use BRD vaccines to
limit respiratory disease in cattle?

Vaccination should be one of multiple tools used to limit respira-
try disease in cattle. When using vaccines, remember basic
principles relevant to vaccine use: 1) animals need time to respond
immunologically to a vaccine—roughly 3 to 14 days, depending
on which aspect of the immune response is needed (serum or
mucosal antibody, T cell responses, etc.). Don’t expect much from
vaccines given right at the time animals are exposed to infection.
IBRV vaccines have been shown to provide protection within 3 to
5 days of administration, but this has not been evaluated for most
other BRD vaccines. 2) Pay attention to recommendations on the
vaccine label; if the label says a booster vaccine is required, optimal
immunity will not be induced if the booster is not given. Many
vaccines are labeled for single dose administration, but this is not
universally true. 3) If an animal is already infected with a pathogen,
vaccination against that pathogen is unlikely to be helpful. Think
about when cattle are at risk for infection and time vaccination to
occur at least 2 to 4 weeks before infection. 4) If an animal is vac-
cinated for a pathogen but it gets infected with a different pathogen,
vaccination can’t help. Many agents can cause BRD, so BRD in
vaccinated cattle can be due to infection with other agents not
included in vaccines. Vaccines for some pathogens that can cause
BRD are not commercially available (e.g., respiratory coronavirus).

In addition to selecting vaccines appropriate to the infection risks
expected for a given group of cattle, and using those vaccines
appropriately, biosecurity should also be used to limit the risk of
BRD. A foremost principle of biosecurity is that newly introduced
animals should be isolated from the rest of the herd for 2 to 4
weeks after purchase; this relatively simple concept is surpris-
ingsly rarely observed. Additionally, limit the age range in a group
of cattle; a common finding in herds with BRD problems is the
mixing of animals with a wide range of ages. In such situations,
older juveniles can amplify infectious agents and then spread
them to younger or older naïve individuals—somewhat like a
child from daycare coming home and giving the whole family a
cold! Evidence-based guidelines are lacking, but limiting the age
range of calves in a group to no more than 60 days may help
decrease the amplification and spread of BRD agents. Producers
should also limit crowding to control BRD; BRD is often a problem
when an unusually large numbers of cattle are brought together
in close contact (due to severe weather, problems with facili-
ties, etc.—not necessarily just due to new introductions).

In working to limit BRD, remember to control non-respiratory
infections; animals with other infections (such as infectious
diarrhea in calves) are more susceptible to BRD. Ensure optimal
transfer of maternal antibody from colostrum: transferred maternal
antibody is very important in limiting BRD (and other disease) in
calves. If calves within the first month or two of life are developing BRD, pay particular attention to maternal antibody transfer in the herd, and factors that can affect it such as dystocia, severe weather, and dam nutrition. Nutrition of calves can play a role in BRD susceptibility through the impact of nutrition on immune responsiveness. While supplementation of nutrients (such as vitamins and minerals) in excess of required amounts has not reliably been shown to prevent disease, correction of deficiencies of protein, energy, and certain vitamins and minerals has been shown to improve immune responsiveness. What is being fed to cattle may be important; some evidence indicates that high energy diets can increase the susceptibility of cattle to BRD (4).

Evaluating Reports of Vaccine Efficacy

Vaccines approved for sale in the United States must be proven by the manufacturer to be safe, potent, stable, and efficacious. Duration of immunity is an additional parameter that manufacturers are beginning to be required to address. Efficacy is usually determined by experimental challenge, using defined methods. For many bovine respiratory pathogens (e.g., BHV-1, BRSV, M. haemolytica), experimental challenge protocols have been developed which can induce disease of reasonable severity. To be approved for sale, vaccines need to prevent to a significant degree clinical signs associated with such experimental challenge. However, while protection against experimental challenge is a useful indication of the possible efficacy of a vaccine under field conditions, few experimental challenge protocols closely model the field situation in terms of simultaneous occurrence of other stresses and concurrent infection with other pathogens. Thus, it is ideal to evaluate vaccine efficacy through evaluation of an appropriately designed field trial. The design of field trials has generally improved greatly in recent years, but in the case of some vaccines (e.g., Mycoplasma bovis vaccines), no high quality published field trials evaluating the efficacy of vaccination for prevention of BRD appear to exist.

Veterinarians can arm themselves with useful information by evaluating published studies of vaccine efficacy in a critical manner. In some studies, animals are vaccinated but never exposed to the pathogen, although antibody or cell-mediated immune responses are measured. Such studies indicate that an immune response has occurred, but they do not guarantee protection from disease when animals are infected. More information can be gained when measurements of immune function are evaluated after natural or experimental infection. In experimental infection (challenge) studies, relatively small groups of animals are vaccinated and later purposely infected with the pathogen in the vaccine. While limitations of experimental challenge studies were addressed above, certain characteristics characterize a high quality experimental study. Some questions to ask include: Was a nonvaccinated control group, identical to the vaccine group in all ways except vaccination status, included? Were control animals tested at the same time as vaccinated animals? If factors which could impact vaccination was present (e.g., maternal antibody), were affected animals divided equally between control and vaccine groups? Did experimental challenge result in disease in the control group? If not, it is impossible to say if vaccination had an effect on challenge. Were investigators who evaluated clinical or pathological signs of disease kept unaware of (blinded to) the treatment status of the animals? This removes an important source of bias which otherwise can make data, particularly subjective data such as “depression” or “dyspnea”, suspect. Were statistical tests used to compare results of vaccine and control groups, and was the “p value” given to indicate likelihood that differences were due to chance alone? Other questions which can help determine the relevance of the experimental study to the field situation include: Was disease resulting from challenge clinically and/or pathologically similar to that seen in field cases? Was the vaccination regimen similar to that used in the field? How soon after vaccination were animals challenged? Did the time between vaccination and exposure mimic the field situation?

Field trials are characterized by allocation of animals in a “field” situation to either vaccine or control groups. Animals are treated accordingly and then followed for variable periods of time to determine whether disease occurs in vaccinated animals and, if so, whether vaccinated animals have disease less often, have disease that is less severe, or have improved production characteristics (e.g., averaged daily gain) following vaccination. When evaluating a field trial, consider the following questions to determine the value of the study: Were animals randomly allocated to control or treatment groups? This is critical; if there is no mention of randomization, it is difficult to gain useful information from the study due to the many types of bias that can impact the outcome. Were concurrent controls used, as opposed to historical controls? Historical controls (untreated animals examined in past months or years) are of much less value in determining vaccine efficacy, as many factors can affect disease outcome in a group of animals from year to year. How many animals were included in the study, and for how long did the study run? In general, trials with larger numbers of animals are more likely to reveal differences between vaccine and control groups. Were evaluators of disease blinded to the treatment groups to remove their bias in interpreting outcomes? Did disease occur in the control animals? One weakness of field trials is that natural disease must occur in the animals under study to determine the effect of vaccination on disease; the investigators have no control over this aspect of the study. If disease did not occur in at least the control animals, the vaccine cannot be evaluated for protection against the disease. Also, ask what outcomes were measured as evidence of protection against disease. In most cases of feedyard trials, BRD or fibrinous pneumonia morbidity and mortality are measured. If so, identify how cases were identified, and determine if the definition is accurate. In many trials, total morbidity and
mortality is also measured as an outcome. This may be considered a less reasonable outcome; for example, it may not be reasonable to expect BHV-1 vaccination to decrease deaths due to rumen acidosis. Production characteristics, such as rate of gain or feed efficiency, are also often evaluated, and net cost of vaccination, including estimated losses due to disease or loss of production, may be calculated. In these cases, evaluate how costs were estimated and determine if estimates appear to be accurate and reasonable. Finally, veterinarians may want to consider whether the field trial was conducted under conditions similar to that seen in their practice; if so, the results may be more relevant to the needs of their clients (adapted in part from reference 5).

Case example

History: A 65 head cow-calf operation is experiencing problems with BRD in nursing calves at 3 to 4 months of age. The cows are Angus and Hereford cross. The producer reports that approximately 30% of the calves have been found with signs of respiratory disease and 2 calves have died. Necropsy of one calf revealed BRSV and Mannheimia haemolytica, the other calf was not necropsied. Body condition scores of the cows are adequate and the pasture stocking density is acceptable. The calving season lasts for approximately 5 months. The cows are not vaccinated routinely but the herd was vaccinated once with an inactivated 5-way (IBRV-BRSV-PI3-BVDV1-BVDV2) 2 years ago.

Suggested actions: Depending on where the herd is in the calving season, it may be too late to prevent BRD in this year’s calves that are not already affected. With 30% of the calves showing signs of BRD as recognized by the producer, it is advisable to get the entire herd up so that all calves can be treated with a long-acting antibiotic to treat or prevent any bacteria that may be contributing to the pneumonia. In order to prevent BRD in calves in subsequent years:

1. Institute an appropriate vaccination program against respiratory viruses in the cows, taking care to administer one dose of inactivated or approved modified live 5-way vaccine within a few months of calving to boost antibody titers in the colostrum. Given an erratic vaccination history in the cows, ensure that all cows in the herd receive appropriate initial vaccination (including a booster if required per label instructions) before they are started on an annual program of boosting in late pregnancy.

2. Undertake actions to shorten the calving season. Having calves in a 5-month age range housed together gives the younger calves the opportunity to become infected by pathogens amplified in the older calves. Contact between cows and calves has been shown to be a risk factor for BRD in dairy calves (6), so it is likely that beef cows can also be a source of infection to calves. Thus limiting the group sizes may also be helpful to decrease contact between cows, older calves, and younger calves. An alternative to shortening the calving season is pasturing cows in groups that contain calves of no greater than a 60-day age range; if this is undertaken, remember that groups should not be pastured with fenceline contact, so that transmission of infectious agents among groups is still possible.

3. Consider instituting a practice of vaccinating calves; since calves in this herd are developing pneumonia at a time (3 to 4 months of age) when maternal antibodies are beginning to wane, vaccination of calves may be helpful. Vaccination of calves at 3 to 5 months of age in the face of moderate to low levels of maternal antibody has been shown to be helpful (7); in general, this has most often been shown for calves vaccinated with modified live virus vaccines against IBRV and BVDV. If vaccination of calves is instituted, it is ideal to give the calves 2 doses of vaccine timed so that the second dose is administered 2 to 4 weeks before BRD cases are expected. In this herd, it would be ideal to vaccinate calves at 1.5 months of age and 2.5 months of age in the next calving season. If logistics prohibit the administration of 2 doses of vaccine, give one dose of a MLV vaccine labeled for single dose administration when the calves are 2 months of age. If calves are in contact with pregnant cows, remember to use an MLV vaccine approved for use in pregnant cows. Inactivated vaccines have not been as reliable as MLV vaccines for immunizing calves with maternal antibody, but a few products have been shown to be effective. Ask the company for data supporting this before choosing an inactivated vaccine to immunize calves with maternal antibodies. Intranasal vaccination may be superior to intramuscular or subcutaneous vaccination in the face of maternal antibodies, but more evidence is needed to prove this. Given the findings on postmortem of the single calf in this example, vaccination of calves in this herd with 5-way viral and also a Mannheimia haemolytica vaccine would be rational.
References


BRSV: What’s the Latest?
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BRSV: The Virus and the Disease
Bovine respiratory syncytial virus (BRSV) causes respiratory disease in cattle, sheep, and goats around the world. Serologic surveys indicate that the vast majority of cattle are infected at some point in their lives. Infection with BRSV can cause a wide range of clinical signs in cattle. Disease may be inapparent; it may be nonspecific and difficult to distinguish from respiratory disease due to other causes; or it may be severe and fatal, with signs including respiratory distress and subcutaneous emphysema in affected animals. Cattle that are 2 to 6 months of age are usually most severely affected; thus BRSV is most likely to be a problem for calves still nursing their mothers, stocker cattle, and lightweight cattle entering feedyards.

One of the reasons that BRSV is a persistent problem for cattle is that natural infection does not induce long-term immunity. While serum antibodies and cellular immunity induced by previous infection can make subsequent disease less severe, cattle can be expected to be reinfected with BRSV multiple times over the course of a lifetime. In fact, adult cattle which have had ample opportunity for previous infection can sometimes have quite severe disease; this is likely due at least in part to aspects of the immune response (possibly modified by genetic factors) in specific individuals. BRSV can cause severe disease alone, or it can participate with other viruses or bacteria to cause pneumonia. Co-infection of cattle with BRSV and bovine viral diarrhea virus (BVDV), Mannheimia haemolytica, or Histophilus somni have all been shown to cause more severe disease than that caused by any of the agents acting alone.

How Does BRSV Cause Disease?

BRSV Transmission
BRSV is easily transmitted among susceptible calves or cattle, with clinical signs seen in most animals in a group within 1-2 weeks of the first case identified. Based on extrapolation of information regarding the closely related human RSV, transmission by direct contact or short distance droplet transfer is more important than long distance aerosol. Thus transmission can be minimized by preventing direct contact between infected calves or cattle and at-risk animals. Equipment such as bottles or buckets, and clothing of caretakers, are likely additional important means of transmission. Thus, caretakers should wear separate coveralls and gloves while handling infected calves to prevent spread of BRSV, and bottles and buckets should be disinfected after use. Because BRSV is an enveloped virus, it is does not persist in the environment for long periods (> 24 hours is unlikely), and it is susceptible to many disinfectants. A recent study in Norway showed that calf respiratory disease was more often a problem in herds where calves shared housing with cows during the first week of life, where more than an 8 week age difference was present among calves housed together in group pens, and where calves were left with the dam for over 24 hours after birth (1). These are all factors that are likely to increase BRSV transmission among calves.

Pathology Caused by BRSV
Gross lung pathology induced by BRSV includes cranioventral collapse or consolidation of lung lobes, often with expansion of the dorsocaudal lobes (dorsocaudal lung may fail to collapse when thoracic cavity is opened). The dorsocaudal lung may have a heavy, rubbery consistency due to interstitial pneumonia. In severe cases emphysema is present. Because other etiologies can cause similar gross findings, involvement of BRSV can be confirmed by immunohistochemistry or reverse transcriptase-PCR (RT-PCR) for the virus in affected lung. Histologic findings seen in BRSV infection include bronchiolitis, alveolitis, and the presence of syncytial cells in bronchioles and alveoli. A mixture of mononuclear cells and neutrophils is usually seen in bronchioles and alveoli of affected animals. In the most severe cases, acute interstitial pneumonia may be seen, with edema, emphysema, and alveolar hyaline membrane formation.

Role of Host Immune Response in Protection and Disease
An important feature of BRSV infection is that some aspects of the immune response can sometimes contribute to disease; this is termed immunopathogenesis. However, certain aspects of the immune response are also important for decreasing disease due to BRSV infection. Calves with moderate levels of serum neutralizing antibodies tend to be resistant to more severe disease, although this is not invariably true. The ability of calves to rapidly produce IgA on mucosal surfaces has repeatedly been shown to limit disease due to BRSV. Also, CD4 helper T lymphocytes and CD8 cytotoxic T
lymphocytes are both important for clearance of virus once calves are infected, and for limiting disease. Vaccines that can induce serum neutralizing antibodies, mucosal IgA, and/or T helper and T cytotoxic responses should thus help calves resist disease, although they may still become infected. While it is possible to limit disease with vaccination, it is likely to be difficult if not impossible to completely prevent BRSV infection using any vaccination strategy.

Aspects of the immune response that may lead to more severe disease include strong T helper type 2 (TH2) responses, which can sometimes lead to production of IgE. TH2 cells produce cytokines (chemical messages) that direct B lymphocytes to produce a profile of antibodies that includes IgE, and IgE has been associated with severe disease in BRSV infected cattle. Because IgE also mediates allergy and anaphylactic reactions, it is not surprising that IgE produced following BRSV infection can be associated with severe disease. It is not entirely clear why some cattle mount TH2 responses with IgE production following BRSV infection, but genetics and the presence of other infectious agents or allergens in the environment at the time of BRSV infection is likely important. For example, calves infected with BRSV after exposure to allergenic fungi sometimes produced higher levels of IgE directed against the virus, and had more severe disease, than calves exposed to BRSV but no allergenic fungi (2). Recent studies of severe disease in children infected with human RSV showed that expression of certain receptors for chemokines (chemical messengers that modify inflammatory responses) was related to disease severity (3). Also, production of antibody that does not neutralize (block viral infection), which may be induced by a strong TH2 response or by certain vaccines, has also been linked to disease of increased severity (4).

When is BRSV Likely to be a Problem For Cattle?

In the field, BRSV most often causes important disease in 2- to 6-month-old calves. Thus, BRSV is most likely to be a problem in nursing calves, recently weaned calves in stocker or backgrounding operations, and lightweight calves entering feedlots. However, calves less than 2 months of age can sometimes develop disease when exposed to BRSV, and the relative frequency of disease in calves older than 1- to 2- months of age may simply be due to the presence of maternal antibody that can more often limit disease in younger calves. Additionally, severe fatal disease has been reported in adult beef and dairy cattle, so cattle of any age or type can potentially be affected by BRSV. BRSV infection commonly occurs after cattle have been mixed and transported; however, BRSV infection has also been found to occur repeatedly over the course of one to two years in closed dairies (5). Thus, BRSV infection can occur in groups of cattle with no recent introductions; whether this is due to long term persistence of infection in certain animals, or continuous, low-level transmission among animals, has been debated (6). While respiratory disease with its related effects on growth, productivity, and animal well-being is the most important concern in BRSV infection, a recent study showed that bulls experiencing BRSV infection had poorer sperm morphology 6 months after infection, as compared to bulls that were not infected. This finding was associated with a small but statistically significant impact on fertility (7).

Do BRSV Vaccines Work? What Type of Vaccines Should be Used?

Much research has evaluated the efficacy of BRSV vaccines, with some evidence supporting the use of some vaccines which are currently commercially available (8,9,11-15). To summarize the evidence briefly, BRSV vaccination of both beef and dairy calves and feedlot cattle has been shown to decrease disease measured either by seroconversion or producer- or veterinarian-diagnosed respiratory disease in some but not all studies. Improved outcomes such as decreased respiratory morbidity and mortality and improved productivity are found in some studies (9), but not others (10). The majority of studies showing benefit have used modified live vaccines, but two commercially available killed vaccines have protected calves from disease due to experimental BRSV exposure (11, 12). It appears that BRSV vaccination can be associated with meaningful improvements in cattle health and productivity, but positive effects cannot be guaranteed in all cases. Recent attention has focused on BRSV vaccines for intranasal (IN) delivery. One study showed that a commercially available multivalent (IBR/BVD/PI3/BRSV) vaccine for IM use protected calves from disease due to BRSV when administered IN (13). This extralabel route of administration protected calves with no BRSV antibodies from disease when they were exposed 8 days later; however, calves did shed the IBR and BVD virus in the vaccine for several days after vaccination. Because rapid onset of nasal IgA production has been associated with resistance to disease due to BRSV, nasal vaccination for BRSV is logical. In some cases IN vaccines have been effective when administered in the face of maternal antibody (16-18). However, no field trials are currently published which compare IN BRSV vaccines to those given IM or SQ in large numbers of cattle in a natural setting, so more research is needed to determine whether IN BRSV vaccination is safe, effective, and superior to IM or SQ BRSV vaccination.

Because adult cattle can develop significant and sometimes fatal disease associated with BRSV infection, adult cows and bulls should be revaccinated annually for BRSV.
Are There Any Downsides to BRSV Vaccination? What Else is Important For Preventing BRSV Infection?

While currently available BRSV vaccines marketed in the U.S. have been shown to be safe and effective, in the 1980’s and 1990’s occasional reports described severe disease that seemed related to BRSV vaccination. Both inactivated and modified live vaccines were linked to adverse outcomes in vaccinated cattle. These events were likely related to the specific formulation of the vaccines in question. No disease enhancement has been reported for any vaccines commercially marketed in the U.S. in at least 10 years, but because of past events of vaccine-enhanced BRSV disease, the use of mainstream vaccines which are well-characterized from use in large numbers of cattle must be emphasized. Given the risk that vaccine formulation can be related to enhanced disease, the use of autogenous vaccines for BRSV is ill-advised.

The question is sometimes raised regarding whether BRSV vaccines should be administered in the face of a natural BRSV outbreak. There is little information to allow an evidence-based decision regarding this question. However, because one reported outbreak of severe disease seemed to be related to vaccination of calves in the early stages of BRSV infection (19), this author does not recommend BRSV vaccination in the face of an outbreak.

As noted earlier, based on extrapolation from research regarding human RSV, transmission should be controlled by preventing nose-to-nose contact between infected and at-risk individuals, and contact between contaminated fomites such as bottles and buckets. Transmission on the clothing of people in contact with infected calves is also likely, so clothing should be changed after working with infected cattle and before working with susceptible but uninfected cattle. The incubation period of BRSV is approximately 5 days, so isolation of newly introduced cattle or calves for 2 weeks should provide adequate protection. However, BRSV infection has been shown to cycle in closed operations (5), so it may be difficult or impossible to completely prevent the occurrence of BRSV-induced disease in groups of cattle.

Are There Any Specific Therapies for BRSV?

There are no specific therapies for BRSV infection; therapy is aimed at supportive care and prevention of secondary bacterial pneumonia. Children infected with RSV typically recover uneventfully, but certain individuals with other underlying health problems are at risk for severe disease which can be fatal. Such children are treated with hyperimmune globulin or humanized monoclonal antibody, which is very expensive. A specific treatment which is a current focus of research is the use of small inhibiting RNA molecules (siRNA), which have been shown in experimental settings to suppress viral infection by causing targeted destruction of messenger RNA molecules for viral proteins (17). While evaluation of this strategy is at an early stage, it may be applicable to the control of bovine viral diseases such as BRSV in the future.
References


Evidence for and Against Antimicrobial Resistance (AMR) in Animals

Jay T. McClure, DVM, MS, Diplomate ACVIM

Clinical importance in veterinary medicine

A. Limits our selection of effective antimicrobials for treatment

B. Antimicrobial selective pressures affects all microbes in the body
   1. AMR pathogens
   2. AMR in resident microflora that can be opportunist or can transfer resistance to newly introduced pathogens

Public health concerns

A. Resistant bacteria in animals may reach humans
   1. Food contamination
   2. Environmental contamination (manure spreading)- Crops, water
   3. Direct contact- food animals & companion animals
   4. Vectors (birds, rodents, ect) transporting it to different locations

Evidence of AMR from animals

**Avoparcin** is a glycopeptide antimicrobial that was used in many European countries as a growth promoter in the 1990's. An association was made between use of this drug, primarily in swine and poultry, and vancomycin resistant enterococci (VRE) in people. Countries that used avoparcin had a 2-4% VRE rate where as the VRE rate was <0.1% in countries that did not. VRE was also identified more frequent from swine farms that used avoparcin. Vancomycin is also a crucial antimicrobial for treatment of “super” infections such as methicillin resistant *Staphylococcus aureus* (MRSA) and unresponsive *Clostridium difficile*. It is for this reason that glycopeptides are prohibited for use in food animals in North America.

**Enrofloxacin** has been banned in poultry in the USA because of a temporal association of fluoroquinolone resistant campylobacter in people and the labeled use of enrofloxacin for treating *E. coli* infections in chickens. There was also evidence of fluoroquinolone resistant campylobacter being isolated from the intestine of chickens. In 2001 a risk analysis estimated that this resistance adversely affected the treatment of ~14,000 Americans. The US ban was official in 2005. Enrofloxacin and danofloxacin is currently labeled for respiratory infections in cattle in Canada. Use of these drugs in an extralabel manner in food animals in the USA is ILLEGAL. In Canada extralabel use is strongly discouraged but not illegal.

**Ceftiofur**, a frequently used 3rd generation cephalosporin commonly used in food animals, is potentially linked to extended spectrum B-lactamase (ESBL) resistance in enteric organisms, particularly salmonella and *E. coli*. Most of this resistance is due to a cephamicinase gene, *bla*<sub>APR1</sub>, that is found on plasmids. Many of these organisms have plasmids with multiple drug resistant genes (up to 9-10 antimicrobials). The *bla*<sub>TEM3</sub> gene also confers resistance to ceftriaxone, a human 3rd generation cephalosporin, that is the antimicrobial drug of choice for treating invasive Salmonellosis in children. Also 3rd generation cephalosporins are widely used for many severe invasive infections in humans including meningitis. In the first 4 months of 2002, 47 people were identified with resistant *Salmonella newport* which resulted in 17 people being hospitalized and one person died (immunosuppressed). This outbreak was associated with consumption of lean ground beef. Since then the
total number of multi-drug resistant *S. newport* isolates from cattle has increased. *Bla$_{TEM}$* has since been found in other pathogenic salmonella and *E. coli* isolates from food animals. *Bla$_{AMR}$* has been found to be present in non-pathogenic *E. coli* that make up the normal gut flora in healthy calves have been found to harbor this multidrug resistant plasmid as well which may act as a breeding ground for transfer of these resistant genes. However this form of resistance from generic *E. coli* is commonly found in poultry but very uncommon in cattle from the Canadian Integrated Program for Antimicrobial Resistance Surveillance (CIPARS) surveillance data. Based on 2003 and 2004 data, the CIPARS noted that in Quebec 63% of the *Salmonella heidelberg* cultured from retail chicken and 33% *S. heidelberg* from human patients were resistance to 3rd generation cephalosporins. On investigation it appeared the poultry industry in Quebec had increased its use of ceftiofur for *E. coli* omphalitis because another drug used to treat this condition became unavailable. The Quebec poultry industry voluntarily discontinued ceftiofur use in 2005-2006 and the number of resistant *S. heidelberg* decreased dramatically, 7% in retail chicken isolates and 8% in human isolates in 2006. In 2007, Quebec partially lifted their voluntary ban on ceftiofur. In 2008, the resistant isolates in retail chicken had increased significantly 18% and the percent of resistant isolates from humans are trending upward (12%). (Dutil et al, Emerg Infect Disease 16:48-54, 2010)

In the fall of 2008 USA FDA proposed banning extra-label use of ceftiofur in food animals. This proposed ban has been withdrawn and further review of the data is ongoing.

**Apramycin** is an aminoglycoside that is approved for oral use in pigs. A recent study out of Denmark has correlated the use of apramycin in swine with increase *E. coli* gentamicin resistance from the intestinal tract of swine. Gentamicin is a drug used in severe, often life-threatening, Gram-negative bacterial infections such as septicemia.

**Methicillin Resistant Staphylococcus aureus (MRSA)** - MRSA has been a problem primarily of human beings but in the last 20 years there has been an increasingly identified in animals. Initially it was seen primarily in horses and dogs and was considered by most to be likely an example of reverse zoonosis (human to animal transmission). In the last five years the identification of a large animal (MRSA ST398) strain that is predominately seen in pigs and occasionally in ruminants and horses. This large animal MRSA strain is rarely seen in people and most human cases are associated with direct contact with affected animals (pig farmers, veterinarians). This food animal MRSA most likely emerged with in the pig population although transfer of the resistant gene from human MRSA cannot be ruled out. Also recently the emergence of methicillin resistant *Staphylococcus pseudintermedius* infections in small animals is likely an example of emergence of antimicrobial resistance in an animal pathogen as this bacteria is extremely uncommon in people.

**What is the role of agriculture?**

It is hard to obtain the actual amount that agriculture has contributed to AMR. Most of the antimicrobial resistance found in human bacterial pathogens comes primarily from overuse of antimicrobials in human medicine. One estimate is that agriculture use accounts for 2-3% of all antimicrobial resistance but that is hard to define. Why?

1. It is hard to determine where the resistance originated- human versus animal.

2. Resistant bacteria or genes generated from human antimicrobial use may find its way into animals and then animals act as reservoirs and/or vectors for transmission of this resistance back to humans as well as other animals.

3. Many blame the use of antimicrobials in feed at subtherapeutic doses for growth promotion and/or disease prevention as a major cause of AMR and point to the fact that almost ½ the weight of all antimicrobials made in a year end up being fed to animals in North America. On the other hand they do not point to the fact that these are mainly antimicrobials that are of limited use (due to resistance) in human medicine such as ionophores, penicillin and tetracycline. However there may be a role of these antimicrobials selecting for multidrug resistant organisms because they process a plasmid that contains resistant genes to tetracycline (for example) and as well as other antimicrobials.

4. There is good evidence that the cephalosporin resistance in *E. coli* and *Salmonella* seen in animal isolates likely is a result of cephalosporin use in veterinary medicine, there is less evidence that these resistant genes are effectively transmitted to the human population. Still this resistance is not just a concern from a human transmission standpoint but it is also a concern to veterinarians as increasing resistance will leave some infections very difficult to treat.

5. Largely ignored to date but companion animals may play a major role in developing or acquiring antimicrobial pathogens and then transmitting them to people. This risk of animal to human transmission has not been looked at to date.
Prudent Decision Making in the Selection of Antimicrobials: Basic Principles

Jay T. McClure, DVM, MS, Diplomate ACVIM

There are several factors that go into making appropriate antimicrobial therapy decisions. These include knowing the pathogen involved and its susceptibility to the antimicrobials being considered, the pharmacokinetic properties of the antimicrobial being used as well as its pharmacodynamic properties. Knowledge of these principles will allow the practitioner to use antimicrobials in a manner that will optimize successful therapy and minimize antimicrobial resistance. For food animals there are other considerations including public safety from exposure to drug residues and cost of therapy. When considering therapeutic cost one must consider not only the cost of the drug but the cost of lost of production from withdrawal times (discarding milk) and the cost of labor for administration. Because of this many of the antimicrobials have labels for food animals that tend to minimize cost by using a minimal concentration for a short period of time that has been shown to be effective for the disease on the label. However this does not mean that this label dose is appropriate for treating other conditions not on the label (extra-label drug use ELDU) nor is the dosing regimen on the label necessarily always maximizing efficacy for the disease that is on the label. Thus it is important that the practitioner understands some basic pharmacology principle so they can maximize the likelihood therapeutic efficacy.

Antimicrobial susceptibility

Antimicrobial sensitivity data has been commonplace in veterinary medicine for decades and has proved to be useful when making decisions on appropriate antimicrobial therapy. It is ideal to make antimicrobial decisions based on sensitivity data from a bacterial culture. This information usually takes 3-5 days from the time the culture is taken and waiting this long to start antimicrobial therapy is not appropriate in most cases. Thus it is essential that a practitioner knows the likely pathogens to be involved in the disease process and to have a good idea of what the sensitivity patterns are for this pathogens so that empirical therapy can be instituted responsibly. This knowledge is part of a veterinarian’s education and experience as well as published reports. One needs to keep in mind however that information derived from one region may not be the same for another region as well as data published several years ago may not reflect today’s reality. So when available it is best to use data that is recent and comes from a similar geographical region. As part of the development of the CVMA Commodity Specific Antimicrobial Prudent Use Guidelines, Provincial diagnostic laboratories were asked to provide sensitivity data for the most common aerobic pathogens for cattle, swine, and poultry. The tables on the next page are the data collected from PEI and Alberta for 2004-2005. When you compare the respiratory pathogens H. somni, M. haemolytica, and P. multocida it appears that the H. somni is about two times more prevalent in Alberta (26%) compared to PEI (13%). When you compare the toxigenic E. coli sensitivity data, the susceptibility of trimethoprim-sulfa is better in the Alberta isolates (78%) than it is in PEI (44%). Although one must be careful when interpreting this as there is a lot of inherent bias to voluntary culture submission to diagnostic labs, this type of regional data can impact your antimicrobial decisions.
### Bovine Non-Mastitis Isolates, AVC Diagnostic Lab 2006-2009

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>2006-2008 n (avg)</th>
<th>CEF/FOX</th>
<th>ERYTHROMYCIN</th>
<th>FLORFENICOL</th>
<th>OXYTETRACYCLINE</th>
<th>PENICILLIN</th>
<th>STREPTOMYCIN</th>
<th>TILMICOSIN</th>
<th>TRIM/SULFA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arcanobacterium</td>
<td>4</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>50</td>
<td>100</td>
<td>75</td>
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<td>E. Coli</td>
<td>36</td>
<td>75</td>
<td>75</td>
<td>75</td>
<td>11</td>
<td>0</td>
<td>6</td>
<td>22</td>
<td>42</td>
</tr>
<tr>
<td>Hemophilus</td>
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<td>100</td>
<td>100</td>
<td>83</td>
<td>100</td>
<td>67</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Listeria</td>
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<td>0</td>
<td>100</td>
<td>88</td>
<td>100</td>
<td>100</td>
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<tr>
<td>Mannheimia</td>
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<td>14</td>
<td>100</td>
<td>90</td>
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<tr>
<td>Pasteurella</td>
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<td>100</td>
<td>22</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>12</td>
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<td>0</td>
<td>100</td>
<td>100</td>
<td>100</td>
<td>0</td>
<td>0</td>
<td>100</td>
</tr>
</tbody>
</table>

### Bovine Mastitis Isolates, AVC Diagnostic Lab 2006-2009

<table>
<thead>
<tr>
<th>Pathogen</th>
<th>2006-2008 n (avg)</th>
<th>CEF/FOX</th>
<th>CEPHALAXIN</th>
<th>CLOXACILLIN</th>
<th>NEOMYCIN</th>
<th>OXYTETRACYCLINE</th>
<th>PENICILLIN NOVOBIOCIN</th>
<th>PRLIMYCIN</th>
<th>TRIM/SULFA</th>
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</thead>
<tbody>
<tr>
<td>E. Coli</td>
<td>92</td>
<td>97</td>
<td>53</td>
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<td>91</td>
<td>83</td>
<td>2</td>
<td>0</td>
<td>98</td>
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<tr>
<td>Klebsiella</td>
<td>39</td>
<td>95</td>
<td>92</td>
<td>0</td>
<td>87</td>
<td>69</td>
<td>5</td>
<td>3</td>
<td>97</td>
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<tr>
<td>Coag+ Staph</td>
<td>175</td>
<td>100</td>
<td>99</td>
<td>100</td>
<td>94</td>
<td>98</td>
<td>100</td>
<td>99</td>
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</tr>
<tr>
<td>Streptococcus</td>
<td>171</td>
<td>96</td>
<td>100</td>
<td>98</td>
<td>13</td>
<td>78</td>
<td>99</td>
<td>85</td>
<td>98</td>
</tr>
</tbody>
</table>
Data from *in vitro* sensitivity data does not always correlate with therapeutic outcome. Treatment failures occur with antimicrobials that should be susceptible to the bacterial isolate according to the sensitivity panel. Treatment successes occasionally occur although the sensitivity report indicates that the antimicrobial you empirically prescribed prior to culture results was resistant to the isolated pathogen. What causes such discrepancies between the *in vitro* sensitivity testing and the *in vivo* treatment results?

The most commonly reported antimicrobial sensitivity information is qualitative data that classifies isolates as sensitive, intermediate, or resistant to the antimicrobial drug being tested. This is usually determined by agar disk diffusion test where the subsequent diameter of the zone of inhibition of bacterial growth that develops around the antimicrobial disk is inversely correlated with the minimum inhibitory concentration (MIC) of drug that inhibits bacterial growth. For each antimicrobial breakpoints zone of inhibitions have been predetermined by a panel of experts. If the zone of inhibition is smaller than this breakpoint it is considered resistant and if it is larger than this breakpoint it is considered sensitive. The report the veterinarian reads gives no quantitative information as to how sensitive or resistant they are. A veterinarian may choose a susceptible antimicrobial that was close to the breakpoint and results in less effective treatment than if he/she had chosen an antimicrobial with a more sensitive MIC.

The breakpoint concentrations used to determine the sensitivity of many of the older antimicrobials, are often based on human data and not animal data. Consequently, it is assumed that the bacterial isolates, pharmacokinetics and dosage forms used in veterinary species are similar to humans but this is not always the case. A good example of this is ampicillin whose sensitivity breakpoint, based on human data, is 8 µg/ml for *E. coli*. If you are administering sodium ampicillin intravenously you will easily achieve drug concentrations above this breakpoint. However, if you are using the food animal dose of 6 mg/kg IM, peak serum concentrations of only 2-3 µg/ml will be achieved. Thus sub-therapeutic dosing occurs for many *E. coli* isolates that appear susceptible on a sensitivity panel. Although not commonly performed in veterinary medicine, one can request MIC testing where you get the actual concentration that bacterial growth is inhibited. This along with knowledge of the pharmacokinetics and pharmacodynamics of the antimicrobial can lead to more informed antimicrobial dosing decisions.

The *in vitro* conditions of an antimicrobial sensitivity test are quite different than the *in vivo* environment where the bacterial infection is taking place. *In vitro* testing requires exposing a standardized concentration of bacterial inoculum to a constant concentration of drug for a fixed period of time under consistent pH, oxygen tension and temperature conditions. *In vivo*, the antimicrobial drug concentrations are continually fluctuating. An infection, such as an abscess may have a low pH, limited oxygen tension, and/or have purulent material that can inhibit the activity of many antimicrobials. Furthermore physiological barriers, such as the central nervous system and mammary gland, or pathological barriers, such as the wall of an abscess, can limit the penetration of many antimicrobials. Alternatively, the affects of an intact immune system or the *in vivo* sub-MIC therapeutic effects of some antimicrobials may result in a therapeutic success. Finally the result of therapeutic failure may be simply due to short duration of therapy.

**Pharmacokinetics**

It is important to know some basic pharmacokinetics when determining appropriate dosing regimens for antimicrobials. Often this information can be found on the inserts in the drug package of newer antimicrobials but is often absent from older antimicrobial packages. There are several textbooks that are useful references for finding this information. Two very useful textbook references are:

- **a.** Antimicrobial Therapy in Veterinary Medicine, 4th ed; Giguere, Prescott, Baggot, Walker, Dowling, Blackwell Publishing, 2006; This is an excellent source for veterinary antimicrobial pharmacology and points out differences between Canada and USA.

- **b.** Veterinary Drug Handbook, 6th ed; Plumb, Wiley Publishing 2008 (USA focused)

**There are also websites that you can obtain some of this information:**

- **a.** American Academy of Veterinary Pharmacology and Therapeutics (AAVPT) http://www.aavpt.org/USPmonographs.shtml

- **b.** Drugs.com http://www.drugs.com/vet/ has Canadian package inserts of some drugs but is not complete.

- **c.** Also on can search for manuscripts focused on the pharmacokinetics and pharmacology of specific antimicrobials in databases like Pubmed.

Specific pharmacokinetic data that is important to consider is the peak concentration, the distributions into specific tissues and half-life of elimination. For many of the antimicrobials developed for beef cattle, they have been formulated to have a slow absorption phase which results in a longer absorption half-life than the elimination half-life resulting in what is referred to flip-flop kinetics. Essentially flip-flop kinetics results with a longer drug elimination than the true elimination half-life. If you know the
peak concentration and the relative half-life of elimination, you can predict approximately what the concentration of the drug will be in the body fluid/tissue studied for any particular time. For each elimination half-life the drug concentration drops in half. For example if the peak concentration is 8mg/l and the elimination half-life is 2 hours. After 2 hours after dosing the concentration should be ~4mg/l and 4 hours after dosing the concentration would be 2 mg/l. After 5 elimination half-lives (10hrs) >95% of the drug is gone and the concentration would be 0.25mg/l.

Another useful parameter to look at is the volume of distribution (Vd). The Vd gives you some insight into how the drug distributes in the body. If the Vd = 1 L/kg of bodyweight you know the drug distributes through all body fluids (both intracellular and extracellular) equally. Drugs that are large molecules or cations or anions (hydrophilic but not lipophilic) tend to have limited penetration into cells and privileged body sights such as the CNS and mammary gland and thus have a Vd < 0.3L/kg which means it is limited to the extracellular fluid. On the other hand if the Vd > 1 L/kg it indicates this drug concentrates intracellular and/or in specific tissues. Also if you know the Vd and the dosage regimen, you can predict the likely peak concentration of drug in the blood. For example if you are dosing cefiofur at 1 mg/kg and the Vd = 0.3L/kg, you can estimate that the peak concentration will be ~3 mg/L (1mg/kg ÷ 0.3L/kg). For drugs with flip flop kinetics the actual peak concentration will be lower than this calculation because of the prolonged absorption; it is best to refer to package insert information or from research data.

It is important to understand basic pharmacokinetic principles as well as the MIC to use pharmacodynamic principle in devising appropriate dosage regimens, especially when using antimicrobials in an extra-label manner.

**Pharmacodynamics**

The pharmacodynamic properties of antimicrobials should also be considered when determining an effective dosage regimen. One of the most important pharmacodynamic properties of antimicrobials is the relationship of the concentration of that drug at the site of infection to the MIC of the pathogen(s) involved in the infection. Most antimicrobials’ efficacy is related to the amount of time they spend above the MIC of the bacterial pathogen between dosing intervals. In other words, once the drug concentration is above the MIC concentration there is not much enhanced bacterial killing by increasing the drug concentration. However, the bacterial killing of a few antimicrobial classes, namely the fluoroquinolones (enrofloxacin), aminoglycosides (gentamicin), and metronidazole exhibit significant concentration-dependent killing and are most effective when drug concentrations are 8-10 times the MIC of the bacterial isolate. Thus appropriate dosage regimens are best determined by having antimicrobial MIC data on the pathogen of concern. Since it is currently uncommon to get MIC information from clinical isolates, we often have to refer to published data that gives MIC information for specific pathogens. Often these studies report the MIC50 and MIC90 for a particular pathogen. The MIC50 is the concentration of antimicrobial that 50% of the isolates tested were inhibited from growing and the MIC90 is the concentration in which 90% of the isolates are inhibited. Ideally one would want to use the MIC90 when determining doses on empirical antimicrobial therapy.

Post-antibiotic effect (PAE), is another important pharmacokinetic property of some antimicrobial-pathogen combinations. Drugs exhibiting PAE prevent bacterial regrowth after the drug is removed or falls below MIC for a period of time, often for several hours. Concentration dependent killing antimicrobial classes such as the aminoglycosides and fluoroquinolones exhibit significant PAE when exposed to most sensitive Gram negative isolates. The macrolides also have been shown to have PAE against many bacterial isolates. Antimicrobials that exhibit PAE can be dosed at an interval greater than the time the drug concentration falls below MIC concentrations. One limitation is it is difficult to predict how long the PAE will last in vivo. The penicillins and cephalosporins do not possess any PAE thus their concentrations should ideally remain above the MIC of the pathogen throughout the duration of therapy to maximize efficacy. These principles will be address in the next session using case scenarios.
Public Health Concerns

Using these principles to derive efficacious dosage regimens, especially for off-label indication can result in dosing regimens that are not consistent with the label. In such situations the meat and milk withdrawal times should be adjusted. It is the prescribing veterinarian’s responsibility to provide appropriate withdrawal times. This can be estimated or preferably based on published data. This is not something most practitioners are comfortable in calculating and finding supporting research data can be time consuming. Canadian gFARAD (http://www.cgfarad.usask.ca/) is a service that veterinarians are encouraged to use for guidance for appropriate withdrawal times for extra-label drug use. Canadian gFARAD does not provide withdrawal information for compounded drugs and APIs (active pharmaceutical ingredients).

Another public health concern is the overuse of antimicrobials in veterinary medicine especially those of great importance to human medicine. Health Canada has developed category list of antimicrobials based on their importance in human medicine with Category I being those that are highly important to human medicine and Category III being of medium importance in human medicine. Most of the antimicrobials labeled for food animals fall in Category II (Important). Ceftiofur (3rd generation cephalosporin), enrofloxacin (fluoroquinolones), and polymixin (component of Special Formula mastitis treatment) are Category I drugs. Category III drugs labeled for food animals include tetracycline and florfenicol.

With increased emphasis on prudent antimicrobial usage by veterinarians, decisions on which antimicrobial to choose to treat an infection becomes more complicated. It is recognized that veterinarians have the expertise to make such decisions. However this can be time consuming for a busy practitioner to keep up on. It is best to know a lot about a few antimicrobials and use them primarily that to know a little about a lot of antimicrobials and use a large repertoire. The CVMA has recently produced prudent use guidelines for antimicrobial use for dairy, beef, poultry and swine to assist veterinarians with these difficult decisions. These were mailed to all large animal veterinarians that are members of the CVMA and are available on the members only area of the CVMA website. These guidelines will be referred to in session 3 case scenarios.
Rationale Decision Making in the Selection of Antimicrobials: Practical Applications in Bovine Practice

Jay T. McClure, DVM, MS, Diplomate ACVIM

Scenario #1

You are called to examine a group of feeder steers recently purchased from the local auction. You pick out one steer to examine. He is breathing 60 times per minute with head and neck extension. Purulent nasal discharge and salivation is present. His temperature is 41.5°C and mucus membranes are pale with a CRT 3 ½ sec. On auscultation you hear tracheal rattles, loud bronchial sounds in the cranial-ventral region and harsh bronchial sounds in the caudal dorsal region.

a. Is there likely a bacterial infection that requires antimicrobial therapy?

b. If so what are the likely pathogens?

c. What would be your first line drug choice? WHY?

i. Sensitivity pattern of the likely pathogens

ii. Ability to get to the site of infection

iii. Category of the antimicrobial class for importance in human medicine

d. What would be your recommended dosage regimen (dose, route, and duration of therapy)? (Indicate if it is label or extralabel)
Scenario #2

You are asked to examine a 5 day old dairy heifer that has been scouring for two days. The producer has told you that this calf received 2 liters of colostrum when it was born and the calf has been treated for the last day with oral electrolytes but seems to be getting worse. The calf’s temperature is 40.6°C and she is recumbent and appears listless with very little suckle reflex but her lungs sound clear.

a. Is there likely a bacterial infection that requires antimicrobial therapy?

b. If so what is/are the likely bacterial pathogen(s)? And what body system(s) are likely affected?

c. What would be your first line drug choice? WHY?
   i. Sensitivity pattern of the likely pathogens
   ii. Ability to get to the site of infection
   iii. Category of the antimicrobial class for importance in human medicine

d. What dosage regimen (dose, route, and duration of therapy)? (Indicate if it is label or extralabel)
Scenario #3

You are called out to exam a 3-year-old Holstein cow. The cow was found recumbent and severely depressed at the evening milking. She calved 5 days ago and had a retained placenta for 2 days post calving. She was BAR and milking well until this morning’s milking where her milk production was decreased.

On physical examination the cow is severely depressed, tachycardic, tachypneic and her temperature is subnormal. She has 1 weak rumen contraction every 2-3 minutes on auscultation. Clinically she appears to be 7% dehydrated. Her manure is watery but not excessive in quantity. The left front quarter of the mammary gland is warm and swollen. The milk in this quarter is watery with white flecks and CMT is 3+. The other 3 milk quarters are within normal limits. Rectal palpation reveals an involting uterus.

a. Is there likely a bacterial infection that requires antimicrobial therapy?

b. If so what is/are the likely bacterial pathogen(s)?
   And what body system(s) are likely affected?

c. What would be your first line drug choice? WHY?
   i. Sensitivity pattern of the likely pathogens
   ii. Ability to get to the site of infection
   iii. Category of the antimicrobial class for importance in human medicine

d. What dosage regimen (dose, route, and duration) would you recommend? (Indicate if it is label or extralabel)
Protocols for Synchronization of Ovulation in Lactating Dairy Cows

Paul Fricke, PhD

Introduction

A long standing goal of reproductive physiologists was to develop a hormonal synchronization program that could overcome the problems and limitations associated with visual detection of estrus in dairy cattle. This goal was realized in 1995 with the publication of a hormonal synchronization protocol that combined GnRH and PGF$_{2\alpha}$ to control ovarian physiology and is now commonly referred to as the Ovsynch protocol (Pursley et al., 1995). The Ovsynch protocol synchronizes follicular development, luteal regression and ovulation such that artificial insemination can be conducted at a fixed-time without the need for estrus detection, commonly referred to as timed artificial insemination (TAI). Subsequent studies that repeated this work soon verified the results of the original publication (Burke et al., 1996; Pursley et al., 1997a,b), and dairy producers and veterinarians began to implement the Ovsynch protocol as a tool for reproductive management on commercial dairies. In the nearly 15 years since this first publication, today many dairy farms in the U.S. and around the world have adopted systematic synchronization protocols as a routine strategy for submitting cows for first and greater postpartum AI service (Caraviello et al., 2006).

Publication of the original Ovsynch protocol represented a paradigm shift for both the industry that began to implement it as well as for reproductive physiologists who began to focus efforts to modify the original Ovsynch protocol to improve fertility to TAI. This active area of research has resulted in a proliferation of synchronization protocols referred to using nonstandardized nomenclature such as Ovsynch-56, Presynch, Cosynch, Double-Ovsynch, G6G, and Resynch-32. It takes time for researchers to sift and winnow ideas and data to reach a consensus on which protocols should be recommended for use on production dairy farms. At certain points in time, a consensus among scientists, farmers, veterinarians, and consultants cannot be reached. Furthermore, because this is an active area of research, new data hold the potential to change longstanding recommendations. In the mean time, dairy producers and their advisors are left with the task of deciding which protocols to implement on their dairies.

The purpose of this report is to make available to the industry a list of protocols for synchronizing ovulation in lactating dairy cows. While many options exist for synchronizing ovulation, this list of protocols was developed based on published research data and use in the industry by the Dairy Cattle Reproduction Council. This group comprises representatives from academia, the AI and pharmaceutical industries, bovine practitioners, and dairy farmers.

Timing of AI Relative to Synchronized Ovulations in Lactating Dairy Cows

For a recent review of timing of AI relative to behavioral estrus and synchronized ovulations in lactating dairy cows see Fricke (2008). Because most cows submitted for TAI after hormonal synchronization show little or no outward signs of estrous behavior upon which to base timing of insemination, a new line of research has arisen to optimize the timing of induced ovulation (accomplished using GnRH analogs) after induction of luteal regression (accomplished using PGF$_{2\alpha}$ or its analogs) within an Ovsynch protocol as well as timing of AI in relation to the induced ovulation.

It is common for farms to choose to adopt a TAI schedule that represents a variation in which the TAI is performed during the same cow-handling period as the second GnRH (i.e., Cosynch), thereby eliminating one cow-handling period compared with the first reported Ovsynch protocol (Pursley et al., 1995). An initial field trial conducted on two dairies in Kansas compared various combinations of Presynch + Ovsynch and Presynch + Cosynch (Portaluppi and Stevenson, 2005). Although results from this experiment indicated a conception rate advantage for the 72 h Cosynch protocol, several subsequent experiments have not supported these results (Brusveen et al., 2008; Nebel et al., 2008). Although the timing of insemination in a Cosynch protocol may not maximize conception rate to TAI (Pursley et al, 1998; Dalton et al.,
use of Cosynch allows for cows to be handled at the same time of the day on different days, thereby allowing for cows to be restrained in self-locking head gates or a palpation rail after a specified milking in 3X milking systems in which cow-handling periods are dictated by the milking routine rather than by pre-selected protocol intervals. Simplification of reproductive management protocols may also improve overall compliance to the protocol, a major determinant of the overall effectiveness of a synchronized breeding program (Fricke et al., 2003). Nonetheless, protocols resulting in superior fertility lead to improved reproductive performance that may easily justify the increased level of management required to comply with the optimized protocol.

Ovsynch56

The optimal timing of the second GnRH injection and TAI in an Ovsynch protocol was tested by Brusveen et al. (2008) by comparing two Cosynch protocols (i.e., Cosynch-48 and Cosynch-72 vs. Ovsynch-56). Lactating Holstein cows (n = 927 cows; n = 1,507 TAI) were blocked by pen on a commercial dairy, and pens were rotated among the three treatments. All cows received GnRH followed 7 d later by PGF$_{2\alpha}$ and then received one of the following treatments: 1) GnRH + timed AI 48 h after PGF$_{2\alpha}$ (Cosynch-48); 2) GnRH 56 h after PGF$_{2\alpha}$ + timed AI 72 h after PGF$_{2\alpha}$ (Ovsynch-56); or 3) GnRH + timed AI 72 h after PGF$_{2\alpha}$ (Cosynch-72). Overall fertility was similar for the Cosynch-48 (27%) and Cosynch-72 (23%) treatments, whereas cows receiving the Ovsynch-56 treatment had a greater fertility (36%) compared to Cosynch-48 or Cosynch-72 cows. A subsequent experiment conducted in 3 herds of lactating cows (n=739) confirmed the results of Brusveen et al. (2008). Cows receiving Cosynch-72 had lower fertility than cows receiving Ovsynch-56 (Nebel et al., 2008).

Based on results from these two experiments as well as an understanding of timing of AI in relation to ovulation, Ovsynch-56 (Table 1) is strongly recommended over Cosynch protocols which do not optimize the timing of AI in relation to ovulation. The Ovsynch portion of all subsequent protocols in this report should include the timing of the Ovsynch-56 protocol for the second GnRH injection and TAI.

Table 1

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Setting up Lactating Dairy Cows for First Postpartum Timed AI

Presynch-Ovsynch

The first results with Ovsynch (Pursley et al., 1995) indicated that all nonpregnant cows could be enrolled into the protocol regardless of their stage during the estrous cycle. Subsequent results from Vasconcelos et al. (1999) using lactating dairy cows, and those of Moreira et al. (2000a) using dairy heifers showed that initiation of Ovsynch between days 5 to 12 of the estrous cycle may result in improved conception rate over the original Ovsynch protocol. Presynchronization of cows to group randomly cycling cows to initiate Ovsynch between days 5 to 12 of the estrous cycle can be accomplished using two injections of PGF$_{2\alpha}$ administered 14 days apart before initiation of the first GnRH injection of Ovsynch. A presynchronization strategy in which two injections of PGF$_{2\alpha}$ administered 14 days apart preceded initiation of Ovsynch by 12 days improved conception rates in lactating dairy cows compared to Ovsynch alone (Moreira et al., 2000b). This presynchronization strategy has become known as Presynch-Ovsynch (Table 2). Lactating dairy cows were randomly assigned to receive Ovsynch (n=262) or Presynch (n=264) for their first postpartum TAI, which was conducted 16 h after the second GnRH injection. The first and second PGF$_{2\alpha}$ injections for Presynch cows were administered at 37 and 51 days in milk, respectively, and all cows received a TAI at 73 days in milk. One possible hormone injection and timed AI schedule based on this research is shown in Table 1. For cycling cows, conception rate increased from 29% for Ovsynch to 43% for Presynch cows; however, no statistical treatment difference was detected when all cows (cycling and anovular) were included in the analysis. Thus, use of Presynch for programming lactating dairy cows to receive their first postpartum TAI can improve first service conception rate in a dairy herd.
A common question regarding the original Presynch data from Moreira et al. (2000b) pertains to the importance of the 12-day interval between the second PGF2α injection and the first GnRH injection. If this interval could be extended to 14 rather than 12 days, the first four injections could be scheduled to occur on the same day during successive weeks. Although Navanukraw et al. (2004) showed that a 14-day interval between the second PGF2α injection and the first GnRH injection increased fertility compared to Ovsynch alone, Galvao et al. (2007) directly compared Presynch protocols using an 11-day vs. a 14-day interval between the second PGF2α injection and the first GnRH injection in a Presynch-Ovsynch protocol (Table 3). Reducing the interval from 14 to 11 days increased ovulatory response to the first GnRH injection of Ovsynch and improved fertility by about 6%.

Taken together, these results support a shortened interval (i.e., 10–12 rather than 14 days) between the second PGF2α injection and the first GnRH injection of a Presynch-Ovsynch protocol. Based on data from Galvao et al. (2007), farms implementing this change can expect an increase in conception rate of about 5%.

**Presynch-Ovsynch with detection of estrus and CIDR devices**

Despite the widespread adoption of synchronization protocols, accurate detection of estrus behavior continues to play an important role as a part of the overall reproductive management program on most dairies today (Caraviello et al., 2006). By contrast, recent large field trials have reported that 20 to 30% of high producing lactating Holstein cows are anovular at 60 to 75 DIM (Gümen et al., 2003; Silva et al., 2007), a time coinciding with the end of the voluntary waiting period and onset of AI breeding to detected estrus and/or TAI after synchronization of ovulation in many herds. Because anovular cows represent a substantial population of cows within a herd that cannot be inseminated based on detection of estrus, studies have evaluated methods for differentially treating anovular cows in an attempt to improve their fertility to TAI. One such strategy is to expose anovular cows to exogenous progesterone (P4) to try to resolve the anovular condition before TAI.

The objective of this experiment was to determine the effect of exogenous P4 during an Ovsynch protocol on pregnancies per AI.
Lactating cows (n = 3,338) from 7 commercial dairy herds were submitted to a presynchronization protocol (2 injections of PGF$_2\alpha$ 14 d apart; Presynch), and cows in estrus after the second PGF$_2\alpha$ received AI (EDAI; n = 1,652). Cows not inseminated by 12 to 14 d after the second PGF$_2\alpha$ injection were submitted to a TAI protocol (GnRH on d 0, PGF$_2\alpha$ on d 7, GnRH 48 to 72 h after PGF$_2\alpha$, and TAI 0 to 24 h after GnRH). At onset of the TAI protocol, cows were assigned randomly to receive no exogenous P4 (control, n = 815) or a controlled internal drug releasing (CIDR) insert containing 1.38 g of P4 from d 0 to 7 (CIDR, n = 871). Although cyclic cows had greater fertility at 40 (38.3 vs. 28.9%) and 65 (35.2 vs. 25.8%) days after AI compared to anovular cows, inclusion of a CIDR insert increased fertility for both anovular as well as cyclic cows by about 6% (Chebel et al., 2010).

For herds wanting to incorporate heat detection into their reproductive management strategy, inseminating cows to a detected estrus after the second PGF$_2\alpha$ injection of Presynch followed by Ovsynch-56 with inclusion of a CIDR insert between the first GnRH and PGF$_2\alpha$ injections is a viable alternative (Table 4). Based on data from Chebel et al. (2010), farms can expect about a 5% increase in conception rate for cows not detected in estrus after the second PGF$_2\alpha$ injection of Presynch and receiving a CIDR insert during the Ovsynch-56 portion of the protocol. This protocol has therefore been referred to as the G6G protocol (Table 5).

### New Presynchronization Strategies

Two limitations of the currently-used used prostaglandin-based presynchronization strategy (i.e., Presynch-Ovsynch) are that 1) PGF$_2\alpha$ alone does not likely benefit anovular cows or resolve the anovular condition before cows initiate the first GnRH injection of the Ovsynch-56 portion of the protocol (Moriera et al., 2000b; Souza et al., 2008), and 2) follicular growth is not precisely synchronized after treatment with PGF$_2\alpha$ alone (Souza et al., 2008). New presynchronization strategies are now being developed and tested to address these two limitations in an attempt to improve fertility to TAI. Although there are only a few published studies at this point, data from large field trials which directly compare these newer presynchronization strategies with a standard Presynch-Ovsynch protocol are underway.

### G6G

A recent study by Bello et al. (2006) used a presynchronization strategy which replaced the two PGF injections of Presynch with a PGF$_2\alpha$ injection followed 2 days later by a GnRH injection. The Ovsynch protocol was then initiated 4, 5, or 6 days thereafter to determine which interval resulted in the highest ovulatory response to the first GnRH injection of the Ovsynch-56 protocol. Ovulation to the first GnRH injection of the Ovsynch-56 protocol increased circulating progesterone at the time of the PGF$_2\alpha$ injection, reduced variation in the size of the ovulatory follicle, and increased synchronization rates during the Ovsynch-56 protocol (Bello et al., 2006). Of the three treatments compared, the 6-day interval resulted in the highest ovulatory response to the first GnRH injection, the highest luteal regression rate to the PGF$_2\alpha$ injection, and the highest conception rate to TAI after the Ovsynch-56 protocol based on about 100 cows per treatment. This protocol has therefore been referred to as the G6G protocol (Table 5).
Although a large field trial was not conducted as a part of this initial experiment, the results support the concept that increased synchronization of follicular waves and luteal function may improve outcomes during the Ovsynch protocol. While this protocol has been adopted by some farms, large scale, controlled field trials are needed to compare the G6G protocol with other presynchronization protocols (i.e., Presynch-Ovsynch and/or Double-Ovsynch).

**Double-Ovsynch**

A recent study by Souza et al. (2008) evaluated using an Ovsynch protocol as a presynchronization strategy before an Ovsynch-56 protocol. This novel presynchronization strategy has been termed Double-Ovsynch (Table 6). The first Ovsynch protocol within Double-Ovsynch has been termed the “pre-Ovsynch” part of the protocol and incorporates a 72-hour interval between the PGF\(_{2\alpha}\) and second GnRH injections. The second Ovsynch protocol within Double-Ovsynch has been termed the “breeding Ovsynch” part of the protocol and is an Ovsynch-56 protocol as described previously.

A field trial was conducted on two commercial dairies in Wisconsin to compare a Double-Ovsynch protocol with a Presynch-Ovsynch protocol. Cows receiving the Double-Ovsynch protocol had increased fertility compared to cows receiving a Presynch-Ovsynch protocol (50% vs. 42%, P = 0.03). Interestingly, there was a treatment by parity group interaction in which the Double-Ovsynch protocol increased fertility only in primiparous (65% vs. 45%; P = 0.02) and not in multiparous (38% vs. 39%) cows. Souza et al. (2008) concluded that future studies using a larger number of cows are needed to further test the hypothesis of higher fertility when using a Double-Ovsynch protocol and to elucidate the physiological mechanisms that underlie the apparent increases in fertility (Souza et al., 2008).

Taken together, G6G and Double-Ovsynch protocols offer new strategies to improve fertility to first postpartum TAI in lactating dairy cows. Both strategies aim to improve fertility by resolving the anovular condition before the TAI, more tightly controlling follicular development and luteal regression, and initiating Ovsynch on approximately Day 6 or 7 of the estrous cycle when an ovulatory follicle is present and when P4 is increasing during growth of the new follicular wave. Although some dairies have already adopted these protocols for managing first postpartum TAI, more data is needed to make strong recommendations for their use. Based on data from Souza et al. (2008) farms adopting a Double-Ovsynch protocol can conservatively expect a 5% increase in conception rate compared to a Presynch-Ovsynch protocol.

**Table 5**

Possible hormone injection and timed artificial insemination schedule for the G6G protocol based on the results of Bello et al., 2006.  
PGF = prostaglandin \(F_{2\alpha}\); GnRH = gonadotropin-releasing hormone; TAI = timed artificial insemination.

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**Table 6**

Hormone injection and timed artificial insemination schedule for the Double-Ovsynch protocol based on the results of Souza et al., 2008  
PGF = prostaglandin \(F_{2\alpha}\); GnRH = gonadotropin-releasing hormone; TAI = timed artificial insemination.

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Comparison among Protocols for First Postpartum Timed AI

The information in (Table 7) is a guide to the amount of published information on the various protocols and a rough estimate of reported conception rates for use of each protocol for synchronizing lactating dairy cows for first postpartum TAI.

Setting up Lactating Dairy Cows for Second and Greater Postpartum Timed AI

For a recent review of resynchronization strategies for lactating dairy cows see Fricke (2006). Although reliance on synchronization of ovulation and TAI for improving service rate to first AI service reduces the impact of poor estrous detection, the improved AI submission rate to first TAI often is followed by a time lag exceeding 60 d before cows failing to conceive are detected and reinseminated. Because conception rates to TAI for dairy cows managed in confinement-based systems in the U.S. are reported to be 40% or less (Pursley et al., 1997a,b; Fricke et al., 1998; Jobst et al., 2000), 60% or more of the cows will fail to conceive and therefore require a resynchronization strategy for aggressively initiating subsequent AI services. Although studies have been conducted to resynchronize behavioral estrus among groups of previously inseminated cows (Chenault et al., 2003), the objective of this review is to overview strategies for resynchronization of ovulation that allow for TAI of cows failing to conceive to a prior AI service.

One of the first field trials to directly compare intervals from first TAI to resynchronization of ovulation on a dairy incorporating transrectal ultrasonography for early pregnancy diagnosis was reported by Fricke et al. (2003). Lactating dairy cows (n=711) on a commercial dairy farm were enrolled into this study after a Presynch-Ovsynch protocol and were randomly assigned to each of three treatment groups for Resynch. All cows (n=235) in the first treatment

Table 7
Comparison among protocols for first postpartum timed artificial insemination in lactating dairy cows.

<table>
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<tr>
<th>Protocol</th>
<th>First Peer-Reviewed Publication</th>
<th>Total Peer-Reviewed Publications</th>
<th>Current use in the field</th>
<th>Pregnancies per AI (%) literature estimate</th>
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<tr>
<td>Ovsynch</td>
<td>1995</td>
<td>Many</td>
<td>++++</td>
<td>~35</td>
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<tr>
<td>Presynch-Ovsynch</td>
<td>2000</td>
<td>Many</td>
<td>++++</td>
<td>~42</td>
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<tr>
<td>G6G</td>
<td>2006</td>
<td>1</td>
<td>+</td>
<td>Not assessed</td>
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<tr>
<td>Ovsynch56</td>
<td>2008</td>
<td>2</td>
<td>++</td>
<td>&gt; than Cosynch</td>
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<tr>
<td>Double-Ovsynch</td>
<td>2008</td>
<td>1</td>
<td>+</td>
<td>~47</td>
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<tr>
<td>Presynch-Ovsynch + ED &amp; CIDR</td>
<td>2010</td>
<td>1</td>
<td>+++</td>
<td>CIDR = +5%</td>
</tr>
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Table 8
One possible hormone injection and timed artificial insemination schedule for the Resynch-32 protocol for second and greater TAI based on the results of Fricke et al. 2003.

PGF = prostaglandin $F_2\_\alpha$, GnRH = gonadotropin-releasing hormone, TAI1= first postpartum timed artificial insemination, TAI2 = second and greater postpartum timed artificial insemination, PG = pregnancy diagnosis

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<td>PG + PGF</td>
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(Day 19) received a GnRH injection 19 d after TAI and continued the Ovsynch protocol if diagnosed nonpregnant using US 26 d after TAI. Cows (n=240) in the second (Day 26) and cows (n=236) in the third (Day 33) treatments initiated Resynch if diagnosed not-pregnant using US 26 or 33 d after TAI, respectively. Overall P/Al to Resynch was 32% and was greater for D26 and D33 cows than for D19 cows (Fricke et al., 2003). Thus, the most aggressive Resynch interval tested in this experiment resulted in unacceptably poor fertility compared to delaying Resynch by 7 to 14 d.

Although coupling a nonpregnancy diagnosis with a management strategy to quickly reinitiate AI may improve reproductive efficiency by decreasing the interval between AI services, early pregnancy loss and the effectiveness of Resynch initiated at certain physiologic stages post breeding may limit the effectiveness of the early Resynch strategies tested thus far. Thus, a justifiable Resynch strategy is a Resynch-32 protocol (Table 8) in which all cows are pretreated with GnRH 7 d before pregnancy diagnosis 32 days after TAI, identify cows failing to conceive to TAI and administer PGF₂α to cows diagnosed not-pregnant 39 days after TAI and complete the Resynch protocol (Table 8).

This recommendation is based on data in which the earliest Resynch intervals of 19 or 26 d after TAI do not yield the greatest fertility (Fricke et al., 2003; Sterry et al., 2006) and the notion that assessment of pregnancy status should be delayed until the latest possible time after TAI and during Resynch to ensure that diagnostic outcomes using transrectal ultrasonography are not confounded by subsequent pregnancy loss (Silva et al., 2007b). New resynch strategies are being tested to reduce the interval between TAI while also improving fertility and may displace the Resynch-32 protocol as the protocol of choice in the dairy industry.

References


Crohn’s disease (CD) is a chronic inflammatory bowel disease (IBD) of unknown cause, the incidence of which is on the increase in high-income countries.1 A number of theories regarding the aetiology of CD have been proposed with most agreeing that it is a multifactorial syndrome with genetic and environmental contributions. Diet, infections, other unidentified environmental factors and immune disregulation, all working under the influence of a genetic predisposition, have been viewed with suspicion.

Although CD has traditionally been considered an auto-immune disease, there is increasing evidence that there may be an infectious cause. Since the first description of the similarities between Crohn’s disease and Johne’s disease in cattle in 1913,2 it has been argued that Mycobacterium avium subspecies paratuberculosis (MAP), which causes Johne’s disease, might also be a cause of CD, and that the dysregulated immune responses are a secondary phenomenon.3–5 Conversely, critics of the mycobacterial theory argue that MAP is a secondary invader rather than a causal factor.6 The association of MAP with CD is supported in part by identification of MAP in patients with CD, but not in appropriate controls. The gold standard for detection of MAP is based on isolation of the organism through culture methods.2,7–10 However, this method is time consuming because of the organism’s fastidious nature and slow growth. Molecular and serological methods are widely used alternatives, including immunocytochemistry,11 nucleic acid hybridisation,12 and PCR techniques.13–15 ELISA is commonly used to investigate the immunological evidence of a MAP infection.16–19 A causal association of MAP with CD would have important implications for both prevention and therapy, and is a continuing matter of concern for public health agencies. Since viable MAP organisms are occasionally isolated from commercial pasteurized milk,20 the efficacy of some heat-treatment procedures of milk would have to be assessed and improved. Additionally, the search for effective treatment regimens against MAP would need to be intensified.

There has been numerous review papers written that both favor the causation of CD by MAP21–23 and others that are either more precautionary or unconvincing that it is a convincing causation.5,24,25 However, there does seem to be a ubiquitous theme that there is compelling evidence to say that there is an association. Some of the more compelling evidence that there is a causal link between MAP and CD has been due to the success in culturing MAP from human intestinal tissues6 but it was typically in only about 5% of people with CD and often after incubation for many months.26–28 The ability to find MAP DNA using IS900 PCR raised the detection rates of MAP in CD gut tissue to about 30%29,30. Since then, there have been 18 peer reviewed reports of similar studies using a variety of sampling processing and PCR procedures, nine of which could identify MAP in CD some or most of the time and nine of which could not.31 Other evidence provided for causation has been the ability to culture MAP from breast milk samples of lactating mothers diagnosed with CD.31 Some of the evidence that is casts doubts on the causation link between MAP and CD include the serologic evidence of antibodies for MAP in CD patients. It was first one of the strong evidence points that CD patients had a higher prevalence of antibodies than in control patients, but a recent meta-analysis has shown that overall that in fact is unclear, as many studies have found the opposite.32 Arguments have also been made that some of the studies showing a serologic response may be due to the fact that these antibodies are non-specific to MAP and in fact the reaction may be due to other environmental Mycobacterial species.25 Other reasons occasionally given to refute the causation link have been that MAP bacteria are not seen in high numbers microscopically using standard staining techniques in tissue samples from CD patients.5 Sometimes considered even more compelling is the fact that in most antibiotic clinical trials, CD cannot be cured, therefore how can it be a bacterial cause? The apparent value of immune modulation and identification of a Crohn’s related gene (NOD2/CARD15) are additional reasons to question bacterial culpability.25,26 For a number of years, investigators have postulated that CD may result from a mycobacterial infection of the basis of histopathologi-
mal similarities between these conditions\textsuperscript{5}. Although this hypothesis has led to a large number of epidemiologically based association studies, the cumulative evidence from these studies has neither confirmed nor refuted such an association\textsuperscript{33}. It has been suggested by Behr that conflicting results has polarized researchers into 2 separate camps and that perhaps going forward researchers should look beyond epidemiological studies using technically demanding assays and instead ask whether the experimental and genetic data on CD are consistent with, or contrary to, our evolving understanding of the host response to mycobacterial infections.\textsuperscript{25}

In summary, based upon two recent meta-analysis of the published research, neither identify strong evidence for the zoonotic potential of MAP\textsuperscript{32,34}. The association of MAP and CD has been well established in case-control studies, but due to their most of the research is done in case-control settings, it is not possible to ascertain causation. However, detection of MAP in milk, along with other potential exposure routes (water, meat, and environment) needs to be continually evaluated. Additionally, currently initiated large scale longitudinal studies may resolve the issue with causation.

References


Update on Controlling Paratuberculosis: A Canadian Perspective

Shawn McKenna, DVM, PhD
Update on Diagnostic Testing for Paratuberculosis

Shawn McKenna, DVM, PhD

No Paper Available
An endurance ride is a marathon race for horses. It is run over a pre-marked, premeasured trail. There are only a few simple rules:

- An endurance ride must be at least 50 miles in length per day. Rides less than 50 miles are considered a limited distance ride. (While endurance rides are races, limited distance rides are won by the first horse across the finish line that reaches pulse criteria.)

- The horses are under the control of veterinarians experienced with horses or endurance rides.

- The ride is open to any breed or type of horse or mule.

- Equines entered in the ride must be at least five years old in an endurance race, and at least four years old in a limited distance ride (25 miles).

- There is no minimum time for completion, but there is a maximum time allowed.

- All riders who successfully complete the ride must receive an award.

- The winner is the rider who successfully completes the ride in the fastest time as long as the horse is judged to be “fit to continue”.

### Pre-Ride Check In

When a rider arrives at the ridecamp, they will check in with ride management and pick up their ride packet and number. Using a livestock marker, the horse’s number is marked on one or both sides of the hindquarters. When ready, the rider presents to the veterinarian for the pre-ride check in. The Rider Card standardizes the examinations to improve risk control on endurance rides. It encourages systematic evaluation of the parameters that best reflect the metabolic and mechanical costs of endurance effort: cardiovascular fatigue, dehydration, muscle fatigue, heat retention, and lameness. The pertinent parameters are listed in logical order, beginning at the nose and proceeding to the tail of the horse. Rate each parameter as “A”, “B”, “C”, or “D”. Although not every veterinarian will draw the same distinctions between grades at the same point, every other examiner will know whether the sign was rated superior (A), acceptable (B), cause for concern (C), or unacceptable (D) and cause for elimination. On the front side of the card are pre- and post-ride examination areas. Both examinations use the single set of outline drawings above them for recording pre- and post-ride marks and lesions. A contrasting colour pen should be used at the post-ride examination to denote problems exacerbated by or occurring during the ride. Time and staff permitting, every ride veterinarian should see every horse before the ride. Efficient organization permits you to examine one horse every three minutes. Concentrate on the essentials. You are not conducting a prepurchase exam; you are looking for evidence that the horse is fit to participate. It’s the veterinarian’s job to assess the readiness of each horse to withstand the stress of this ride under these particular conditions. Rigorous screening at this point prevents many problems during the ride. Horses that are fat or emaciated or lack evidence of appropriate fitness can be excused without explanation. Lameness is graded on a 5 point scale (from the AAEP):

- Grade I: Difficult to observe. Not consistently apparent regardless of circumstances.
- Grade II: Difficult to observe at a walk or trot in a straight line. Consistently apparent under certain circumstances (eg, weight carrying, circles, inclines, hard surfaces).
- Grade III: Consistently observable at the trot under all circumstances.
- Grade IV: Obvious lameness; marked head nodding, hitching or shortened stride.
- Grade V: Minimal weight bearing in motion and/or at rest.
Horses that are Grade III or IV lame, or have mechanical problems (joint, tendon, ligament) should be excused with a brief explanation and prognosis. The details of any shortcomings in any area are not important at the time of this examination, but out of courtesy, you may discuss your findings with the disappointed rider when the time permits. The horse can be jogged for soundness first, either simply out and back or in large circles in either direction. It is especially important for veterinarians to watch every horse in motion to help in evaluating soundness and impulsion later during the ride. If the horse is slightly unsound, ask the rider to return with it at the end of all the examinations for further evaluation and a decision. Many seasoned campaigners will have slight gait aberrations that are not a true lameness. If the horse is Grade III or worse, it should be eliminated on the spot. For jogging the horses, firm and consistent footing is desirable. Severe manoeuvres such as tight circles or sharp turns should not be used in routine examinations. Form a general impression of fitness and capabilities while the horse is jogging. The fit endurance horse, although lean, has a bright coat, elastic hide, and bright eyes expressing alertness and interest. Examine the heart and lung fields with your stethoscope after the horse returns from jogging. This examination gives a much better evaluation of the heart’s fitness than when the horse is completely at rest. The rapid decrease of the pulse rate from the slightly elevated rates achieved at the jog will allow you to evaluate the efficiency of the heart. Complete the metabolic examination according to the order on the rider’s veterinary card. If you practice it from the first horse, it will become second nature, and easy to complete rapidly during the ride at vet checks.

Start at the nose and mouth, assessing mucous membranes and capillary refill time. Press over the jugular vein for venous refill time. Pinch up a fold of skin on the point of the shoulder (a more consistent location than the side of the neck) to assess hydration. Any delay is noteworthy, and a second or longer is definitely significant. As you walk toward the rear, ballotte the triceps, glutei, and hamstring muscles for tone and reaction. Palpate the back muscles for soreness. Stop and listen to the gut sounds; they do not need to be numerous or loud to be normal. Step to the rear and lift the tail, evaluating tail muscles and anal sphincter tone. Not every parameter will be an “A” at the pre-ride examination, especially mucous membrane colour, capillary refill, dehydration, muscle efficiency, attitude, impulsion and gait. But this is the time to note individual peculiarities in these parameters, so that decisions during the ride can accurately utilize changes occurring on the trail. Because this is a sound horse jogging, the legs are examined in a cursory fashion. Inspect them for signs of previous significant pathology, such as enlarged joints, tendons or ligaments, recent wounds, or severe interference marks. If necessary, quickly feel suspicious areas, but avoid deep palpation or strenuous manipulations of the legs. Pick up each foot and check the adequacy and type of shoeing. Similarly, check the saddle, girth and bit areas.

Check all the items on the pre-ride area of the Rider Card and sign your name alongside in the space provided. Return the card to the rider. At the subsequent scheduled vet checks, use the consecutive numbered boxes to record your findings.

After completing all routine examinations, re-examine horses of questionable soundness in more detail. Here a more rigorous examination is justified, but it should not exceed what is minimally necessary to determine the nature and prognosis of the problem. If a joint, tendon, wound or other acute injury is the source of the lameness, the likelihood of further damage during the ride may be estimated. Many experienced, high-mileage endurance horses have mild chronic discomforts that improve with work. They may be given the benefit of the doubt to start, with notation on the card to be alert for exacerbation of the problem. Before signing the card of such an entry, notify the rider of your concern and intent to follow the horse closely.

Rider Briefing

After the pre-ride examinations, ride management will call a meeting to brief competitors. This is your chance to establish the relationship between the veterinary staff and the riders and it should be taken seriously and systematically. The riders must understand that the veterinary control team is in partnership with them to produce a high completion rate with reasonable risk. First, be sure to introduce yourself and your assistants. Then describe your control procedures and criteria, and detail the flow patterns through each vet check. Describe any special concerns that you have about the trail and weather that you feel should affect the rider’s strategy and pacing. Describe the post-ride examination for completion and Best Condition award. Tell the riders how to handle a seriously tired or lame horse on the trail between vet checks.

Vet Checks During the Ride

Horses should be examined regularly and often during the ride. At least once during a 25 mile ride, two to three times during a 50 mile ride and five to six times during a 100 mile ride. There are several types of vet checks used in endurance rides. The most preferred is the “gate” into a timed hold. When the rider arrives at the vet check (like entering a “gate”), his time is recorded. Whenever the rider decides that his horse meets the criteria for continuance on the trail (usually 60-64 BPM), he presents the horse to the Pulse & Respiration team for measuring pulse, and respiration (and temperature, if required). This presentation should ordinarily occur within 30 minutes after the horse reaches the check point from the trail. The recovery values achieved more than 20 minutes after rest are of little value in estimating fitness to continue. The fittest horses achieve pulse rates below 70 BPM within five minutes of rest. These
horses rarely show other significant signs of metabolic fatigue. A recovery rate of 70 within 10 minutes should be expected. A recovery of 66 within 20 minutes and 60 within 30 minutes are reasonable expectations. If, when the horse is presented to the P&R team, it does not meet the criteria, it must be represented to the P&R team. The control veterinarian may allow as many rechecks as the rider requests, or may set a mandatory time penalty that must be observed before the horse is rechecked. At congested check points, a horse that does not meet criteria may be required to go to the end of the line of horses waiting to be checked. After the first check, a rate 4-6 beats per minute lower should be expected at each successive check after a 10 minute rest. The palpable and auscultable pulse should be regular and full, not wandering, labile, thin or “slapping”. If the horse does not reach criteria by 30 minutes of arrival at the P&R check, then it is disqualified and treated if necessary. Once criteria are met, the “hold” time officially begins. At some time during the hold period (usually within 30 minutes) the horse must be presented to the veterinarian for examination.

Otherwise, the rider is free to use the hold time as they sit fit to take care of themselves and their horse. The “gate into a hold” method provides an advantage to the metabolically fit horses. They recover more quickly and are able to begin the mandatory hold period sooner than less fit horses. Horses are released from the vet checks at a time appropriate to their individual fitness levels and “hyper” horses are prevented from being run into the ground. Other methods of vet checks include a stop and go check and a trot by. A stop and go check only requires the horse to be at or below criteria before continuing on the trail. A veterinarian must be present at this type of check but further veterinary evaluation may or may not be carried out. This method of check is problematic when several horses present for examination at the same time. Often there will not be enough P&R personnel or veterinarians to process them all simultaneously. Even a minute lost to a hold up at a vet check can make a difference in final placings. The most simple vet check is a trot by, where the veterinarian merely observes the horses and riders in motion as they pass by on the trail. Only horses showing obvious lameness are pulled for further evaluation. At a vet check, the veterinarian may elect to use the Heart Rate Recovery Index (HRRI) (also known as the Ridgway Trot for veterinarian/rider Dr Kerry Ridgway) for fitness evaluation. The horse’s pulse is taken and then the rider jogs the horse to a marker 125 feet away and then back again. The veterinarian starts a one minute timing as the horse starts to jog. As the horse jogs down and back (usually takes 25-30 seconds) observe the gait, attitude, and impulsion. Grade and record each on the A-D scale. When the horse returns, begin the metabolic exam by examining the mucous membranes and capillary refill, proceeding in order to successive parameters until the timed one minute is up. Take the pulse again and record. A rate at or below the initial rate taken 1 minute previously is characteristic of a horse that is fit to continue. Rates 10% above the initial pulse rate suggests some fatigue. Rates more than 10% higher suggest significant fatigue. Additional evidence should be sought in other metabolic factors such as mucous membrane colour, capillary refill time, anal tone, tail trembling, poor impulse, skin tent, etc.) A poor HRRI may signal an impending colic, exertional rhabdomyolysis, or musculo-skeletal pain. Repeat the HRRI whenever in doubt. Watch all horses with an elevated HRRI until their healthy recovery is certain. The horse’s respiratory recovery will vary with weather conditions. Horses with an oxygen debt move relatively large volumes of air; horses without an oxygen debt may use respiration as a method of heat dissipation under hot and humid conditions. For this reason, it is important to distinguish between gasping respirations which are deep and frequent and move large volumes of air and panting horses which are moving small volumes of air over their turbinates at rates often in excess of 100 breaths per minute. Under normal cooling conditions, the respiratory rate will subside parallel to and below the pulse rate. Since endurance horses generate tremendous body heat, panting in hot, humid conditions is entirely consistent with optimal performance. If pulse and other signs of recovery are prompt and progressive, panting horses with a rectal temperature of 39.4 C (103 F) are merely devoting respiratory effort to further cooling within the physiological range. Panting horses with core temperatures above 39.4 C (103 F) should be closely monitored for other fatigue signs and need to reach and maintain a lower temperature to remain in competition. Horses with temperatures above 40 C (104 F) should receive supplemental cooling with water, and horses with temperatures above 40.5 C (105 F) should be cooled by emergency methods with ice water on subcutaneous vessels and enemas. Thick, sticky or scanty sweat, prolonged skin tenting, dry and injected mucous membranes, sinking of the eye with drooping of the eyelid are all signs of dehydration. Capillary refill times over two seconds signals low blood volume and/or hypotension. Poor capillary refill often corroborates findings of dehydration and fatigue. The diversion of blood in the over-worked horse from visceral to muscle circulation causes ileus and diminished gut sounds. Reduced gut sounds in an otherwise fit, alert horse are of little concern. Behavioural changes are related to the horse’s personality and are expected in moderation toward the end of an endurance ride. Any loss of elasticity, power and length of stride should be moderately progressive over the trail, but the horse should always look eager to continue. Sleepy eyes, droopy ears indicate some fatigue. Loss of attentiveness is significant. If the horse is normally a good eater, loss of appetite is alarming. The loss of elasticity, power and length of stride are proportional to muscle fatigue and should be moderately progressive over the trail.

Exertional rhabdomyolysis (“tying up”) can occur early in the ride due to breed-associated myopathy or later in the ride due to electrolyte derangements. Urine may be scant or discoloured due to circulatory and muscle problems. Lameness of Grade I or II can usually continue with caution unless the pain clearly is from a tendon or ligament, where further use could cause irreparable damage. Progressive lameness and all lameness greater or equal to
Grade III should not be allowed to continue. When in doubt about fatigue, pull the horse. When in doubt about lameness, let it go. Don’t rely on numbers alone. Many horses with satisfactory recovery numbers can be in serious metabolic fatigue based on other parameters such as impulsion, alertness and elasticity. Disqualification of a horse is never pleasant; it is much easier with the experienced competitor who is concerned about the health and safety of their horse. However, some competitors will be less than cooperative. In all cases where disqualification (“pulling”) of the horse is necessary, the veterinarian should give the rider an assessment of findings to make him aware of the condition of the horse. Once the rider understands the deteriorating physical status of the horse, he should be given the opportunity to voluntarily withdraw. This allows the rider to feel better about the decision and to recognise that they have exercised good horsemanship in the decision to pull. If the rider does not concur, it is always wise to get a second veterinary opinion. But in some cases it is not possible to obtain a second veterinary opinion, and under these circumstances the rider must abide by the control veterinarian’s decision. Especially if there are additional veterinarians involved in the evaluations, it is important to use the Rider Card to communicate your findings to other veterinarians. It is very helpful to use a highlighter to call attention to any questionable parameters or comments that will be of value to the next veterinarian. A highlight can be placed on the box of the next vet check to draw attention to parameters that should be closely examined. Also, the Rider Cards should be reviewed for pertinent comments as soon as they are received. All cards should be signed by the examining veterinarian at each check point. There are no conditional releases from a vet check. If the horse is allowed to pass the vet check, then the rider is free to continue to ride as they see fit. The veterinarian cannot stipulate that the horse may continue only if the rider will follow certain verbal or written conditions. But this acceptance does not mean that the veterinarian should not communicate their thoughts or recommendations to the rider. Advice is invaluable in increasing the knowledge and awareness of the rider. The rider is always ultimately responsible for the welfare of their horse, because the veterinarian only sees the horse for brief time periods during the ride. The more informed the rider is, the better off the horse will be. Once the vet check is passed, the timed hold period the rider is free to use the remainder of the hold time as they see fit. The hold times are predetermined by ride management in consultation with the veterinarian, taking into account the distance the horses have gone, the logistics and facilities at each vet check point, and the weather conditions. This hold allows horses time for eating, drinking and rest, and allows the rider to do the same. During the timed hold, the veterinarian also has the opportunity to reassess control decisions, such as the progress of a slight lameness or relief of dehydration, before the horse returns to the trail. At the end of the timed hold, the rider is free to continue on the trail.

Completion Examination

All horses must undergo a post ride examination to determine eligibility for awards and points, but most importantly to evaluate the horse’s health prior to leaving the ride while veterinary assistance is still available. The veterinarian’s criteria and procedures should be no more stringent at the completion examination than they were at any previous veterinary examination. A veterinary evaluation of soundness should be performed as soon as the horse finishes and the rest of the completion examination should be performed soon after the horse finishes and absolutely within one hour of finishing. Horses must recover to pulse is determined as the time that the horse reaches pulse criteria, not the time that it crosses the finish line. All horses should finish an endurance competition in an adequate metabolic and soundness state so that they would be considered “fit to continue”. Allow for wear and fatigue that is reasonable for the trail and conditions. Horses should be certified for completion if they could be safely ridden further even if at a reduced speed, have stable vital signs, and show subjective and objective signs of recovery, and do not require medication for fatigue or injury. Horses judged to be fatigued and in need of veterinary care should be referred to the treatment veterinarian and eventually to the rider’s own veterinarian. The post ride examination should consist of a final evaluation of hydration parameters, pulse rate, and evidence of progressive recovery on horses exhibiting marginally acceptable parameters. The HRRI is very useful in determining recovery (and progressive recovery when repeated in 15 to 20 minutes on marginal horses). Each horse should be evaluated in motion, followed by gentle palpation of muscles, joints, tendons, ligaments and body lesions for any injury that might progress or require treatment. Using the “fit to continue” criteria, the veterinarian may deny completion to any horse if the horse is significantly lame or metabolically unstable, or if the horse has severe body or mouth lesions that were caused by tack. Denial of completion is a duty and a power not to be taken lightly or abused. Veterinarians must always do their best to eliminate horses with problems before the final examination.

In Conclusion

Participating in the sport of endurance and competitive trail riding as a veterinarian can be a fun and rewarding experience. Planning ahead and working with ride management to make sure the veterinarian’s needs are met increases the efficiency and accuracy of the veterinary judging. A good working relationship between veterinarians, ride management and the riders will ensure that the sports of endurance and competitive trail riding remain “Fit to Continue”!
In endurance rides, horses compete over distances from 25 to 100 miles in one day on a variety of trails and a range of environmental conditions. It is considered an extreme equine sport and it has been highly scrutinized from an equine welfare perspective. Historically, the first endurance races were 100 miles long with few rules and no veterinary involvement. With the formation of official sanctioning organizations, such as the American Endurance Ride Conference (AERC) and bad publicity from some tragic horse deaths during rides, veterinary involvement and control in the sport began and continues to evolve to this day.

This presentation will highlight the efforts of the American Endurance Ride Conference, the Australian Endurance Riders Association (AERA), Endurance Canada, the Federation Equestre International and other organization to address the welfare aspects of endurance riding. Research (“What went wrong?”) and education (“How can we prevent what went wrong from happening again?”) have been coupled initiatives to reduce the need for treatment and prevent equine fatalities occurring during or after endurance rides. No other horse sport has such a comprehensive welfare and risk management system as endurance riding. Most recently, the AERC has endorsed the American Horse Council (AHC) Welfare Code of Practice. The AHC drafted the Welfare Code of Practice, which outlines in generic terms what it means for an organization to be committed to the responsible breeding, training, care, use, enjoyment, transport, and retirement of horses. The AHC’s Welfare Code of Practice has already been supported by the American Association of Equine Practitioner, the American Quarter Horse Association, the Kentucky Thoroughbred Association, the National Thoroughbred Racing Association, the U.S. Equestrian Federation, and the U.S. Trotting Association. The Code is not intended to replace or pre-empt those activities or any rules and regulations specific to a segment of the industry. Rather it is hoped that the endorsement of a broad, more generic Welfare Code of Practice by as many organizations as possible will be another indication to the public, the media, federal and state officials and within the horse community that the horse industry “Puts the Horse First.”

Welfare Code of Practice
American Horse Council

Introduction
American society has grown away from its agrarian roots of only a few generations ago. The horse, which was once a staple of American agriculture and general transportation, is now used primarily for breeding, competition, sport, recreation and entertainment, although there are still many horses used for work on farms and ranches, and in urban areas and exhibitions.

The horse industry is committed to the safety, health, care and welfare of all horses and to always “Put the Horse First.”

We address equine welfare and responsible care (1) by supporting a uniform Code of Practice regarding the responsible breeding, training, competing, care, use, enjoyment, health, transportation, and retirement of horses; and (2) by initiating communication with the public, the media, federal and state officials and within the horse community regarding these issues.

Our Commitment to all Horses and the Horse Industry
The organizations listed below are committed to the principle that the welfare and safety of the horse is the guiding principle in the decision-making process for all owners, service providers, organizations, events and activities.

- WE ARE COMMITTED to the dignity, humane care, health, safety and welfare of horses in all our activities and care. These are our highest priorities. We are the stewards of our horses and must be firm in the standards and practices that guide us. Our first principle is:
  - The welfare, safety and stewardship of the horse is the guiding principle in the decision-making process for all segments for the horse industry.
  
- WE ARE COMMITTED to promoting responsible breeding practices and to produce better horses, not just more horses.
• WE ARE COMMITTED to responsible training techniques. All training should be done with the maturation and ability of the horse considered. Horses should be prepared for competition with proper training and conditioning methods. Excessive disciplining methods, whether in stables, training areas, or during competition, will not be tolerated.

• WE ARE COMMITTED to educating owners, trainers, veterinarians, competitors, exhibitors and recreational riders to ensure that they know and respect their horse’s abilities and limits, and their own, so as to not push the horse or themselves beyond their ability level.

• WE ARE COMMITTED to making all competitions fair and ensuring all competitors an equal opportunity to succeed. Performance-enhancing drugs, practices or equipment have no place in competitions or exhibitions. Effective drug testing by accredited laboratories is essential to the safety and welfare of our horses and the public support of competitions, with appropriate penalties levied for violations. The welfare of the horse must take precedence over the demands or expectations of owners, breeders, trainers, sellers, buyers, organizers, sponsors, officials, or spectators.

• WE ARE COMMITTED to the welfare of the horse as paramount during competition. The horse industry should invest in the infrastructure, environment and facilities to provide a safe environment for all horses in all activities, whether breeding, competing, or simply riding. Any facilities that house horses should be committed to the appropriate care and treatment of all horses while in their facility, and should be designed with the environment and the intended use of the horse in mind.

• WE ARE COMMITTED to minimizing injuries to horses during training, competition, use, or work. Whenever possible injury data should be collected, documented and reported to the governing body of the competition or any other injury database for analysis in order to ensure a safer environment.

• WE ARE COMMITTED to the continual review, evaluation and improvement of all rules, regulations, policies and practices in all equine activities, based on science (where indicated). When warranted, they should be refined or changed. This includes existing practices to ensure they are not being perceived as acceptable, particularly if new research has called them into question.

• WE ARE COMMITTED to providing continuing education on all activities involving horses and eliminate inhumane practices as well as strengthening sanctions for non-compliance.

• WE ARE COMMITTED to educating all people who own or work with horses to ensure they are knowledgeable in the proper husbandry, care, and handling of horses. Each horse should be observed frequently to ensure that they are healthy. In consultation with a veterinarian, all such individuals should develop a sound health care program, appropriate to the facilities, environment and needs of the horses.

• WE ARE COMMITTED to providing an environment in which anyone aware of equine cruelty or neglect is willing to report it to the proper local, state or federal authorities. Should an incident occur at an event it should be reported to judges, stewards, responsible authorities or the sanctioning organization.

• WE ARE COMMITTED to improving the health and welfare of horses through scientific research, collaboration, advocacy and the development of appropriate rules. The industry should continue to support and work with the many individuals, universities, veterinarians and foundations doing and funding equine health and welfare research in order to reduce injuries and improve health.

• WE ARE COMMITTED to horse owners and caretakers ensuring horses in their care are current on vaccinations and following best practices to minimize infection and disease. When a disease outbreak occurs horse owners and events must act quickly and responsibly, monitor the horses, report the outbreak to, and cooperate with, veterinarians, authorities, facility management and all stakeholders to bring a rapid resolution to the outbreak.

• WE ARE COMMITTED to ensuring that our horses will have an opportunity to transition to additional careers, uses or activities as the need arises. When necessary, owners and veterinarians may have to consider end-of-life decisions. The welfare, safety and dignity of the horse must continue to be the guiding principle in deciding how and when to provide a humane death.

• WE ARE COMMITTED to being transparent about our activities in order to ensure the public, the media, federal, state and local officials and the various segments of the horse community understand what we do, why we do it, and support it.
Musculoskeletal Welfare
Aspects of Sporting Dog Activities

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As dog sports become more popular, owners of these athletic dogs look to animal health care practitioners to answer their sport-specific questions, assess their sport-specific performance issues or non-specific lamenesses, and provide effective treatments applicable to their sporting animal. For the welfare of these performance animals, it is imperative that the veterinary community be better prepared to meet the needs of this unique population.

Survey of Injuries in Dog Sports

Dog owners can participate in a wide variety of dog sports. Agility, Hunting, Track Racing, Lure Racing, Obedience, Rally-O, Flyball, Scent Hurdle, Earth Dog, Weight pulling, Field Trialing, Sled Dog Racing, Skijoring, Freestyle, Carting, Tracking, Frisbee Dog, and Herding are some common dog sports. Of these, little is written or reported regarding musculoskeletal injuries associated with these sports, and only 5 papers could be retrieved that endeavoured to study these types of injuries.

Agility trialling made its debut as entertainment for spectators at the Crufts Dog Show in England in 1978-79. (Holmes 2005; Levy et al 2009) Since this time, it has become the most rapidly growing dog sport. (Gauntt 1996) Two surveys could be found pertaining to agility, one British and one American. (Holmes 2005, Levy et al 2009) The Holmes study reported that 19% of respondents suffered agility-related injuries, whereas the Levy study reported a 33% injury rate amongst respondents. Commonalities in the surveys revealed that border collies, followed by working hounds, were the breeds most commonly participating in the sports, and Levy et al (2009) found that these dogs were the most commonly injured as well. Both studies reported injuries occurring primarily during competition or practice. Injuries occurred due to direct contact with objects (A-frame, teeter, dogwalk, or bar jumps), turning or twisting during jumps, slips and falls, or chronic overuse. Both studies found that injuries were predominantly soft tissue injuries (including sprains / strains), and the Holmes study reported additionally that non-specific lameness was commonly reported. Personal communication with Holmes elucidated further that 48% of the injuries were undiagnosed lamenesses. The Levy study additionally found that 78% of injured dogs had their diagnosis confirmed by a veterinarian, and that injuries to the shoulder and back were most common, with a lesser amount of injuries occurring to the hips, stifles, carpi, thumbs, and phalanges. Forty six percent of injuries lasted less than 6 weeks, and 42% required greater than 6 weeks to resolve according to Levy et al. Whereas Holmes found that 54% of injured dogs resumed normal activity within 4 weeks and 46% returned to sport within 10 weeks. Both studies found that many dogs retired from the sport directly due to an injury acquired during performance of the sport. Additionally, Holmes reported that 26% of the injured dogs were referred to physiotherapy, of which, 78% reported to be significantly improved and the remaining were cured.

Gundog lameness and injuries over a two year period in Great Britain were studied via survey by Houlton (2008). The incidence of injuries / lameness was found to average at 25% of dogs per season, of which, only 47% were treated by a veterinarian. The author commented that veterinarians may not necessarily be the best informed about the nature of such injuries associated with hunting activities since many dogs are treated by knowledgeable and experienced owners / trainers. From the data collected, the primary kinds of injuries were firstly grouped together as wound, (excluding foot injuries), as well as tail injuries. The second most common injuries were those to the pads, nails and webbing of the feet. Thirdly, ocular injuries and miscellaneous conditions were reported, and lastly, articular pathology, fractures and muscular injuries were identified. Four and a half percent of the injured dogs were diagnosed with a shoulder-related lameness, of which 80% were thought to be strains. Additionally, there was a highly significant association between tail injuries and undocked Springer and Cocker Spaniels.

Two studies have endeavoured to survey racing injuries in greyhounds. (Prole 1976; Sicard et al 1999) The 1976 study reported that shoulder injuries made up 18.5% of the total injuries, with the primary lesion being a strained triceps muscle (affecting the right limb more so than the left). Injuries to the carpus comprised 11% of the total, with the primary lesion being sprains (affecting the right limb more so than the left). Ten percent of the total injuries were strains of the flexor tendons at the metacarpus region (this time affecting the left limb more than the right). Sprains of the forelimb toes accounted for 33% of all injuries (again affecting the left more than the right). Within the hind leg, muscle injuries accounted for only 8% of total injuries, with the primary lesion occurring to the gracilis muscle (right greater than left). Six percent
of injuries were to the tarsus, with nearly all of these being fractures to the right side. The pes suffered 9% of all injuries, primarily affecting a toe and showing fairly even distribution between the right and left sides. The Sicard et al study in 1999 reported an average of 4.4% injury rate per race. That study noted that 20% of injuries occurred during the first turn of the race, that 8.3% occurred during the second turn, and that while injuries were reported to have occurred at the third and fourth turns, 45.5% occurred at unknown locations on the track. Of the injuries reported, muscle was injured most, followed by hock injuries, toe injuries, carpal injuries, and metacarpal / metatarsal injuries.

These surveys when interpreted together reveal some commonalities, and bring forth a number of questions. All studies could beg the question, ‘how can injuries be prevented’? Can slips and falls or collisions with obstacles in agility be avoided? While wounds, foot trauma and ocular injuries could simply be hazards of working in bush or unmanicured fields, can muscular injuries be prevented in gundogs? Is there any correlation between the high incidence of muscle injuries and the high incidence of injuries occurring at the first turn of a greyhound race? Can this too be prevented? As well, since many of the dogs in all groups suffered muscular injuries of the shoulder region, can veterinarians be better informed regarding assessment and treatment of muscle injuries in this area? Lastly, what is the best way to return an injured animal to sport? The remainder of this paper will attempt to answer these questions.

Injury Prevention Strategies

Stretching

Stretching is a popular prescription among health care professionals, athletic trainers and in fitness/coaching personnel, all of whom have an interest in improving flexibility in both healthy and injured clientele. Stretching has been touted to enhance athletic performance, prevent musculotendinous strain injuries and reduce delayed onset muscle soreness. However confusion and controversy exists over when stretching is most effective, and some claims and common uses of stretching are not supported by research. (Decoster et al 2005)

Stretching has been shown to be effective in increasing joint mobility about the knee, hip, trunk, shoulder and ankle joints including muscle length and flexibility. (Davis et al 2005, Decoster et al 2005; Knudson 1999; Magnusson et al 1998; Power et al 2004; Thacker et al 2004) Studies have shown that regular stretching can improve eccentric and concentric force production, velocity of contractions, maximal volitional contractions, counter-movement jump height, 50 yard dash and athletic performance. (Hunter et al 2002; Shrier 2004) One study found that regular stretching was able to induce hypertrophy in immobilized muscles and another speculated that this effect may actually improve performance in the long term.(Coutinho et al 2004; Shrier 2004)

Results varied for the optimum time required to obtain the most favourable muscle lengthening/joint range of motion (ROM). Studies found that passive stretches of 15 to 30 seconds were more effective than stretches of shorter duration and just as effective as stretches of longer durations. (Decoster et al 2005; Thacker et al 2004) Reports of other studies found that the overall time of stretching was most important and found that 6 repetitions of 10 seconds each was just as effective as 2 repetitions of 30 seconds. Three sets of 15 second stretches were effective as well. The greatest gains in flexibility were made if stretching occurred on a regular basis over time. (Decoster et al 2005) One study reported that a static stretch of 1 repetition for 30 seconds, 3 days a week for 4 weeks, significantly increased hamstring length/flexibility. (Davis et al 2005) Consensus was that passive static stretching was more effective than proprioceptive neuromuscular facilitation techniques, active assisted or dynamic stretching protocols in improving muscle length. (DeCoster et al 2005; Shrier 2004)

It is in the use of stretching immediately before exercise or testing where the adverse effects of this technique are seen. It has been shown in human studies, that acute bouts of stretching does not improve maximal volitional force output, jump height, running speed, static balance, reaction time, or movement time. (Behm et al 2004; Knudson 1999; Power et al 2004; Shrier 2004; Thacker et al 2004) In this format, stretching results in a decrease in isokinetic performance, velocity of contraction, muscle force produced with contractions, musculotendinous unit compliance and a reduced ability to store elastic energy in the eccentric phase. (Fletcher et al 2004; Shrier 2004) These negative effects have been reported to last up to 1 hour following stretching. (Thacker et al 2004) A reduction in running economy has been reported as a result of stretching. (Thacker et al 2004), however Nelson et al (2001) dispelled that assumption in their study by showing that VO2peak was not affected by a chronic stretching program.

Several studies and reviews have looked into the use of pre-event stretching to reduce the risk of injury. (Hart 2005; Herbert & Gabriel 2002; Pope et al 2000; Thacker et al 2004; Witvrouw et al 2004) Most have shown or reported that pre-event stretching does not reduce the risk of injury. Witvrouw et al (2004) suggested in their review that pre-event stretching was useful in preventing injuries in sports with high stretch shortening cycle movements (i.e. football or soccer), however stretching was always incorporated with an active warm up, which may have contributed to the reduction of injury. As well, studies of athletes that suffered muscle lesions were found to have less muscular flexibility than those without injury. One should not make the assumption that pre-event stretching would have benefited these athletes more or less than a regular stretching program.
It should also be reported as a fallacy, that stretching before or after exercise does not confer protection from muscle soreness. (Herbert & Gabriel 2002) Additionally, it has been suggested that in sports that do not require burst or flexibility (i.e. jogging or cycling) that a certain amount of stiffness in the musculo-tendinous structures would in fact be beneficial. (Witvrouw et al 2004)

In any sprint activity, muscle flexibility should be adequate enough to allow the full range of joint motions required for the activity (deVries 1986), but not overt flexibility which would impede the immediate transference of musculotendinous forces to the bones and potentially reduce the speed of movement. (Witvrouw et al 2004) However, a recent study of racing greyhounds found that dogs that had received race training had greater flexibility, possibly due to training having an active stretching role on muscles, tendons and other structures limiting the hip. (Nicholson et al 2007) Presumably, stretching to gain flexibility would not be necessary for injury prevention in endurance athletes such as sied dogs, as the gait and speed at which an endurance race is run only utilizes the mid ranges of the extremity joints. (Witvrouw et al 2004)

**Warming-Up**

Warming-up the animal prior to racing or exercise is of great importance to achieve superior performance and prevent injuries. (Tyler et al 1996; Steiss 2003) In horses, a warm-up of 5 to 10 minutes is more beneficial for improving oxygen kinetics than a shorter warm-up period. (Tyler et al 1996) There are conflicting citations however, as to whether warming-up has any effect on performance in sprinting activities in people. (deVries 1986) A recent human study however revealed that both active warm-up (10 minutes at VO2max) or passive warm up (hot water submersion) were superior to no warm up for speed. (Brown et al 2008) Some literature also cites that endurance athletes perform better with five minutes of vigorous high-intensity warm-ups that include some sprinting. (deVries 1986) Essentially, heating of muscle tissues can improve musculotendinous extensibility and may thereby reduce its susceptibility to strain injury. (Stickler et al 1990)

**Growth Concerns**

Animal owners and their health care practitioners should be aware of a few growth issues that could potentially impact the long term health of a young canine athlete. Many owners that engage in dog sports acquire puppies and begin to train in aspects of their specific sport at a very young age. From the aspect of motor skill acquisition, this can be a very desirable process (Helton 2007), however other aspects should be considered.

It has become a known factor that a greater percentage of dogs that rupture their cranial cruciate ligament are altered dogs of both sexes. (Slauderbeck 2004; Powers YM 2005,) It has additionally been shown that early neutering is a significant risk factor for development of excessive tibial plateau angles in large breed dogs with cruciate ligament disease. (Duer et al 2007) Another recent study showed that dogs spayed or neutered before 5 1/2 months had a significantly higher incidence of hip dysplasia than those spayed or neutered after 5 1/2 months of age. (Spain 2004) Since an athletic dog is destined to put more forces and strains on its body, it may be prudent for the informed veterinarian to discuss the risks mentioned above with owners who participate in dog sports, or at the least to support the sporting dogs’ owners’ decision to delay spaying or neutering.

Growth plates are susceptible to repetitive stress in children. A review of human literature revealed that growth plate injuries have been reported in the proximal humerus in throwers and a badminton player, and in the proximal tibia of a runner. (DiFiori 1999) In gymnastics, repetitive loading of the wrists can injure the distal radial growth plate. It appears that metaphyseal ischemia inhibits mineralization within the zone of provisional calcification, prolonging chondrocyte life. This, together with continued division of chondrocytes in the proliferative zone, results in widening of the growth plate. Physeal injuries may produce partial or complete growth arrest. Hence advice on adequate, and not excessive training in young athletes is important, however guidelines to provide justification for appropriate recommendations do not yet exist. A recent series of postings on an animal rehab chat group revealed many suggestions based on personal experiences and opinions:

**PLAY:** should be limited to small amounts or free exercise regularly and that play should be with same age puppies or calm adult dogs (who do not play aggressively). The thought is that the puppy will self monitor its own activity level under these parameters.

**Exercise:** no forced exercise, endurance, or aerobic activities (i.e. running, jogging, hiking) until the growth plates are closed (<14 months and not until 18 months if the animal was neutered prior to 6 months of age). So as to not inadvertently over-exercise puppies, owners need to pay attention to the exercise variables (speed, duration, frequency, difficulty, and complexity). Specifically training a puppy with proprioceptive exercises can be important.

**Sport Training/Agility Training:** before the growth plates are closed, limit training to low impact ground training, recalls, start-line, lowered obstacles, weave poles split, movement on the flat (following instruction), low jumps (no higher than carpal height), and general strengthening. After growth plates are closed, jumps can be increased to elbow height.

**Identification of Soft Tissue Injuries of the Shoulder**

Soft tissue injuries are often an under-diagnosed source of canine lameness. (Breur & Bevins 1997; Fitch et al 1997; Steiss 2002) Sporting and working dogs may be particularly at risk of
suffering acute traumatic muscle strains, ligamentous sprains or chronic overuse degenerative tendinosis lesions resultant from poor healing of repetitive strain injuries. Less conditioned animals may also be at risk when performing infrequent burst activities or endurance tasks, much like the phenomenon known as weekend-warrior-syndrome in humans. Physical therapy skills and knowledge lend the ability to systematically assess, diagnose and conservatively treat soft tissue injuries in the canine patient.

Background

Muscle strains may be caused by poor flexibility, inadequate warm-up, fatigue, sudden forceful contraction or forced extension/flexion, strength imbalances, intense interval training, insufficient breaks and overtraining. The potential for certain muscles to be strained or torn is greater for some muscles than others. Multi-joint muscles are those that cross two or more joints and are at greatest risk for strain because they can be stretched by the movement at more than one joint. A strain may also occur when high forces are put through tendons and muscles, as occurs during eccentric muscle contractions (where a muscle is contracted during a stretch), when forces are applied quickly and obliquely, or during an explosive burst of movement. Muscle strains most often affect the muscle origin or insertion, typically at the musculotendinous and tenosseous junctions but can occur within the muscle belly as well. (Steiss 2002; Fitch et al 1997; Nielsen & Pluhar 2005) A strain may also occur when high forces are put through tendons and muscles, as occurs during eccentric muscle contractions (where a muscle is contracted during a stretch), when forces are applied quickly and obliquely, or during an explosive burst of movement.

Tendon injuries may be secondary to acute trauma or repetitive loading. The designation of tendon pain as “tendonitis” is often a misnomer as it implies inflammation. Tendinopathy is a better generic descriptor that can be used to include all pathologies that arise in and around tendons (i.e. tendonitis, tendinosis, or paratenonitis). (Khan et al 1999)

There is a lack of good quality histological data from symptomatic tendon disorders of short duration to unequivocally state that tendon lesions are actually inflammatory in nature. (Rees et al 2006) Marr et al (1993) described an inflammatory reaction in superficial digital flexor tendon injuries in horses, but only within the first 2 weeks. Other animal models suggest that an inflammatory reaction is present in acute situations but that a degenerative process soon supersedes this. Classic inflammatory changes are not frequently seen in chronic athletic tendon conditions, and it has been suggested that the time at which the tendon becomes symptomatic for pain does not coincide with onset of pathology. (Magannaris et al 2004; Rees et al 2006; Wilson & Best 2005). On a practical note, in clinical practice most tendinopathies are chronic (tendinosis lesions) by the time the patient (or animal owner) seeks medical attention. (Khan et al 1999; Wilson & Best 2005) So perhaps, the clinician should place only minimal, if any, focus on inflammation for tendon pain conditions.

Paratenonitis occurs when a tendon rubs over a bony protuberance and is alternately known as peritendinitis, tenosynovitis and tenovaginitis. It is clinically characterized by acute edema and hyperaemia of the paratenon with infiltration of inflammatory cells and within hours or days, fibroin exudates fills the tendon sheath. Despite these results, pathologists and scientists in this field argue that inflammation of the paratenon is a rare occurrence. (Khan et al 1999)

Tendinosis describes intratendinous degeneration without clinical or histological signs of an inflammatory response. This form of tendinopathy is typically considered an overuse injury that involves excessive loading of the tendons, frequent cumulative micro trauma and subsequent mechanical breakdown of the loaded tendon. In order to mediate the repair process, local tenocytes must maintain a fine balance between extracellular matrix network production and degradation, an unless fatigue damage is actively repaired, tendons will weaken and eventually rupture. (Sharma & Maffulli 2005) In humans, tendinosis is a common problem that is characterized by persistent, localized, activity related pain and swelling associated with common calcaneal (Achilles), patellar, and supraspinatus tendons. (Fransson et al 2005) The histological appearance of tendinosis is that of collagen disorientation, disorganization, and fibre separation with an increase in mucoid ground substance, increased prominence of cells and vascular spaces with or without neovascularisation and focal necrosis or calcification. (Clancy 1990; Sharma & Maffulli 2005) Additionally, affected tendons are characterized by fibrocartilaginous metaplasia of tenocytes and hypercellularity. (Fransson et al 2005) On visual inspections of affected portions of a tendon, they are lacking their normal glistening-white appearance and have been reported to have a gray-brown or pink-yellow appearance. (Fransson et al 2005; Sharma & Maffulli 2005)

Barring a direct trauma muscle strain, it is more likely that a soft tissue injury is a tendinosis lesion, and the practitioner should be aware of the pathology.

Specific Canine Shoulder Injuries

Problems specific to the canine shoulder joint include tendinopathies of the supraspinatus, and subscapularis muscles, bicipital tenosynovitis or bursitis, medial shoulder ligamentous instability, and strains of the teres major muscle. Supraspinatus calcification as well as tendinosis has been reported in veterinary literature. (Fransson et al 2005; Long & Nyland; Muir...
et al 1996; Laitinen & Flo 2000; Flo & Middleton 1990; SosloWSky et al 2000; Bardet 1998) Calcification has been reported to be a cause of unilateral forelimb lameness in dogs, with an incidence of 2.8 – 7% in all clinically lame dogs. (Long & Nyland 1999) The indicated treatment is surgical excision. (Muir et al 1996; Laitinen & Flo 2000) However, mineralization of the supraspinatus tendon is a common finding in asymptomatic limbs, and while improvement in symptoms is reported following surgery, long term follow up reveals that supraspinatus tendon mineralization can recur within a 5 year post-operative period. (Laitinen & Flo 2000; Flo & Middleton 1990) Tendonosis lesions of the supraspinatus tendon have been described in dogs and may precede or be associated with calcium deposits in the tendon. (Fransson et al 2005; Long & Nyland 1999) Additionally, it has been shown that overuse injuries of the supraspinatus can be induced in an animal model with a simulation of repetitive eccentric muscle activity created by running rats on a decline treadmill. (Soslowsky et al 2000)

The biceps tendon is a major stabilizer of the canine shoulder joint impacting cranial, medial and lateral translations of the humerus relative to the glenoid cavity. (Sidaway et al 2004) Bicipital tenosynovitis is a commonly reported pathology of the biceps tendon. (Gilley et al 2002; Long & Nyland 1999; Kramer et al 2001; Bruce et al 2000; Davidson et al 2000) Biceps tenosynovitis has been described as an inflammation of the biceps tendon or origin, its tendon sheath and the bicipital bursa within the intertubercular groove in the proximal humerus. (Davidson et al 2000) While inflammatory pathology of this tendon does exist, in some dogs, this disease may be the result of a degenerative process rather than an inflammatory process. (Gilley et al 2002) Other disease processes localized to the biceps tendon include but are not limited to calcification, osseous metaplasia, bone chip in the tendon sheath, and osteophyte formation in the intertubercular groove or supraglenoid tubercle. (Davidson et al 2000; Gilley et al 2002; Long & Nyland 1999; Kramer et al 2001) Clinical evaluation of the biceps tendon includes the biceps tendon test (positioning the forelimb into shoulder joint flexion with the elbow extended), pain on focal digital pressure applied directly to the biceps origin and/or intertubercular groove and the biceps retraction test. (Bruce et al 2000; Davidson et al 2000; Gilley et al 2002) As well a history of chronic and/or progressive weight bearing lameness that is worse after exercise and affecting active middle-aged or older medium to large breed dogs is common. (Bruce et al 2000; Gilley et al 2002; Davidson et al 2000) However, Bardet (1998) proposed that the biceps tendon test appears to be more of an indicator of generalized shoulder joint pain than a pathognomonic sign of biceps tendon disorders. Common veterinary treatments for bicipital tendonopathies include oral administration of non-steroidal anti-inflammatory (NSAID) medications, local injection of corticosteroids or tenotomy or tenodesis of the tendon. Yet animal model studies have revealed that NSAID administration or corticosteroid injections inhibit or delay collagen repair of muscles / tendons following NSAID administration and if inflammation is not the source of the pathology, then their use would be unwarranted. (Almekinders & Gilbert 1986; Obremsky et al 1994; Fransson et al 2005) As well, with the finding that the biceps tendon has a significant role in passive stability of the shoulder joint, the question as to whether any long-term adverse effects such as osteoarthritis development in the shoulder joint may be caused by a mild instability after tenotomy or tenodesis has not been answered. (Sidaway et al 2004)

Shoulder instability appears to be a common cause of lameness in medium and large-breed hyperactive dogs with a chronic permanent or intermittent foreleg lameness. (Bardet 1998) Other clinical signs of shoulder subluxation include atrophy of the shoulder muscles, non-weight-bearing lameness, spontaneous cries, signs and symptoms of disc disease or a ‘wobblers walk’ presentation, as well, abnormal cranio- caudal or mediolateral translations (drawer tests) are reported to be consistent indicators of shoulder joint instability in dogs subsequently diagnosed by arthroscopic evaluation. (Bardet 1998) This same author suggested grading of the direction and degree of the drawer translation: Grade 1 – when the translocation of the head of the humerus on the glenohumeral joint is not appreciated; Grade 2 (Mild) – when the translocation is appreciated but is not enough to allow the head of the humerus to rise up on the rim of the glenoid cavity; Grade 3 (Moderate) – when the head of the humerus is appreciated but is not enough to allow the head of the humerus to rise up on the rim of the glenoid cavity; Grade 4 (Severe) – when the head of the humerus courses over the rim of the glenoid cavity and is dislocated. Additional clinical findings may include pain with the biceps tendon test and pain on shoulder joint hyperextension. (Bardet 1998) Cook et al (2005a) described clinical diagnostic testing utilizing measurement of shoulder abduction angles. In dogs diagnosed with instability, the mean abduction angles (53.7 ± 4.7° measured goniometrically) were significantly larger than for all unaffected shoulders (32.6 ± 2.0° measured goniometrically). They proposed that the difference between angles is substantial enough to suggest that a visual observation of this asymmetry may be all that is required to make a preoperative diagnosis of medial shoulder joint instability in dogs. Medial shoulder instability is attributable to pathology of the medial aspect of the joint capsule, the subscapularis tendon and or the medial glenohumeral ligaments and may precede glenoid cartilage or humeral head cartilage wear or defects and eventual degenerative joint disease. (Bardet 1998; Cook et al 2005a) A demonstrated treatment for this condition is radiofrequency-induced thermal ‘shrinkage’ of the lax tissue to induce tightening of the joint capsule followed by post operative care involving a Velpeau sling and physiotherapy treatments. (Cook et al 2005b) The teres major muscle originates from the caudal angle and caudal edge of the scapula and inserts into the eminence on the proximal 1/3 of the medial surface of the humerus and shares a common
tendon of insertion with the latissimus dorsi. (Evans 1993) The teres major muscle is reported to flex the shoulder joint, however in analyzing the origin and insertion of teres major in the canine, this muscle can not only flex the shoulder but should also adduct and internally rotate the shoulder when the front limb is in an outstretched position. (Edge-Hughes 2004b) The proposed mechanism of injury would be an exaggerated extension, abduction and external rotation which could occur when a dog is running at high speeds and makes a sudden turn. (Edge-Hughes 2004b) Clinical presentation is of acute or chronic forelimb lameness that improves with rest but returns when allowed to resume normal activities. (Edge-Hughes 2004a) Physical examination reveals mild discomfort with full shoulder extension, inclusive of scapulothoracic movement, an increase in discomfort with the addition of abduction and external rotation, and moderate to severe tenderness (patient yelp or muscle twitching) on palpation of the teres major muscle or its tendon of insertion located in the ‘roof’ of the caudal aspect of the axilla. (Edge-Hughes 2004a)

Return to Sport Rehabilitation

Treatment of a tendinopathy lesion

What may look like an Acute tendinopathy may be a well-advanced failure of a chronic healing response in which there is neither histologic nor biochemical evidence of inflammation. (Magra & Maffulli 2006) When an athlete first notices tendon pain, tissue damage may already be advanced. (Kahn et al 1999) Chronic ligamentous (or musculotendinous) injuries will have random collagen orientation, wound contracture, restrictive adhesions / scars and may be degenerative. (Kahn et al 1999; Maganaris 2004; Sharma & Maffulli 2005) Treatments for tendinosis would utilize alternate treatment strategies to that of tendinitis / acute tendon lesions.

Prolonged immobilization may have detrimental effects such as a tendon atrophy, decrease in tensile strength and strain at failure, decreased water and proteoglycan content of tendons and an increase in number of reducible collagen cross links. (Sharma & Maffulli et al 2005) Therefore a proper balance between guided activity and relative rest is imperative.

Stretching has been shown to increase collagen synthesis and improve collagen fibre alignment, resulting in higher tensile strength. (Sharma & Maffulli 2005) A recent study compared eccentric exercise training versus a stretching regime for Achilles tendon pain and found that both groups exhibited marked improvement in symptoms but no significant difference between the groups. (Norregaard et al 2006)

Hands on manual therapy treatments, such as deep transverse frictions have been proposed for soft tissue lesions. (Cyriax 1982) Studies have been unable to show a consistent benefit over control groups for improvement of pain. (Rees et al 2005) Massage, which is thought to increase blood supply and therefore promote healing, is another form of manual therapy that has not been adequately studied in these cases. Therefore, neither of these treatment methods have proven efficacy as therapies of choice for tendinosis.

Cryotherapy is believed to decrease blood flow and tendon metabolic rate and hence reduce swelling and inflammation. (Rees et al 2006) While tendinosis lesions are not inflamed, this therapy could help if paratenonitis is present or for its analgesic effects. (Kahn 1999; Rees et al 2006)

The use of modalities such as ultrasound, laser and pulsed electromagnetic field may be beneficial in the treatment of muscle and tendon lesions. Both laser and ultrasound have been studies and proven to have a beneficial effect for tendon healing. (Demir et al 2004) One recent study found that 5.4 Joules per point of a 904 nm wavelength infrared, 20 mW laser was effective in increasing pressure pain threshold and reducing prostaglandin E2 concentrations in Achilles tendon lesions. (Bjordal et al 2006) Pulsed Magnetic Field Therapy (at 17 Hz) has been shown to improve collagen fibre alignment and increase the force to breakage, yet other studies failed to find improvement in adhesion formation with use in lacerated tendons. (Sharma & Maffulli 2005)

Perhaps the strongest evidence for treatment of tendinosis lesions lies in eccentric strength training protocols. Mechanical loading has been identified as an accelerant of tenocyte metabolism. (Kahn et al 1999) Eccentric muscular training is described as an event where the muscle contraction is purposely less than the opposing outside force, hence allowing for a slow controlled lengthening of the muscle or musculotendinous unit. (Magee 1987) Eccentric training consisting of twice daily exercises of several repetitions for 12-weeks was able to produce a decrease in tendon thickening, resolution of neovascularization, and an increase in patient satisfaction in the Achilles tendons as well as showing similar improvement with patellar tendinopathy and supraspinatus lesions. (Alfredson et al 1998; Ohberg et al 2004; Rees et al 2006) Gravare et al (2001) utilized a 12 week program aimed at increasing local blood circulation, improving ROM, plus balancing and gait exercises and specific eccentric exercises, which graduated in intensity to eventually incorporate quick rebounding exercises to address an Achilles tendinopathy. This program was shown to provide subjective improvement in symptoms over a training program that utilized concentric exercise training. An additional study for patellar tendinopathy utilized eccentric squats on a decline board, doing 3 sets of 15 reps, twice a day for 12 weeks, while adding 5kg increments of weighting to progress the exercise. (Bahr et al 2006) This study did allow for a resumption of cycling, jogging on a flat surface, or exercise in water at the 8 week mark is pain was not involved in the exercise. Yet another study of Achilles tendinosis in soccer players, utilized 12-weeks of heavy resistance eccentric training while allowing the participants to continue with their
regular soccer training so long as the pain did not increase in doing so. (Langberg et al 2006) This study did find an increase in collagen synthesis rate with this protocol despite the lack of relative rest prescribed in many other protocols. A few of the studies cited above mentioned the importance in acceptance of pain during eccentric loading in order to obtain excellent results with this therapy. (Bahr et al 2006; Langberg et al 2006; Norregaard et al 2006) With this information, all muscular and tendinous lesions should undergo eccentric training at some point in time during their rehabilitation.

Extracorporeal Shockwave Therapy (ESWT) has been shown to create neovascularization and nitric oxide synthesis and may promote healing of experimentally induced Achilles tendinopathy lesions in rats. (Sharma & Maffulli 2005) ESWT may be useful for calcifying tendinopathies of the shoulder and possibly heel pain but there is little evidence of benefit for others. (Rees et al 2006) Users of ESWT should be warned that there may be the possibility of dose dependent tendon damage including fibrinoid necrosis, fibrosis and inflammation. (Rees et al 2006)

Prolotherapy can be an effective treatment for tendinosis. It has been shown to be beneficial in Achilles tendinosis lesions, its use when combined with eccentric exercise provided more rapid improvements. (Yelland et al 2009) Stem cell therapy may also be an effective option. This technique has been used in both small and large animal models; for example, mesenchymal stem cells do promote healing in a rabbit Achilles tendon, and using autologous bone marrow-derived stromal cells, researchers have developed a treatment for the management of injuries to the digital flexor tendons in horses. (Rees JD et al 2009)

Treatment of acute muscle injuries would simply follow traditional therapeutic principles. Incorporation of rest, ice, compression and ‘protection’ (since elevation is not always applicable) is utilized in the very acute stages. Following is the regeneration phase, which requires therapies to address tissue regeneration and promote newly constructed fibres to align properly, increasing circulation and restore coordination and body awareness. Lastly in the remodelling phase, strengthening, muscle extensibility, joint mobility, advanced neuromuscular retraining and return to sport conditioning are needed. Details on all of these components are beyond the intention of this paper. and return to sport for canine athletes to be instructed in and led through this kind of reconditioning following an injury or surgery. Considerations for this ‘end-stage / advanced level’ or rehabilitation may include the following (Edge-Hughes c): Exercises up and down hills, trotting exercises, acceleration / deceleration activities, cutting or rapid turning exercises, jumping exercises, concentric strengthening, eccentric strengthening, plyometrics, endurance, static balancing, and dynamic balancing. Reintroducing and retraining sport-specific movements is imperative to successful reintegration to an athletic career.

Conclusion

It is important for veterinarians and other animal health care practitioners to be aware of canine sporting injuries. Knowledge of stretching, warming up, and soft tissue injury identification and treatment options for the canine shoulder will benefit the welfare of canine athletes, as veterinarians become more able to offer suggestions for injury prevention and management to this unique clientele.

Return to sport following injury or surgery

Neuromuscular retraining

Neuromuscular retraining is a term coined to describe the rehabilitative retraining of coordination, skill training, and higher levels of strengthening. It is imperative to the end functioning

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The Importance of Differential Physical Therapy Diagnostics for the Canine Lumbo-Pelvic Hip Region to the Welfare of Canine Athletes

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The lumbo-pelvic-hip region, also known as the pelvic girdle consists of 7 lumbar vertebrae, a sacrum, two innominate bones, and the two femoral heads. Although described as separate entities in anatomy textbooks, they are interdependent in transferring weight between the torso and rear legs, and are interconnected via soft tissue structures. The signs and symptoms of subtle lesions in any of these areas can be similar and sometimes confusing for the practitioner to differentiate. A systematic physical manual exam as utilized in human physiotherapy practice would be of benefit to veterinary practitioners in dealing with canine athletes with a lesion in this region.

Potential Pain Generators in the Lumbo-Pelvic-Hip Region

To best approach pain localization and/or the root cause of the pain in the lumbo-pelvic-hip region, it is useful to identify potential pain generators in the area. Essentially, the practitioner is attempting to either localize or ‘theorize’ the lesion to be within joint, muscle, ligament, fasciae, bursae, nerve, dura, disc, or bone. As a general rule, physical therapy diagnostics first work to identify or rule out whether there are ‘red flags’ (cancer, aneurysm, compression fracture, spinal infection, visceral pain), which may manifest as a general history of sleep problems, inability to urinate or inability to hold urine, or if the patient is a smoker). Next, ‘yellow flags’ need to be considered, such as depression, fear-avoidance, or pain catastrophizing. It is hard to say whether ‘yellow flags’ would be detectable or have any implications in veterinary medicine. If the human patient is clear of red flags; and yellow flags are managed (by referral to the appropriate professional or by modifying the assessment/intervention approach), then the problem can be further classified. For ‘low back pain’, physiotherapy research has identified via retrospective studies, subgroup classifications of patients that respond successfully to different groupings of treatment approaches. These treatment classifications are grouped into one of the following treatment groups: manipulation, stabilization, centralization phenomenon, spinal stenosis, and neurodynamic. The qualifications for each grouping in humans do not directly translate to the animal, but it is perhaps worthwhile to consider these 5 subgroups of back pain never-the-less. Pain in or around the sacroiliac joint, is further classified by the dysfunctional component and/or treatment technique needed: form closure (as it relates to the joint position or stiffness/hypomobility of the joint), force closure (the ability of the muscles to hold the joint in the proper position), and motor control and timing (the ability for the surrounding muscle groups to contract at the precise time that they should when confronted by movement or weight bearing). The hip is then categorized by the type of disorder, for example, soft tissue, joint, osseous, fractures/dislocations, nerve entrapment syndrome, and paediatric disorders. For the purpose of this discussion, only minor lesions that would create performance issues in canine athletes will be discussed.

The Lumbar Spine

Just a Little About Anatomy & Biomechanics

There are 7 lumbar vertebrae, and in comparison to the thoracic spine, the vertebral bodies are longer. The transverse processes are long and angle cranially and slightly ventrally. Accessory processes are present from L1 to L3/4 but are absent at L5 or L6. Accessory processes limit lateral bending. The facet joints in the lumbar spine are involved in weight bearing and load transmission. They are mostly vertically aligned from L1 to L6/7, thus flexion and extension are the primary motions in this region, and any rotational motions are generally accompanied with flexion. In the presence of lordosis, the caudal articular surfaces can adapt and the facet joints remodel in response by creating larger articular surfaces. With the loss of a natural kyphosis in this region, the lumbar spine can develop ventral facet joints and/or caudal facet joints. These additional facet joint surfaces function as an attempt for the body to resist motion (rotation and extension) in extreme lordosis. However, when present together, both ventral and caudal facets result in the formation of a ‘ball and socket joint’ which would reduce spinal stability, leaving nothing to effectively resist axial rotation. Thus
intersegmental muscles would provide the stability and overall stiffness of the vertebral segments in rotation and lateral bending, and quadratus lumborum (which may replace the role of the iliolumbar ligament in humans) may serve to fixate and restrict lateral bending in the lumbar portion of the vertebral column. A theory on motion coupling has been proposed based on the orientation of the facet joints: It is logical that lateral bending induces axial rotation and axial rotation can, in turn, induce lateral bending.

There are four major factors relating to motion pattern; disc height, facet joint angle in the transverse/horizontal plane, facet joint angle difference between levels in the transverse plane, and length of lever arm. Higher intervertebral discs, less sagittally-oriented facet joint angles, and smaller cross-sectional area to height ratios allowed more flexion and extension range. The lumbosacral junction is prone to stenosis (especially affecting German Shepherd Dogs), characterized by intervertebral disc degeneration, disc herniation, osteophyte formation, and thickening of the ligaments and facet joint capsule. German Shepherd Dogs (GSD) have been observed to have a straighter (more vertically aligned) facet joint orientation at L5/6 and L6/7 and a larger angle difference between the lumbar and lumbosacral facet joints compared with control dogs. This inefficient facet geometry suggests a mechanical imperfection resulting in a significantly higher prevalence of osteophyte formation and an increase in facet joint surface area, which may predispose GSD to the development of lumbosacral stenosis. Mechanically induced damage to the disc is an important factor in the development of disc degeneration. In the cervical spine, facet joint orientation that permits a greater amount of rotation is thought to be associated with a higher degree of stenosis, instability and degeneration of synovial joints and discs. However, in humans it has been shown that lumbar disc degeneration in an early stage causes segmental instability, but in severely degenerated discs, motion is reduced. In dogs, it has been proposed that if materials of the nucleus pulposis herniated, then the intervertebral disc becomes more flexible. Thus, a greater amount of overall motion does not lead to degenerative disc disease, but rather degenerative disc disease can lead to greater mobility. As well, a large facet joint orientation divergence from L6/7 to L7-S1 may restrict overall mobility, which may lead to mechanical adaptations and movement strategies that put the disc at risk of degenerative changes. From this rationale, a correlation between degenerated facet joints in dogs and degenerative lumbosacral disc disease is that it is more likely that the disc disease precedes other osteologic changes or pathology.

### Spinal Nerves of the Lumbar Spine

The cord segments of the eleventh thoracic (T11) to the third lumbar (L3) sit within the vertebra of the corresponding number, whereas cord segments L4 – L7 sit within the fourth lumbar vertebra, the cord segments S1 – S3 sit within the fifth lumbar vertebra, and all five caudal cord segments lie within the sixth lumbar vertebra. Table 1 outlines the peripheral nerves, nerve roots and muscle innervations pertaining to the lumbar spine and hind limbs.

**Table 1. Segmental Nerve Roots, Peripheral Nerves and Thoracic Limb Muscle Innervation**

<table>
<thead>
<tr>
<th>Nerve</th>
<th>Nerve Roots</th>
<th>Muscle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Femoral Nerve</td>
<td>L4, L5, L6</td>
<td>Iliopsoas, Quadriceps complex, Sartorius</td>
</tr>
<tr>
<td>Obturator Nerve</td>
<td>(L4), L5, L6</td>
<td>External obturator, Pectineus, Gracilis, Adductor</td>
</tr>
<tr>
<td>Cranial Gluteal Nerve</td>
<td>L6, L7, S1</td>
<td>Middle Gluteal, Deep Gluteal, Tensor Fascia Lata, Piriformis</td>
</tr>
<tr>
<td>Caudal Gluteal Nerve</td>
<td>L7 (S1, S2)</td>
<td>Superficial gluteal, (Middle gluteal), (Biceps Femoris), (Semitendinosus)</td>
</tr>
<tr>
<td>Sciatic Nerve</td>
<td>L6, L7, S1, S2</td>
<td>Biceps Femoris, Semimembranosus, Semitendinosus, Obturator internus, Quadratus femoris, Gemelli</td>
</tr>
<tr>
<td>Common Peroneal Nerve</td>
<td>a/a</td>
<td>Peroneus longus, Lateral digital extensor, Long digital extensor, Cranial tibial</td>
</tr>
<tr>
<td>Tibial Nerve</td>
<td>a/a</td>
<td>Gastrocnemius, Popliteus, Superficial digital flexor</td>
</tr>
<tr>
<td>Pudendal Nerve</td>
<td>S1, S2, S3</td>
<td>External anal sphincter, the external genitalia</td>
</tr>
</tbody>
</table>

![Figure 2.1](https://example.com/figure2.1.png)  
*Figure 2.1* shows the dermatomes for the entire body, including the rear limb of the dog.
An understanding of neuroanatomy is important in order to make sense of neurologic signs and symptoms. Things to take into consideration are whether the animal is exhibiting upper motor neuron (UMN) or lower motor neuron (LMN) signs and symptoms, reflexes in specific muscle groups, lick or chew patterns along dermatomal regions, limb use, etc. The meaning of these findings will be discussed later in this paper.

**Selected Muscles of the Canine Lumbar Spine**

In addition to the abdominals (internal and external obliques, transversus, and rectus abdominis), the spinalis, iliocostalis, longissimus, multifidus, psoas muscles and quadratus lumborum are key muscles in the region of the lumbar spine. While the majority of these epaxial and hypaxial muscles are important ‘movers’ of the lumbar spine, it has been shown in humans that the multifidus and transverse abdominal are key to stabilization as well. Table 2 outlines important selected muscles of the lumbar spine.

<table>
<thead>
<tr>
<th><strong>Muscle</strong></th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Psoas Minor</strong></td>
<td>Originates from the vertebral bodies of T9/10 to L3/4 and inserts onto the ilium, blending with quadrates lumborum. It acts to dorsally (posteriorly) tilt the pelvis.</td>
</tr>
<tr>
<td><strong>Psoas Major</strong></td>
<td>Originates from the vertebral body of L3/4 to L7 and inserts with iliacus onto the lesser trochanter of the femur. It acts to flex the hip.</td>
</tr>
<tr>
<td><strong>Quadratus Lumborum</strong></td>
<td>Originates from T9 – T13 and rib 13 and the transverse processes of the lumbar vertebra. It inserts onto the pelvic surface of the wing of the ilium. It acts to fixate the lumbar spine.</td>
</tr>
<tr>
<td><strong>Multifidus</strong></td>
<td>Multifidus is an important muscle for control of intervertebral motion and fixation of the vertebral column. It is reported to have 4 different fascicles (including sacrocaudalis dorsalis medialis), originating as caudally as the sacrum and terminating as cranially as C2. Fascicles cross anywhere from 2 or more vertebral segments from mammillary or articular processes of caudal vertebra to spinous processes of cranial vertebra.</td>
</tr>
<tr>
<td><strong>Transverse Abdominal</strong></td>
<td>Deepest of the abdominals, it originates from the lumbar vertebrae and the thoracolumbar fascia, as well as the medial sides of ribs 12 and 13 and the costal cartilages of ribs 8 and 11. It inserts onto the linea alba and abdominal aponeurosis to the pelvis. Its actions are the same as for the obliques. It has been found in humans to have a significant role in control of intra-abdominal pressure, tensioning of the thoracolumbar fascia, stabilization of intervertebral motion, as well as support for the sacroiliac joint.</td>
</tr>
</tbody>
</table>

**Table 2. Examples of pathogen factors affecting diseases of cattle**

<table>
<thead>
<tr>
<th><strong>Muscle</strong></th>
<th><strong>Origin</strong></th>
<th><strong>Insertion</strong></th>
<th><strong>Action</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Psoas Minor</td>
<td>Vertebral bodies of T9/10 to L3/4</td>
<td>Ilium, blending with quadrates lumborum</td>
<td>Dorsally (posteriorly) tilt the pelvis</td>
</tr>
<tr>
<td>Psoas Major</td>
<td>Vertebral body of L3/4 to L7</td>
<td>Iliacus</td>
<td>Flex the hip</td>
</tr>
<tr>
<td>Quadratus Lumborum</td>
<td>T9 – T13 and rib 13</td>
<td>Pelvic surface of the wing of the ilium</td>
<td>Fixate the lumbar spine</td>
</tr>
<tr>
<td>Multifidus</td>
<td>Vertebral column</td>
<td>Sacrum to C2</td>
<td>Control intervertebral motion, fixation of the vertebral column</td>
</tr>
<tr>
<td>Transverse Abdominal</td>
<td>Lumbar vertebrae and thoracolumbar fascia</td>
<td>Linea alba, abdominal aponeurosis</td>
<td>Stabilization of intervertebral motion</td>
</tr>
</tbody>
</table>

**Physical Therapy Assessment of the Lumbar Spine**

Human patients with low back pain (LBP) may present with similar symptoms but form a heterogenous group based on their response to treatment. Some patients are more likely than others to respond successfully to specific interventions by physical therapists. Prediction rules have been developed to identify patients with low back pain responding to manipulation, stabilization exercises and traction. The treatment-based classification system has been developed by clinical experts to guide the prescription of treatments for low back pain. The use of LBP-classification grouping of patients has been shown to lead to improved patient outcomes. The treatment classification system is comprised of 5 groups: Manipulation; Stabilization; Centralizing Phenomenon; Lumbar Spinal Stenosis; Neuromdynamic. Since evidence and clinical commentary on physical therapy for canine lumbar pain is lacking in the literature, this author proposes a systematic approach based on likely symptoms and clinical finding for various lumbar pathologies (Table 3).
### Table 3. Differential Diagnoses for Lumbar Spine Lesions

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Clinical Signs</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>FACET JOINT DYSFUNCTION</strong></td>
<td>• Reduced athletic performance or postural adaptations</td>
<td>• Unilateral epaxial muscle hypertrophy in an acute lesion or atrophy in a chronic lesion</td>
</tr>
<tr>
<td></td>
<td>• Human symptoms include referral of pain to the back, buttock, lower abdomen, groin, or legs.... so animals may display and exaggerated kyphosis (roach), excessive lordosis, spinal curvature or favouring a limb</td>
<td>• Discomfort to direct dorso-ventral pressure over the spinous process</td>
</tr>
<tr>
<td></td>
<td>• Animal may groan to move, be slow to rise, avoid certain movements / activities (i.e. jumping into the car)</td>
<td>• Discomfort to lateralized pressure to the side of the spinous process (often more painful in one direction than the other)</td>
</tr>
<tr>
<td></td>
<td>• Human studies show a high correlation with lower limb sporting injuries (i.e. hamstring tears, ligament injuries, tendonopathies)</td>
<td>• Discomfort to flexion testing</td>
</tr>
<tr>
<td></td>
<td>• Unilateral epaxial muscle hypertrophy in an acute lesion or atrophy in a chronic lesion</td>
<td>• Hypomobility/Stiffness to mobility testing (i.e the ability of the joint to fully open or close properly)</td>
</tr>
<tr>
<td><strong>DISC LESION</strong></td>
<td>• Antalgic posture</td>
<td>• Very reactive on palpation of the spinous processes or adjacent soft tissues (L1 – L4/5)</td>
</tr>
<tr>
<td>In the region of Cord Segments</td>
<td>• Avoidance behaviours</td>
<td>• Muscle spasms impede mobility testing (i.e. for facet joint stiffness)</td>
</tr>
<tr>
<td></td>
<td>• Slow to rise, displays of discomfort with certain activities</td>
<td>• Bilateral epaxial muscle spasms</td>
</tr>
<tr>
<td></td>
<td>• Neurological signs (if present)</td>
<td>• Possibly poor balance on displacement</td>
</tr>
<tr>
<td></td>
<td>• Reduced coordination and/or balance on displacement</td>
<td>• Possibly sluggish, slow, or diminished placing reflex</td>
</tr>
<tr>
<td></td>
<td>• Scuffing</td>
<td>• Possibility of a crossed extensor reflex (if lesion is cranial to L4)</td>
</tr>
<tr>
<td></td>
<td>» UMN bowel/bladder is more likely if lesion is cranial to L4</td>
<td>• Possibility of hyper-reflexia of tendon/muscle reflexes</td>
</tr>
<tr>
<td></td>
<td>» LMN bowel and bladder is more likely if lesion is caudal to L4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Very reactive on palpation of the spinous processes or adjacent soft tissues (L1 – L4/5)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Muscle spasms impede mobility testing (i.e. for facet joint stiffness)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Bilateral epaxial muscle spasms</td>
<td></td>
</tr>
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<td></td>
<td>• Possibly poor balance on displacement</td>
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<td>• Possibly sluggish, slow, or diminished placing reflex</td>
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<td></td>
</tr>
<tr>
<td>Lesion</td>
<td>Clinical Signs</td>
<td>Test</td>
</tr>
<tr>
<td>------------------------</td>
<td>-------------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------</td>
</tr>
<tr>
<td>DISC LESION</td>
<td>• Antalgic posture</td>
<td>• Very reactive on palpation of the spinous processes or adjacent soft tissues (L5/L6 – L7/S1)</td>
</tr>
<tr>
<td></td>
<td>• Avoidance behaviours</td>
<td>• Muscle spasms impede mobility testing (i.e. for facet joint stiffness)</td>
</tr>
<tr>
<td></td>
<td>• Slow to rise, displays of discomfort with certain activities</td>
<td>• Bilateral epaxial muscle spasms</td>
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<tr>
<td></td>
<td>• Scuffing</td>
<td>• Possibility of hypo-reflexia of tendon / muscle reflexes</td>
</tr>
<tr>
<td></td>
<td>» LMN bowel and bladder is more likely if lesion is caudal to L4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>» Depending upon severity of the damage and location caudal to L4, may probably have paraesthesia (manifesting as off-loading of a limb, and/or licking and chewing, or a lick granuloma)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>» Depending upon severity of damage and location caudal to L4, may have a root signature stance</td>
<td></td>
</tr>
<tr>
<td>DISC DEGENERATION</td>
<td>• Signs and symptoms are likely to look similar to a disc lesion affecting cord segments or the cauda equina</td>
<td>• Discomfort to direct dorso-ventral pressure over the spinous process</td>
</tr>
<tr>
<td></td>
<td>• Adaptive shortening may be detected by stretching the associated muscles and comparing from side to side and to other muscle groups</td>
<td>• Discomfort to lateralized pressure to the side of the spinous process (symmetrically affected)</td>
</tr>
<tr>
<td></td>
<td>• Trigger points can be manually palpated and are felt to be tight, reactive, fibrous bands within a muscle</td>
<td>• Bilateral epaxial muscle atrophy</td>
</tr>
<tr>
<td></td>
<td>• Facilitated muscle segments are often bilateral, and found to be hypertonic and painful on palpation.</td>
<td>• Depending upon severity and location, may show UMN or LMNL signs if disc degeneration is cranial to L4 or LMNL signs if lesion caudal to L5</td>
</tr>
<tr>
<td></td>
<td>• Manual stimulation (i.e. massage) may increase the tone in the ‘facilitated’ muscle</td>
<td></td>
</tr>
</tbody>
</table>
### Lesion

**MUSCLE**
- Weakness of the abdominals
- Atrophy of multifidus and epaxials
- Adaptive shortening of abdominals, psoas muscles and/or latissimus
- Myofascial trigger points in iliocostalis, quadratus lumborum or iliopsoas
- Facilitated muscle segments (affecting iliocostalis, & iliopsoas)

### Clinical Signs
- Are muscle issues in the lumbar spine every primary? Not likely.
- Weakness of the abdominals is often seen in association with a chronic lumbar spine dysfunction, sacroiliac joint lesion, or in juvenile or adult dogs with poor body awareness and conditioning
- Adaptive shortening is more often associated with postural compensations for other pain or injuries (i.e. off-loading of a limb)
- Myofascial trigger points are primarily activated by acute overload, overwork fatigue, direct impact trauma and by radiculopathy or secondarily by existing trigger points, visceral disease, arthritic joints, joint dysfunctions and by emotional distress. (Quadratus lumborum facilitation is found with suspected L/S instability)
- Facilitated muscle segments may be the result of irritation to the nerve root at a particular vertebral level, causing excitation and a reactive spasm in the adjacent muscle or those peripheral muscles supplied primarily by the affected nerve root (iliopsoas facilitation is often found with any form or lumbar spinal pain)

### Test
- Abdominal weakness may display as an inability to hold the trunk and pelvis in a level position when one rear leg is slowly taken off the ground (which improves when the abdominal muscles are stimulated to contract)
- Adaptive shortening may be detected by stretching the associated muscles and comparing from side to side and to other muscle groups
- Trigger points can be manually palpated and are felt to be tight, reactive, fibrous bands within a muscle
- Facilitated muscle segments are often bilateral, and found to be hypertonic and painful on palpation.
- Manual stimulation (i.e. massage) may increase the tone in the ‘facilitated’ muscle

**NERVE**
- Nerve root inflammation
- Mechanical deformation &/or damaged nerve root
- Irritation of dural sleeve
- Movement impairment or adverse mechanical tension

### Clinical Signs
- Antalgic posture
- Active movement dysfunction (i.e. poor cranial swing of the rear limb with sciatic nerve involvement)
- Root signature stance or simple off-loading of a limb
- Possible lick granuloma and/or licking or chewing at a limb

### Test
- Adverse responses to neural tissue provocation tests which relate specifically and anatomically to the suspected nerve / nerve root.
- Passive movement dysfunction (i.e. poor mobility or resistance throughout range to ‘stretch hamstrings’ for a sciatic nerve issue)
- Pain on palpation of specific nerve trunks which relates specifically and anatomically to the suspected nerve/nerve root.
- Evidence of a local cause for the neural tissue mechanosensitization disorder
- Abnormal tendon reflexes (hyper or hypo-reflexes)

**BONE**
- Spondylosis
- Osteophytes
- Facet arthrosis

### Clinical Signs
- Spondylosis may manifest with no signs and symptoms or may appear just as a degenerative disc (with the symptomatic lesion being at a mobile site adjacent to the spondylosis)
- Osteophytes are likely to present as a nerve lesion
- Osteophytes may be detected by placing the facet joints on the suspected side into full extension/compression (which might recreate the signs and symptoms of a nerve root impingement)

### Test
- Spondylosis will yield no movement (with a boney end feel) to any manual movement tests.
The Sacroiliac Joint

Sacroiliac joint dysfunction is a term used in humans to describe pain in or around the region of the sacroiliac joint (SIJ) which is presumed to be due to misalignment, abnormal movement or insufficient stabilization of the joints. Dysfunctions and/or lesions affecting the canine sacroiliac joint have received limited attention in veterinary research and/or clinical practice. However, the sacroiliac joint in the dog and possibly in other small animals is similar enough to that in humans to argue that sacroiliac joint lesions and dysfunctions may well be a potential source of back, pelvic and/or hind limb pain. Subsequently, similar diagnostics and treatment techniques or therapies to those used in humans may be applied to dogs.

Just a Little Sacroiliac Joint Anatomy and Biomechanics

The SIJ in dogs is formed by both synovial and cartilaginous elements and is stabilized by the dorsal and ventral sacroiliac ligaments as well as the sacrotuberous ligament. It is capable of an average of 7° of rotation. No other motions (shears or translations) have been tested. The canine SIJ has asymmetric and varying angles of dorsoventral slope, medial-lateral slope and concavity between and within breeds. As well, the SIJ articular surface and the ligamentous attachment sites are proportionately smaller in large dogs, which may theoretically indicate that higher forces are exerted on the SIJs in large-breed dogs. Radiographic appearance of calcification may occur in more than 50% of dogs by the age of

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Clinical Signs</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>LIGAMENTOUS</td>
<td>• Instability (i.e. spondylolysis and lumbosacral disc disease</td>
<td>• Spondylolysis and L-S disc disease can demonstrate LMN lesion signs described above in the cauda equina section</td>
</tr>
<tr>
<td></td>
<td>• Poor dynamic muscle control (and excessive lordosis)</td>
<td>• Pain on palpation of the suspected vertebra, worsened with increasing pressure that creates extension</td>
</tr>
<tr>
<td></td>
<td>• Ligamentous ‘creep phenomenon’ from prolonged postural positioning</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Ligamentum flavum hypertrophy</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Spondylolysis animals may be excessively lordotic or hold themselves in a kyphotic position to reduce discomfort associated with extension postures</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Animals with poor muscle control may just appear clumsy and lacking in coordination, with or without exaggerated lordosis</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• While it is possible to experience ligamentous creep in a human, it may not be a causal factor in canine back pain.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Ligamentum flavum hypertrophy is likely to present just as a degenerative disc disease</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• A chronic spondylolysis lesion will result in hypotonicity of the adjacent epaxial muscles</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Exaggerated tail extension or hip extension (beyond the physiologic range of pure hip extension) may result in pain</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Poor muscle control can be tested by challenging balance in stance and ambulation.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• See degenerative disc disease for testing when suspecting ligamentum flavum hypertrophy</td>
<td></td>
</tr>
</tbody>
</table>

Lesion Clinical Signs Test
1.0 – 1.5 years. Chronic overuse, microtrauma from daily repetitive activities, or hormonal relaxation in females are proposed etiologic factors which might precipitate calcification. Calcification could also potentially suggest that in dogs, SIJ dysfunctions or inflammation may have been present at some time during the animal’s life. However, in contrast, Vleeming et al 1990 reported that with standard X-rays, the cartilage covered ridges and depressions in humans can easily be misinterpreted as pathologic because of the well-known over projections in SIJs, but that these cartilage covered ridges are not pathologic. Table 4 shows the anatomical comparisons between the canine and human sacroiliac joints.

**Sacroiliac Joint Innervation**

Human and (mostly) animal studies have shown the following regarding innervations of the SIJ:

- The SIJ is innervated by the dorsal rami of the first, second, and third or fourth sacral nerves (S1 to S3 or S4)
- The SIJ has sensory innervation from the dorsal root ganglia of the first lumbar (L1) to the third sacral (S3)
- The cranial part of the dorsal side of the SIJ could be the part most associated with pain in the SIJ
- 29 mechanosensitive afferent units have been identified in the SIJ and adjacent tissues: 26 were in the capsule and 3 in the adjacent soft tissues; 28 of the 29 were classified as nociceptors and only 1 of the 29 was classified as proprioceptive.

Thus sacroiliac joint dysfunctions may also cause pain beyond the pelvic or back region. Human studies have found sacroiliac joint pain referral patterns that range from the upper lumbar spine to anywhere in the lower limb. Additional studies have shown that sciatic pain (in patients clear of lumbar disc or stenotic issues) may arise from the extravasation of sacroiliac joint fluid near the lumbosacral plexus or the presence of Substance P in the periarticular tissues of the SIJ.

### The Piriformis Muscle as it relates to the Sacroiliac Joint

Human studies have suggested that sciatica may ensue secondary to compression as a result of piriformis tension (known as piriformis syndrome). Any lesion of the sacroiliac joint may cause an inflammatory reaction of the piriformis muscle and its fascia since their origin lies at the capsule of the sacroiliac joint (and the mechanisms mentioned above for sciatic nerve inflammation). This phenomenon has been classified as secondary piriformis syndrome or pelvic outlet syndrome in cases where there is buttock pain with or without radiation down the leg (as this depends on the location of the pathology in relation to structures adjacent to the sciatic notch) provided that spinal pathology is excluded. In a recent study and review, it has been speculated that piriformis syndrome may be as common as herniated discs in the cause of sciatica. In dogs, the origin of the piriformis muscle is reported to be the lateral surface of the third sacral (S3) and first coccygeal vertebrae (Cxy1) and/or the border and ventral surface of the sacrum and sacrotuberous ligament. The phenomenon of piriformis spasm and tension has been hypothesized to occur in association with sacroiliac joint dysfunctions in the dog.

### The Hip

**Anatomy and Biomechanics of the Canine Hip**

The hip joint is a ball and socket joint formed by the head of the femur articulating with the acetabulum which is further deepened by the acetabular labrum. The joint capsule has various thickenings but there are no definite ligaments external to the joint. Securing the joint however, is the ligament of the head of the femur, which is a heavy flattened cord from the fovea in the head of the femur to the acetabular fossa. The transverse acetabular ligament traverses the acetabular notch. The available ranges of motion will include 45° flexion, 165° extension, 120° abduction (with hip flexed and stifle flexed), 65° adduction (with stifle flexed), 55° internal rotation and 50° external rotation.

<table>
<thead>
<tr>
<th>Canine</th>
<th>Human</th>
</tr>
</thead>
<tbody>
<tr>
<td>The SIJ is both synovial and cartilaginous.</td>
<td>The SIJ is both synovial and cartilaginous.</td>
</tr>
<tr>
<td>Ligamentous stabilizing structures: dorsal, ventral and interosseous SIJ ligaments, sacrotuberous ligament (note: the sacrospinous ligament &amp; iliolumbar ligament are not mentioned in canine anatomy texts).</td>
<td>Ligamentous stabilizing structures: dorsal, ventral and interosseous SIJ ligaments, sacrotuberous ligament, sacrospinous ligament, &amp; iliolumbar ligament.</td>
</tr>
<tr>
<td>Mean rotation at the SIJ is 7° with a 95% confidence interval between 4° and 13°.</td>
<td>Mean rotation at the SIJ is reported between 2° and 9°.</td>
</tr>
</tbody>
</table>
Development of the Canine Hip

All puppies are born with normal hips, but by 2 weeks, changes may have already occurred that predispose to excessive joint laxity and changes in the shape of the femoral and pelvic components of the joint. Adaptive remodelling has been shown to cause an abnormal hypertrophy of the femoral heads in 5-week old puppies where the femoral head was manually dislocated and left luxated. The same study found that luxation and repositioning did not impact the shape or size of the femoral heads at a 4-week follow-up. It is said that joint incongruity leads to osteoarthritis and varying degrees of dysfunction and pain. However, passive laxity is not independently sufficient to cause degenerative joint disease (DJD), yet there is a greater probability of developing DJD if greater laxity is present. Canine literature reports that greater pelvic muscle mass is associated with a reduced incidence of canine hip dysplasia (CHD). Diminished muscle mass and altered muscle fibre size and composition is a key finding in dysplastic dogs as early as 8-weeks of age. Environmental factors, including diet have been shown to have significant effects on the incidence and severity of DJD in dogs with CHD. There is a natural tendency for many immature dysplastic dogs to overcome acute hip pain as they mature. This is perhaps due to fibrosis of the joint capsule and acetabular remodelling that increases stability, and the healing of microfractures.

Legg-Calve-Perthes (LCP) is a heritable developmental disease of the canine coxofemoral joint manifesting as avascular necrosis of the femoral head. It appears more in small and toy breeds of dogs. Puppies can experience a reduced blood flow rate of the femoral head with either traction or compression applied at one half body weight to the hip joint. The same occurrence does not occur in adult dogs. The plausible proposed theory is that there is occlusion of the dorsal retinacular blood supply perhaps due to excessive jumping on their rear legs. Roentgenographically detectable changes in the affected femurs may be observed 2 – 3 weeks before onset of lameness and muscle atrophy in the corresponding limb. Lameness may not appear until the bone collapses, and the optimal congruity at the hip joint is lost, which leads to degenerative changes.

Muscles Causal to or Impacted by Hip Pain

Spasm and tension in the pectineus muscle spasm has been documented to occur in conjunction with canine hip dysplasia. However, spastic activity of the pectineus muscle when stretched can occur in both dysplastic and normal dogs, and at least one study confirms that a pectineus tenotomy does not prevent CHD and may indeed result in added pathologic changes within the hip. In man, the pectineus muscle has connections to the inferior hip capsule. Similar origination has not been reported in the dog, however this author has found that a pain response on compression palpation of the canine pectineus muscle correlates well with clinical hip pain. Atrophy of the gluteal muscles in response to hip pain, dysfunction, and following hip surgeries is a common occurrence in both

Table 5. Differential Diagnosis for a Sacroiliac Joint Lesion

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Clinical Signs</th>
<th>Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sacroiliac Joint Dysfunction</td>
<td>Not all of these signs may be present in all dogs with suspected SIJ dysfunction:</td>
<td>Human studies for SIJ dysfunction validate the use of a 3/5 rule for testing the SIJ. Proposed tests are as follows:</td>
</tr>
<tr>
<td></td>
<td>• Reduced athletic performance</td>
<td>• Pain on palpation of the piriformis muscle</td>
</tr>
<tr>
<td></td>
<td>• ‘Crooked’ sitting</td>
<td>• Pain on palpation of the dorsal SIJ ligaments</td>
</tr>
<tr>
<td></td>
<td>• Slowness on walks</td>
<td>• Presence of gluteal atrophy (if chronic)</td>
</tr>
<tr>
<td></td>
<td>• Exaggerated kyphosis / ‘tucking under’ of rear end</td>
<td>• Pelvic asymmetry</td>
</tr>
<tr>
<td></td>
<td>• Yelp when getting up from lying</td>
<td>• Asymmetric stiffness or hypermobility on translation tests (joint glides) or rotational tests at the SIJs</td>
</tr>
<tr>
<td></td>
<td>• Gait alterations, mild off-loading of a limb, lameness, or root signature stance</td>
<td>• Specifics Tests</td>
</tr>
<tr>
<td></td>
<td>• May lick or chew at a limb or present with a lick granuloma</td>
<td>» Thigh thrust technique</td>
</tr>
<tr>
<td></td>
<td></td>
<td>» SIJ Distraction technique</td>
</tr>
<tr>
<td></td>
<td></td>
<td>» FABER test (flex, abd, ext rotation) of the hip</td>
</tr>
<tr>
<td></td>
<td></td>
<td>» Trendelenburg sign (dropping of the pelvis/torso during a 3-leg stand)</td>
</tr>
</tbody>
</table>
man and animal. In humans, adaptive muscle shortening of certain muscles around the hip (i.e. tensor fascial latae, rectus femoris, iliopsoas, and adductors) may also impact ideal hip functioning. As well, myofascial trigger points may be causal or resultant of pain in and around the hip region. One canine study described the presence of trigger points specifically in the quadriceps, pectineus, ilio-costalis lumborum, semitendinosus, semimembranosus, tensor fasciae latae and gluteus medius muscles. This author would add iliopsoas, sartorius and deep gluteal to this list based on clinical findings. Lastly, strain injuries pertinent to the canine hip have been reported in the iliopsoas, pectineus, gracilis, sartorius, tensor fasciae latae, rectus femoris and semitendinosus muscles.

Nerve Injuries
Gait problems and lameness could result from nerve injuries near the canine hip. It has been documented that due to the close association, the femoral nerve can be impacted by iliopsoas injuries. Sciatic nerve injuries have been reported in association with hip dysplasia, and hip or pelvic surgeries. No studies could be found implicating isolated obturator nerve injuries in dogs.

Table 6. Differential Diagnoses of the Canine Hip

<table>
<thead>
<tr>
<th>Lesion</th>
<th>Clinical Signs</th>
<th>Test</th>
</tr>
</thead>
</table>
| Muscle strain or tendonopathy       | • Lame after resting, resolution after warm up, & lame again with too much exercise  
• Gait alterations  
• In rare cases, LMNL neurologic signs | • Pain on palpation  
• Pain to stretch (see table 7)  
• Pain to resist a muscle contraction or movement that uses the affected muscle  
• If neurologic involvement, LMNL reflexes  
• If neurologic involvement, adverse responses to neural tissue provocation tests which relate specifically and anatomically to the suspected nerve / nerve root. |
| MUSCLE IMBALANCES                   | • Gait alterations  
• Adaptive postures  
• Distal limb injuries | • Adaptive shortening may be detected by stretching the associated muscles and comparing from side to side and to other muscle groups  
• Trigger points can be manually palpated and are felt to be tight, reactive, fibrous bands within a muscle  
• Gluteal weakness may display as an inability to hold the trunk and pelvis in a level position when one rear leg is slowly taken off the ground (which improves when the gluteal muscles are stimulated to contract) = Trendelenberg sign |
### Lesion | Clinical Signs | Test
--- | --- | ---
**JOINT** | • Juvenile dog  
• Reduced exercise tolerance  
• Slow to rise / lie down  
• Preference for sitting compared to prolonged standing  
• Bunny hopping gait  
• Poor coordination  
• Lameness after exercise  
• Audible click in hip with walking | • Positive Barlow, Barden, & Ortolani test  
• Hips may click with balance on displacement  
• May have gluteal weakness & a Trendelenberg sign (see description above)  
• If painful: atrophied gluteals  
• If painful: pain on deep palpation of pectineus or deep gluteal muscles  
• If painful: pain to compress the joint (craniodorsally and/or medially)  
• If painful: pain on isolated hip extension and/or medial rotation

**JOINT** | • Juvenile dog  
• Small or toy breed, Terrier, or Australian Shepherd  
• Gait alterations or lameness | • Pain with hip ROM  
• Pain on hip joint compression  
• May have gluteal weakness and a Trendelenberg sign (see description above)

**JOINT** | • May occur following trauma or developmental disease at any stage of life  
• Lame after resting, resolution after warm up, & lame again with too much exercise | • May have gluteal weakness & Trendelenberg sign (see description above)  
• If painful: atrophied gluteals  
• If painful: pain on deep palpation of pectineus or deep gluteal muscles  
• If painful: pain to compress the joint (craniodorsally and/or medially)  
• If painful: pain on isolated hip extension and/or medial rotation

### Table 7. Description of stretches designed to target specific muscles.

<table>
<thead>
<tr>
<th>Muscle</th>
<th>Stretch</th>
</tr>
</thead>
<tbody>
<tr>
<td>Iliopsoas</td>
<td>Extend the hip with internal rotation (the stifle should be extended as well).</td>
</tr>
<tr>
<td>Pectineus</td>
<td>Abduct the hip (The stifle should be flexed)</td>
</tr>
<tr>
<td>Gracilis</td>
<td>Flex the hip with the stifle extended. Add abduction of the hip to focus the stretch.</td>
</tr>
<tr>
<td>Sartorius</td>
<td>Extend the hip with the stifle flexed.</td>
</tr>
<tr>
<td>Rectus femoris</td>
<td>Extend the hip with the stifle flexed (Note: During multiple dissections, this author has attempted to stretch the rectus femoris muscle using this method, but has found that the tension produced in the sartorius muscle did not permit any stretch of the rectus femoris muscle. It was found that rectus femoris could not be put into tension with any combination of leg movements.)</td>
</tr>
<tr>
<td>Semitendinosus</td>
<td>Flex the hip with the stifle extended. A very slight abduction of the hip and/or flexion of the tarsus may accentuate the stretch.</td>
</tr>
<tr>
<td>Tensor fasciae latae</td>
<td>Adduct the limb with the hip in a slightly extended position.</td>
</tr>
</tbody>
</table>
References


Social Policy in Food Production Integrating citizen concerns for Farm Animal Welfare

Terry Whiting, DVM, MSc

The contemporary reality of food, farming and animal use is a result of changing relationships between the state, the market and civil society, caused by processes of globalization, industrialization, privatization and individualization. In postmodern society a plurality of policy arrangements around food, animals and agriculture suggest that solutions to future problems of animals and society will not be addressed with the previous tools used to develop agricultural policy. In 2005 Agriculture and Agri-food Canada reorganized to eliminate the then embryonic infrastructure that would have allowed for the inclusion of farm animal welfare in national policy (Canadian Agri-food Research Council, The expert Committee Structure, specifically the Expert Committee on Animals). Social policy related to farm animal welfare in Canada currently must be pursued at the sub-national level for political and legislative action and directly at the industry level for consumer action.

INTRODUCTION

For centuries, the world has been divided into sovereign Nation States, most of which in the last 100 years pursued a national policy of self-sufficiency in food production, with an ongoing goal to be independent of the food supply of other countries. To reach this goal, most nations subsidized agricultural food production where under-producing sectors were encouraged, and overproduction was always paid for by the government or was exported often supported by subsidy. Catastrophic world wars and resultant food shortages solidified food security as a core national program. Farmers in most countries up until the recent past have been buffered somewhat from volatility of the market: everything they produced was bought by somebody (overproduction mostly by the state). Society communicated its support for this arrangement by continuing to elect governments that pursued this agenda. Society communicates with industries via legislation; individuals communicate to industries via purchasing patterns. Veterinarians have been heavily involved in increasing production of human food of animal origin and in certifying those products and animals for export. During the last two decades, dramatic changes in the social, political and economic environments have had considerable impacts on the society’s and the consumers’ view on food and on the way food, especially foods of animal origin, are produced.

The major events that had and still have an indirect or direct impact on the production of food with or from animals are:

• The BSE, Avian Influenza and FMD outbreaks in UK and Europe resulted in the massive killing of animals and a growing suspicion in the public that something essential to modern industrial food production was fundamentally wrong, wrong to animals, wrong to the environment, contrary to the nature of food, and contrary to the human-animal contract upon which pastoral, agrarian and modern society had developed and flourished.

• The emergence of H5N1 poultry-human influenza in Hong Kong, recent emergence of “swine flu” in Mexico possible swine-human variant.

• The brake-down of the communistic block in the late 80’s and early 90’s, with free-market principles (neo-liberalism) replacing plan-economy prescriptions. This contagious Thatcherism has initiated the globalization of almost all economies especially in agriculture trade.

• The creation of the WTO (World Trade Organization) replacing the GATT (General Agreement on Tariffs and Trade) in 1994 leading to a growing liberalization of the trade in food and raw materials for food including animals and animal products:

  » led to the fact that food retailers and grocery chains can theoretically buy any food from anywhere in the world, and national food supplies are not any longer something that retailers are dependent on.

  » led to the fact that food producers/processors and retailers can buy (where the consumer discriminates) on qualities other than price like “freely traded” or eco-friendly products.

  » emerging oligopolists such as Wal-Mart, demanding large volume of standardized produce at cut throat prices greatly decreasing the margins available to producers, especially small producers.

  » Group actions like the “Battle in Seattle” bringing international trade policy and agriculture policy into the living room.
The enlargement of the EU on May 1, 2004 with 10 new EU members emphasized a new trend in blocking of the globe into trading blocs as opposed to trading nations.

In Europe there has been increased politicization and market action of the “ideas” related to GMO’s (genetically manipulated organisms) in plant production, and animal welfare in livestock production.

North America, by serendipity has been spared the brunt of the majority of the critical issues that have affected Europe. Notable exceptions have been the BSE scare, environmental concerns related to concentrated livestock feeding operations and most recently movements to regulate livestock production methods at the State (sub-national) level in the USA.

These demanding changes in society, the market and legislative restriction on production are not only provides challenges for the animal producers, but also for the veterinary sciences. The rapid changes in expectations from society may significantly impact on the future work of the veterinary profession.

Democracy and Freedom

There are differences between countries in the way in which agriculture policy and animal welfare is dealt with politically. These can be partially explained by differences in political traditions, systems and cultures and/or differences in policy or regulatory styles. For example Canada is a parliamentary democracy while the USA has a presidential system with strong sub-national legislatures. In the EU agricultural policy is common and pan-national. Within Canada, there are differences in the way animal welfare policy is developed and implemented and in the respective roles of the state, market and civil society.

Animal protection can be considered as a policy domain as society has an interest in regulating severely unbalanced relationships between sentient beings; relationship between human animals (responsible for their actions, and with all the power) and non-human animals (not culpable for their actions, and generally powerless). The government constrains human treatment of animals from two significantly different moral justifications. In legal discourse on animal “cruelty”, the animal suffering can be largely considered irrelevant as the intent of law is to control the evil behaviour of the human perpetrator. Cruelty is about human intent not about objective animal experience. Animal protection laws on the other hand are focused on the negative animal experience and not necessarily concerned with human intent. In animal protection legislation, the intent is to place restrictions on human behaviour because the suffering of animals is an evil that society has a valid interest in avoiding. Some legislative instruments are muddy and fail to clearly separate these two social issues. Regardless; to restrain human choice and behaviour, society must come to some policy arrangement to decide what is not allowed or what must be done.

Animal Welfare A “Private” Good

In democratic societies, laws reflect the broader societal attitudes which in essence Parliament and legislatures represent. Current laws to protect animals from suffering are of two types; firstly, laws where affirmative acts of infliction of suffering are prohibited (prohibition of cruel treatment of animals), and secondly, laws that impose an affirmative action, for example to require person to provide a certain level of care for animals. Imposing an affirmative act has always been considered more burdensome, difficult to articulate and difficult to enforce than prohibiting an action (Favre & Tsang, 1993).

Animal protection law made major strides in the mid 1800’s in Britain and North America. In early animal protection legislation the primary societal attitude was concern regarding the moral state of the human actor rather than the suffering incurred by the animal and was an expression of what came to be known as a new Victorian ethic. This focus on human behavior, and even more specifically on the malicious intent of human behavior, placed early animal protection laws within criminal codes, where in Canada it remains today largely unchanged since the 1880’s.

Criminal law is intended to inform the conduct of individuals and to protect society from criminal activity. Enforcement of criminal statutes is universally paid for by the state through uniformed police services and the court prosecution and penal infrastructure. Enforcement of emerging social standards in Victorian times was problematic as the setting of “social” standards and informing the public of these standards was a new function of government. Previous to this era policing largely focused on property protection and had not generally been identified as contributing to the public good. Animal protection like the protection of children was initially placed outside the arena of public good and therefore public funding and was relegated to caring and philanthropic citizen groups. For most jurisdictions in North America, where the legal protection of animals is only extended to companion animals, the taxpayer has generally not footed the bill for enforcement. Private policing of animal welfare statutes through non-profit animal protection associations has become a popular model in North America for animal welfare enforcement in the context of non-criminal regulatory offences (Whiting et al. 2006). The non-profit organizations generally pay for identification and policing of offences from revenues collected by voluntary donation.
Farm Animal Welfare

In Canada animal welfare policy of non-livestock is purely a provincial or municipal affair. When animal welfare policy extends to livestock, due to Section 95 of the Canadian Constitution Act, the policy falls within the broader domain of national agricultural policy, which is set in a context of commodity free trade. In considering the extension of a duty of animal care from companion animals to commercial, especially farm animals in Canada; a new policy arrangement is required.

A 'policy arrangement' refers to the temporary stabilisation of the organisation and substance of a policy domain at a specific level of policy. It is characterised by a specific composition of (supporting and challenging) policy coalitions, their (differential) access to power and resources and opportunity to (re)define the rules of the game. The study of the formal policy discourses reveals how policy issues are framed and constructed and thereby explicitly addresses the changing interactions between the state, the free market and civil society, a key dynamic in agriculture policy and in animal welfare policymaking.

An appreciation of the dynamics of a changing policy arrangement and resulting political dance can be obtained by review of the attempts to update the Criminal Code animal cruelty provisions which started in earnest in 1998. After multiple variations of proposed amendments to the Criminal Code being introduced into the House of Commons, the discourse on this issue has been effectively halted or at least stalled by the passage of Senate Bill, S-213 on Dec 7, 2007 (AFAC, 2010).

Arts et al. (2000 a, b) distinguish between several policy arrangements including corporatist, statist and liberal (Figure 1). Under a corporatist arrangement, policy is made through the interaction and negotiation of established interest organisations, whereas in the statist arrangement the state plays a more dominant and regulatory role. The liberal arrangement is characterised by the greater involvement of private parties, such as companies, but also non-governmental organisations who may also contribute to resolving a problem and a decreased participation of the citizen as represented by the elected government. Canadian national policy is largely corporatist in nature with provinces and national organizations co-operating to arrive at solutions no one likes but all can live with.

Corporatism is a system of economic, political, or social organization where corporate groups such as agricultural, business, ethnic, labour, military, patronage, scientific, or religious groups are joined together into a single body in which the different groups are mandated to negotiate with each other to establish policies in the interest of the multiple groups within the body. It is predicated on the strength of the nation state, and the independence of states.

Corporatism has many critics as it is extremely susceptible to negotiated corruption, but, it does allow for the existence of a national policy. Countries that have corporatist systems typically utilize strong state intervention to direct corporatist policies and to prevent conflict between the groups. Examples of Corporatist intervention are subsidies in USA agriculture policy and Supply Management, with import control policies in Canada. Globalization with the growing strength of multi-national or better described as non-national corporations in agriculture product marketing works to minimize Nation based corporatism. For example, Europe is unable to legally discriminate against North American beef products (produces with hormone implants) on the basis of their prohibition of hormone implant use within Europe.

The profession of veterinary medicine is organized on a corporatist model as are all self-regulated professions in Canada. A changing public ethic related to access of foreign trained professionals to work in Canada is challenging this policy arrangement. Corporatism can occasionally be very ineffective as any individual group can block progress of the policy discussion by willful obstruction. In Canada, the Canadian Food Inspection Agency has been in corporatist negotiations with the livestock industries for more than 15 years to update the Humane Transport of Animals section (Part XII) of the Health of Animals Regulation. There has been little, to no discernable progress in this essentially a social contract issue. Recently Alexandra Mendès, Member of Parliament for Brossard –
La Prairie, PQ introduced a private members bill [Bill C-468 (CAN)] October 28, 2009 to effect by legislative means what the CFIA could not negotiate by co-operative means. This was a dramatic shift from a Corporatist policy negotiation to a Statist model in response to perceived failure of the previous negotiation to responsibly proceed.

The Liberalization of the Global Trade in Food

The current quantity-oriented food production (agricultural bulk-commodity supply of agricultural raw products into the food production chain) that guarantees the nutrient supply for a nation is changing into an international quality-oriented food system (vertical supply chains for the production of identity preserved food). The main driver of this development was and is without doubt the never-ending chain of food safety break-downs: *Salmonella* Enteritidis in eggs, BSE in the UK, *E. coli* O157:H7, the emergence of multi-drug resistant *Salmonella* Typhimurium DT104, the dioxin in animal feed scandal in Belgium, and the BSE-scares in several countries, including Canada. These events led to an increasing demand for transparency, traceability, and quality management in the entire food production chain, including agricultural primary production. In Canada there are emerging traceability and method of production certification for food safety in all commodities and for animal welfare in swine (Canadian Pork Council) and chicken production (Chicken Farmers of Canada).

Citizens have two methods of communicating convictions related to food and animal use or demonstrate they have opinions on farm animal welfare. Citizens may or may not be consumers. Consumers can communicate directly with production chains by boycotting product or preferentially paying for specialty certified product. Citizens who are not consumers (Vegan-vegetarian in this context) can not communicate by purchase patterns. Citizen concerns are communicated by voting patterns and sometimes by participation or support of political agendas and non-profit political organizations.

Consumers, who won’t voluntarily pay more for specific production practices as an individual, will often vote with non-consumers to make everyone pay more as demonstrated in recent political campaigns in the United States (*Table 1*) (Tonsor et al. 2009). In addition governments are often willing to constrain economic development in agriculture if supported by citizen concerns (Auger et al. 2003, Bill 17 Manitoba 2008).

The modern animal production industry, characterized by export competitiveness, efficiency of scale, intensification, and technology has been working diligently to do more to educate the public on scientific issues of modern livestock production and the advantages and benefits of modern production practices. A significant portion of the public remains unconvinced that these benefits sufficiently outweigh the costs of modern livestock production. A recent study by Lusk and Norwood (2008) in the USA suggested that people’s philosophic views on animal welfare are not likely to be strongly influenced by education campaigns; especially if those views are based in existing moral and ethical conviction. The attitudes of the population are not highly influenced by many science based measures of productivity and animal welfare. The results imply that science based animal production industry extension programs are unlikely to change public opinion about perceived welfare issues in modern production.

Although counter incidents can be identified, the overall power shift in the way policy is decided in Canada and other western democracies are in two distinct ways. There is a strong shift from the producer making decisions on his/her farm to the retailer describing the method of production. In the trade arena, there is a strong shift of power from society (The Nation State) to multinational corporations (Thompson et al. 2007). Some have argued that the use of the term multi-national is misleading, and that the adjective un-national better reflects a business practice model characterized by disregard, distrust and demeaning of any attitude that would try to balance the self-determination or social convictions of peoples with the profit and efficiency of trading groups.

Can Agriculture Afford More Animal Friendly Systems?

Nineteen cents of every dollar spent on U.S. grown food goes to the farmer for the raw food inputs, while the other 81 cents covers the cost of transforming these inputs into food products, promoting them and getting them to our grocery shelves and lunch counters (ERS 2004). Relatively expensive changes to methods of production on farm have small incremental costs in final product. Bornett et al. (2003) estimated that moving from slatted floors to deep straw feeder pig production would increase the cost of production about 30% (farmer bankrupt if unilaterally implemented) but increase the cost of pork only 4%. Increasing the floor space of feeder pigs, (partially slatted floor system) from 7.5 ft² to 8.6 ft² increased full chain cost of pork production 1% (in Holland) but reduced on farm profitability by 45% (den Ouden, 1997).

Within the supply chain from farmer to consumer, the cost of shelf segregation of product by label greatly exceeds the cost of on farm implementation of significant improvements in animal welfare. From a holistic utilitarian perspective, using labels to segregate product based on method of production is a very inefficient tool to improve the welfare of the average production pig.
Conflicting Thinking within the Livestock Industry

The big social concerns triggering intervention in livestock production in the 1990s have been environmental cost, food safety and animal welfare. Industry and government approach in food safety has been to seduce farmers to implement food safety and traceability programs on farm and internalize the increased cost. This has been accompanied by the conceptual/semantic conversion of farmers from livestock producers to food producers; the “I am a beef producer” campaign. The “Beef Producer” identity has had very poor uptake in the beef cattle industry, especially cow calf production where the farmers remain self-identifying as “cattle producers”. This phenomenon has also been documented in Europe (Skarstad et al. 2007).

Conversion from the zoophite language “cattle producer” to the sachrophite language “beef producer” is a powerful tool to commodify the production chain. Commodify: to turn into or treat as a commodity; make commercial; is consistent with the food safety dogma. In animal welfare evangelistic credo, commodification of livestock is associated with innate corruption and industrialization of livestock production.

From the food safety perspective; commodification of livestock production is the salvation of mankind and our hope for the future while from the moral obligation to animal care perspective; it is the original sin.
Livestock Industry Proactive and Reactive Initiatives

The major holistic proactive tools recognized for adopting standardized animal welfare production procedures at farm level is the implementation of on-farm measures based on the principles of HACCP (Hazard Analysis Critical Control Points) and on the principles of quality management (QM-Systems) and certification programs (Quality Assurance) such as ISO 9000:2000. The need to improve the method of production of food animal production in response to the consumers’ and the society’s expectations has been realized and addressed for at least 10 years. These changes are most evident in countries with a developed pork production, especially in countries that export pork (Denmark, The Netherlands, Belgium, the USA and Canada). These countries have, in slightly different ways, developed standards for swine production that are driven by the producer associations (the Canadian Pork Quality Assurance System, and the PQA System of the U.S. National Pork Producer Council), or by industry associations (the Danish Quality Management System for pork, the Quality Assurance System of the UK Meat and Livestock Council, the Dutch Produktchapt voor Vee and Vlees with its renowned IKB-program (Integrale Keten Beheersing), and the German QM-System for food from feed to retail (QS-System) (Blaha 2005).

The major re-active tools to deal with social concerns in agriculture have been legislative at the national or sub-national level including the “Phasing-out” of the most egregious components of management systems, in the EU but not treating the management system as a whole. In late January 2007, the world’s largest producer and pork processor Smithfield Foods, Inc. (Smithfield Foods, Inc. SFD) announced voluntary plans to replace gestation stalls at its 187 company-owned sow farms. In January 2008 Maple Leaf Foods Inc., reported that they also will also phase out the use of sow gestation stalls in favour of group housing at all its hog production operations within the next 10 years.

Canadian Government Actions

The Canadian Agri-Food Research Council (CARC) (1974-2006) was the most important national advisory body influencing agri-food research and policy. Now disbanded, it was funded by the Research Branch of AAFC, and had a small full-time staff in Ottawa. Its membership included representatives from AAFC and each provincial government (only one for Atlantic Canada), a representative from universities with colleges of agricultural and/or veterinary medicine, representatives of a number of national organizations (such as the Canada Grains Council, the Canadian Federation of Agriculture, the Canadian Forage Council, the Canadian Pork Council, the Agricultural Institute of Canada and the Canadian Horticultural Council), and chairs of four national “Canada committees.” The latter were the Canada Committees on Crops, Animals, Natural Resources, and Food; each of these committees met at least once a year to formulate recommendations to go to CARC.

The four “Canada committees” were the apex of a series of national or regional committees, generally referred to as “expert committees.” For example, the Canada Committee on Animals formed a sub-committee, the “expert committee on animal welfare” which met annually to identify research needs in both science and policy. Each expert committee involved representation from various provinces; the meat processing industries, the national livestock associations, veterinary and animal science universities and the Canadian Federation of Humane Societies; the producer representation was generally minimal on most of these committees.

CARC maintained an inventory of agri-food research in Canada, and assisted various sectors in developing national research and development strategies. For example, research strategies were developed for dairy and pork. The CARC web site, www.carc-crac.ca is now defunct; a search of AAFC website returns no reference or history that to suggest CARC ever existed and there is no successor group at the federal level to replace the functions of CARC. One could suggest that the federal government simply went out of the animal welfare consulting business; or effectively removed animal welfare from the federal agriculture ministers’ agenda.

CARC was a classic instrument of corporatist policy negotiations. It facilitated connection and communication between social and institutional power blocks involved in agriculture policy. Its dissolution represented a decisive policy decision that moved the process of policy making to a statist or liberal approach. An approach with less communication with society at large can be viewed as more Statist, even though in Agriculture policy is largely about subsidies, and what industries and programs qualify for public support.

On June 2001, the federal, provincial and territorial Ministers of Agriculture took an additional dramatic new approach to the participation of society in agriculture. New agriculture policy development would be a shared and integrated process based on 5 year plans. Costs for agriculture policy would continue to be jointly shared by the federal and provincial governments. The first 5-year plan was called the Agriculture Policy Framework (APF). It was primarily a business plan to try and keep farmers profitable. Topics made it onto the agenda if they could affect farm profitability; such as the areas of science and food safety. Environmental stewardship, which essentially is a social policy concern and the sole purvey of the Provinces under the Canadian Constitution, was also included in the scope of the APF. This in part may be explained as most provinces in Canada had implemented environmental protection legislation related to manure management between 1995 and 2000, making consideration of the environment a cost of production.
Animal welfare may be a social issue similar to environmental protection, but, was clearly excluded from the APF agenda and therefore no program related to farm animal welfare was eligible for funding under APF. The other dramatic change in overriding policy arrangement was the federal government through AAFC, would no longer fund new programs only the start up process of such programs. This reflects a markedly liberal conviction with a movement towards non-involvement of the government (society) in the business and economy of agriculture.

The new 5-year plan started in 2008 and is called the “Growing Forward” policy framework with $1.3 billion in federal funding. Farm biosecurity and livestock traceability were added to the agenda of approved initiatives but, animal welfare is not in scope with the exception of a side agreement signed with Alberta. If farm animal welfare as a true citizen concern, it is not a concern of the current FPT (Federal-Provincial-Territorial) policy arrangement. Provincial farm animal welfare initiatives can not be funded jointly with the federal government as other agriculture issues are, but, are the sole initiative of the province.

Some Hope

There are three emerging organizations on the Canadian landscape that give some hope for a future where farm animal welfare can be reflected in public policy. The first is the National Farm Animal Care Council [http://www.nfacc.ca/]. Agriculture and Agri-Food Canada’s Advancing Canadian Agriculture and Agri-Food (ACAAF) Program provided initial funding to establish a national Council on farm animal care. This is a non-government organization with a mandate to provide a national coordinated approach, to promote responsible farm animal care. The Council is composed of and funded primarily by the livestock industry. This organization will replace the function of developing farm codes of practice previously delivered by CARC. The NFACC must become self funded by the agricultural business interests in the near future as there is no method of funding this organization under the current FTP policy arrangement. The lack of government funding can be interpreted as a lack of independent greater societal participation in this issue.

Secondly, the provinces departments of agriculture have all reorganized to appoint a Chief Veterinary Officer and there is a consultative council of CVO’s in Canada. It is clear that in the near future improvements to farm animal welfare and legislation related to improved welfare will be in the jurisdiction of the provinces, possibly at variance with Section 95 of the Canadian Constitution. One of the early projects of the CCVO group was to participate in the development of a national Farm Animal Health and Welfare Strategy (Anon 2009).

Counter to all regular Agriculture policy of the Federal Government; on Friday Nov 6th 2009, The House resumed consider-

Conclusions

It can be expected that the organizational pattern of animal production will change considerably during the next decade characterized by increased industrialization and concentration as has been witnessed in poultry and swine production. Trade with food of animal origin will be increasingly dominated by vertically integrated supply chains which are market leaders for certain products and compete with other supply chains. Horizontal competition between single companies will be substituted by a competition of vertically integrated production systems.

Competition is good for the consumer as it will not only add to the quality and safety of the product, reduce the risk of the introduction and dissemination of infectious diseases as the flow of material and information can be documented and controlled, and decrease the price to the consumer. Markets dominated by a small number of buyers who are able to collectively exert control over the supply (oligopoly) tend to squeeze the profit margin of the producer and force increased industrialization and economy of scale.

Canada, as a moderate socialist country has a long history of citizen participation in the development of agriculture programs and a cohesive national public policy related to social contract issues. There is no current evidence that this tradition will continue in dealing with farm animal welfare as an emerging social issue. Citizen participation in policies around farm animal welfare may be increasingly difficult to actualize in Canada as a nation.
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Growing concern about industrial animal production, massive disease eradication exercises in Europe, coupled with recurring food scares, largely explains the increasing attention given to method of livestock production as a specific object for Public Policy in Europe. Simultaneously society has moved away from an economic model of companion animal ownership. Within the veterinary profession it is difficult to envision a unified ethic that can embrace the instrumental use of animals in food production and the affective use of companion animals. This paper entertains the idea that this divide can be explained by the partial replacement of the utilitarian ethics of the modern era with the emerging paradigm of postmodernism. Oxford English Dictionary refers to postmodernism as “a style and concept in the arts characterized by distrust of theories and ideologies and by the drawing of attention to conventions”.

INTRODUCTION

Hobbes describe the life of the average man in his natural state as solitary, poor, nasty, brutish and short (Hobbes 1651). The industrial revolution, introduced the period of “modernity” where the quality of life of the individual human was greatly improved because of increased cooperation, more efficient means of production, expanded mining of the earth for resources especially energy, increased freedom from pernicious labor and “stuff” became much more available, pervasive and cheap.

Some of the new readily available and inexpensive “stuff” was high quality foods of animal origin thanks to the new and efficient agro-industrial complex. Another manifestation of “stuff” was that increasing personal wealth and leisure time allowed more individuals and families to keep pets (Spencer 2006). Veterinarians are heavily involved in both consumer streams of modernism; 1. related to animal health and productivity for future use as food and, 2. related to animal use as sport, leisure, entertainment and as pet/companions. With these specialized areas of service, veterinary medicine has become a reasonably profitable profession for most.

The contemporary veterinary profession is trapped half in the modern world of industrial production and consumption, and half in the postmodern world constructed from individuals’ perceptions and expectations. The contemporary veterinarian must provide services with species representing many facets of societal values. Society values low priced eggs and simultaneously abhors the presence of puppy mills. The reflective veterinarian may experience ambivalence a she is tasked to serve the best interest of both the human client and the animal patient; the animal society as a whole; the human society as a whole; and of course his own monetary plight (de Graaf, 2007).

Modernism, Postmodernism

Sociological texts on animals and modern to postmodern society emphasise simultaneously the distancing between humans and livestock animals and the increasing human affinity for companion animals. Both changes in distance is thought to have intensified during recent modernity as a result of urbanisation, the replacement of animal by machine power and the industrial concentration of livestock production.

In this paper I use modernism as an approximation of Fordism as it has become culturally applied to livestock production. Others use “industrialization” essentially to identify a similar idea (Frazer 2005, Gyles 2010); however industrialization conveys a process where modernism better reflects a belief and lifestyle system. Fordism refers to the Utopian system of mass production and consumption characteristic of highly developed economies during the 1940s-1960s. Under Fordism, mass consumption was combined with mass production to produce continual economic growth and widespread material advancement. The 1970s to present have been a period of slower growth and increasing income inequality and some loss of blind confidence in the Fordist-Utopian paradigm. The Fordist conviction of “more, bigger, faster, cheaper,” in agriculture has driven the industrialization of livestock production and in consumerism the emergence of the big-box store. A characteristic mark of modernism is the attitude that no matter what the problem, with technology, we can fix it, whatever “it” may be.

Postmodernism is largely a reaction to the assumed certainty of scientific, or objective, efforts to explain reality. If you consider the blinded controlled clinical trial as the gold standard for evidence based medicine, then EBM clearly is a modernist instrument (Stolberg 2006, Hróbjartsson et al 1998). In the postmodern understanding, interpretation is everything; reality only comes into being through our interpretations of what
the world means to us individually. Perhaps an inaccurate example but consistent with postmodern thought; alternative veterinary medicine may be a “good” although no measurable effect on the animals’ health can be detected.

Public health, food safety and agricultural production are located in a purely modernist social geography. Modernist activities employ a significant portion of the veterinary profession. In the production of animals for human consumption, the profession is the body that assures the animals are drug free, pain free and healthy prior to slaughter and the product is wholesome at the end of processing.

The Welfare Quality® project is a large European Union research initiative directed toward the integration of animal welfare in the food quality chain. The intent is to provide a method to respond to public concern to improve farm animal welfare and communicate the improvements back to the citizen/consumer in a transparent manner. The project aims to accommodate both societal concerns and market demands, to develop reliable on-farm monitoring systems, product information systems, and practical species-specific strategies to improve farm animal welfare.

Some of the early studies, which are now published [see British Food Journal 2007:109(11)] investigated the attitude of farmers to farm animal health. The project aims to accommodate both societal concerns and market demands, to develop reliable on-farm monitoring systems, product information systems, and practical species-specific strategies to improve farm animal welfare.

The Welfare Quality® project is an interesting mix of postmodern thought expressed through classic modernist approach and application. The process is modern in that it has the format and structure of social science research; they start with working hypothesis; then measure, count, and test, do data analysis and make inferences. However, what they are attempting to measure is the human attitude towards animal welfare and agricultural production systems. The actual welfare of the production animal, from the perspective of the animal is not really of interest. It is not a question of whether sows prefer group housing over individual stall housing it is a question of how people would like sows to be housed. The Welfare Quality® project is an example of reality created by the interpretation of the viewer.

Postmodern View of Animals

The Merriam-Webster dictionary describes postmodern as “of, relating to, or being a theory that involves a radical reappraisal of modern assumptions about culture, identity, history, or language”. Bauman in Postmodern Ethics describes postmodern reality as a manifestation of urban living and the tyranny of individuality. Bauman’s compulsory individuality is depicted as a very difficult way to live, especially due to the stress that results from every individual being responsible to develop their own coherent set of moral convictions in isolation from any fixed community. Bauman also discusses the largely urban inter-personal practices, the way people in highly concentrated urban populations treat each other, which organise and underpin different ‘social spaces’, cognitive, aesthetic and moral. In his discussion of cognitive urban space he looks at sociological accounts of how people relate to ‘strangers’.

Bauman describes what he calls ‘the arcane art of mistreatment’. This refers to a set of techniques for living with strangers’ which involves relegating them to the background context of interaction, treating them as ‘non-admitted existence’ and allocating them to ‘the sphere of disattention’. In this constructed realm of non-engagement, there is no emotional cost and the social space is ethically empty.

Social techniques are used to ‘evict’ others from one’s social space, leaving them without recognition of their subjectivity and placed outside the rules of engagement and interaction operating in that space. For example panhandlers are recognized and placed in this realm of non-engagement. A key technique is avoidance of eye contact. Winnipeg has a by-law1 forbidding panhandling within the vicinity of “captive” audiences: bus stops, banks and ATMs, parking lots and parked cars, indoor public walkways, elevators and outdoor patios. This by-law enforces the intent of urban public space to not be social space.

Tovey (2002) considers the Bauman social structure of ‘mismet’ also extends to account of how people deal with modern food animal production. We might say that pets are met [have a name and are recognized as within society], wild animals are unmet [are respected, but are outside the common law], but vermin, food animals and laboratory animals are mismet [have no or limited standing within society].

The Bauman/Tovey theory offers us a way of understanding food animals as creatures that have been re-incorporated into society but under conditions which render them ignored by it or “outside of society”. Farm animals are in a different social space than companion animals and non-pest wild animals. At the farm level, modernity, the rule of man over nature is firmly entrenched (Buller and Morris, 2003). In modernist terms, animal welfare is defined in a negative framework, as the absence of identifiable evidence of non-welfare.

Veterinarians mentalité and the Instrumental use of Animals

Mentalités are “long term, unfocused, and passive popular beliefs about existing society that are not oriented toward action in the public arena” (Conrad 2006). When we speak or write about a topic, some ideas are reproduced, often unwittingly, because they are widely assumed to be true.

1 City of Winnipeg’s Obstructive Solicitation By-law No. 7700/2000,
All veterinarians share a background mentalité regarding the instrumentality of animals. Small animal practitioners believe that the “good life” for a dog includes separation from other dogs, intensive indoor housing often in isolation and castration/neutering; all things designed to modify the animal to better serve its purpose of being an animal companion to people. In zoo practice genetic control of the captive species are desirable which requires for example the creation of permanent bachelor troops of higher primates as part of the greater good (Lewandowski 2003). The zoo workplace requirement for neonates as they bring in the crowds brings problems of animal overpopulation and breeding is controlled by an International Zoological oversight and permit system. The largest user of animal life (perhaps with the exception of commercial fishing) is in the agro-industrial livestock system.

Integrating respect for animals is a growing part of companion animal practice and vegetarian and vegan lifestyle choices are considered consistent with the caring nature of veterinary medicine. The use of analgesia in companion animal practice may have reached the level of reasonable standard of care and a professional duty, while simultaneously analgesic use in food animal practice is not always provided even where the pain is caused by surgical procedures (Hewson et al 2007 a,b).

CONCLUSION

It is possible there is a growing difference in mentalité toward the use of animals between companion animal and food animal practitioners. I would suggest that this difference is far more fundamental and deeply established than a mere differing of opinion. The different mentalité may actually reflect a significant and fundamental difference in world view. Differences in what individuals believe to be morally acceptable and therefore define professional conduct may be problematic within a self-regulated profession where individual belief systems vary widely.

Alternatively it may be that Fordist livestock production systems are morally questionable and the veterinary profession has a leading role to play in finding ways to improve these systems (Gyles 2010).

The Big Questions

The practice of veterinary medicine in Canada is increasingly companion animal in nature (Jelinski et al 2009). This trend has also been identified by both the American Veterinary Medical Association (AVMA) and the Royal College of Veterinary Surgeons (RCVS) report that ≥60% of their practitioners are small animal (SA) practitioners, while 10% to 15% service the food animal industry (AVMA 2008, Robinson & Hooker 2006). Historically schools of veterinary medicine have been aligned with schools of agriculture and supported by departments of agriculture especially in the land grant university system in the United States.

If the primary employment of graduates of postmodern veterinary schools is companion animal practice then there is a real question of why a Ministry of Agriculture should fund veterinary medicine over chiropractic medicine. In Canada our most recent veterinary College in Calgary has aligned with the provincial medical training infrastructure not the agriculture training infrastructure, perhaps reflecting this obvious shift in priorities of the profession.

Perhaps a more sensitive issue is if the profession is grooming its public image to be the companion animal paediatrician (Rollin 2002) can the companion animal political interests continue to tolerate the Fordist approach to animal use in livestock production? For a vicious social criticism of the “pet paediatrician” model of veterinary practice I recommend the portrayal of “Dr. Leslie” in the television program King of the Hill (FOX 2005).

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Veterinarians are in the business of killing animals; killing for food, killing surplus companion animals in shelters and euthanasia. As veterinarians, we also kill surplus zoo animals; consult on pest animal control and the rare dangerous animal. Outside of the veterinary profession, animals are killed in hunting and dragged from the bottom of the seas by the millions to suffocate or be crushed prior to becoming food or animal feed. Simultaneously there has emerged the no-kill shelter movement in the United States and legal prohibitions on killing healthy animals in Europe. This paper will briefly review several issues involving veterinarians and animal killing.

INTRODUCTION

Many people in post-modern times feel that their pet is a member of the family and treat them as such (Ferguson, 2004) and euthanasia is a compassionate service provided by the empathetic small animal practitioner. Modern animal agriculture on the other hand has been heavily criticized for brutal exploitation of sentient beings, global environmental degradation, and fostering a fatal nutritional crisis in the population (Lund & Olsson, 2006). Whether in companion animal, equine, zoo, wildlife or food animal practice, veterinarians either directly kill or oversee and advise on the killing of animals.

Euthanasia and Humane Killing

A person’s emotional attitude towards something can often and easily be influenced by the term that is used to refer to it. A euphemism is a replacement term that is introduced in order to make something less offensive or negative that it would otherwise be. The paradigmatic example of a euphemism is the replacement of the term ‘civilian casualties’ with ‘col-lateral damage’ by war reporters. By contrast, a dysphemism is a replacement term that is introduced to make something seem more offensive or negative that it would otherwise be.

Tom Reagan in his book The Case for Animal Rights argues that painless killing of an animal is harm. Regan maintains that painless killing of animals is not always euthanasia and reserves the term euthanasia for incidents of animal killing characterized by the following; (a) technical; killing must be by the most painless means possible, (b) good; the killing must be in the animals best interest, (c) righteous; the one who kills must be motivated out of a concern for the interest, good, or welfare of the particular animal involved.

Veterinarians are involved in animal killing primarily in slaughterhouses assuring the provisions of humane slaughter legislation and in animal shelters. Veterinarians also provide the Regan definition of euthanasia in terminally ill companion animals and horses.

Killing of animals is unpleasant work and can also be dirty and dangerous. It may be the “dirty work” of the veterinary profession. “Dirty-work” is a term uses in sociology to refer to work that needs to be done but that society as a whole disapproves of (Ashforth & Kreiner 1999, Arluke 2003). Examples include, garbage collection, slaughter house and sex-trade workers.

The Human Animal Bond

Among the important positive contributions of animal agriculture is the effect of the human–animal relationship itself. It adds to the quality of life of many farmers and their families (Lund & Olsson, 2006). In practical farming, all animals are either generated specifically to become food or are salvaged at the end of their productive cycle for human food (dairy cows). Farmers see this cycle as a manifestation of the natural order of things and do not find it offensive. The killing of animals outside this natural order such as occurs in foot and mouth disease eradication is very offensive to farmers (Cohen et al. 2007, 2009). In recent work in Europe, close physical contact with farm animals is a part of the social and cultural environment that allows people to become close with animals (Bock 2007).

The human-animal bond (HAB) (Tannenbaum, 1985) entered popular culture with the publication of a book The Human Animal Bond and Grief in 1994 (Lagoni et al. 1994). The concept of “bond-based” companion animal practice soon emerged both in the practice of veterinary medicine and in the education of veterinary students. Briefly, this business theory suggests that veterinary practices can provide a better service (and be more profitable) if they de-emphasize the goal of dealing with the health of the companion animal and spend more attention to the emotional needs of the companion person. In postmodern companion animal veterinary practice “grief” may be a profit centre for your business (Reinisch, 2009).
Does being strongly bonded to an animal change the ethical relationship? Does one owe more to animals close to oneself than those more socially separated? In 2006, while working for the federal veterinary service, I traveled to a rural farm in Manitoba to kill tuberculin reactor cattle, collect tissue samples and dispose of the carcasses by rendering. The farmer was there to assist in assembling the cattle. Our planned killing process was to shoot the cows by free bullet while the cows were individually restrained in a head gate. Somewhat to my surprise, the farmer insisted he shoot the cows himself, and we could then do what we needed to get our samples. The killing of these cows was clearly a moral concern to this farmer. The full nature of that concern remains unclear to me. However, it was partly (from his statement) because he had more confidence in his technical skills using a firearm than our skills; so cows experiencing death without pain was important to the farmer. I believe there was other more compelling personal motivation for this action. Farmers primarily care for and protect their animals from injury, especially wildlife predation in this particular area of Manitoba. It may be that allowing someone else to kill the cows would have been a violation of the responsibility of protection that was not violated by killing the cows himself. My suspicion is that the issue was not about animal welfare and not about cows at all, but about the farmer and his relationship with cows.

The Postmodern Veterinarian

No other practice demonstrates the modern vs postmodern veterinary paradigm better than the ethical questions around the justification for the killing of animals. One thing the modern and postmodern veterinarian has in common is that we all kill animals, or supervise the killing of animals. The context of killing and the moral justification of killing could not however be more divergent.

Postmodernism is largely a reaction to the assumed certainty of scientific, or objective, efforts to explain reality. In the postmodern understanding, interpretation is everything; reality only comes into being through our interpretations of what the world means to us individually. Perhaps an inaccurate example but; in postmodern thought, alternative veterinary medicine may be a “good” although no measurable effect on the animals’ health can be detected. Postmodernism does not perceive science as objective, or the preferred route to truth especially when applied to areas outside of the physical sciences. Evidence based medicine (EBM) is by definition a modernist approach to improving care of individuals and choosing what medical modalities will be supported by the health care system in human medicine.

Killing Animals to Use Their Parts

In Fordist livestock production, the production system is also the creator of the animal. Almost all piglets and dairy calves are the result of artificial insemination and all poultry life starts at the incubator not the nest. Modern broiler chicken meat production bears very little resemblance to the natural increase in village chicken numbers. In the absence of human technical assistance modern breeds of chickens and turkeys could not reproduce at all.

These creation aspects of technical farming, such as heat detection and artificial insemination bring the farm worker in very close physical contact with the animal. Despite the human attachment to breeding animals, the creation of animals to produce products for human use is based on the mentalité that painless killing in the absence of anxiety or fear does not harm an animal. The phrase that death is not an animal welfare issue (Webster 1995) is considered self-evident by the farming community. Farmers agree with Tooley (1972) an organism possess a serious right to life only if it possesses the concept of a self as a continuing subject of experiences and other mental states, and believes that it is itself such a continuing entity.

Modernization of livestock production has provided many more millions of animals to the slaughter and processing for food complex than was possible before the modern era. Industrialization of agriculture is seen by some as an independent evil spawned by the most pernicious characteristics of free enterprise capitalism. However, industrialization of agriculture can also be explained by a more “natural” economic/technical evolution and need not be explained principally as the result of corporate takeovers, excessive profit taking and the disappearance of animal care values. Frazer (2005) has postulated that the emergence of refrigeration and efficient live animal transport by modern highways allowed for the concentrating of slaughter facilities and the development of effective oligopolies. An oligopoly in livestock production occurs when there are so few buyers of product (processing plants) that the buyer not the seller sets the prices for live animals. Oligopolies in meat production progressively decreased the profit margin of small producers who were forced out of business or to seek increasing economies of scale in order to remain in business.

Regardless of the cause, how we make things in modern agriculture systems dictates not only how we work (the quality of life of the farmer) but what we buy, how we think, and the way we live.
**Justified Killing**

There are several theoretical justifications for slaughter in animal agriculture in addition to; humans are smarter and chicken is tasty (Might makes Right). In the ecocentric view (Lund et al. 2004), animals are part of the agro-ecosystem, and just as in natural ecosystems, the surplus of the system should be harvested each year, if ecosystem sustainability is to be maintained. For the purpose of system balance, the human takes the role of the predator. Alternatively in the utilitarian application of an overall cost–benefit analysis; provided that the animals are not aware of their own future, and that they are slaughtered painlessly and unknowingly, the harm done to them can be considered less than all the other benefits to the animals and others gained by animal agriculture. Animals that can not plan for and anticipate a future are not harmed by the foreshortening of the future; they are not subjects-of-a-life and are not injured by humane killing.

The criterion for what constitutes a being that is the subject-of-a-life is however, hotly contested. Singer (1975) concludes that farming animals for meat is not an ethical problem if these animals lead a good life, are killed without suffering and are replaced by new individuals. The evil of killing animals at the end of their normal production cycle is mediated and becomes acceptable when animals have a good life before their death (Singer 1975). However, if the right to life is considered as an essential basis of all other rights, animal agriculture remains an ethical problem. From this viewpoint, killing is unacceptable even though no foreknowledge or pain is involved.

The companion animal shelter movement, the backbone of most SPCA organizations is based on the popularity and public support for the saving (prolonging) of animal life. The common terminology is rehoming or rescue, stressing the concept that surplus companion animals have a claim on society, a claim to be rescued from death. Shelter veterinary medicine has emerged as a unique specialty dealing with the problems related to uncontrolled companion animal reproduction (Burns 2006). The Association of Shelter Veterinarians was originally founded as a grass roots group of several dozen shelter veterinarians looking for a means to network. It has now grown into a formal organization consisting of over 600 member veterinarians and 11 student chapters from all around the globe.

Western society has developed social and philosophical techniques to separate how we think about food animals from the social space we give to companion animals.

**Killing Food Animals in Practice**

The last 15 years has been an extraordinary period of farm animal killing for reasons other than the “natural” cycle of producing food for human consumption. Between April 1996 and October 2001, 5,196,274 cattle were killed and destroyed in Britain due to the Over Thirty Month Scheme to control BSE. Also in Britain, as part of the FMD outbreak in 2000 4,230,786 primarily cattle and sheep were killed. To prevent H5N1, bird flu spilling into the human population in December 1997, Hong Kong veterinary authorities emptied the wet bird markets and eliminated 1.4 million geese, ducks, chickens, quail and other birds. A major epidemic of type O FMD in Taiwan (China) in 1997 caused the immediate death of some 184,000 pigs and a further 3.85 million pigs were slaughtered as part of the eradication campaign. The 1997-98 Hog Cholera (CSF) outbreaks in the Netherlands resulted in the deaths of 12 million mostly disease free pigs.

In April 1997 in the Netherlands CSF outbreak the volume of suspect pig meat from disease control and animal welfare operations was exceeding the rendering capacity of the country. Because this problem could be alleviated to some extent by buying-out piglets born in the movement restriction areas shortly after birth instead of at a weight of 25 kg, a scheme for buying-out 3 to 17-day-old piglets was introduced in May 1997. Veterinarians working under contract to the Government killed these young piglets on the farm by lethal injection. Farmer participation in this operation was officially on a voluntary basis, but farmers who declined to participate were not allowed to sell piglets of 25 kg to the buying-out scheme at a later date. The Royal Netherlands Veterinary Association (RNVA), farmers and farming organizations opposed this arrangement on welfare and moral grounds (Pluimers et al, 1999).

More recently in Manitoba, over 6000, 5kg, 21 day old piglets were killed due to seasonal market collapse in the absence of a catastrophic disease or other “natural” disaster (Whiting et al. 2010).

The practice of modern livestock farming appears to predispose livestock populations to massive killing where the use of the carcass for food is prohibited thereby removing the normal justification for the killing. The veterinary profession, by tradition of being seen as the expert and historic participation in the public service has been compelled to participate in these animal killings on a massive scale.

Crisis related to foreign animal disease incursions have changed the way governments in Europe consider what acceptable ways to deal with infected livestock are, and prudent management of at risk animals. Tovey (2002) suggested that postmodern influences in society make the policies of stamping out foreign animal disease untenable. This suggestion has also been supported by social survey research (Cohen 2007, 2009). People witnessing or experiencing the mass killing of healthy animals in disease.
control programs refer to the actions as “sin” conferring a high level of moral engagement in the question (Meijboom 2009).

Despite the wealth of experience and reports from Europe, animal welfare concerns consequential to foreign animal disease eradication are not widely discussed in the veterinary communities in North America tasked with this potential public service. This lack of preparedness may result in unavoidable public criticism of veterinarians as a profession and the official veterinary infrastructure if and when a foreign animal disease interrupts free trade in livestock in North America (Whiting 2008).

Killing in Shelters

The Humane Society of the United States (HSUS) estimates that approximately three to four million pets are killed yearly in shelters across the United States (HSUS 2010). The No Kill Advocacy Center and Alley Cat Allies claim this number is closer to five million (Anon 2010). In their statement on Feral cat Management, The Association of Shelter Veterinarians suggest “colonies should not be considered as an alternate lifestyle for owned or socialized cats, or a dumping ground for unwanted animals” (Sheltervet 2010).

Arluke (2003) in his review of the emergence of “no-kill” shelters in the United States and the social and philosophical rift generated between limited access and open access shelters, suggested that the no-kill movement is not primarily about limiting animal suffering. It is a movement related to personal identity; how the shelter workers identify themselves. Individuals do not volunteer at an animal rescue to kill animals. It is a human issue, human identity concept and the emotive tool of animal killing is used in the debate.

Similar to my farmer colleague killing his own cattle, the issue of shelter killing is at least partly about personal identity and perhaps not primarily about animal welfare.

The “Problem” of Feral Cats

The target standards of well being for farm animals for the past 50 years are based on the five freedoms from the Bramwell Report (Bramwell 1965). Applying this ethical template to the conditions of feral urban cats which exist because of available food from natural or human provided sources, no animal has a better life than the feral cat. The feral cat can free roam, feed, fight, and fornicate with near impunity. However, their lives usually are short in comparison to their neutered, isolated, protected and overfed cousins housed in the companionship of people.

In discussions with, writing, and websites of feral cat protection groups, the problem is complex, but not primarily one of the welfare of the individual feral cat. Most discourse revolves around cat population control. Most organizations advocate sterilization and return of the feral cat to its prior state, suggesting the welfare of the individual feral cat is at least acceptable. The problem is centered in 1) feral cat colonies generate hundreds of kittens that can turn up at local shelters and have to be killed or 2) the presence of feral populations encourages people to abandoned socialized cats. The evil to be avoided is the killing of surplus cats. Killing cats disrespects the cat as a sentient being and does not respect the value of the human animal bond with cats. It is possible that there is also a sense that feral cats are in a state that is “outside of nature” in that the correct social space of the cat is to be a companion to human animals. Spay neuter release programs are intended to eventually lead to the demise of the colony.

Reports of successful control of feral cat numbers are mixed. Reports from Florida have been promising (Hughes et al 2002) while 10 year study in Rome suggested spay/neuter campaigns brought about a general decrease in cat number but the percentage of cat immigration (due to abandonment and spontaneous arrival) is around 21%. This suggests that all these colony management efforts, without an effective education of people to control the reproduction of house cats (as prevention for abandonment) are a waste of money, time and energy (Natoli et al 2006). One would suspect a high risk of reporting bias of positive or promising results by researchers with a vested interest or working within the companion animal rescue culture.

The Case against Killing

Even if painless killing does not harm the healthy animal and therefore not a welfare issue, it may remain morally undesirable because of other collateral harms. If one believes that animals have a right to non-mortal intervention (Reagan 1983) killing is an ethical issue. Others have argued that biological life, “telos” has an inherent value (Rollin 1981). Or that killing represents a lack of virtue, care or respect for sentient beings (McMahan 1995). In these arguments killing is a moral issue if not an animal welfare issue. In addition, the killing of animals can certainly be a hardship on the people providing the killing (Reeve et al 2004, Rogelberg et al 2007)

Yeates (2009) constructs an argument to support the assertion that killing is also an animal-welfare issue as it leads to the exclusion of possible future relevant positive states. A similar argument as to the potential of the unborn foetus has not received strong support in parallel discourse in human medical ethics (Tooley 1972).
CONCLUSION

Animal killing is not primarily an issue about animal welfare although it is often portrayed as such in the media and in political discourse. The concern about animal killing is what the act of killing animals means in current social context. Animal killing is about people, our society and about the relationship of people to animals.

Animal killing can be experienced as a powerful moral issue, especially the killing of healthy animals for economic reasons when due to lack of resources in animal shelters or consequential to foreign animal disease outbreaks. These economic justifications for animal killing are becoming less credible with the general public. The veterinary profession will always be involved in the painless killing of animals, as we are the specialists in animal pain and its avoidance.

In part due to the risk to the public image of the profession, killing of animals should be and remain a primary area of concern for the profession and the profession should maintain a clear dialogue on this topic within the profession and to society at large.

REFERENCES


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There are four elements of emergency management:

1. **Prevention/Mitigation**: Preventing an emergency from happening or reducing the risk associated with an emergency situation.

2. **Preparedness**: Developing plans, procedures and policies for managing emergencies and providing regular training and the opportunity to participate in emergency exercises.

3. **Response**: Actions taken immediately before, during or directly after an emergency occurs.

4. **Recovery**: Efforts taken to repair and restore after an emergency.

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**1. Prevention**

**CFIA Roles and Responsibilities**

**Import Risk Assessment**

The Canadian Food Inspection Agency (CFIA) is responsible for the decision to allow or prohibit importation of animals, animal germplasm and animal-sourced products and by-products (referred to as commodities) because of the associated level of animal disease risk. The CFIA may establish specific conditions under which importation may proceed, e.g., testing with or without quarantine, in order to safeguard the Canadian animal health status.

To establish the above, an evaluation of the disease risk associated with imports must be conducted for the importation of a new species (animal or product) and/or the importation of an already assessed species from a new country. This applies to any commodity/country combination where the CFIA does not currently have import conditions.

The main tools used in this process consist of country assessment and hazard identification. Country assessments provide information on the current status of an exporting country for diseases which have been identified as hazards for the commodity in question, as well as information on the country’s import controls, surveillance procedures, and control measures. Hazard identification is completed for a species or animal product and identifies all the diseases of concern relative to that species/commodity that need to be considered when developing an import protocol to safeguard Canada’s animal population and health status.

**Import Controls**

Commodities are listed as either eligible for importation under certain pre-determined conditions which mitigate the risks associated with the introduction of Foreign Animal Diseases.
(FAD) into Canada, or they are ineligible. In most cases importers need to obtain documentation prior to importation, which could include applying for an import permit and/or obtaining official export documentation, issued by a veterinarian in the country of origin. Import permits not only provide for legal permission to import certain commodities, but also contain the conditions under which the commodity must be imported. Official export certificates are issued by veterinarians in exporting countries, and they certify that the commodity to be imported into Canada meets our import conditions as required.

Federal inspectors and/or veterinarians inspect imported commodities and/or their paper work upon arrival at the various ports and locations and determine whether the commodity may be released or refused. Canada Border Services Agency acts on the CFIA's behalf at ports of entry and refers commodities to CFIA for inspection. Some commodities are subject to further restrictions within Canada, e.g. further testing, quarantine. Commodities that do not meet our import requirements are either rejected until they can be brought into compliance, or outright refused. Commodities that are refused, must either be removed from Canada at the importer's expense or be destroyed at the importers expense.

**Biosecurity Standards**

In 2006, the CFIA established an Office of Animal Biosecurity (OAB) to develop and implement biosecurity strategies and standards for livestock, poultry and aquaculture production. The OAB works in close collaboration with industry groups, the animal health community, governments and academia. The CFIA also oversees animal biosecurity advisory councils comprising of representatives from industry, governments and the animal health community; identifies emerging risks and evolving biosecurity technologies; conducts biosecurity research and sets priorities; provides training on animal biosecurity programs and carries out biosecurity awareness and communications.

**Private Practitioner’s Roles and Responsibilities**

**Biosecurity**

Veterinarians who visit farms pose a risk of spreading disease organisms from one farm to another. By stringently practicing every day biosecurity measures, this risk can be mitigated to an acceptable level. These common, everyday measures include personal hygiene; wearing of clean protective outerwear and footwear; routine sanitation of equipment and instruments; routine sanitation of vehicles; proper storage and disposal of used consumable items; proper storage, cleaning and disinfection of reusable items; safe handling of laboratory specimens; planning and staging of farm visits, e.g. attending to sick animals after all healthy ones have been seen; and observing self quarantine if highly contagious diseases are suspected.

Practising veterinarians play a large role in the planning and implementation of disease prevention and biosecurity programs on the farm. Practitioners can review their clients’ plans to be sure they focus on the basic principles of biosecurity. Client education is an integral part of the practising veterinarian’s role. Producers rely on their veterinarian as a primary source of information. Biosecurity plans should address management of access, animal health and operation risks.

**Early Detection**

Veterinary practitioners may be the first responders to a suspected disease incident on the farm and provide surveillance for FADs. Early recognition and response is critical to human and animal health, food safety and the environment. It is important for veterinarians to maintain current knowledge of the foreign animal diseases most likely to enter Canada, be aware of clinical/necropsy findings which should alert suspicion, routinely include foreign animal diseases in differential diagnoses and immediately report suspected foreign animal disease to the nearest District Veterinarian. Once a firm suspicion is established, veterinarians should remain on the suspect premises until they are relieved by the District Veterinarian and encourage others not to leave the premises. During an outbreak, they should continue to refer suspicious calls to the CFIA.
2. Preparedness

CFIA Roles and Responsibilities

In emergency management, the important factors of success are:

- Having effective lines of communication, both internally and externally;
- Having in place emergency plans that are regularly exercised to develop trust and mutual agreement within the Agency and with federal departments, provincial and municipal governments and industry partners;
- Having emergency plans and a response effort that are an extension of the day-to-day roles and responsibilities of CFIA staff – in an emergency situation, any significant deviation from these roles may cause miscommunication and confusion;
- Having predicted decisions or policies made and procedures developed before an emergency occurs; and
- Having plans and employee services in place to ensure the health and safety of responders and sufficient surge capacity.

Emergency Plans

The CFIA has developed the following preparatory planning pieces:

CFIA Emergency Response Plan

This plan is a generic (all program areas) reference tool for CFIA staff that have a responsibility for emergency management. Depending on the nature of the emergency, the plan is designed to be used in conjunction with other CFIA Functional Plans, Hazard Specific Plans and Procedures. It describes the generic response structure and roles and responsibilities of responders. Also identified in this plan are the legal mandates under which the CFIA is required to respond to emergencies and the stakeholders with which CFIA works collaboratively in response. CFIA’s emergency response team structures are based on the principles of the Incident Command System (ICS). ICS is the combination of facilities, equipment, personnel, procedures and communications operating within a common organizational structure and is designed to assist with the overall emergency response. Its objective is to maximize team efficiency and minimize disruption to normal operating policies and procedures and it can be described as a “function” oriented approach to an emergency. ICS can be used for a wide range of emergencies, from small to complex incidents, and is capable of expanding or contracting to meet the dynamic needs of any situation. ICS training is required to ensure that all who may become involved in an incident are familiar with the principles of ICS. Several international, federal, provincial/territorial and industry organizations have developed emergency response systems based on the principles of ICS.

Animal Health Functional Plan (AHFP)

The CFIA is mandated by Health of Animals Act and other federal legislation to respond to incursions of FADs into Canada. Outbreaks of the most serious of these diseases could cost Canada’s livestock industry billions of dollars in lost production, the loss of international markets through export embargoes, extensive collateral damage, and the costs of control and response activities. As well, there could be a potential threat to the safe supply of certain food commodities.

The AHFP describes the division of labour and responsibilities between the Field, Regional, Area, and National levels to prepare for and respond to animal health incidents. The objectives of the AHFP are to outline the CFIA emergency response program and describe the process for all phases of the incident. This includes defining the responsibilities for preparedness for managers and responders, and defining the structure and responsibilities of the various response units (e.g. destruction, disposal, decontamination) within the CFIA during a FAD incident.

Hazard Specific Plans (HSP)

The HSP outlines the response to be undertaken by the CFIA when there is suspicion of a FAD or when an outbreak occurs. Examples of diseases for which HSPs have been developed include foot and mouth disease and notifiable avian influenza. These documents provide an overview of the disease, authorities and principles of control, and information on initial investigation of the premises, identification of exposed premises, tracing/surveillance, movement control, destruction and disposal, decontamination, zoning, and Proof of Freedom requirements.

Procedures

Several procedures have been developed to provide instructions for CFIA responders to carry out specific tasks related to disease eradication activities. They are validated in field training sessions as opportunity arises. Examples of these procedures are in-barn gassing of poultry and composting of carcasses.

Foreign Animal Disease Emergency Support (FADES) Plans

To be truly effective, emergency response involves collaboration with other federal and provincial departments, municipalities, stakeholders, and public and international agencies. Provincial, territorial, and municipal governments take lead roles in supporting
the CFIA action against FAD outbreaks within their boundaries. All provinces have FADES Plan agreements with the CFIA that set out the roles and responsibilities of provincial and local agencies during a FAD outbreak. Chief Provincial veterinarians, in their respective ministries of agriculture, generally coordinate the activities of their animal health network at the provincial level in close cooperation with the CFIA and industry associations. There are also provincial emergency measures organizations that are responsible for incident response within their provincial jurisdictions for a broad range of natural, man-made, and epidemiological disasters.

The CFIA works with all stakeholders (e.g., livestock industry, veterinary associations, academia, and agriculture services industry) involved in a FAD outbreak. The CFIA will look to industry for technical assistance, cooperation in response measures, advice, and support during an incident response.

**Traceability**

Traceability provides the ability to determine the origin and destinations of animals and therefore the source and spread of an outbreak. Traceability is not new to Canadian agriculture. Tags, tattoos, brands and paper-based logbooks are all elements of traceability that have been employed for many years by both industry and government. There are three main pillars to traceability systems: the identification of animals or products, the ability to follow their movement, and the identification of departure and destination premises. Many industry sectors in Canada have solid traceability systems that have proven to be useful in emergency management.

Regulated animal identification programs currently exist for the beef cattle, dairy cattle, bison, and sheep sectors. The Canadian Cattle Identification Program (CCIP) was introduced in 2001 through an amendment to the Health of Animals Regulations and is applicable to all cattle and bison. The Canadian Sheep Identification Program (CSIP) followed in 2004. Both programs are mandatory in all provinces. Under the CCIP and the CSIP, all bovine, bison and ovine animals must bear a registered ID tag before they leave their farm of origin. Information on cattle, bison and sheep from most provinces is stored in a central database. In the event of a disease outbreak or food safety emergency, the origins of tagged animals involved can be accessed by the CFIA.

Recognizing industry’s leadership and foresight in building the foundation for livestock traceability, federal, provincial and territorial agriculture ministers have committed to phasing-in a National Agriculture and Food Traceability System (NAFTS), beginning with livestock and poultry. Industries and governments in Canada are working together to develop the NAFTS - a secure, sustainable and value-driven system that will provide timely and relevant traceability information that enhances emergency response and market competitiveness in a manner that best serves industries and governments.

**Surveillance and Detection**

Animal disease surveillance supports Canada’s ability to recognize and deal with emerging animal disease problems. Surveillance also plays an important role in providing Canadian livestock and poultry products access to more markets. The CFIA’s surveillance activities are supported by a nationwide network known as the Canadian Animal Health Surveillance Network (CAHSN), which draws on the disease detection capabilities of practicing veterinarians, provincial and university diagnostic laboratories and the federal government.

**CVR Roles and Responsibilities**

The Canadian Veterinary Reserve (CVR) is a joint initiative of the Canadian Veterinary Medical Association (CVMA) and the CFIA. As part of a National Emergency Management Policy and Plan, the CVR will assist governments in responding to animal health emergencies such as disease outbreaks or natural disasters by providing teams of trained private sector veterinarians ready to be assembled and deployed on short notice to areas in need for response and recovery.

In general, governments lead the response in large-scale emergencies and disasters. In Canada, as in most countries, the government (federal, provincial or territorial) may not have sufficient numbers of veterinarians on staff to deal with an animal health crisis of very large proportion. Governments maintain a base of resources that can respond to the most likely occurrences and look to other sources to assist on an “as and when needed” basis (referred to as surge capacity). The CVR has been established to provide this veterinary medical surge capacity in Canada and in the future internationally.

At the request of the federal government and/or provincial/territorial government(s), the CVR may respond to: FAD outbreaks (emergency response led by the CFIA), and natural or man-made disasters referred to as civil emergencies, affecting the health and well-being of large numbers of animals (emergency response usually led by the affected province or territory). Duties of reservists in a FAD response may include field diagnoses, sample collection/submission, quarantine, disposal, biosecurity, decontamination and expert witness in legal proceedings. The establishment of the CVR means that Canada is much better prepared to respond to an emergency in the future. Having the ability to mobilize a much larger veterinary force provides the surge capacity to effectively combat and control the spread of animal disease and to treat animals in distress.
Becoming a Member

CVR members must be veterinarians with: a Doctor of Veterinary Medicine (DVM) or equivalent degree, and a Certificate of Qualification, or must have been previously recognized for general practice by the CVMA, and a provincial general practice license. CVR members should be willing to commit up to 5 days of training per year plus up to 3 weeks duty anywhere in Canada when a crisis occurs. CVR members accept that they may be called to service on very short notice and are expected to be available to serve promptly when called (e.g. within 24-72 hours). Depending on the nature and extent of the emergency, there may be staggered response levels that allow for first responders and subsequent relief teams. In prolonged emergencies, Reservists may be invited (but are not obligated) to return for an additional 3-week cycle of duty after having had a sufficient rest break to preserve their health and well-being. At present they could be deployed anywhere in Canada. In the future, there may also be the opportunity to serve internationally.

Assignment will depend on the specialty of the Reservist and his/her experience with, and training in, particular types of emergencies. Within the CVR membership, veterinary specialties include small and large animal veterinarians, food animal veterinarians, specialists in exotics, epidemiology, etc. In addition to the qualifications required to be a CVR member, experience with food producing animals, in working with governments, previous training in emergency response, knowledge of foreign animal diseases and special diagnostic skills will be assets. Possession of a federal government security clearance certificate and a health certificate will also be required.

Training

The FAD training program is currently offered over a 5-day period and includes orientation sessions on working for the Government of Canada and the CFIA, as well as training in emergency preparedness, specific foreign animal diseases, biosecurity and biocontainment and the duties of veterinary inspectors in emergency response. The training session closes with a tabletop exercise to practice lessons learned during the week.

Private Practitioner’s Roles and Responsibilities

Traceability

Livestock identification is a cornerstone on which the National Animal Health Program is based. The ability to accurately identify and trace the movement of livestock cannot be overstated. It is essential to maintaining and ensuring a healthy livestock population. Industry leaders including private practitioners recognize and communicate the importance of safeguarding our national herd and assuring consumer confidence at home and in our export markets.

Surveillance and Reporting

Canada is one of a few countries which remain free from a number of serious epizootic diseases of animals. It is a high priority of the CFIA that FADs, especially rapidly spreading diseases like Foot and Mouth Disease, be recognized and eradicated as soon as possible. The consequences will depend on the size and nature of the outbreak, and can be greatly minimized by early identification, containment and elimination. Veterinary practitioners are most likely to be the first to encounter and suspect a FAD once it has gained entry into Canada. Early recognition by veterinarians may prevent widespread transmission and great expense to the Canadian public.

FADs of concern to the CFIA are those which are zoonotic or would have severe economic consequences in Canada, primarily associated with the loss of our export markets. It is essential to be aware of the possibility of FADs. The spectrum of pathogenicity of FADs has changed significantly. Traditional expectations of dramatic clinical manifestations of FADs in our highly susceptible livestock must be questioned. Changes in pathogenicity induced by accidental release of modified strains, or alterations induced by passage through partially immune hosts have resulted in a generation of agents whose clinical signs could closely mimic common diseases of Canadian livestock.

The challenge for the clinician then becomes - when do I refer a case to the CFIA District Veterinarian? This must remain the judgement of the attending clinician; however, there are guidelines which may be useful. Firstly, a history of a possible recent contact, such as visitors or people or livestock returning from abroad, should raise suspicions. Secondly, a syndrome which does not follow expected clinical or treatment and response patterns should also be questioned. During the last 30 years, outbreaks of Hog Cholera, Anaplasmosis, Newcastle Disease, and Bluetongue have all occurred in Canada. Although clinicians are unlikely to encounter such diseases, the possibility must always be considered.
The following examples may be useful reminders of some of these:

- Hemolytic anemia with no hemoglobinuria, affecting adult cattle - consider Anaplasmosis.
- Mature cattle affected with oral lesions and diarrhea, morbidity and mortality high or low - consider rinderpest.
- Pigs with severe systemic illness, morbidity high or low and increasing (insidious) - keep the possibility of African Swine Fever and Hog Cholera in mind.
- Reproductive problems in sows - always include Pseudorabies, Hog Cholera and African Swine Fever, at least in initial list of rule-outs.
- Horse with vesicles or papules on tongue - definitely call the CFIA on suspicion of Vesicular Stomatitis.
- Several bred mares return to heat with mucopurulent vaginal discharge; cultures are negative - search in breeding/travel history for possibility of Contagious Equine Metritis.
- Sheep with stomatitis, lameness - suspect Bluetongue or Vesicular Diseases.
- Poultry with depression, neurological signs, head edema, diarrhea, variable morbidity and mortality, hemorrhagic enteritis - consider Newcastle Disease, Avian Influenza or possibly Fowl Typhoid. If restricted to chicks and poults - consider Pullorum Disease.

You can request printed material from your District Office, to keep updated on clinical signs and post-mortem findings of serious Foreign Animal Diseases.

Veterinarians are required by law to immediately notify the District Veterinarian of reasonable suspicion of any serious FAD, regardless of whether it is reportable. Once a firm suspicion is established, it is important that the practitioner remain on the suspect premises until relieved by the CFIA Veterinarian. The danger of disease transmission by veterinarians must be recognized along with the potentially tragic consequences and possible liability to the veterinarian should such an incident occur. Individuals should maintain a list of alternative contacts, in case you are unable to reach your local District Veterinarian (e.g. neighbouring District Veterinarian, Area Office Personnel).

3. Response

CFIA Roles and Responsibilities

The Health of Animals Act ("Act") addresses diseases and toxic substances that may affect animals or be transmitted by animals to persons, and the protection of animals. The purpose of the Act and the Health of Animals Regulations is to prevent the introduction and spread of communicable diseases in Canada. The Act provides, among other things, for the powers over the importation and exportation of animals, the control of infected places, and the regulation of animal transportation.

CFIA veterinary inspectors, inspectors and enforcement officers designated under the Health of Animals Act may enter premises, open receptacles or things, require presentation of animals for inspection, examine any animal or thing, require production of documents, conduct tests or analyses, seize and detain animals and enter a dwelling place with a warrant.

CONTROL ACTIVITIES:

Movement Controls

For highly contagious diseases, spread can only be prevented by rapid destruction of affected animals and strict movement control measures. All epidemiologically linked premises must be quarantined as suspect infected premises and subject to strict movement controls. A Control Area may be declared to enforce movement control, to establish infected zones as well as to define disease free zones within Canada.

Declaration of Infected Place

The objective of a Declaration of Infected Place is to prevent any further disease spread. Upon declaration of infected place status (quarantine), the veterinary inspector will conduct a census of all susceptible animals; inventory all animal products, animal by-products, manure and animal feed and bedding; impose movement restriction on all susceptible animals to or from the premises; require appropriate disinfection at entrances and exits of buildings and security at the entrance of the premises; ensure biosecurity at the gate of the infected premises; order dogs, cats and other non-susceptible potential mechanical vectors be confined; advise owners of adjacent premises to keep susceptible animals away from the perimeter of the declared infected place; order vehicles and equipment be cleaned and disinfected prior to leaving the declared infected place; prevent effluent from draining onto roads, pastures or watercourses; and implement vermin control, feral animal control or wildlife control measures if warranted.
Conspicuous signage indicating the presence of a FAD may be posted at the farm entrance(s) along with the biosecurity provisions including facilities for cleaning. Vehicles, clothes, footwear and things worn or carried by any person entering or leaving a premises affected or suspected of being affected with a communicable disease are ordered to be cleaned and disinfected.

Prior to the Ministerial declaration to define a control area, a general provision exists to individually declare infected places, all premises with susceptible species within 5 km of a premises where a disease has been confirmed. This infected place declaration is an interim measure until control area legislation is enacted and movement control zones defined.

Declaration of a Control Area

Epidemiological assessment of the estimated extent of the outbreak will be factored into the determination of the limits of the control area. Tracing of movements of potentially exposed animals and locating all infected and potentially infected herds will be conducted until the extent of the outbreak can be determined. The initial restriction of movement and conditions on places, risk goods, conveyances and risk activities imposed are prescribed by a Control Area Notice. The standstill will apply until restrictions and conditions can be focused on infected and at-risk places. Some conditions on risk activities will continue at a national level until the declaration of freedom.

Disease Investigation

Tracing investigations include those epidemiologically linked to the infected place. Movement of animals from the infected place (trace-out) since the estimated introduction of the FAD as well as movement of animals into the infected place (trace-in) for a critical period before the estimated first case must be investigated. The critical period is the epidemiologically significant period for tracing purposes on confirmed positive premises, generally the period from the estimated date of introduction or the maximum incubation period before the onset of clinical signs. Priority must be given to animal movements, although the possibility of contaminated fomites such as transport vehicles and human traffic must also be investigated.

RESPONSE ACTIVITIES:

Destruction

Clinically affected animals on infected places will have destruction priority to eliminate virus multiplication. All known exposed susceptible livestock may also be ordered destroyed. Unexposed susceptible animals on an infected place may also be destroyed. Pre-emptive slaughter is the destruction of susceptible animal species in herds on premises which have been exposed to infection by direct animal-to-animal contact, or by indirect contact of a kind likely to cause disease transmission. The Act provides for destruction and disposal of animals or things known to be infected or suspected of being infected and contacts with animals or things known to be infected or suspected of being infected or known to be a vector or suspected of being a vector of a disease.

Disposal

The disposal of carcasses, animal products, feed and manure (where applicable) is carried out with consideration for the environment which is under provincial jurisdiction in Canada. Different disposal methods may be available but consideration is given to those that will minimize both the risk of disease spread and environmental impact. A disposal decision tree is available that considers these two important parameters and work is ongoing between CFIA and provincial environmental authorities to determine optimal local practices.

Decontamination

The persistence of the disease agent in the environment must be considered. Confirmed infected places, vehicles and equipment must be thoroughly cleaned and disinfected. Organic matter may prevent the action of disinfectants; therefore, cleaning before disinfection is critical. If disinfection cannot be achieved effectively and quickly, then contaminated materials and equipment should be destroyed. Animal fluids and excreta should be treated to eliminate infectious virus or buried, incinerated or composted. Cleaning and disinfection (C&D) should be according to internationally accepted standards and is carried out under official supervision according to official instructions.

CVR Roles and Responsibilities

Activation

Since response to FAD in Canada is led by the CFIA, the CFIA would make an official request to the CVMA to mobilize the CVR, indicating the number of Reservists they require and the areas of expertise needed (e.g. beef, dairy, pork, poultry etc.). Reservists who are called to serve in a FAD response become temporary employees of the CFIA, would be designated as Veterinary Inspectors under the Health of Animals Act and are paid by the CFIA for their services. Practice/Business Continuity Compensation has been put in place for this purpose and is available to non-government employers that provide staff for CFIA-led FAD emergencies.
Private Practitioner’s Roles and Responsibilities

In the case of an outbreak of a FAD, a predetermined Emergency Response Team would be mobilized to control the spread and eradicate the disease. Operationally, this Team is made of units having very specific tasks to do such as diagnostics, trace-out, movement control, evaluation, destruction and disposal, and decontamination. Veterinary practitioners could be requested to give assistance in one of these areas.

During an outbreak, practitioners receiving information suggestive of the FAD in question would notify the CFIA in the outbreak area. In the case of a FAD emergency, appropriate information including CFIA contact information, the limits of the control area, movement restrictions, and disinfection procedures would be made available to all practitioners through the appropriate channels. The prescribed personal cleaning and disinfection protocol must be strictly followed. Provision exists under the Act to recover costs incurred in taking measures in respect of a control area from persons who through their fault or negligence caused or contributed to the spread of disease.

Client education is an integral part of the practising veterinarian’s role in FAD prevention and control. Owners will turn to their veterinarian as a primary source of information in the event of an outbreak. Control procedures such as disease reporting, quarantine and disinfection will be effective only with the element of owner co-operation and participation. This results from an understanding of the procedures and their rationale.

4. Recovery

Recovery includes all efforts focused on returning to pre-emergency state, if possible.

CFIA Roles and Responsibilities

Evaluation and Compensation

Efforts are focused on finalizing compensation payments to owners whose animals and things have been ordered destroyed as quickly as possible. Compensation payable is equal to the market value of the animals and things at the time of evaluation but it shall not exceed the maximum amount specified in the Regulations. Compensation paid to owners does not include production losses associated with the destruction of the animal (e.g. egg or milk production, costs associated with farm vacancies due to downtime or procurement difficulties, costs of “Cleaning and Disinfection” of infected premises).

Agriculture and Agri-Food Canada business risk management programs may be more appropriate for losses due to business disruption or for one time disease related costs.

Post Outbreak Surveillance

Post-outbreak surveillance is undertaken to validate the response activities and demonstrate to our trading partners that disease-free status has been regained. This is facilitated by the presence of the CAHSN (currently for NAI, Newcastle disease and Foot and Mouth Disease). The post-outbreak surveillance regime is designed according to the epidemiology of the disease, the nature of the outbreak, scientific principles of surveillance and World Organization for Animal Health (OIE) recommendations.

Restoring Market Access

Canada’s primary policy regarding FADs is stamping-out in order to achieve the requirements for regaining disease-free status as defined in the Terrestrial Animal Health Code of the OIE. These requirements are different for each disease. For example, for NAI, disease free status can be re-established three months after stamping-out operations (including cleaning and disinfection), provided that surveillance has been carried out during that three month period.

Negotiations with our trading partners to achieve recognition of regained disease-free status involve collaborations between CFIA, AAFC and the Department of Foreign Affairs and International Trade (DFAIT). The Import/Export section of CFIA will establish priorities according to the industry’s markets, develop strategies to keep or reopen markets, and negotiate export certificates in accordance with the new criteria.
Recognition of zoning or compartmentalization may be negotiated by Canada during an outbreak.

**Demobilization**

Demobilization is the gradual return of human and material resources to pre-crisis status. This is a process of demobilizing emergency response teams and closing of activated emergency operations centres according to the needs at various levels. Movement and other controls are lifted from all the different disease control zones as authorized in writing by the Minister.
Biosecurity Essentials for All Practice Types in the Face of a Pandemic

Keith Campbell, DVM

No Paper Available
Summary

The Canadian Veterinary Reserve (CVR) was created as an initiative of the CVMA and supported and funded by the Canadian Food Inspection Agency (CFIA) in October 2006, to respond to major emergencies involving animals. The presentation will outline what the CVR is, how private veterinary practitioners across Canada participate, and how Reservists are deployed when there is a large scale emergency. Major animal emergency scenarios and the CVR’s response plans are described.

CVR Definitions

1) Emergency
An abnormal event that:

(a) Impacts directly on animals, and directly or indirectly on humans.

(b) Requires additional resources (“surge capacity”) over and above those which are normally available for emergency response.

2) Foreign Animal Disease (FAD)
A federally reportable animal disease not normally found in Canada. An FAD outbreak has severe animal health, economic and international trade consequences, and sometimes serious implications for human health.

3) Civil Emergency
A natural or manmade disaster involving animals, people, or both. Man-made disasters can be accidental, or intentional.

4) “All Hazards” Approach
An approach to Emergency Management (Planning and Response) that involves both FAD’s and Civil Emergencies.

5) “First Responder”
The person(s), groups, or government departments / agencies initially on site during an emergency (or shortly thereafter), and often responsible for management of that emergency.

1.0 Introduction — What is the CVR, and Why Do We Need It?

The first decade of the 21st century has already seen its share of large scale animal emergencies. Events such as Foot and Mouth Disease in the UK in 2001, Avian Influenza in BC in 2004, and Hurricane Katrina in 2005 are familiar to all of us. In that context, CVMA has recognized that, as a profession, Canada’s veterinarians need to demonstrate due diligence and social responsibility by being ready to help in the response to future FAD outbreaks, or civil emergencies. Consequently, in December 2005, CVMA produced an Emergency Plan. A major component of that plan is the Canadian Veterinary Reserve (CVR).

The CVR is a pool of trained non-government veterinarians ready to assist first responders in large scale animal emergencies. In FAD outbreaks or civil emergencies, the urgent requirement for human resources can sometimes exceed what governments and government agencies can be realistically expected to provide. The CVR, therefore, offers an invaluable “surge capacity” to the people “on the ground” so that animal emergencies can be dealt with as quickly, effectively, and completely as possible. If the crisis is prolonged, government first responders also require relief and rest.

CVMA vetted the concept of the CVR at a workshop in Ottawa in March 2006. From that two day meeting of Canada’s veterinary and animal health leaders came strong agreement on the three founding principles of the CVR:

1. The CVR is valuable and necessary.
2. The CVMA should be the home of the CVR.
3. Because the CVR provides a public good, it should be funded by the public purse.

In the spring and summer of 2006, CVMA immediately proceeded to identify a funding partner for the CVR, and develop plans for how the CVR would operate. The Canadian Food Inspection Agency (CFIA), which has a clear mandate for dealing with reportable FADs, generously stepped forward to provide the fiscal resources to get the CVR up and running. CVMA simply...
does not have the dollars to finance a project as large as the CVR, so without CFIA, the CVR would not be possible.

November 2006 marked the official birth of the CVR. Recruitment of CVR Members began soon after. The response was excellent and by December 2009, over 400 veterinarians from all across Canada had applied to the CVR. Five successful FAD training sessions have been held, 4 in Winnipeg at the National Centre for Foreign Animal Disease, and one in Ottawa at the Fallowfield Laboratory. To date, 200 Reservists have received initial FAD training.

The CVR has adopted an “all hazards” approach in developing the CVR’s veterinary surge capacity. The objective is to develop and maintain a trained veterinary medical capacity to respond to any and all emergencies affecting animal health and welfare.

1.1 CVR Structure and Management:

The CVMA Council and Executive are responsible for the overall direction and policy of the CVR. The CVR Secretariat is responsible for day-to-day management and ongoing development of the CVR. The Clinicians Working Group is a select group of private practice veterinarians who are engaged as needed to help the Secretariat operationalize the activities of the CVR. The CVR Advisory Board and its sub-committees consist of various animal health stakeholders who provide their expertise and advice to the CVR Secretariat and the CVMA Council and Executive.

CVR Members must have a general practice license. Reservists are trained CVR Members. Most Reservists are veterinarians with private practice experience and others are from Canada’s veterinary colleges, or other non-government types of practice. So far, Reservists have been trained by the CFIA, and are paid by the CFIA for service time during travel, training and deployment. Travel, accommodations and food expenses are also covered. The CVMA pays practice continuity compensation to those qualified and continuing education (CE) credits are recognized for Reservists who undertake the CFIA-led training. Full details in regard to all compensation and benefits are included in the Frequently Asked Questions section of the CVR on the CVMA website.

Foreign Animal Disease - The CVR & CFIA

The CFIA is the lead authority for the monitoring, control and eradication of FADs in Canada. The “Big Seven” diseases below are those which the CFIA has identified as having the highest likelihood of occurrence and/or potential negative impact in Canada.

- Highly Pathogenic Avian Influenza
- Newcastle Disease
- Foot & Mouth Disease
- African Swine Disease
- Classical Swine Fever
- Swine Vesicular Disease
- Vesicular Stomatitis

The CFIA has developed up to date science based strategies to deal appropriately with FADs if one is found in Canada. These strategies include organized procedures, structures and resource management that lead to early detection of the disease, prediction of the likely spread, containment, targeted control and elimination with subsequent re-establishment of verifiable freedom from infection. The CFIA works in accordance with the Animal Health Code of OIE (World Organization for Animal Health).
2.1 The CFIA CVR FAD Training Process

Reservists need to be trained in advance so that they can respond rapidly and be more effective. CFIA FAD strategies and many other aspects of working with the CFIA are covered in detail at the CFIA-led FAD training sessions for CVR members. In 2010, we expect a single training session to be held in the fall in Ottawa.

2.1.1 Selection and “Pre-Training Requirements”

Priority for selection for FAD training has been given to veterinarians who have food animal experience. As well, FAD Training is now being made available to companion animal veterinarians. The “Pre-Training” process can be time consuming and requires a lot of lead time so you will be notified well in advance. It is important to act quickly once you have been asked to participate in training. When you confirm your participation is when the process begins. Once selected you can expect the following steps:

1. **Security Clearance**: To qualify for CFIA training, you must pass a security clearance as required by the CFIA.

2. **Health Exam and Health Certificate**: To qualify for CFIA training, you must have a health exam administered by your physician according to the requirements set by the CFIA (various forms are provided by CFIA that the physician must complete).

3. **CFIA “Hiring”**: With security and health clearance completed, you will then receive a CFIA hiring package of forms which, upon completion by you and approval by CFIA, will qualify you as a CFIA “as and when needed” employee. If called to serve, you would be considered an employee of CFIA during the time served, with authorities, rights and benefits of “as and when needed” employees.

2.1.2 CFIA CVR FAD “In Person” Training:

An applicant is eligible for CFIA training once all of the above steps are complete.

Training consists of 4 days of intensive classroom training. The CFIA brings in its senior training personnel and Human Resources staff to ensure CVR members receive excellent training. Topics covered range from Federal Legislation to Virus Biology to the use of Personal Protective Equipment. A Table Top Simulation Exercise is also included. Reservists have been very pleased with quality and scope of the FAD training.

2.2 CVR Activation Protocol FAD Response

The CVMA maintains a continually updated database of member contact information and training status to ensure trained Reservists can be contacted and deployed rapidly in the event of an emergency call up. Trained Reservists are expected to respond positively to a call up request by advising the CVR immediately of their availability to serve.

The CFIA would notify the CVMA of a need to put Reservists on alert or call them up. The CFIA would determine what exactly is needed in terms of numbers, the timing of when they should arrive, and where they are to be deployed. The CVMA would work through its CVR database to contact Reservists and create a list that meets the needs of the CFIA. The CVMA would work with Reservists to ensure they have all the information they need to deploy (including travel etc.) The Reservists would deploy from their home bases and arrive on location where they would immediately work under the direction of the CFIA.

Reservist Roles in an FAD Response could include Forming Teams with CFIA staff; Collection of Initial Epidemiological Information on-site; Diagnostic Investigation (Responding to sick calls, clinical exams, sample collection); Visits to Contact Premises; Surveillance Activities; Euthanasia and Carcass Disposal.
3.0 Civil Emergencies in Canada

Civil Emergency management and public safety responsibilities in Canada are shared by federal, provincial, and territorial governments and their partners, including individual citizens who have a responsibility to be prepared for disasters. Under Canada’s constitution, however, the provinces and territories have lead responsibility for civil emergency management in their respective jurisdictions. Therefore, most civil emergencies in Canada are local in nature and are managed by the municipality or at the provincial or territorial level. Each province and territory has its own emergency preparedness plans and has an emergency measures coordinating office.

A federal government lead response in civil emergencies is limited to some very specific circumstances, such as matters of national security, or events which overwhelm the capacity of a single province’s ability to respond. The federal government also leads development of the emergency management framework by working in partnership with provincial and territorial governments and others to establish an effective coordinating structure for civil emergency management in Canada.

“Other animal diseases” (non Reportable) are included under civil emergencies. These include diseases which may be considered an emergency at the provincial level but may not be ones for which the CFIA is responsible. Other characteristics of civil emergency management in Canada are:

- **Multiple stakeholders:** Responders could include government departments, pet owners, farmers, industry, fire and police departments, the CVR, local veterinarians, veterinary colleges, veterinary associations, SPCAs, and other non-governmental organizations.

- **Principles of cooperation:** Collaboration, coordination and integration are key requirements to ensure the most effective use of resources and execution of activities.

- **Advance Planning:** Emergency response plans are necessary to ensure the activities of multiple responders are effectively coordinated.

- **The Incident Command System:** It is the proven structured, yet flexible, management system which holds an emergency response together.

Unlike the very clear role the CFIA has in regard to FAD emergency response, there is no existing national model or mandated regulator in Canada that addresses the needs of animals in civil emergencies. To be effective, therefore, the CVR needs to develop relationships not only with the emergency measures organizations of the provinces and territories, but also with federal agencies such as Public Safety and Public Health Agency of Canada.

3.1 CVR Civil Emergency Scenarios

The CVR has contemplated a multitude of possible civil emergency scenarios.

Provincial Veterinary Officers also were polled on what they thought were the events with the highest risk of occurrence as well as the highest potential negative impact on animals. This is their list...

- Severe protracted summer / winter storms
- Forest fires
- Traffic accident involving one or more transport trucks, trains or planes carrying animals
- Event in Canada resulting in a comprehensive USA-Canada border closure
- Environmental Incident
- Acts of Terrorism
- Earthquakes, Tornadoes

The magnitude of any of these events can range from small to massive...for example the recent earthquake in Haiti hit in its most populated region with tragic results.

Hurricane Katrina in 2005 taught us many lessons, and led to what is regarded as the largest animal rescue operation in history. More than 250,000 pets were left stranded by the storm’s destruction. Owners, expecting to return a few days later, left food and water for their pets. But days turned into weeks, and pets had to struggle to survive without supplies and the care of their owners. In some cases, because of the strength of the human animal bond, many people were reluctant to evacuate without their pets, resulting in subsequent loss of human life.
3.2 CVR Training for Civil Emergencies - PROPOSED

Civil Emergency Training will include a combination of workshops, web-based learning and exercise simulations (both tabletop and live). Requirements for CVR civil emergencies training include:

- Emergency Management Model (general understanding)
- Incident Command System (general understanding)
- Provincial or territorial mandates and roles
- Federal role
- CVR call up and communications process
- Animal Health Emergency Teams role
- Field and medical triage protocols, V-START techniques and coding systems
- Personal Protective Equipment, disinfection, decontamination of patients, bio-containment
- Hazardous materials and PH risks (+ zoonoses)
- Euthanasia and disposal
- Simulation exercises

3.3 CVR Activation Protocol For Civil Emergency Response: - PROPOSED

The CVR will only respond if requested by the federal, provincial / territorial or municipal government first responder authority. As with the CFIA model, the CVMA confirms the requirements (numbers, training and expertise, timing, etc.) with the requestor and selects from the CVR database those Reservists that meet the criteria. An urgent email is sent to all selected Reservists informing them of the nature of the emergency, what is expected of them and asking them to respond to the CVMA with their availability. From there, the CVMA assembles the response group or teams.

The CVMA is responsible for all arrangements up to and including Reservists arriving at the emergency location.

Once on site at the emergency, Reservists will work in Animal Health Emergency Response Teams (AHERT) under the direction of the Incident Commander or their designate. In an ideal scenario, AHERT teams will include veterinarians, veterinary technologists and technicians, and non-governmental organizations. Triage and medical care are those functions in which veterinarians are most likely to play a role.

Other key functions during a civil response could include Advance Needs Assessment; Animal Search and Rescue; Evacuation (if required); Identification and Tracking; Animal Housing, Care and Feeding; Disease Prevention (because of exposure, co-mingling, and stress); Recovery & Rehabilitation; Veterinary Advice on Public Health Issues (zoonoses, carcass disposal) and Euthanasia.

It is important to note again that this is a concept and that the civil emergency aspect of the CVR is still under development as we seek various funding opportunities to help us further develop this aspect of the Reserve.

4.0 Current and Future Developments for the CVR

Our first CVR goal, developing a robust FAD response capacity, is progressing well. The challenge in this era of budget restrictions is to maintain training levels and provide ongoing refresher training. The CVMA is working with the CFIA, and other stakeholders to develop effective, low cost training modules for Reservists. We will continue CVR recruitment to attain a level of 400 CFIA trained Reservists and maintain their numbers and training readiness. We are working to include Reservists in CFIA work activities, training and live exercises.

Developing the “civil side”, is our second CVR goal. The main challenge is to find and maintain a sufficient level of funding to continue to build the civil capacity of the CVR. Discussions have started with Ontario, Manitoba and other provinces to determine how best to continue development and integration of the animal response into provincial-territorial and federal emergency response plans. The CVR expects to have signed Memoranda of Understanding with provinces and territories defining the terms and conditions for CVR support to major emergencies involving food and companion animals. We continue to work with Public Safety Canada and the Public Health Agency of Canada, both of whom recognize that the CVR will be a valuable component of their emergency preparedness. CVR “all hazards” training, including on-line modules and simulation exercises, are under development.

The CVR is also working with Canada’s five veterinary colleges on student reservist training, curriculum development and ways and means to utilize the resources and facilities of the colleges in collaboration with the CVR.

Our third CVR goal has been to make the CVR a resource providing the capacity to deploy internationally. We will strive to provide a response model for other countries to emulate, provide assistance in both FAD and civil emergency
response, provide opportunities for CVR members to have international experience and work with international animal health organizations and our USA counterpart, the AVMA.

In January, in the immediate aftermath of the massive Haiti Earthquake, the CVR was on standby alert status to offer veterinary assistance. Approximately 50, mainly French speaking CVR reservists, indicated their willingness and availability to work on recovery efforts in Haiti. As of January 23, 2010, this offer of assistance has not yet been required.

The CVR continues to recruit Canadian veterinarians. If you are interested in applying, details and application forms are available on the CVMA website: http://canadianveterinarians.net/index.aspx

The CVR Vision

A nationally and internationally recognized emergency response partner and a competent resource that is available to be called upon in the event of large-scale animal disease outbreaks and civil emergencies affecting animal health and welfare.

“Animals are a Part of Every Emergency!”
An All-Hazards Emergency Exercise to Demonstrate Key Management and Triage Protocols

Gordon Dittberner, DVM

No Paper Available
Foreign Animal Disease Outbreaks and the Impacts on Your Clients and Your Practice

Kevin Millar, DVM

No Paper Available
The Incident Command System for Responding to Emergency Situations

Don Puccini, B.A.Sc, MBA

Summary

This presentation will illustrate how the Incident Command System (ICS) is used by first responder authorities to manage emergencies in Canada. It will include a brief overview of Canada’s emergency management framework and structure including ICS principles and their application, and an example scenario illustrating call-up and deployment of the Canadian Veterinary Reserve to support first responders in handling and treating animals in distress.

1. Introduction

Safeguarding animal health and welfare during major animal disease outbreaks and all hazards emergencies poses unique challenges for emergency management in Canada.

Canada lacks a national emergency animal health strategy and first responders are generally not equipped to address companion animal issues in their emergency plans. There are no specific provincial emergency response plans for dealing with large scale emergencies affecting animals (other than for Reportable animal diseases managed by CFIA). There are inadequate first responder arrangements for ensuring consistency of approach in responding to large-scale animal emergencies. The civil emergencies component of the Canadian Veterinary Reserve is intended to fill this “critical gap” in Canada’s emergency management framework.

The CVR plans to work closely with federal, provincial and local government authorities, non-governmental organizations, and the animal health industry in responding to “all hazards”- type natural and manmade disasters. For this program to be effective, the animal civil response must be fully integrated into federal, provincial and regional/local first responder programs.

The Canadian Veterinary Reserve consists of a roster of more than 400 (mainly private practice) veterinarians ready for call-up and deployment to emergency sites across Canada. The CVR program currently provides for the provision of surge capacity trained veterinarians to be hired by CFIA as term employees on an “as and when needed” basis during major animal disease outbreaks (FAD) in Canada. There are about 200 CFIA-FAD trained CVR reservists. Planning and discussions are underway with provincial governments to provide veterinarians to assist first responder authorities during civil (all hazards) emergencies. Planning to determine the supporting roles of animal health technologists and technicians, non-government organizations, the animal health industry, veterinary students and the Veterinary Colleges in the CVR is underway.

2. Emergency Management in Canada

(Reference: Public Safety Canada. FPT Framework Document)

In Canada, emergency management adopts an all-hazards approach that addresses both natural and human-induced hazards and disasters. These are increasing in both number and frequency across the world, resulting in ever growing human suffering and economic cost. Canada is not immune to these events. Natural and human-induced hazards and disasters have become more prevalent in urbanized societies and terrorist attacks on western targets are likely to persist. These events can have profoundly negative effects on Canadians.

Most emergencies in Canada are local in nature and are managed by the municipalities or at the provincial or territorial level. Moreover, accumulating risks associated with factors such as increased urbanization, critical infrastructure dependencies and interdependencies, terrorism, climate variability and change, animal and human health diseases and the heightened movement of people and goods around the world have increased the potential for various types of catastrophes.

The ultimate purpose of emergency management is to save lives, preserve the environment and protect property and the economy. The protection of life is of paramount importance. In the broadest sense, emergency management raises the understanding of risks and contributes to a safer, prosperous, sustainable, disaster resistant and resilient society in Canada. Emergency management is comprised of four interdependent risk-based functions as follows:
Prevention - to eliminate or reduce the impacts and risks of hazards through pro-active measures taken before an emergency or disaster occurs, for example land-use management, public education and protective structures such as flood dykes.

Preparedness - to be ready to respond to a disaster and manage its consequences through measures taken prior to an event, for example emergency response plans, mutual assistance agreements, resource inventories and training, equipment and exercise programs.

Response - to act during or immediately after a disaster to manage its consequences through, for example, emergency public communication, search and rescue, emergency medical assistance and evacuation to minimize suffering and losses associated with disasters.

Recovery - to repair or restore conditions to an acceptable level through measures taken after a disaster, for example return of evacuees, trauma counseling, reconstruction, economic impact studies and financial assistance.

Traditionally, emergency management in Canada has focused on preparedness and response. The changing risk environment now demands that emergency management also deal with specific risks, hazards and vulnerabilities through prevention and mitigation measures in advance of emergencies and disasters. Greater attention or investment in prevention and mitigation can help prevent disasters or significantly reduce the social, economic and environmental costs and damages when events occur.

**Principles**

The principles described here are at the heart of an emergency management framework for Canada. They reflect the essence of emergency management in Canada and they frame the key underlying beliefs and goals of emergency management.

**Responsibility**

Emergency management roles and activities are carried out in a responsible manner at all levels of society in Canada. Legal and policy frameworks and other arrangements establish guidelines and standards to ensure that due diligence is exercised and accountability is respected in the conduct of emergency management activities. Provincial and territorial governments have responsibility for emergency management within their respective jurisdictions. The federal government exercises leadership at the national level relating to emergency management responsibilities in its exclusive fields of jurisdictions and on lands and properties under federal responsibility.

In an emergency, the first response is almost always by the municipalities or at the provincial or territorial level because disasters occur most often locally. Should a provincial or territorial government require resources beyond their own in an emergency or disaster response, the federal government responds rapidly to any request for assistance by a provincial or territorial government.

**Comprehensive**

FPT governments have respectively adopted a comprehensive approach to emergency management. The approach is proactive and integrates risk-based measures, all-hazards, partners from all parts of society and coordinates and balances efforts across the prevention and mitigation, preparedness, response, and recovery functions.

**Partnerships**

All Canadians are involved in emergency management. Individual citizens, communities, municipalities, and federal, provincial, territorial governments, First Nations, emergency first responders, the private sector (both business and industry), volunteer and non-government organizations, academia, as well as international allies may be involved in emergency management. Good partnerships based on effective collaboration, coordination and communication are a key component of FPT emergency management systems.

**Coherency of Action**

Emergency management requires collaboration, coordination and integration to facilitate complementary and coherent action by all partners to ensure the most effective use of emergency management resources and execution of activities. Complementary emergency management systems at all levels are to provide for concerted efforts to facilitate timely and effective prevention and mitigation, preparedness, response and recovery measures to deal with disasters. Coherency of action relies on the existence of clear and appropriate roles, responsibilities, authorities and capacities of emergency management partners. Collaborative action based on widely shared expectations, understanding and support for these factors are key to coherency of action.

**Risk-based**

A risk-based approach informs the interdependent functions of emergency management in Canada. This approach emphasizes the importance of assessing vulnerability to all hazards at the outset to determine the optimal balance and integration of functions to address vulnerabilities and risks. Recognized, flexible and effective risk-based approaches allow emergency management activities, programs and systems to be tailored to address particular environments and to accept that living with certain risks may be both prudent and safe.
All-hazards
Emergency management adopts an all-hazards approach in every jurisdiction in Canada by addressing vulnerabilities exposed by both natural and human-induced hazards and disasters. Natural hazards and disasters that are relevant to emergency management include extreme natural events such as floods, hurricanes, storm surges, tsunamis, avalanches, landslides, tornadoes, wild-land urban-interface forest fires and earthquakes. Human-induced disasters that concern emergency management include intentional events that encompass part of the spectrum of human conflict, such as terrorist or cyber attacks. They also include electrical power outages or other disruptions to a critical infrastructure sector (for example, finance, water supply and telecommunications) that result from a human or technological accident or failure. In addition, biological hazards, for example animal or human health diseases that risk causing a pandemic influenza, concern emergency management in Canada.

Resilience
Resilience is the capacity of a system, community or society to adapt to disturbances resulting from hazards by persevering, recuperating or changing to reach and maintain an acceptable level of functioning. Emergency management aims to strengthen the resiliency of citizens, responders, organizations, communities, governments, systems and society overall to keep hazards from becoming disasters. Resilience minimizes vulnerability or susceptibility to damage from hazards by creating or strengthening social and physical capacity in the human and built-environment to cope with, adapt to, respond to, and recover and learn from disasters.

Clear Communications
Clear communications by appropriate authorities are a critical and continuous process before, during and after an emergency. Prior to an emergency, communication objectives focus on public education concerning emergency management to enhance awareness of hazards, risks and vulnerabilities; strengthen prevention, mitigation and preparedness measures; and provide information on all aspects of emergency management. Public alerting communicates warning messages that a disaster is imminent. Communications during and directly after a disaster explains and guides immediate response actions to minimize impacts and protect safety. These communications are instructive on the requirements for short, medium and long-term recovery.

Continuous Improvement
Lessons learned and knowledge generated from evidence-based and qualitative information is used to develop improved practices, which are shared widely. After emergencies or disasters occur, a systematic approach is used to learn lessons from the experience, increase effectiveness and improve emergency management practices and processes.

Conclusion
An emergency management framework for Canada is established through FPT governments’ emergency management systems. The framework aims to encourage this important contribution of FPT governments in partnership with others towards the effective functioning of an emergency management framework for Canada enhancing the public safety of Canadians.

3. First Responders and On-Site Emergency Management
Upon deployment of the civil component of the CVR to an emergency location, the following model is proposed for reconnaissance, assessment, and response:

- Reconnaissance/Assessment Team: Advance team of trained experts to assess situation and identify requirements;
- On-Scene Medical-Assistance Rescue: CVR team arrives, coordinates with local Veterinary Officer (VO) and Incident Commander, deploys for animal rescue/retrieval; to tranquilize, treat or euthanize; preliminary ID, transport, referral and/or disposal;
- On-Site Animal Emergency Care Centre: ID recording, medical records, triage, medical or surgical treatment wards, non-medical wards, shelter, and/or release to owner, refer to local facility, or refer for disposal. Pharmacy set up, medical supplies and equipment accessed. Daily review and report on all animals in the emergency facility;
- Animal Emergency Care Centre Annex: Includes administration, facilities and equipment; storage (animal food, medical and non-medical equipment, supplies and telecommunications needs), and liaison staff;
- Off-Site Provincial Emergency Operations Centre: Directs the response, co-ordinates the activities of the various provincial first responders, liaises with federal and national organizations;
- Public Health Agency of Canada, Centre for Emergency Preparedness and Response;
- Federal Government Operations Centre (GOC) at Public Safety Canada: Provides central hub for coordination of national organizations, and other federal departments, and addresses issues and resources to meet all the emergency needs identified;
- Existing National (Stakeholder) Organizations: CVMA, CAHI, CFHS,CAAHTT, and others. Provides the main channel for two way information flow with members, or for seeking assistance from other organizations.
4. The Incident Command System

Introduction
The Incident Command System (ICS) is a model for command, control, and coordination of emergency response at an emergency site. It provides a way of managing the efforts of agencies and resources as they work together toward safely responding, controlling and mitigating an emergency incident.

ICS was developed in the 1970s in response to a series of major wildfires in southern California. It was developed as a direct result of several recurring problems identified during multi-agency responses. These included:

- Terminology used by responding agencies was not standardized;
- The response structure lacked the capability to expand and contract as required by the situation;
- Communications were neither standardized nor integrated;
- There were no consolidated action plans;
- Designated facilities were not available.

Although originally designed as a management structure in response to wildfires, ICS has evolved into an all-hazard system that is appropriate for all types of emergencies.

Much of the success of ICS has resulted directly from creating a common organizational structure and applying key management principles in a standardized way. These management principles have been proven to improve efficiency and effectiveness. First responder organizations around the world use an ICS to manage their emergency response. Consequently, it is critical that CVR reservists have a clear understanding of ICS principles so they can integrate successfully into an emergency response.

Basic Incident Command Structure and Functions

Incident Commander
Single Incident Commander - Most incidents involve a single Incident Commander. In these incidents a single person commands the incident response and is the decision-making final authority.

A Unified Command is used on larger incidents usually when multiple agencies are involved. A Unified Command typically includes a command representative from major involved agencies and one from that group to act as the spokesman. During multiple-incident situations, an Area Command may be established to provide for Incident Commanders at separate locations.

General Staff

Operations Section Chief - The Operations Section Chief is tasked with directing all actions to meet the incident objectives.

Planning Section Chief - The Planning Section Chief is tasked with the collection and display of incident information, primarily consisting of the status of all resources and overall status of the incident.

Logistics Section Chief - The Logistics Section Chief is tasked with providing all resources, services, and support required by the incident.

Finance/Administration Section Chief - The Finance/Administration Section Chief is tasked with tracking incident related costs, personnel records, requisitions, and administrating procurement contracts required by Logistics.

Command Staff

Safety Officer - The Safety Officer monitors safety conditions and develops measures for assuring the safety of all assigned personnel.

Public Information Officer - The Public Information Officer serves as the conduit for information to internal and external stakeholders, including the media or other organizations seeking information directly from the incident or event.

Liaison - A Liaison Officer serves as the primary contact for supporting agencies assisting at an incident.

CVR reservists, upon deployment to an emergency site will work under the Incident Commander’s Operations Chief to provide reconnaissance, needs assessment, recommendations on veterinary resources, infrastructure, supplies, equipment and logistics requirements, triage, emergency veterinary medical services, treatments and care.

Onsite, the CVR reservists working individually or in teams as the circumstances dictate will coordinate and work with other animal health stakeholder groups such as;

- Provincial Chief Veterinary Officers and their staff
- Local humane societies and SPCAs
- Animal pharmaceutical industry
- Animal food industry (livestock & pets)
- Livestock Industry (Canadian Animal Health Coalition)
- Provincial Veterinary Medical Associations
- Provincial Veterinary Registrars
5. Alerting, Call-Up and Activation of the CVR

The civil emergencies roster of the CVR is activated by request to the CVMA. This request may come from Public Safety Canada, the Public Health Agency of Canada or provincial/territorial EMO. Trained civil reservists can be quickly deployed anywhere in Canada, are activated upon request of the first responder organization, function under the general direction of the CVMA’s Executive Director and under the field supervision of the first responder’s Incident Commander.

Reservists are expected to be on site and operational within 48 hours of the initial call-up. Each team of CVR reservists will have sufficient back-up resources to be able to function 24/7 for periods of up to six to eight weeks in the field, with provision for rotation of personnel every 3 weeks.

CVR teams will be comprised initially of veterinarians, but planning is underway to add veterinary technologists and technicians, and other trained NGO personnel. They will train and work together as emergency/medical animal care teams. Their mandate, under the authority of the provincial Chief Veterinary Officer, will be to provide emergency veterinary medical assessments, treatments or surgeries to alleviate animal welfare concerns for all animals affected by the emergency.

Each team will be under the field leadership of a senior, experienced veterinarian trained in incident command. They will be established regionally as MOUs are signed between the CVMA and provincial/territorial first responder government authorities. The core teams will supplement the resources of local and provincial responder and non-government organizations (NGOs). The provincial EMOs, using provincial NGOs, will provide animal rescue, transportation, shelter, food, logistics and other non-medical supplies working in collaboration with the CVR teams. Tentative locations for these teams will be in each of the provincial capitals, or in proximity to the provincial CVO.

CVR Deployment Needs/Conditions

- Air/ground transportation
- Assurance of physical security on-site
- Basic food, potable water, health services, accommodation, etc. met
- Means of communication, data or voice, to home city/town
- Accidental medical, death and disability insurance
- Compensation - wages and practice continuity (about $600/day)
- Assurance of professional liability insurance
- Clearance/accreditation from local veterinary authorities to enter jurisdiction and practice
- Suggested tour of duty - rotational/3 weeks
- Deployed reservists will be provided the CVR Personal & Business Preparedness Checklists before they are deployed.
- Veterinary Equipment & Supplies (list of essential carry-on items, instruments, pharmaceuticals, clothing, etc.)
- CAHI and other AH industry to be consulted re provision of bulk quantities of basic AH pharmaceuticals, other medical supplies and pet food/animal feed.

6. Example Scenarios – Civil Emergency and FAD

Ultimately, when fully operational, it is anticipated that the CVR will respond to four types of animal disasters:

6.1 Animal Disease Outbreaks in Canada (CFIA lead)

Normally the CFIA will activate its agency-wide emergency plan in conjunction with its disease-specific arrangements or plans (e.g. Foot and Mouth Disease Strategy, CFIA, 2003). The CVMA’s role is to coordinate activation of the Canadian Veterinary Reserve in support of the emergency response. The Executive Director of the CVMA and/or a designated CVMA emergency coordinator will maintain close liaison with the appropriate CFIA response officials in providing veterinarians from the CVR to CFIA.
6.2 Large-Scale Natural and Technological Emergencies in Canada

For major national emergencies, the federal Emergency Management Act with Public Safety Canada as the lead federal department provides for the deployment of federal and national resources to the affected area. The Executive Director of the CVMA and/or a designated CVMA emergency coordinator will maintain close liaison with the appropriate federal response officials and the federal Government Operations Centre. The CVR will be activated in this situation to be on standby for deployment of veterinarians at the request of first responder authorities in the affected areas.

6.3 Provincial or Local Animal Health Emergencies

The vast majority of emergency situations are handled by provincial and local authorities. In cases where the impacts overwhelm local resources and capabilities, (e.g. earthquake in British Columbia, major floods in southern Manitoba, etc.), the CVR can be activated and deployed upon request by the first responder authorities.

6.4 International Animal Health Emergencies

During large scale international emergencies, Canada is often called upon to provide financial, technical and professional assistance to the affected communities. The CVR can be activated and deployed internationally subject to memoranda of understanding regarding the transferability of professional credentials to other jurisdictions, compensation, insurance, legal liability and other cross-border issues. CFIA has a number of international agreements in place for cooperation during animal disease outbreaks.

Demonstration Scenario and CVR Alerting/Activation/Call-Up:

An example scenario illustrating call-up and deployment of the Canadian Veterinary Reserve to support first responders in handling and treating animals in distress.

To be presented at the CVMA Calgary Convention, July, 2010.

Summary and Conclusions

For the civil program, the CVR is nearing completion of online training modules and the main challenge is to find and maintain a sufficient level of ongoing funding support to continue to build the civil capacity of the CVR. With the assistance of the Training Sub-Committee of the CVR Advisory Board face-to-face and online training modules will be ready for delivery later in 2010.

Subject to the availability of CFIA funding, the CVR will continue to engage a large number of reservists on an ongoing basis in training programs for FAD that will ensure the ready availability of the CVR to be deployed when requested by the federal government to provide surge capacity veterinary resources.

Discussions are underway with Ontario, Manitoba and other provinces to determine how best to continue development and integration of the animal response into provincial-territorial and federal emergency response plans.

The CVR is working with Canada’s five veterinary colleges on student reservist training, curriculum development and ways and means to utilize the resources and facilities of the colleges in collaboration with the CVR.

At the federal level, Public Safety Canada and the Public Health Agency of Canada recognize that the CVR will be a valuable component of their emergency preparedness.

Next Steps for the Canadian Veterinary Reserve

- Brief all provincial/territorial EMOs on the CVR with a view to signing MOUs defining the CVR arrangements during a first response;
- Continue development of the civil training program using a combination of learning webinars, other online training tools, tabletop and field exercises;
- Explore feasibility of providing some training online, and other cost-effective training delivery options including participation in exercises by first responder groups, e.g. HUSAR;
- Explore options for veterinary technologists and technicians to join the CVR;
- Explore options for working with NGOs such as OERS, Vets Without Borders, SPCAs and other animal welfare stakeholders;
- Participate in national, provincial, regional and international EM exercises;
While there are many types of personal protective equipment (PPE) this presentation will confine itself with the most common used in veterinary practice; Gloves, Coveralls, Boots and Respirators.

Many of us use protective equipment without much thought. Perhaps it has always been part of our professional life or we received little or no explanation as to its proper use and care.

Yet, to be protective, the equipment must be used properly. Further, the fact that it is termed “personal” protective equipment implies the consideration that individuals vary greatly in size, experience and preference. This leads us to several tenants for the use of PPE:

• PPE must be appropriate for the task and in compliance with applicable regulations,

• Employees must be trained in the proper use, maintenance and storage of protective equipment,

• PPE must be sized to fit the individual so as not to become a hazard in itself,

• PPE should be comfortable and functional or compliance will decrease.

When choosing equipment brands, styles, or material consider the following;

• Environment in which the work will take place (temperature, rough surfaces, or sharp projections),

• Any chemicals (disinfectants) to be used, especially with respect to glove material compatibility.

• Length and intensity of the work to be done.

• Hazards (biological, chemical, physical)

An example of the environment influencing PPE choice or compliance is with a respirator. The disposable N95 filtering face piece disposable respirator may be comfortable when worn in cool dry air, for tasks requiring light to moderate effort. The same respirator it is unusable (significant respiratory resistance and resulting leakage around the face seal) if the air is hot, moist and dusty or if the wearer is engaged in moderate to heavy work. The disease hazard may not change but the respirator used might need to be changed because of the conditions of work or the work environment.

Gloves

If graded on a pass or fail for leaks by American Society for Testing and Materials D5151 standard test, there are no significant differences in failure rates among the vinyl, latex and nitrile gloves when tested directly out of the box. However, studies show virus and bacterial break through after ½ hour of regular clinical use in up to 83% of vinyl gloves and 35% of regular latex but only 3-4% of nitrile. A single layer of latex gloves was found to be less protective than a double layer of vinyl.

Latex allergy incidence is estimated to be 8-13% of workers in settings were glove use is common. Latex allergy is now known to be a leading cause of occupational asthma. For these reasons I would generally recommend the use of nitrile gloves. Latex gloves are acceptable for general laboratory or clinical use- if no specific disease is suspected or confirmed. However, when dealing with a known or highly suspected disease (especially a zoonotic one), nitrile gloves should be worn because of the leakage issues described above.

Respirators

Prior to wearing any respirator, including disposable N95 half face piece respirators, you should receive, or supply your employees with; respiratory protection training, fit testing and a medical assessment.

Before a respirator can be selected for a particular task, a workplace hazard assessment must be carried out by a “competent person”. Generally this would be an industrial hygienist, environmental health officer or other trained professional. The risk assessment must include a review of the work environmental factors, O2 levels, and work intensity level and duration as well as an assessment of the classes and levels of respiratory hazards present.

Specific respirator program requirements vary according to provincial legislation. However, a number of provinces
have adopted or refer to the Canadian Standards Association Standard z94.4-2002 Selection Use and Care of Respirators. The Standard defines the requirements for the following components of a respiratory protection program:

- Roles and Responsibilities
- Hazard Assessment
- Respirator Selection
- Respirator Fit Testing
- Training
- Use of Respirators
- Cleaning, Inspection, Maintenance, and Storage of Respirators
- Health Surveillance
- Program Evaluation
- Recording Keeping

While an opening clause of the Standard states “This standard is not intended to address the selection of respirators for use against a) infectious agents; and b) nuclear biological chemical (NBC) agents”, all other aspects of the standard apply.

A number of safety supply companies offer respirator selection assistance, training and fit testing services.

There are two main types of respirators; air-purifying respirators (APRs) and supplied-air respirators. Air-purifying respirators remove contaminants from the ambient breathing air by filtering out particulates (e.g., dusts, metal fumes, mists, etc.) or by adsorbing gases or vapours in a specialised cartridge or canister. Supplied-air respirators supply clean air from a compressed air tank or through an air line. The air supplied from tanks or compressors must meet standards set for purity and moisture content (CSA Standard Z180.1-00: Compressed Breathing Air and Systems).

Filters are designed and labelled as to specific contaminant filtering ability. For chemical type filters, it is the responsibility of the user to determine the “change out” requirements of the filter. That is to say, depending on the level of contaminant, how often the filters need to be changed to avoid saturation and then exposure of the worker to break through of the contaminant in question.

For particulates, filters are designate “N” or “P”. N type filters can only be worn with dry or aqueous based aerosols. P type filters can be used for dry, aqueous and oil based aerosols. The most commonly known respirator is the “N95” – properly named a disposable one half face piece filtering respirator.

Disposables respirators come in many filtering capacities (the amount of particles 0.3 µm and greater it will remove), with 85, 90, 95 and 99.97% being the most common.

The issue with these respirators is the difficulty in maintaining adequate face seal and the increase in respiratory resistance in conditions other than stationary or light work at normal room temperature conditions. The greater the resistance to respiratory effort, the greater the degradation of efficiency (leakage) of the respirator. As a result, this type of respirator is often not appropriate for a number of large animal veterinary applications.

Disposable respirators can cost from 1.50 to 10.00 each. A silicone reusable respirator costs $20 to $30 and filters are in the range of $9 to $30 per pair. The filters (especially particulate ones) do not necessarily have to be changed every use. They do have to be capped up for decontamination as water / disinfectant etc will ruin the filter medium. Depending on the frequency of use disposable respirators may not be the cheaper option.

Coveralls

Cotton coveralls and lab coats: The most common issue with the use of lab coats or coveralls is the failure to maintain separation of clean and dirty; wearing coveralls worn on a farm in your vehicle, a used lab coat to the reception area, or hanging coats/coveralls on top of each other will spread contamination to other areas of the practice, and to worker’s clothing and personal effects. Separation of clean and dirty is discussed further on.

Disposable Coveralls: A common non woven material is Tyvek. It is a simple mat of fibres like paper and has no tensile strength or resistance to liquids. Plain Tyvek may not be good choice for work with high hazard zoonotic agents. There are other fabrics have more resistance to fluids and better particle hold out.

To identify these materials look for material test ratings. ASTM F 1670 is a pass/fail test for visual penetration of a synthetic blood solution at 2 psi applied pressure. ASTM F 1671 is a pass/fail test incorporating a viral surrogate solution at 2 psi applied pressure. However, while the material of the garment may pass either of these standards, seams may leak if sewn. If preventing any liquid penetration is necessary, look for sealed, or heat bonded seams.

One might use ASTM F 1670 certified materials work with low/moderate risk pathogens where significant liquid contamination is not an issue. Extra protection can be provided at pressure points such as through the use of knee pads or aprons or the higher rated materials with bonded seams should be used.

A note on the difference between impermeable and impenetrable. Often we see in a biosafety advisory that “imperme-
able" overalls are required. This may be correct but more often is the misuse of a word that has a very specific meaning in the PPE industry. Protective clothing is rated on permeability, which is the diffusion of a chemical, held at a preset pressure for a specific time period, though the material. Penetration however, is the movement of liquid through the natural gaps in the substrate. A material like Nufab, Proshield and others may be perfectly water resistant, yet not technically “impermeable” from the industry and standards point of view.

Boots

Many boots have treads that are difficult if not impossible to clean and disinfect. Look for wide tread with smooth, rounded corners on the tread angles when choosing boots. Some people use boot covers as a time saver. But unless judicious zoning is used, boot covers will track greater amounts of contamination, further, than rubber soles.

When using boot baths or boot covers, the most common failure of contamination control is through improper separation of clean and dirty – for example, standing beside the wash pail, scrubbing the bottom of a boot, then putting the foot down in the same area as before washing it.

Separating Clean and Dirty

In contamination control; industrial, chemical or biological – there is the convention of the 3 zone system. This system can be applied to an area as large as a city, or to one as small as a corner of a room. Ideally one has physical barriers, such as a door, between the zones to contain contamination but the zones can be simply marked out with chairs, tape on the floor or lines drawn in the dirt.

The cold zone is a clean area were no PPE or precautions are required. The hot zone is an area considered to be contaminated or potentially contaminated with the hazard. The warm zone is a transitional area between the hot and cold zones, where decontamination control steps take place. It is important to understand that the warm zone is contaminated albeit at a lower level than the hot zone. A person or object is not considered clean until they have completed all the required decontamination steps and step into the cold zone.

Establish a sequence of events and a specific location for each to take place. Then everyone follows the same procedure. This means that all staff know, at any given time the (clean/dirty) status of every object on the premise or in the isolation area. Items can be soaked in disinfectant at the transition lines or bagged for decontamination later.

If worn, outer gloves and coveralls are removed at the “hot/warm” line and boots are disinfected/ boot covers removed. Clothing should be handled carefully so as not to contaminate the in(clean) side or clothing worn underneath. If only one pair of gloves is worn, they should be sprayed with disinfectant. Lab coat or single coveralls are taken off just before exiting the warm zone after all samples, waste and equipment are disinfected. Always leave on gloves to the last. See presentation for full explanation.

Maintenance and Shelf Life

While many manufacturers often list up to 2-3 years as a shelf life for gloves, this is for unopened boxes only, stored away from light at room temperature (approximately 22°C). Gloves become more porous with age. Once open, throw away a box of gloves after 6 months (or less, if sticky, inelastic or fragile when stretched). It is good practice to writing the date of opening on a box of gloves.

Most PPE- gloves, respirators, and synthetic coveralls are degraded by UV light. Store in a cool, dark, dry place. Check expiry dates for all items of PPE before use.

Respirators vary quite a bit in chemical compatibility so check as to what pH or type of product can be used to disinfect and clean them. Do not store respirators lying on the face seal – over time it will distort. Always clean before storage and check parts before donning.

Keep disposables respirators in their original packaging – folding, wrinkling or wetting will degrade the level of protection provided.
A Canadian Veterinarian’s Experience from Involvement in Hurricane Katrina

Carin Wittnich, DVM

Hurricane Katrina – we all saw the images and the devastation and realized that it can happen in “our own back yard”. The hurricane struck and what was left behind exceeded anyone’s imagination. Thousands upon thousands of people were left stranded. Many of those people who stayed behind, did so because they would not abandon their animals. At that time, no provisions had been made to include animals and rescue efforts were directed solely to the human element. Animals were considered extra baggage and over the course of days the scene of people being forced at gun-point to leave their animals at the mercy of the elements as they boarded buses, boats or helicopters was repeated numerous times. Only occasionally, at the discretion of the rescuers, were they allowed to bring their pets, only to have them taken away at the end of their journey upon arrival at the evacuation site. The scenes then shifted to these abandoned and helpless friends of man left behind.

At this point animal rescue groups throughout the US and Canada began to step in. My deployment was through a Canadian registered charity organization, the Oceanographic Environmental Research Society (Disaster Response Division). They were contacted and asked to assist at the request of local and state authorities under the US Federal Emergency Management Agency (FEMA). The rest of this talk will focus on the OERS teams’ experiences both before and during the 2 week deployment including: getting to the disaster area, rendering assistance at Hattiesburg and Gulfport, Mississippi, the return home and lessons learned.

OERS-DRD always ensures its team members are adequately protected and prepared for any deployment. This includes personal health, supplies, appropriate personal protective equipment (PPE), medical and other items. For the Katrina deployment that included all the usual vaccinations as well as protection from exotic diseases that the team might be exposed to while in the disaster region. Neglect of this aspect would have put everyone on the team in jeopardy. This would not be tolerated by our Director of Operations. This then meant team members had to make rapid visits to travel medicine clinics to get vaccinations and prophylaxis meds prescribed. Then came further logistics as the team was kitted and the van filled with all the necessary items to ensure the team would have what they needed for floodwater rescues in a region where potable water, food and basics of living might not be available. Then once this was secured, the appropriate paperwork had to be processed to ensure the travel into a foreign country, the US, was made as smooth as possible. This included confirming veterinary licensure issues with the state and travel documents for the border. During this time, team members arranged their personal affairs to allow their departure as soon as was feasible. The OERS logistics co-ordinator also planned the travel route, secured sleeping accommodations for the long 3 day drive down and coordinated with the US agencies expecting the team’s arrival. They in turn informed OERS on the best most passable routes to use. Then, 10 days after the hurricane had initially hit, departure day arrived and the team was off.

Travelling as a team is the safest and most secure mode of travel. One has the logistics support of the organization’s home base, with whom the team maintained constant contact. As the team drove into the devastation area, this issue became much more critical as highway signs and markers did not exist and GPS and cell communications were the only way to navigate. Finally the team arrived at their destination, Hattiesburg, Mississippi, on the outskirts of the worst devastated area. The fairgrounds served as the staging area for all rescue efforts in the affected region (including military, fire rescue, animal rescue and the animal housing compounds).

Once on site, the OERS-DRD team worked under FEMA with the Humane Society of the United States, State Department of Public Health as well as local animal shelters to rescue and care for the thousands of animals in need – including a wide variety of species from household pets, wildlife and domestic farm animals. The situation on scene was that it was seriously understaffed; so with a quick round of introductions, the team set to work, leaving the set-up of their accommodation to later. This does not mean this aspect should be neglected, as ensuring safe accommodations with security provided, as it was for the team, is ultimately also crucial. Because of the extent of the staging area, the team was housed in large army tents, with air conditioning in the sleeping and mess tents and access to latrines and portable showers. Given that temperatures reached 110 degrees Fahrenheit during the day, this was amazing relief. As well the army medics had set up ‘misting tents’ for staff to use if they felt the heat was overpowering. This makes the point that to work within the system can have its benefits. However, one can recognize that this is not always possible as the team found out on their second leg of their deployment. More on that in a little bit.
The logistics of conducting daily triage of hundreds of animals, decontaminating them from floodwaters and providing medical care and housing under adverse conditions will now be covered. The team had many responsibilities, including the screening and medical checks of animals prior to their admission to the facility, ensuring appropriate decontamination was done even prior to that. Then preventative vaccination and treatments for any parasites both internal and external as well as health issues needed to be done. It is well beyond the scope of this talk to detail these. However, one can image local issues such as the endemic nature of heartworm in this region which is often untreated or even diagnosed continued as an issue. Also the fact that injuries of every kind, dehydration and heat stroke was always an issue as was malnutrition or GI disorders from ingestion of contaminated foods. These all needed attention, and had to be handled, often with less than the ideal medications or equipment. Quick thinking and common sense were two of the most valuable skills constantly used by our team to adapt on the fly. For example to avoid further heat stress to the animals, all shipping of animals was conducted after sundown, whenever possible, when the temperatures were closer to 90 degrees. During the day, firefighters, who were waiting to be staged to their assigned area, used fire hoses to pour water on the roofs of the barns to try to keep the heat down. During the teams stay, over 150 animals were admitted to the facility per day. The 3 existing fairground barns that remained intact were made of cement and metal, were located on the highest ground in the entire area, and housed over 1500 animals each. Not only were the newly rescued animals requiring care but the animals already on site needed to be closely monitored for any new health issues as well as requiring ongoing medical care for their existing conditions. To make this work, there also needed to be a lot of effort placed on sanitation and hands on care for all the animals. The OERS-DRD team was only a very small cog in the much larger effort where over 60 volunteers at any one time carried out all the needed duties. These ranged from daily cage washing and disinfection, regular feeding and walking and cage maintenance to ensure the animals were kept clean, well watered and as cool as possible. How does one keep cats cool? Why let them rest on bags of ice with fans circulating air around them of course. The heat and extent of the work that needed to be done were such that you could easily overdo it. If you did, you became another casualty, putting even further stress on an already taxed system. The team wisely paced the work that needed to be done were such that you could easily overdo it. If you did, you became another casualty, putting even further stress on an already taxed system. The team wisely paced the work that needed to be done were such that you could easily overdo it. If you did, you became another casualty, putting even further stress on an already taxed system. The team wisely paced the work that needed to be done were such that you could easily overdo it. If you did, you became another casualty, putting even further stress on an already taxed system. The team wisely paced the work that needed to be done were such that you could easily overdo it. If you did, you became another casualty, putting even further stress on an already taxed system. The team wisely paced the work that needed to be done were such that you could easily overdo it. If you did, you became another casualty, putting even further stress on an already taxed system. 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Those, along with others who had irrevocable serious behavioral issues that rendered them a threat to anyone coming in close proximity, had to be euthanized. This later aspect was not understood by many ‘on scene’ volunteers who did not have the kind of training we had in disaster response. That said, one of our first acts upon arrival on scene the first day, was the triage of a lovely yellow Labrador that had been hit by a vehicle and left by the side of a highway. Passersby who witnessed the accident stopped and picked up the dog who was trying to drag himself away from the road and brought him to us. My first case, and triage identified broken ribs, hemorrhagic shock most likely from internal bleeding, a fractured pelvis and possibly spinal trauma. Had this been a normal situation at a veterinary trauma centre where all equipment and intensive care possible, this lovely animal could have made it. The folks who picked up the animal begged us to try everything, offered all the money they had but that was not the issue. This was not a normal situation and despite my deep desire to treat aggressively and never give up, despite my deep bond for Labrador retrievers, I had to agree with the Chief Veterinarian’s (the person who’s job I would fill the following day) decision that euthanasia was the only reasonable and kind thing to do. This was incident command and it was done, my first act upon arrival at a disaster triage center. Stay clear if you are faint of heart. We witnessed a number of volunteer veterinarians and their technicians arrive with the best of intentions and depart within 12 hours as the stress and apparent chaos was too much. The toll emotionally these deployments take even for the experienced and trained team is often not immediately apparent as the job required action and very little time for reflection. However, upon return home, this is when the magnitude may hit and follow-up debriefing, routinely done by our OERS-DRD teams, is another critical component to a deployment. This supplements the daily debriefings that were done to deal with immediate issues. Note that once home, the drain both physically and emotionally is far from over.

Now on to practical lessons learned that impact on current CVR preparedness:

- Deploy with a group that knows what it is doing and works within the system
- Where-ever you go ensure you are equipped to do the job.
- Have experience or training in disaster response
- Take care of yourself, ensure you are adequately protected
- Do not assume all your needs will be taken care of
- Expect hardship and difficulties
- Learn to work without, adapt
- Understand and appreciate the ICS
- Know your limitations
- Expect nothing, hope for the best
Evidence-Based Physiotherapy

Laurie Edge-Hughes, BScPT, MAnimSt(Animal Physiotherapy), CAFCI, CCRT

History of Physiotherapy in Canada

Human physical therapy in Canada began in and around the First World War, when large numbers of wounded servicemen began arriving back home from overseas and found themselves unable to cope with life’s demands. Intensive one-year physical therapy training courses were set up in 1916 as the nation realized that medical care and surgery were not enough to restore severely wounded men into healthy, functional members of society. Physical therapists in those days administered light and heat therapy, as well as hydrotherapy, electrical treatments, massage, and passive, active and resisted exercises. These early physical therapists were registered and monitored by the Canadian Association of Massage and Remedial Gymnastics (a forerunner of the Canadian Physiotherapy Association – CPA) for the maintenance of high standards of education, quality of treatment, and professional conduct. As demand grew, educational advances progressed, and the first school of physiotherapy was established in 1929 at the University of Toronto, offering a two-year diploma course. McGill University offered the first baccalaureate degree in 1954, but it was not until the 1970’s that the 3 to 4-year baccalaureate degree was established as the minimum educational requirement in order to practice physiotherapy on human patients. The current entry-level educational requirement for a physical therapist to practice in Canada is a 2.5-year full time, year-round Masters degree (following a prerequisite Bachelors degree), and by 2020, all university physical therapy programs will change their curriculum to a Doctorate in Physical Therapy (DPT), which will be the minimum educational requirement for an entry level physical therapist. The 3+ year DPT is currently being offered in the United States and transitional-DPT courses are available for practicing therapists who want to upgrade to this degree. Newly graduated physiotherapists (a term widely accepted as being interchangeable with the title of physical therapist) possess extensive knowledge and understanding in human anatomy, physiology, psychology, orthopaedics, manual therapy (soft tissue mobilization, and joint mobilization, manipulation, and stabilization), kinetics, bio-mechanical sciences, neurology, cardio-respiratory sciences, therapeutic techniques and tools, and exercise prescription. In addition, recent physiotherapy graduates are adept in client health management and case management, as well as in research evaluation, design, and implementation. Currently in the field of human physical therapy, a professional competency exam must be passed upon graduation, and continuing education is a mandatory requirement for licensure. Most physical therapists seek to expand their knowledge in a specialized area of their profession by means of continuing education opportunities. Some of these special interests are reflected in (but not limited to) the official divisions of the CPA: orthopaedics, sports, neurosciences, cardiorespiratory, women’s health, private practice, leadership, acupuncture, seniors’ health, paediatrics, international health, pain sciences, oncology, and animal rehabilitation. Human patients in Canada have direct access to physical therapist in private practice settings, in other words, they do not require a physician’s referral to be able to seek services from a physiotherapist. Regardless of this autonomy of practice, physical therapists continue to work collaboratively with medical doctors and other allied health care professionals in order to ensure that all aspects of a patient’s health care are addressed in a patient-centred model of care provision. Physical therapists are authorized and capable of making a clinical diagnosis prior to administration of treatment. In hospital settings, physiotherapists treat patients by physician referral due to practical and policy reasons, but they also carry out physical diagnoses, establish problem lists, and set therapy goals and treatment plans. Physical therapist diagnoses are often of a different nature than those of medical doctors (or veterinarians), and may include our impression of muscle imbalances, joint or spine dysfunctions, identification of specific soft tissue lesions, and the creation of a list of functional impairments to be addressed in treatment.
History of Animal Rehabilitation in Canada

The Animal Rehab Division of the Canadian Physiotherapy Association, formerly named The Canadian Horse and Animal Physical Therapists Association (CHAP), was first established in 1994 as an organized group of physical therapists interested in using their professional skills to treat animals. In 1994, CHAP was the third such group of its kind in the world, following the lead of Great Britain in 1984 and the Netherlands in 1989. In 2004, the Animal Rehab Division was officially recognized as a special interest group of the CPA. Currently, there are 12 countries with animal physiotherapy groups/divisions which form part of their national physiotherapy association. Other countries include South Africa, Sweden, Spain, Finland, Australia, the United States, Switzerland, Ireland, and Belgium. All of these animal physiotherapy associations fully recognize that additional educational is necessary for a physical therapist to engage in the practice of animal rehabilitation. Each of these countries has (or is) taken the initiative of creating its own educational system and setting of standards to train physical therapists in animal rehabilitation / physiotherapy / physical therapy. In North America, there are three certification programs available to train physiotherapists to apply their skills to animal patients. The two existing programs in the United States are offered to physical therapists, veterinarians, and animal health technicians or equivalent. These programs attempt to bolster the different aspects of knowledge of each professional group of students. The Canadian animal rehabilitation program (offered by the Animal Rehab Division of the CPA) limits its enrolment to physiotherapists, and focuses entirely on teaching this single group of professionals, animal principles (mostly canine and equine) such as anatomy, biomechanics, pathology, clinical conditions, common veterinary surgical and clinical interventions, and handling skills, as well as physiotherapy assessment and treatment techniques for these species. England (and previously Australia) boasts the world’s only post-graduate (Masters) degree programs. In the case of The Royal Veterinary College in England, the program offered is a Master of Science in Veterinary Physiotherapy, whereas at the University of Queensland in Australia, the program previously offered a Masters of Animal Studies in Animal Physiotherapy. The Australia program is currently not running and is proposed to be re-established at another university. These courses have limited their enrolment to physiotherapists, and are two-year course-based programs with a research component and a publishable clinical thesis requirement. No similar university level program currently exists in North America, and only a handful of North American physical therapists have completed either one of these Masters programs abroad.

The type of clinical practice in the field of animal rehabilitation is quite diverse. In general, physical therapists have established collegial partnerships with veterinarians throughout North America. Some therapists work out of veterinary clinics, others do house-calls (or barn-calls), some have a home-based office/barn, and others operate businesses or are employed by stand-alone rehabilitation referral centres. Regardless of the type of clinical setting, the Animal Rehab Division strongly advocates that rehabilitation of animals be provided by properly trained physical therapists upon veterinary referral if an animal is lame, injured, or requires post-surgical services. Physiotherapy in the human health care field is often complimentary to other health care services, and the Animal Rehab Division believes that it is with this same professional approach and conduct that animal rehabilitation should be delivered. Members of the Division can obtain professional liability insurance specific to the treatment of animal patients, and the Division encourages direct communication between the referring veterinarian and the physiotherapist providing animal rehabilitation services to their patients in order to ensure that both professional health care providers are aware of the clinical conditions, advice, treatments, and/or prescriptions provided by the other. At the present time, the practice of animal rehabilitation is not regulated by any Canadian provincial physiotherapy regulating group. For this reason, the Animal Rehab Division is in favour of discussions with various provincial veterinary regulatory bodies, to work towards the establishment of guidelines for the delivery of animal rehabilitation by physical therapists in order to provide the best and most professional services possible to ensure the well being of animal patients. At this time, the terms and titles of physiotherapy / physical therapy and physiotherapist / physical therapist are restricted to licensed physiotherapists engaged in the practice of human physical therapy, and hence the term animal rehabilitation is currently used to describe the practice of physical therapy in animals. However, the term animal rehabilitation is not a protected term, and lay persons engaged in massage, chiropractic, and aquatic therapy have been applying this term to their practices, causing confusion for the public as well as for referring veterinarians.

Veterinarians and veterinary technicians have entered the field of animal rehab as well. Some utilize their rehab training to provide rehab services to their own patients, to referred patients, and some vets find the rehab training enables them to provide more thorough musculoskeletal evaluation and some additional knowledge to prescribe advice or exercise therapies to their regular clients. Animal rehab is being included at veterinary conferences internationally, and especially in the USA, where there is even discussion of adding Animal Rehab as a potential boarded specialty in the future. As well, the International Veterinary Academy of Pain Management felt that rehab was such an important part of pain management, that they have instituted a component of their certification exam process that encompasses knowledge of rehab, and they have invited and welcomed physical therapists to become members of their organization.

As advances in veterinary medicine take place and as more
refined diagnostic tools and techniques become available to animal patients and more sophisticated surgical techniques are developed, greater emotional and financial investments are generally placed on animal ‘family members’ in our society. Therefore, expectations of longer animal life-spans and increased quality of life are being demanded by the general public. The addition of animal rehab and alternative therapies to the more traditional veterinary medicine practices for the treatment of animal patients appears to be an important step towards improving the overall quality of life and life-span of these animals.

So What Can Human Physiotherapy Research Tell Us?

When searching the literature for ‘physiotherapy’ or ‘physical therapy’, it is hard to find evidence for its use, however, when one searches different specific techniques or interventions (assessment or treatment, etc), then the research that underpins physiotherapy practice becomes more evident. Physiotherapy can be broken down into categories and subcategories. If we use the CPA Divisions as categories of practice (i.e. Orthopaedics, Neurosciences, Cardiorespiratory, etc), then we can further divide these into the subcategories of therapies or skills provided. For the purpose of time management, this lecture will cover only therapies addressed in the practice of orthopaedic physical therapy. These can include, but are not limited to, manual therapies, exercise prescription, and modalities. Other forms of orthopaedic treatments (not discussed within this lecture) can include fitting of assistive devices (splints, slings, or bandaging), biomechanical analysis (work place or sporting), acupuncture and dry needling, or first-responder duties (in the terms of sports physiotherapy) to name a few.

Manual Therapy

Manual therapy is actually a very broad topic itself. Included within this spectrum, are mobilization, manipulation, traction, range of motion, stretching, trigger point release/myofascial release, massage, and occasionally various osteopathic techniques (i.e. craniosacral therapy).

Mobilization and manipulation are often categorized together, while a manipulation is a high velocity low amplitude thrust, a mobilization is a gentler coaxing of a movement by passive rhythmical oscillations performed at the beginning, within or at the limit of range. Both techniques are generally utilized for the treatment of joint stiffness and/or joint pain (spinal or extremity). (Maitland et al 2005) Bronfort et al (2004) conducted a systematic review on the efficacy of spinal manipulation and mobilization for low back pain and neck pain and concluded that recommendations can be made with some confidence regarding the use of spinal manipulative therapy and/or mobilization as a viable option for the treatment of both low back pain and neck pain. However this paper and many others have stated that future studies need to be conducted on homogenous sub-classifications of neck or back pain patients. Not all spinal pain is the result of the same type of lesion, so to treat all lesions with just one treatment technique is bound to yield mixed and inconclusive results. Flynn et al (2002) studied a clinical prediction rule for identifying patients with low back pain: duration of symptoms less than 16 days; absence of symptoms below the knee; a Fear Avoidance Beliefs Questionnaire work score of less than 19; lumbar segmental hypo-mobility (as judged by downward pressure on the spinous process; and greater than 35 degrees of hip medial rotation in at least one hip. When these factors were taken into consideration (four of five of these variables), then the probability of success with manipulation went from 45% to 95% within one week of intervention! This clinical prediction rule was further validated by a randomized controlled trial by Childs et al (2004). The moral of the story is that if you manipulate all patients with spinal pain, you get so-so results, but if you manipulate the ‘right’ patients, you can get great results. Physical therapy research has recently shown very positive results in demonstrating prescriptive validity for a Treatment Based Classification system. The resulting treatment classification subgroups were specific exercises, manipulation, stabilization, and traction. Fritz and Brennan (2007) reported a Treatment Based Classification for neck pain, which categorized neck pain into groups of mobility, centralization, exercise and conditioning, pain control and headache. While this information is promising, more studies are needed.

Traction is a common technique employed for the treatment of disc disease in humans. Randomized controlled trials (RCT) on traction are frustrating to read. Again, unless patients with spinal pain are subclassified, then results are always inconclusive. Fritz et al (2005) found that patients that responded to lumbar traction are characterized by the presence of leg symptoms, signs of nerve root compression, and either peripheralization with extension movements or a crossed straight leg raise. Traction may have an important role in breaking the “cycle of pain” in cervical radiculopathy caused by herniated discs. The cycle begins when nerve roots are entrapped within the intervertebral foramina. Irritated nerves produce a reflex response to the patient’s cervical muscles, causing those muscles to contract, further narrowing the foramina and increasing neck pain. Intermittent traction helps relieve the inflammatory reaction of nerve roots by improving the circulation and reducing swelling to surrounding tissues. Gentle alternations of stretching and relaxation of soft tissue structures (such as with gentle traction) in the neck prevent the formation of adhesions of the dural sleeve. Human patients with radiculopathy symptoms lasting more than 12 weeks show less favourable improvements with traction, and early intervention is believed to be more successful. Exposure of a herniated disc material in the cervical spine (C/S) to the vascular...
environment of the epidural space contributes to its resorption and regression. Large extruded discs have wider exposure to resorption mechanisms and tend to regress more rapidly. The response to early therapeutic intervention in cases where there is a large extruded disc is therefore more favourable (Constantoyannis et al 2002; Malanga & Nadler 1999). Treatment protocols that include traction appear to be highly effective in individuals with lumbar pain related to a confirmed herniated lumbar disc with radiculopathy. Reports indicate that a treatment protocol which partly included traction as well as other physiotherapy interventions resulted in 90% good or excellent outcome and a in 92% return-to-work rate in 64 patients with CT scan-proven herniated lumbar disc and EMG-proven radiculopathy (Sal & Saal 1989). In another study, lumbar traction was most likely to be beneficial in patients with acute radicular pain of less than 6 weeks duration and concomitant neurological deficit (Krause 2000). As well cervical traction has been shown to have a positive impact (in combination with electrotherapy) in patients with radiculopathy. (Joghataei et al 2004)

Stretching and range of motion have been shown to be effective in increasing joint mobility about the knee, hip, trunk, shoulder and ankle joints including muscle length/flexibility. (Davis et al 2005; Decoster et al 2005; Knudson D 1999; Magnusson et al 1998; Power et al 2004; Thacker et al 2004.) Studies have shown that regular muscle stretching can improve eccentric and concentric force production, velocity of contractions, maximal volitional contractions, countermovement jump height, 50 yard dash and athletic performance. (Hunter & Marshal 2002; Shrier 2004)

One animal study even found that regular stretching can induce hypertrophy in immobilized muscles and another speculated that this effect may improve performance in the long term. (Coutinho et al 2004; Shrier 2004) Cook et al (2007) found that Labrador retrievers demonstrated a goniometric increase in osteoarthritic joint range of motion utilizing daily passive stretching. Technique may impact efficacy, as Meroni et al (2010) found that active stretching exercise was more effective than static stretching for increasing flexibility and maintaining the flexibility gains. A study on racing greyhounds found that dogs that had received race training had greater flexibility, possibly due to training having an active stretching role on muscles, tendons and other structures limiting the hip range of motion. (Nicholson et al 2007) Poor muscle flexibility has been equated with human knee injuries (Messier et al 2008) and reduced preseas on hip and knee range of motion has been correlated with a statistically higher risk for a muscle strain injury in soccer players. (Bradley & Porta 2007) There are no systematic reviews or RCTs that investigate the use of stretching as a stand-alone treatment for tendinopathies, muscle strains, or myofascial pain syndromes although its use is common in clinical settings.

Additional muscle therapies may include trigger point/myofascial release and/or massage. Myofascial trigger points (MTrP) have been described as "...a hyper-irritable spot in skeletal muscle that is associated with a hypersensitive palpable nodule in a taut band. The spot is tender when pressed, and can give rise to characteristic referred pain, motor dysfunction, and autonomic phenomena..." (Simons et al 1999) Several possible mechanisms can lead to the development of MTrPs, including low-level muscle contractions, uneven intramuscular pressure distribution, direct trauma, unaccustomed eccentric contractions, eccentric contractions in unconditioned muscle, and maximal or submaximal concentric contractions. (Dommerholt et al 2011) Research has show there is an increase in muscle activity, as recorded on EMG, in active myofascial trigger points. (Kuan et al 2007) Furthermore, muscles with active trigger points show changes in biochemical markers – increases in substance P, calcitonin gene-related peptide, bradykinin, interleukin-6, interleukin 1β, tumor necrosis factor–α, serotonin, and norepinephrine, and decreases in pH. (Shah et al 2008; Shah et al 2005) Using clinical diagnostic methods (tender

Table 1. Treatment options for Myofascial Pain

<table>
<thead>
<tr>
<th>Treatment technique</th>
<th>Finding</th>
<th>Reference</th>
</tr>
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<tbody>
<tr>
<td>Stretching</td>
<td>Passive stretching along with fluoromethane vapocoolant spray decreased pain and increased pressure pain threshold in people with myofascial pain (uncontrolled study)</td>
<td>Jaeger &amp; Reeves 1986</td>
</tr>
<tr>
<td>Dry Needling</td>
<td>Dry needling combined with active stretching exercises produced greater reduction in pain compared to active stretching alone or a no-treatment control.</td>
<td>Edwards &amp; Knowles 2003</td>
</tr>
<tr>
<td>Ischemic pressure</td>
<td>Ischemic pressure of a myofascial trigger point when combined with active ROM exercises has an immediate effect on reducing pain, increasing pressure pain threshold and tolerance and improving range of motion</td>
<td>Hou et al 2002</td>
</tr>
<tr>
<td></td>
<td>Both ischemic pressure and transverse friction massage significantly reduce pain intensity</td>
<td>Fernández-de-las-Peñas et al 2006</td>
</tr>
<tr>
<td>Massage</td>
<td>Both Thai massage plus stretches and Swedish massage plus stretches show significant reductions in pain and disability measures</td>
<td>Chatchawan et al 2009</td>
</tr>
</tbody>
</table>
Modalities

Several modalities exist in physical therapy practice. Some of the more commonly utilized modalities are laser, ultrasound, electrical muscle stimulation, TENS, and pulsed electromagnetic field therapy. Low-level laser therapy (LLLT) uses laser light to aid tissue repair, relieve pain, and stimulate acupuncture points. (Woodruff et al 2004; Enwemeka et al 2004; Siendentopf et al 2002) It’s general effectiveness can be attributed to anti-inflammatory mechanisms (which can be similar to pharmacological agents such as celecoxib, meloxicam, diclofenac and dexamethasone), the ability to reduce oxidative stress, improved angiogenesis, augmentation of collagen synthesis, and skeletal fatigue and inhibition of transmission at the neuromuscular junction. (Bjordal et al 2006; Chow et al 2009) Research into the use of LLLT for pain reduction and tissue repair spans more than 30 years, but only more recently have high quality reviews and meta-analyses been able to elucidate details on laser types and dosages that yield positive results. Chow et al 2009 revealed that for acute and chronic neck pain, the optimum dose per point for an 820-830nm laser was 5.9 Joules with an irradiation time of 39.8 seconds and using a 904nm laser, it was 2.2 Joules delivered with an irradiation time of 238 seconds. The number of repetitions and treatments per week were variable. Data from the reviewed trials suggested that positive effects were immediate and could be maintained for up to 3 months after treatment ended. Bjordal et al 2006 determined that LLLT at high doses (7.5 J/cm²) at the target tissue in the first 72 hours (to reduce inflammation) followed by the lower doses (2.1 J/cm²) at target tissues in subsequent days (to promote tissue repair) was most advisable. Each of these two authors noted that studies that reported negative results, also utilized inadequate doses, and poor laser exposure technique. As well, systematic reviews that did not employ procedural assessment of factors, such as dosage and exposure technique, should be disregarded.

Ultrasound is by far the most widely used physical agent currently available to clinicians in the US, Canada, Australia, England, and the Netherlands. (Belanger 2003) Ultrasound studies are also plagued with dosage and parameter insufficiencies. Alexander et al 2010 determined that studies that showed beneficial effects of ultrasound for shoulder pathologies typically had 4 times longer total exposure times and applied much greater ultrasound energy per session compared with studies that showed no benefit of ultrasound. A Cochrane Review paper concluded that ultrasound yields significant benefits to the healing of leg ulcers (Al-Kurdi et al 2008), and therapeutic ultrasound may have an effect on pain and loss of function in osteoarthritis. (Rutjes et al 2010) On the other hand, evidence for the effect of low intensity pulsed ultrasonography on healing of fractures is moderate to very low in quality, however overall results are promising. (Busse JW et al 2009) Additionally, there has been some encouraging results with low intensity pulsed ultrasound to promote healing in various soft tissues such as cartilage, inter-vertebral disc, and tenosynovial junctions, but the role of low intensity pulsed ultrasound in treating tendinopathies is questionable. (Khanna et al 2009)

Electrical muscle stimulation is used in clinical practice to strengthen muscle. However one research review has shown that use of electrostimulation is less effective on its own as compared to a superimposed or combined therapy with active exercise. (Dehail et al 2008) As well, for strength gains, it yields no higher benefits than traditional strengthening methods. The use of neuromuscular electrical stimulation to prevent muscle atrophy associated with prolonged muscle immobilisation following ligament reconstruction surgery or injury has been extensively studied. NMES has been shown to be effective in preventing the decreases in muscle strength, muscle mass and the oxidative capacity of thigh muscles following knee immobilisation. (Lake 1992) In animals, the primary use is the treatment of muscle atrophy, to underpin re-education of muscle function, and muscle strengthening. (Baxter & McDonough et al 2007) Rodent studies have identified morphological and histological properties of muscle with the use of electrical stimulation to prevent muscle atrophy. Boonyarom et al 2009 concluded that low-intensity, low-stimulation frequency (20Hz) could prevent atrophy of slow-stitch muscle fibers, and that short periods of low-intensity, high-stimulation frequency (30Hz) could prevent atrophy in fast-twitch muscle fibres. Dupont Salter et al (2003) revealed that remediation of disuse atrophy may be accomplished using unphysiologically low rates of motor-unit activation (2 Hz and 10 Hz). Transthecutaneous electrical muscle stimulation (TENS) is a modality utilized for pain relief. The two main mechanism by which electrostimulation produces pain relief are segmental inhibition through pain-gating mechanisms, and via descending inhibitory mechanisms. Animal models have produced studies that demonstrate that different frequencies of TENS produce analgesia through action on different neurotransmitters and receptors. (Sliuka & Walsh 2009) Essentially high frequency / conventional TENS (>60Hz) relies on the selective stimulation of larger diameter fibres in peripheral nerves, which in turn helps to ‘block’ nociceptive activity in smaller...
Exercise Therapy

General aerobic exercise has been demonstrated to produce hypoalgesia in healthy subjects (animal and human). However, duration and intensity of exercise is important (75% of VO2 max for 30 minutes) to achieve these results, and animals that run more (spontaneously) have higher thresholds compared with animals that run less. (Bement 2009) However, in chronic muscle pain, low-intensity exercise produces hypoalgesia through activation of the opioid system. (Bement & Sluka 2005) Strengthening exercises can be performed at a lower intensity than aerobic exercise to produce hypoalgesia. Systematic review show that exercise is beneficial for a variety of pain conditions including neck pain, chronic low back pain, pelvic pain, osteoarthritis, patellofemoral pain, intermittent claudication, fibromyalgia, rheumatoid arthritis, and tendonitis. (Bement 2009)

Specific strengthening and exercise prescription is a keystone in physiotherapy practice. A meta-analysis showed that pre- and in-season neuromuscular training with an emphasis on plyometrics and strengthening exercises was effective at preventing ACL injury in female athletes. (Yoo et al 2009)

Strengthening exercises, specifically, have been shown to be efficacious for hip osteoarthritis. (Hernandez-Molina et al 2008) Exercise combined with mobilization/manipulation, and exercise alone demonstrated either intermediate or long term benefits for mechanical neck disorders. (Gross et al 2007)

The Expanding Role for Physiotherapists in Human Health Care

Physical therapists have evolved their role as movement specialists. (Studer 2007) Studies have shown the benefits of pre-operative physiotherapy before total hip replacement or total knee arthroplasty to reduce hospital length of stay and modifying discharge conditions, and accelerated early functional recovery of patients immediately after total hip replacement. (Coudeyre et al 2007; Vukomanovic et al 2008) It is becoming more common for advanced practice physiotherapists to take on additional duties beyond those of a regular physiotherapist in order to screen patient pre- and post-operatively, triage patients for surgery, prescribe conservative management and monitor patients on an ongoing basis. (Aiken et al 2009) One Canadian study reported that advanced practice physiotherapists can effectively manage over 30% of patients referred to a surgeon for hip or knee replacement surgery because these patients do not require surgery; rather, they require conservative management. (Aiken et al 2009) An Australian study found that nearly two-thirds of patients with non-urgent musculoskeletal conditions referred by their GPs to one public outpatient orthopaedic department did not need to see a surgeon at the time of referral and were appropriately assessed and managed by experienced, qualified physiotherapists. (Oldmeadow et al 2007) Another Canadian study evaluated agreement between physiotherapists and orthopaedic surgeons for management of patients with hip and knee problems. (MacKay et al 2009) The researchers found that there was an agreement in 91.8% of cases, and in discordant cases, the physiotherapists tend to refer for consultation. Patients with hip and knee pain referred to orthopaedic surgeons can be appropriately referred for orthopaedic consultation by physiotherapists working in extended roles.
Conclusion

Physiotherapy is a profession, with a history of collaboration within the medical community. It strives to provide evidence-informed therapies and treatments and is active in research and knowledge translation. Physiotherapists are practicing in advanced roles in human practice and are contributing meaningfully to the expansion of veterinary medicine. Physical therapists are not only practicing animal rehabilitation collaboratively within the veterinary industry, but are leaders in educating veterinarians, veterinary technicians, physical therapists, and physical therapy assistants in how to practice animal rehabilitation. (Edge-Hughes 2009) Both physical therapists and veterinarians with training in animal rehabilitation are enhancing the health and well being of animals.

References


Among the first details for this session to address must be: “What exactly do we mean by the term Integrative Medicine?” You won’t find it in your printed dictionaries, but the online version of Webster’s includes this: “medicine that integrates the therapies of alternative medicine with those practiced by mainstream medical practitioners.” This is a fair, if incomplete definition, and a quick review of the semantics of our topics today may be helpful.

“Alternative” implies you can have one or the other: that both patient and practitioner must choose conventional medicine or Ayurveda, Traditional Chinese Medicine, or Osteopathy, for example. And if the phrase Alternative Medicine suggests an obligatory and unavoidable choice, then “Complementary” connotes that a therapy is a kind of therapeutic afterthought; that we may use when time, money, and convenience permit, but the conventional is still the main source of healing. The phrase “Integrative Medicine” is closer to the mark, and has the signal virtue of recognizing that the practitioner has conventional medical training, but also has additional knowledge and skills which can be used where deemed appropriate. Some prefer to discard these categories altogether by saying that: “There is no alternative medicine. Medicine either works or it doesn’t, and if it works it is no longer alternative.” Fair enough, but there will always be the difficulty of obtaining agreement on how we decide if a therapy works or not. The principles of evidence-based medicine will be of considerable help to us in sorting this out.

Our profession must be careful that in labeling new therapies and entire systems of medicine with which we are unfamiliar as “Alternative or Complementary” that we do not make the mistake of equating them: this has been done too often in the past, and you may still see examples of it today. Our own association’s (CVMA) Position Statement continues this error, and unwittingly lumps the sciences of acupuncture and physiotherapy with homeopathy and aromatherapy.

Today we will be hearing about the current state of published evidence for four of the most frequently-encountered kinds of therapy that very few of us were taught during the years we spent in veterinary school (although this is changing): Acupuncture, Chiropractic, Herbal Medicine, and Physiotherapy. Each of these is in the process of becoming an established part of health care for animals, and perhaps it will be helpful to review the essentials of who is doing these, where they learned it, and how the knowledge and standards for each discipline is developing.

A cautionary note: in the summaries below, I have given numbers and percentages regarding how many people do these things, which are probably no better than close approximations. There are multiple avenues for training and certification in all the disciplines being discussed here today, and reliable information regarding our profession’s level of interest and application of these therapies can be a little hard to come by. These figures may also be confounded by semantic as well as opinion differences. We may not agree about what constitutes a textbook in a particular discipline, for example.

In the U.S. there are 482 veterinarians in general practice who identify “Alternative / Complementary as their primary medical discipline.(1) Not a large number, almost exactly 1% of the total in general practice, but still, this number is greater than those whose principle activity is in animal behavior, dentis cardiologists, or dermatologists, and a number of other specialties. The number of veterinarians who use alternative or complementary therapies as a part of their practice, in other words who practice Integrative Medicine, is probably much higher.
Acupuncture

**World / National View:** This is a mature health discipline, and is widely recognized for use in humans around the world, though North America has been arguably the last region to embrace it. While there is substantial acceptance of its validity in human health care, it is often regarded as a treatment for pain only.

**Typical case:** Aged animals with chronic orthopedic pain, especially those which cannot tolerate NSAID therapy.

**Approximate advent into veterinary medicine:** late 1970’s.

**Who does this?** Mostly veterinarians; though there appears to be a degree of interest by human-licensed practitioners; most jurisdictions do not permit nonDVM practice without referral.

**How Many?** North America: Estimated 2,571 or 3.9%(3,4)

Note that AAVP surveys indicate 16% of its membership performs acupuncture, and more than twice that many refer clients to another who does. (2)

**Training:** annual courses in many countries (alternate years in Canada), all under either the IVAS or the Chi Institute syllabus.

**Qualifications:** 120 hours didactic and laboratory

**Textbooks:** 2

**Academic Status:**
- Preclinical: 10 North American Veterinary Colleges as a component of an introductory course in CAVM
- Clinical / Applied: 4 N.A. veterinary colleges

**Certification:** by written and practical exam under the auspices of the International Veterinary Acupuncture Society or the China National Society of Traditional Chinese Veterinary Medicine.

**Associations**
- Canada: AVAC
- U.S.A.: AAVA
- International: IVAS

**Summary**

Acupuncture is the longest-known, most established, and most accepted of the things called alternative in veterinary medicine. It is possible that the level of awareness and acceptance has reached the point that acupuncture is on the verge of being accepted as conventional medicine, or at least will likely be there in the next decade or so.

Chiropractic

**World / National View:** A mature health discipline, well-known throughout North America, somewhat less so in U.K., much less evident throughout Europe and Asia. Usually regarded as a treatment for pain or trauma of the spine. Accepted in all N.A. jurisdictions as an “entry portal” for human health care.

**Typical case:** Younger to middle-aged horse with an abnormal gait not due to lameness, or a performance problem which training fails to correct.

**Approximate Advent into Veterinary Medicine:** late 1980’s.

**Who does this?** Approximately half of animal chiropractic is provided by veterinarians, and half by chiropractors: this reflects a deliberate effort to develop the discipline by combining the knowledge base of each profession at the entry level.

**How Many?** North America: estimated 1,301 (1% of DVMs, about 1.2% of DCs). (5)

**Training:** annual courses in U.S., Canada, Germany. Nearly all under the Options for Animals syllabus.

**Qualifications:** 220 hours didactic and laboratory

**Textbooks:** none

**Academic Status:**
- Clinical / Applied: 5 North American veterinary colleges

**Certification:** by written and practical exam and provided by either the International Veterinary Chiropractic Association or, the American Veterinary Chiropractic Association).

**Associations**
- none in Canada;
- U.S.A.: AVCA
- International: IVCA

**Summary**

Chiropractic is not widely known among small animal owners, but is rapidly becoming established in almost any sport use of horses. Although it is gradually becoming more accepted by veterinarians at the general practice level, especially for horses, it will face continued resistance to recognition by the national professional associations due to 1) the “chequered past” it has in regard to political conflict with conventional human medicine, and 2) the long-established acceptance of DCs into its ranks of animal practitioners, both in training courses and certification.
Physiotherapy - Rehabilitation

World / National View: Although the most recent discipline to find application in animals, the well-established value of physiotherapy in human beings and the considerable infrastructure which it enjoys probably qualifies this group of therapies as having the highest recognition factor among the general public. Also, owners of animals which have had recent orthopedic surgery are more likely to be referred for physio and rehab due to the historical, and cooperative relations between surgeons and therapists. There may be a better awareness of physiotherapy available for animals in Europe than in North America.

Typical Case: canine athletes and post-op orthopedic surgery

Approximate Advent into Veterinary Medicine: early 1990’s.

Who does this? Mostly graduate physiotherapists, some veterinarians and veterinary technicians.

How Many?
Canada: 130 (6)
North America: 560 (member composition not known).

Training: 200 hours, canine or equine, in two recognized courses in U.S.: University of Tennessee, and the Canine Rehab Institute, Florida.

Qualifications: numerous options specific to canine or equine, and in part based on previous education/qualifications. All require animal-specific training.

Textbooks: 2, plus handbooks on specific equipment and techniques.

Academic Status: not taught in any Canadian veterinary college; preclinical introduction and use in the teaching hospital of 4 American colleges.

Certification:
DVMs: by the sponsoring educational program, or
Physiotherapists: by the Canadian Physiotherapy Association

Associations:
Canada: CPA – Animal Rehab Division
International: IAVRPT

Summary
Although physiotherapy and rehabilitation are two very different concepts, their close combination is logical and routine, and so they are often combined in training and practice. As animal owners obtain orthopedic surgery for their pets and athletes, and as surgeons gradually acquire an understanding of the benefits of physiotherapy, this field will continue to grow and become more available.

Other Considerations

Patient insurance: all the major providers will cover costs for acupuncture, chiropractic, and physiotherapy when performed by a veterinarian, or by employees of and within a practice. It is more difficult to know if coverage is provided when these therapies are done outside of the VCP relationship, and this detail should be confirmed with the specific carrier prior to start of treatment.

Provider insurance: Of more immediate concern may be that professional liability insurance can be very difficult for the lay practitioner (DC, physiotherapist) to obtain to practice on animals. If injury occurs to an animal following referral to a nonveterinary practitioner, your own liability insurance probably will not cover the situation, while legally your practice license does. The result may be a personal financial liability without insurance coverage of any kind.

An Introduction to Evidence-Based Medicine (EBM)

The concept of EBM is simple to state, though may be very difficult to apply in practice. There is no agreed definition for the term EBM, and there are important differences between those which may be found today.

Evidence-based medicine intends to bring the best available evidence gained from the scientific method to medical decision making. This does not exclude informed opinion or extensive clinical experience, for these are based on a kind of informal hypothesis-testing that is the basis of the scientific method. It is important to remember that evidence-based medicine is a goal which has not been achieved by either mainstream or alternative medicine. EBM is something we reach for, but will never achieve due to the constant introduction and mixing of new treatments with the established. For example, an understanding of pharmacogenetics or psychoneuroimmunology will ensure that EBM will never have an endpoint. It is enough that it is our goal, and though we may wish to practice by its principles, most of the time this won’t be possible.

All the varieties of EBM have in common the desire to bring the best information about the practice of medicine to the specific patient or client, and most definitions include the obligation to consider the values and preferences of the patient or client.
The conceptual and practical difficulties of EBM have been recognized in human medicine, if less well in veterinary medicine. The additional complications we face include the interposition of an animal owner between us and our patients, and of practicing medicine on patients which may not have the interests we think they do. Newer concepts which address some of these conflicts are being developed, and in the future you may encounter them as EBPractice, or EBTreatment, and ESupported Treatment.

Evidence Based Medicine requires collection of the available evidence, and sorting it by its particular value. This collection and weighing forms the basis to write a recommendation for therapy, or against a therapy, which may then be brought to the larger consideration of the particular patient’s circumstances. There are at present at least three different systems for rating the evidence on a particular point of medical procedure or therapy.

The US Preventive Services Task Force assigns each opinion or publication to one of five categories, with clear criteria for each.

**Level I:** Evidence obtained from at least one properly designed randomized controlled trial.

**Level II-1:** Evidence obtained from well-designed controlled trials without randomization.

**Level II-2:** Evidence obtained from well-designed cohort or case-control analytic studies, preferably from more than one center or research group.

**Level II-3:** Evidence obtained from multiple time series with or without the intervention. Dramatic results in uncontrolled trials might also be regarded as this type of evidence.

**Level III:** Opinions of respected authorities, based on clinical experience, descriptive studies, or reports of expert committees.

An understanding of the available evidence is converted to recommendations for clinical action according to the following scale.

**Level A:** Good scientific evidence suggests that the benefits of the clinical service substantially outweighs the potential risks. Clinicians should discuss the service with eligible patients.

**Level B:** At least fair scientific evidence suggests that the benefits of the clinical service outweighs the potential risks. Clinicians should discuss the service with eligible patients.

**Level C:** At least fair scientific evidence suggests that there are benefits provided by the clinical service, but the balance between benefits and risks are too close for making general recommendations. Clinicians need not offer it unless there are individual considerations.

**Level D:** At least fair scientific evidence suggests that the risks of the clinical service outweighs potential benefits. Clinicians should not routinely offer the service to asymptomatic patients.

**Level I:** Scientific evidence is lacking, of poor quality, or conflicting, such that the risk versus benefit balance cannot be assessed. Clinicians should help patients understand the uncertainty surrounding the clinical service.

A similar, but somewhat more clear description comes from the UK National Health Service

**Level A:** Consistent Randomized Controlled Clinical Trial, cohort study, all or none clinical decision rule validated in different populations.

**Level B:** Consistent Retrospective Cohort, Exploratory Cohort, Ecological Study, Outcomes Research, case-control study; or extrapolations from level A studies.

**Level C:** Case-series study or extrapolations from level B studies.

**Level D:** Expert opinion without explicit critical appraisal, or based on physiology, bench research or first principles.
The third system is from the GRADE Working Group [Grading of Recommendations Assessment, Development and Evaluation] offering, which differs from the first two for having a somewhat more clinically-oriented approach to reaching recommendations for action.

The overall quality of the published literature which evaluates a treatment will be categorized as high, moderate, low or very low, according to the following scale.

**High:** further research is very unlikely to change our confidence in the estimate of effect

**Moderate:** further research is likely to have an important impact on our confidence in the estimate of effect and may change the estimate

**Low:** further research is very likely to have an important impact on our confidence in the estimate of effect and is likely to change the estimate

**Very Low:** any estimate of effect is very uncertain.

The GRADE group then summarizes the view of the evidence for a treatment in a simple statement in one of two forms, the weak or the strong recommendation. It is important to note the inclusion of treatment risks, health service costs, and “patient burden” are a part of the final consideration of a treatment. The other two systems are deficient in one of these important determinants.

"**Strong recommendation:** Based on the available evidence, if clinicians are very certain that benefits do, or do not, outweigh risks and burdens they will make a strong recommendation.

**Weak recommendation:** Based on the available evidence, if clinicians believe that benefits and risks and burdens are finely balanced, or appreciable uncertainty exists about the magnitude of benefits and risks, they must offer a weak recommendation. In addition, clinicians are becoming increasingly aware of the importance of [owner] values and preferences in clinical decision making.” (8)

In the end, the practice of medicine is replete with daily examples where commonly used treatments lack objective support, and yet often obtain exactly the result we hope for. As previously stated, this situation should serve as motivation for us to continue to refine our knowledge of clinical practice through research. At the same time, we must accept that for most circumstances experience, opinion, and measured clinical estimates will always form the basis for many of our treatment decisions. In this regard, Integrative Medicine is no different from the conventional, it merely requires more of this kind of decision-making of its practitioners because they have more to consider in their decisions.

**References**

1. Professional Activity Analysis, 2009. From the American Veterinary Medical Association, internal documents.


Also: Grateful acknowledgement is offered here to Dr. Jim Kenney and Laurie Edge-Hughes for specific details of material presented above: Thank-you.
Overview of Veterinary Chiropractic Today

Animal chiropractic is among the more recent of human medical disciplines to be presented to us for consideration of its clinical utility in veterinary medicine. Chiropractic is one of the techniques to be found under the umbrella of manual or manipulative medicine, which includes osteopathy, Bowen technique, shiatsu, and perhaps even massage therapy. Chiropractic for humans is largely a North American discovery, and has been developed over the last century to include a number of modern techniques with a common purpose: mobilization of joints with concurrent proprioceptive inputs to the CNS which together may resolve problems related to local pain and neurologic dysfunction.

The history of chiropractic which eventually led to the respect and acceptance which it has today is a colorful one, including a spirited and ultimately successful defense against the open hostility of the allopathic medical community in the first half of the last century. It was toward the end of the 1970’s that Dr. Sharon Willoughby, a graduate of first the MSU veterinary college and subsequently the Palmer Chiropractic College, developed a modern curriculum for teaching veterinary chiropractic that continues to this day.

In the present day, chiropractic for animals is well-known in the horse world and by veterinarians who work with performance horses of almost any kind. In terms of number of animals treated, I suspect that at least as many small animals are treated for chiropractic lesions as are horses, but there is simply less awareness of it than is the case among horse owners.

In its present form, if not always its past, chiropractic is founded upon most of the same basic sciences which we all learned at least part of in our first two years of veterinary school, including the same anatomy, neurophysiology, and biomechanics which we require in conventional medicine. Chiropractic is much less concerned with pathology, however, than with function, and especially that of animal gait, motion, and any disability that may be a result of chronic musculoskeletal pain related to the spinal column.

A typical chiropractic consultation may vary from 15 minutes to an hour, and will include a specific hands-on evaluation of the condition of a number of soft-tissues and joints which are not ordinarily examined in the same way. An animal’s standing posture, static tone of epaxial and other paraspinal muscles, responses to dynamic movement and perhaps even a pain challenge will all provide important detail of the problem.

The essential feature of chiropractic therapy is the “adjustment” – a century-old term for a specific therapeutic manipulation of the patient which has been the subject of much laboratory and clinical research through the intervening years. An adjustment is a very brief impulse applied by the clinician’s hand or with an instrument, which is specific in direction, amplitude, force, and timing; adjustment is nonpainful. The chiropractic adjustment uses short-levers, and is high-velocity and low-amplitude. It provides 1) mobilization of a joint which is restricted in range of motion or in its biomechanical relationship with other, usually adjacent joints, 2) a significant local proprioceptive input through local muscle spindle cells. In combination and when applied to intervertebral joints, these two mechanisms will alter local neuromuscular tonus and change clinical signs of local pain or restrictions of normal movement.

The increasing use of chiropractic in human medicine is being matched by increasing research and acceptance. Research into the underlying pathology of vertebral subluxation complex and the effects of the manual adjustment is improving our understanding of how chiropractic achieves restoration of function and reduction of pain of spinal origin. And while the same may be said for veterinary clinical chiropractic, it must be acknowledged that before 1990 there was very little to be found in the modern published literature in this regard.
Current Veterinary Literature

Authors/Title:
Dyson & Murray: Pain associated with the sacroiliac region: a clinical study of 74 horses

Source:

Study Design:
Descriptive survey, selecting for horses with signs of sacroiliac region pain, with supporting scintigraphic or diagnostic local anesthesia signs

EBM Scale:
USPSTF: not applicable
NHS: not applicable

Author's Conclusions:
Careful clinical examination combined with scintigraphic evaluation of the SI joint region and local anesthesia can enable a more definitive diagnosis of SI joint region pain than has been previously possible.

Clinical Agreement: High

Clinical Relevance:
description of affected populations (Warmbloods, most cases are >163 cm in height, used in dressage and showjumping, numbers of racehorses are few.
The sacroiliac joint is the most often found restricted and adjusted.
Response to local anesthesia may be sufficient for diagnosis of SI joint pain

Authors/Title:
Haussler, Ayturk, Hill: Sacroiliac joint and pelvic deformation in horses

Source:

Study Design:
Descriptive survey, laboratory measurement of biomechanical features of cadaver equine sacropelvic articulations.

EBM Scale:
USPSTF: not applicable
NHS: not applicable

Author's Conclusions:
1) the equine pelvis is not a rigid structure,
2) the sacral axis of rotation is near to the SI joints,
3) pelvic displacement in response to loading was similar in three-axes of movement,
4) asymmetric pelvic deformation occurs during most sacroiliac joint movements.

Clinical Agreement: Unclear

Clinical Relevance:
important confirmation of clinical chiropractic theory with respect to elasticity and mobility of sacropelvic joints

Authors/Title:
Gomez Alvarez, Bobbert, Lamers, et.al.: The effect of induced hindlimb lameness on thoracolumbar kinematics during treadmill locomotion

Source:

Study Design:
Controlled, descriptive, instrumented gait measurements

EBM Scale:
USPSTF: not applicable
NHS: not applicable

Author's Conclusions:
1) Even subtle hindlimb lameness provoked detectable changes in thoracolumbar kinematics,
2) changes of thoracolumbar motion include increased range of motion and hyperextension,
3) hindlimb lameness also resulted in decreased range of motion of the lumbosacral segment and of the normal pelvic rotational motion

Clinical Agreement: Consistent with current opinion

Clinical Relevance:
High, supporting clinical impressions of back pain and dysfunction consequent to chronic hindlimb lameness
Authors/Title:
Haussler, Hill, Puttlitz, et al.: Effects of vertebral mobilization and manipulation on kinematics of the thoracolumbar region

Source:

Study Design:
Controlled, laboratory, randomized-crossover

EBM Scale:
USPSTF:  I
NHS:     B

Author’s Conclusions:
1) Passive vertical mobility of the trunk varies from cranial to caudal,
2) spinal manipulative therapy increased the amplitudes of dorsoventral displacement and applied force, indicative of increased vertebral (sic) flexibility and increased tolerance to pressure in the thoracolumbar portion of the vertebral column.

Clinical Agreement:
consistent with clinical observations and theory

Clinical Relevance:
if increasing thoracolumbar range of motion represents improvement of a clinical disorder, then chiropractic is a valid means of achieving such improvement (in support: 1) the specific treatment manipulation procedures used in this trial were high-velocity, low-amplitude, and 2) applied to vertebral dorsal spine contact points).

Author/Title:
Gomez Alvarez, L’Ami, Moffatt, et al.: Effect of chiropractic manipulations on the kinematics of back and limbs in horses with clinically diagnosed back problems

Source:

Study Design:
Prospective, observational, case series

EBM Scale:
USPSTF:  II-3
NHS:     C

Author’s Conclusions:
The main overall effect of the chiropractic manipulations was a less-extended thoracic back, a reduced inclination of the pelvis, and improvement of the symmetry of the pelvic motion pattern.

Clinical Agreement:
consistent with some clinical opinions

Clinical Relevance:
Chiropractic manipulations elicit slight but significant changes in thoracolumbar and pelvic kinematics. Some of the changes are likely to be beneficial, but clinical trials with increased numbers of horses and longer follow-up are needed.

Authors:
Sullivan, Hill, Haussler: The effects of chiropractic, massage and phenylbutazone on spinal mechanical nociceptive thresholds in horses without clinical signs

Source:

Study Design:
Prospective, controlled, clinical trial

EBM Scale:
USPSTF:  II-1
NHS:     B

Author’s Conclusions:
Chiropractic treatment and massage therapy increased spinal mechanical nociceptive thresholds within horses not exhibiting signs of lumbar pain: phenylbutazone did not.

Clinical Agreement:
Not applicable

Clinical Relevance:
Pressure algometry provides an objective tool to evaluate the effects of commonly-used, but currently unproven treatment modalities on spinal mechanical nociceptive thresholds.

Summary of Chiropractic Literature

The peer-reviewed veterinary literature describing the effects and clinical features of animal chiropractic has shown promising results from the recent interest of competent investigators. What cannot be known is the extent of any publication bias which prevents our knowing of unpublished research that might contradict anything described in the literature summarized above. What we do know is that the collected findings to date, though a small number, suggest that our profession will want to encourage further development of this discipline. If the concept of “one medicine” holds true for manipulative therapy, as it appears likely it will, we may well find as much to offer our patients in the future as humans have benefitted from chiropractic in the past.
References


Evidence-Based Acupuncture

Steve Marsden, DVM, ND, MSOM LAc, Dipl. CH CVA AHG

Introduction

The term acupuncture comes from the Greek term acus, meaning needle, and pungare meaning to pierce. It is the insertion of needles into specific points on the body to cause a desired healing effect. Acupuncture has played an important part in medicine in China for 2500 years, but appears to have been independently discovered and practiced as long as 5000 years ago in Western Europe. Acupuncture points contain no unique histological structures but do contain free nerve endings. The needles are extremely fine and metallic. A small electrical current is commonly applied to the needles to amplify their stimulation of afferent impulses in the free nerve endings.

The main healing effects sought by acupuncture are the relief of pain and modulation of inflammation. These are mediated by the central nervous system, the brain in particular, and can be classified as super segmental effects. Segmental effects also occur, and consist of immune modulation and the alteration of blood flow to locales reflexively linked to the needle site.

While not yet a recognized specialty, veterinary acupuncture has a significant following, with over 1700 practitioners being registered by its de facto certification body, the International Veterinary Acupuncture Society (IVAS; www.ivas.org). IVAS, based in Fort Collins, Colorado, is also the chief provider of training in the discipline worldwide.

While interest in veterinary acupuncture is substantial, and the technique widely practiced, clinical trials supporting the efficacy of acupuncture in animals are surprisingly lacking. A systematic review of 31 clinical trials of acupuncture in animals concluded that encouraging evidence exists for the management of pain and diarrhea. Positive intergroup differences were seen for spinal cord injury, Cushing’s syndrome, lung function, hepatitis, and rumen acidosis. Overall, however, the authors concluded that evidence from clinical trials supporting veterinary acupuncture was not compelling (Habacher et al, 2006).

Despite a lack of compelling research to date, interest in acupuncture among veterinarians is growing as more practitioners experience its positive effects first hand, either by having the technique performed on them, or by observing the successes of their veterinary acupuncturist colleagues. Most of these colleagues are not specialists in complementary medicine, but are general veterinary practitioners like themselves. Ultimately, the development of veterinary acupuncture originated from, and continues to be driven by, an ever-increasing public demand for the service (Scott, 2001). Certainly its very low rate of adverse effects (NIH, 1997) has facilitated experimentation with acupuncture and the acquisition of positive clinical experiences. To confirm and fully understand these suspected therapeutic benefits, we must look to medical research.

Acupuncture in Human Medicine

After reviewing the extensive body of research into the efficacy of acupuncture and its mechanisms, the National Institutes of Health (NIH, the main source of medical research grants in the United States) concluded as early as 1997 that acupuncture was a promising medical technique with “sufficient evidence of … value to expand its use into conventional medicine and to encourage further studies of its physiology and clinical value” (NIH, 1997). Research support for acupuncture has grown since then, with recent systematic evaluations of clinical trials reporting its apparent efficacy in:

• Chronic pain (Madsen et al, 2009)
• Obesity (Lin et al, 2009)
• Depression (Wang et al, 2008)
• Acne (Li et al, 2009)
• Tension headaches (Linde et al, 2009)
• Stroke rehabilitation (Shiflett, 2007)
• Knee pain (White et al, 2007; Bjordal et al, 2007)
• Osteoarthritis (Kwon et al, 2006)
• Low back pain (Manheimer et al, 2005)
In some conditions, acupuncture was recently found to be equivalent to drug therapy, namely:

- **Migraine prophylaxis** (Linde et al, 2009)
- **Insomnia** (Chen et al, 2007)
- **Post-operative nausea and vomiting** (Lee et al, 2009)

Almost invariably, however, the systematic evaluations qualify their positive assessments of acupuncture, citing a general tendency towards weakness and heterogeneity in study design that clouds interpretation and precludes meta-analysis. All papers call for confirmation of these positive trends through better designed clinical trials. For some conditions, these study weaknesses were prevalent enough to render inconclusive acupuncture’s value in:

- **Allergic rhinitis** (Roberts et al, 2008)
- **Dysmenorrhea** (Yang et al, 2008)
- **Erectile dysfunction** (Lee et al, 2009)
- **Vascular dementia** (Peng et al, 2007)
- **Smoking cessation** (White et al, 2006)

Interestingly, some of these are disorders the NIH consensus statement originally listed acupuncture as effective for, suggesting that design problems continue to plague even recent studies. Finally, acupuncture was found to lack support for use, again in contrast to earlier findings, in the treatment of fibromyalgia (Mayhew and Ernst, 2007) and intra-operative analgesia (Lee and Ernst, 2005). The latter is especially ironic, and difficult to accept, since it was the demonstration of acupuncture’s intra-operative analgesic properties in the 1970’s that originally sparked the interest of western scientists.

An interesting phenomenon that confounds both study design and conclusions of acupuncture’s effectiveness is its equivalence to placebo in many pain studies. Placebo in acupuncture studies is created by needling sham acupuncture points, as opposed to known true acupuncture points. The assumption was that if true points couldn’t relieve pain better than sham points, the improvements were not genuine. Recent studies, however, using positron emission tomography (PET) and functional magnetic resonance imaging (fMRI) have conclusively demonstrated that sham acupuncture has therapeutic pain-relieving benefits (Cho et al, 2006). In the figure below, (a) on the left is an fMRI image of cortical activity in response to a pain stimulus. The same stimulus is applied in (b), along with an analgesic acupuncture treatment. Figure (c) shows the same stimulus when applied simultaneously with a sham acupuncture treatment. Note the similar and only slightly lesser suppression of cortical activity in (c) relative to (b), illustrating even sham acupuncture’s substantial effectiveness at analgesia. Any of the above reviews that concluded acupuncture to be ineffective in pain management because of its equivalence to placebo require revision in light of this evidence.

### Analgesic Mechanisms of Acupuncture

Many of the systematic reviews supporting acupuncture efficacy are for painful conditions. There are two main mechanisms by which acupuncture effects analgesia. Both of these mechanisms have been extensively investigated and are quite well understood, and are enhanced with the passage of small amounts of electrical current through the acupuncture needle. These mechanisms are known as Spinal Gating and Descending Inhibition.

#### Spinal Gating

Spinal Gating takes advantage of the fact that there are three main first order neurons carrying afferent signals from acupuncture points to the spinal cord:

- **Aβ fibers** are myelinated fibers, and therefore fast conducting. They are responsive to thermal and mechanical stimulation. They help the organism react quickly and avoid damage from noxious stimuli, and synapse with second order neurons that propagate the pain stimulus to the thalamus and cerebral cortex. Once these signals have arrived in the cortex, the organism becomes conscious of pain.
  - **Aβ fibers** are also myelinated, and conduct signals quickly. They respond to lower thresholds of stimulation than Aβ fibers. They synapse on interneurons that inhibit the passage of pain signals in the second order neurons stimulated by Aβ fibers. Effectively, then, ‘close the gate’ to pain signal transmission. The benefits of Aβ fibers are especially clear when compared to the activity of C fibers.

- **C fibers** are unmyelinated and conduct at slow speeds. They lead to the perception of aching throbbing and burning pains that are stimulated by the presence of inflammatory compounds like substance P and platelet aggregating factor. They are the fibers of chronic pain, which almost invariably is due to inflammation.
Since Aβ fibers are faster than C fibers, stimulating them can reduce the discomfort associated with chronic pain through the same gate-closing mechanism, allowing a degree of desensitization to the pain of unresolved inflammation. In transcutaneous electrical stimulation (TENS), these non-nociceptive fibers are selectively stimulated by using particular frequencies. TENS, as an analgesic technology, was directly inspired by the earliest investigations of acupuncture’s pain-relieving effects.

The mechanisms behind Spinal Gating, and its interface with the other major analgesic mechanism of acupuncture, Descending Inhibition, are summarized in the figure below.

**Descending Inhibition**

Descending inhibition is an innate mechanism that facilitates the development of pain tolerance. Serotonin and noradrenergic mechanisms are involved. Acupuncture merely accentuates the effectiveness of this desensitization mechanism.

In Descending Inhibition, signals from nerves stimulated by acupuncture needles travel the same spinal cord pathways to the brain that pain signals traverse. As they pass through the periaqueductal grey matter (PAG) en route to the thalamus, descending pathways are activated by acupuncture that send signals back down the spinal cord. Here they both directly and indirectly inhibit further general nociception in the spinal cord laminae (see ‘E.’ in the above figure)

At the same time as inhibitory signals are descending the cord, endogenous opioids are released locally in the PAG that depress the pain response even if nociception in the laminae is not successfully dampened.

Not all forms of AP are equally effective for providing analgesia. Electro-acupuncture seems to best deliver stimuli that activate these powerful opioid and non-opioid analgesic mechanisms. Strong analgesic effects take perhaps a half hour to develop, but are relatively short-lived, often disappearing within minutes, and sometimes in a day. Yet studies of acupuncture efficacy in chronic knee pain demonstrate enduring relief of pain (White et al, 2007) even several months after treatment. Knowing the importance of C fibers and inflammation in mediating chronic pain, it becomes apparent that the transient mechanisms of Spinal Gating and Descending Inhibition are not alone responsible for acupuncture’s analgesic effects. In recent years, attention has begun to turn towards how acupuncture might have an anti-inflammatory effect as well.
Anti-Inflammatory Effects of Acupuncture

There is more direct evidence that acupuncture has an anti-inflammatory effect than its enduring effects in inflammatory conditions. Several studies have shown statistically significant declines in erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP, an inflammatory mediator) in clinical trials of acupuncture efficacy in rheumatoid osteoarthritis (Wang et al, 2008). In addition, traditional acupuncture compared favourably with sham acupuncture in the treatment of Crohn's disease, an autoimmune syndrome. While both patient groups perceived improvement, only traditional acupuncture was additionally able to lower levels of alpha 1 glycoprotein, a by-product of inflammation (Joos et al, 2004). In another study of the use of acupuncture in pelvic inflammatory disease, acupuncture was likewise found to be effective in reducing the level of inflammation in patients, as determined by significant declines in ESR and serum IgM (Wozniak et al, 2003). Other studies have shown clear benefits of acupuncture in the reduction of inflammation associated with asthma and rhinitis (Freek et al, 2003).

Despite ample suggestive data, an anti-inflammatory effect has been more difficult to consistently show in systematic reviews. Not all rheumatoid arthritis and asthma trials show the same anti-inflammatory effects, even when well designed. Unlike with analgesia, where a benefit is fairly easy to demonstrate with even sham points, modulation of inflammation may require much more careful point selection to consistently demonstrate. This introduces yet another confounding variable in the interpretation of acupuncture efficacy, in that points used in clinical trials are frequently chosen somewhat arbitrarily, and commonly vary even within the trial from patient to patient.

Recently, however, a method of point selection has been identified that is not determined by bias or opinion, but which seems to result in improved clinical efficacy. The technique stems from the fact that, in addition to free nerve endings, acupuncture points also contain an independent blood supply. Local accumulations of nitric oxide enhance the blood flow of points relative to surrounding tissues, allowing them to stand out on infra-red imaging. Not all points are injected with blood simultaneously, but particularly, it seems, those that are among the most therapeutic.

Two studies of the benefits of acupuncture in treating Bell's palsy, a facial paralysis condition arising from facial nerve inflammation illustrates the benefits of this point selection technique. In the study, one group of sixty patients had their acupuncture points chosen using infra-red imaging. Points showing a one half Celsius degree difference between one side of the face and the other were needled on the affected side, with re-evaluation at each treatment. Efficacy of the acupuncture treatment was then compared to outcomes in 120 control patients where points were chosen arbitrarily by practitioners, as in a typical experimental protocol. Patients with points chosen using thermography showed a significantly higher cure rate (68%) compared to the control group (46%) over the study duration (Zhang et al, 1991).

A dramatic difference was also seen in the speed of recovery for those cured cases, and the potential benefit of acupuncture made more clear, in a follow up study. Per the NIH fact sheet on the condition (http://www.ninds.nih.gov/disorders/bells/detail_bells.htm#109673050), Bell's palsy typically requires three to six months to resolve, regardless of treatment. The average treatment duration for acupuncture to affect a cure when points were selected using thermography was 6 weeks (requiring 25 sessions). The control group required an average of 24 weeks (requiring 79 sessions), suggesting minimal benefit in accelerating improvements (Zhang, 2007).

Cholinergic Anti-inflammatory Effect

The mechanisms behind the relief of inflammation using acupuncture have not yet been as extensively researched as its mechanisms of analgesia, since it was the analgesic mechanisms that first attracted the interest of scientists. The main anti-inflammatory mechanism receiving research attention is the cholinergic anti-inflammatory effect. This is once again a super-segmental effect and is an innate mechanism for the control of inflammation.

The dorsal vagal complex and the dorsal motor nucleus of the vagus nerve possess receptors for inflammatory mediators, including tumor necrosis factor (TNF), interleukin-1b (IL-1b) and high mobility group B-1 (Cho et al 2006). Binding of these cytokines promotes neural outflows via the autonomic nervous system and vagus nerve which negatively feedback on the inflammatory process to control its extent. Parasympathetic nerve endings release acetylcholine (ACh), which has numerous effects including the suppression of macrophage activity and IL-1b release.

Acupuncture to somatic afferents, rather than the vagus nerve directly, can likewise invoke this same mechanism. In addition, it can activate the hypothalamic-pituitary-adrenal (HPA) axis to increase cortisol secretion, resulting in a further dampening of inflammation.

Feng et al (2007), in their discussion of the use of acupuncture in the treatment of asthma, provide further clarification on how acupuncture to somatic afferents can invoke the anti-inflammatory cholinergic effect.

In asthma, neural growth factor (NGF) is one of the many compounds released by inflammatory and epithelial cells. NGF stimulates afferent nerves from the lungs to release substance P within the dorsal root ganglion. As the primary effective substance in airway neurogenic inflammation, Substance P then triggers efferent signals that result in a reflexive increase in airway inflammation and bronchoconstriction.
Acupuncture can modulate this effect, possibly in a manner similar to Spinal Gating. Stimulation of points on the dorsum (Bladder channel) may result in afferent signals to the dorsal root ganglion that activate inhibitory inter-neurons and block the effects of substance P in promoting neurogenic inflammation.

Even more likely is the possibility that substance P levels are depleted through repetitive acupuncture stimulation of the dorsal root ganglion through somatic afferents. This mechanism, proposed by Freek et al (2003), is likely the most important mechanism by which acupuncture limits inflammation, namely the alteration of blood flow.

**Reduction of Inflammation through Changes in Blood Flow**

Given that acupuncture is not immune suppressive, enduring relief of pain by acupuncture in autoimmune conditions like rheumatoid arthritis is likely achieved simply by regulating circulation through inflamed areas. Improved blood flow to areas of chronic inflammation allows removal of inflammatory mediators that otherwise stimulate C fibers to produce sensations of chronic pain. Conversely, decreasing blood flow can help limit damage arising from acute inflammation. Both objectives can be readily achieved with acupuncture, simply by altering the duration and intensity of needle stimulation. The mechanism behind this effect is likely quite simple, as summarized by Freek et al (2003), and involves only three chemical compounds, all of which are released from afferent nerve endings in response to acupuncture stimulation. The compounds are beta-endorphin, calcitonin gene related peptide (CGRP), and substance P. Their effects on blood flow are as follows:

- **CGRP** released from nerve endings increases local and systemic blood flow. Menopausal women have direct experience with this effect. During menopause, low gonadal hormone levels result in an up-regulation of CGRP receptors, resulting in numerous vascular menopausal complaints such as headaches (migraines) and ‘hot flashes’.

- **Substance P** released from nerve endings likewise increases blood flow, by triggering the release from efferent nerve endings of neurotransmitters that activate inflammatory cascades. In addition, nitric oxide is synthesized, heightening blood flow through the tissue. The pro-inflammatory effects of Substance P are somewhat self-regulating, with increases in nitric oxide inhibiting further mast cell degranulation, and with Substance P itself feeding back negatively on the release of CGRP by the neuron.

- **Beta-endorphin** is also released from nerve endings, but usually has an inhibiting effect on blood flow by activating T Helper cells to produce interleukin-10, a compound which interferes with vasodilation. Beta-endorphin also inhibits pain sensation.

These neurotransmitters are not all released equally upon afferent stimulation, resulting in an ability to manipulate vascular effects through the level of stimulus provided. For example, high frequency, repetitive, higher intensity (so-called sedative) acupuncture treatments will exhaust nerve ending supplies of Substance P and CGRP but increase beta-endorphin release. Beta-endorphins will thus dominate the vascular effect, limiting blood inflow and allowing control of acute inflammation.

Conversely, short duration, intermittent, low intensity (so-called tonic) treatments increase Substance P and CGRP levels, resulting in increased blood inflow. This can be an advantage in re-ordering the vasculature of chronically inflamed tissues; removing inflammatory mediators; and supporting tissue oxygenation and repair. Indeed, CGRP, as a mediator of angiogenesis (Toda et al, 2008), is perhaps crucial in fostering the re-orientation and organization of vascular beds following the mess made by acute inflammation, and likely has a critical role in ensuring the import of fresh supplies (e.g. fibroblasts) with which to repair tissues.

These vascular effects of acupuncture can be felt in sites distant to the point of needling, which Chinese medicine depicts as linked to the point itself by acupuncture channels or meridians (Zhang, 2007). It is to this effect on circulation that the Chinese medical classics repeatedly ascribed the beneficial effects of acupuncture on health, to the point on relying on pulse palpation and the color of the complexion as its most important patient assessment tools. Given this long assumption of its importance, and its more recent validation through medical research, this impact of acupuncture on blood flow may ultimately emerge as its most crucial effect.

**Summary**

Acupuncture, an effective analgesic, also shows promise as a highly safe anti-inflammatory treatment. The mechanisms behind this anti-inflammatory effect are still being elucidated, but the long tradition of use of acupuncture as a medical modality is increasingly validated by scientific research. Future investigations will likely further illumine these mechanisms and confirm its effectiveness in clinical trials for various painful and inflammatory disorders. Trials are especially likely to be successful if they incorporate recent discoveries of the effectiveness of even sham acupuncture in pain relief; and if they utilize infra-red technology and other objective tools in selecting the points to be treated.
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Evidence-Based Herbal Medicine

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Introduction

Interest among veterinarians in herbal medicine is growing, driven ultimately by dissatisfaction of pet-owners with conventional medical outcomes. In some cases, veterinarians are turning to herbal medicine of their own volition, to avail themselves of therapies that have heard have a reputation for success in the problems they are frustrated by, but where formal evaluation of efficacy is not yet apparent. Such conditions include conditions for which pharmaceutical therapy produces too many adverse events; and conditions for which effective drugs simply don’t exist. Others seek herbal medical training simply to be more informed of the therapies pet owners are taking it upon themselves to try.

The pharmaceutical industry has also stepped up research into herbal therapies, which are a logical source of new drugs. As an example, the National Cancer Institute screens thousands of plants annually, looking for new anti-neoplastic agents. Many of the plants are selected based on their use in traditional medicine. Neoplasia is a constant threat to plants, given the extreme oxidative stress they incur during the process of photosynthesis. As eukaryotes, plants share many of their basic metabolic pathways with animals, allowing compounds that work well in plants to have similar effects in animals.

In addition to cancer, plants face many other threats shared by animals, including the constant risk of bacterial and viral infection. In addition to antimicrobials, plants have an immune system with many similar traits to those of animals including the use of pattern-recognition receptors to identify pathogens. Like antibodies in animals, the complement of patterns recognized by plants is a heritable trait. Often, the same patterns are recognized as pathogenic, regardless whether the host is a plant or animal. Binding of antigens triggers the synthesis of chemicals that travel through the plant and generally rally plant immunity by triggering release of defensive compounds. These defensive compounds regulate the same types of chemical cascades that characterize animal immune systems. As in animals, invocation of these cascades results in the plant sustaining collateral damage in the process of warding off the invading pathogen. These commonalities in immune function between animals and plants support the notion they share a common origin, and also allow compounds used in plant immunity to have related effects in animals.

Many of these immune regulating compounds within plants are familiar to veterinarians and include steroids, salicylates, and nitric oxide regulators. Unlike in drug therapy, however, plants do not make large amounts of any one of these compounds, but exhibit an inherent economy of nature, in which small quantities of individual compounds play multiple physiological roles, and interact synergistically to create these effects. These synergies among trace levels of compounds help explain the general safety of plant medicines (see ‘Herb-Drug Interactions’ below), since toxicity is usually a dose-dependent effect of an individual molecule. It also explains the common inability by pharmaceutical companies to isolate active ingredients that perform as well as when they are in the herb. Often, then, to use the agents with maximum effectiveness, practitioners are left with no option but to use the herbs themselves.

Herbal Medicine in Companion Animals

While herbs are commonly used by small animal veterinarians, very few recent high quality clinical trials of their efficacy in dogs and cats exist. The situation is little better for horses, although some studies are available. The research can certainly be described as early and incomplete, but some conclusions are able to be drawn. Naturally most of the investigations in equine herbal medicine have centered around finding well-tolerated and efficacious alternatives to anti-inflammatory drugs. Ginseng has been found to exert an inhibitory effect on pro-inflammatory cytokines and cyclooxygenase-2 expression and holds potential as a herbal anti-inflammatory. Ginger has also been explored and found effective to minimize inflammatory post-exercise. Echinacea has been reported to have anti-inflammatory and antioxidant properties in the horse, while Yucca contains steroid-like saponins which produce anti-inflammatory, antioxidant, and anti-spasmodic effects. In race horses, care must be taken to ensure herbs don’t cross react with tests for prohibited substances. Regarding adverse events, few have been reported of a significant nature. It has been somewhat uselessly determined, however, that horses fed garlic at the rather extreme dose of greater than 0.2g/kg per day will develop Heinz body anaemia.
Research Challenges

In the absence of high quality clinical trials for small animals, veterinarians have looked to human herbal lore and research for inspiration. The amount of research articles pertaining to herbal medicine is comparatively extensive and includes clinical trials, laboratory studies, and case reports.

Herbal medical research is not without its challenges, however. For example, quality, origin, preparation, and dose are frequent confounding variables in interpreting whether a product that failed to show efficacy in a clinical trial is due to a lack of efficacy of the herb itself. As an example, consider Echinacea, an herbal extract frequently used for the common cold. It exhibits clear evidence of antimicrobial and immune-regulating effects in bench studies, but showed only weak effect in clinical trials, due at least in part to variability in manufacturing methodology. It is always important for researchers to completely characterize product and dose, to help differentiate studies where an herb has failed to perform from studies where a version of the herb is ineffective.

Another challenge to herbal medicine research is a very clear and acknowledged tendency to publication bias, even in peer-reviewed conventional medical journals. For example, a 2002 review identified more than 1400 randomized clinical trials (RCTs) and 47 systematic reviews of pediatric complementary alternative medicine (CAM) treatments. Formal evaluation has suggested that the quality of these RCTs is as good as those of conventional medicine, and the quality of systematic reviews of CAM exceeds that of systematic reviews of conventional medicine. Despite this high quality of research, reverse publication bias resulted in only negative studies being accepted for publication in well-known journals. Meanwhile, while foreign language journals are more likely to be publish positive studies, showing the bias goes in both directions. Naturally, however, the negative studies carry more weight because of the reknown of the journals where they are published.

Herb Drug Interactions

Publication bias is also quite evident in even a cursory review of the literature pertaining to herb-drug interactions. The highest level of evidence for efficacy and safety of a therapy is demonstration of its value across multiple clinical trials. These trials are subjected to scrutiny in meta-analyses and systematic reviews, in order to determine with the highest level of assurance whether research support truly exists for use of the therapy. A search for systematic reviews of herbal medicine on the NIH pubmed database does not result, however, in a list of reviews of the thousands of randomized clinical trials that have been performed on herbal medicine products. Instead a long list of literature reviews of potential herb-drug interactions is generated, many of which have never been observed in clinical settings or in vivo, but are demonstrable only in in vitro studies. Many of these articles are repetitive, citing the same studies and case reports of questionable relevance, rather than adding new information. The better of these articles acknowledge that the clinical significance of many of these proposed interactions are still uncertain at best and often fail to stand up to scrutiny.

For example, over half of the articles citing adverse herb-drug interactions pertain to the supposed anticoagulant effects of ‘the four G’s’: Panax ginseng, Garlic, Ginger, and Ginseng biloba. These herbs are believed to interfere with blood clotting, potentially raising the risk of hemorrhage intra-operatively or in patients receiving anticoagulant therapy. The number of studies citing this belief number in the hundreds, yet critical review reveals these effects are seldom clinically observed. Where case reports of the effect have been supposedly identified, scrutiny has ended up attributing the hemorrhagic effect to other influences. In the spirit of reverse publication bias, never amongst these articles is the speculated anticoagulant effect identified as potentially beneficial (for example, as an alternative to anticoagulant therapy or to inhibit platelet aggregation in patients prone to heart disease), but only as a risk to patients receiving surgical and anticoagulant therapy.

Perhaps the most clinically important proposed herb-drug interaction identified to date is the effect of Hypericum perforatum (St. John’s wort; SJW) on the induction of cytochrome p450 activity. This is rarely a concern as yet in veterinary medicine, since the main use of the plant thus far is as an anti-depressant. Its efficacy has been clearly demonstrated, and safety and tolerability studies have revealed that St. John’s wort preparations have better safety and tolerability profiles than synthetic antidepressants.

SJW does, however, induce the metabolism of other drugs, heightening their rate of hepatic clearance. Levels of cyclosporine, serotonin re-uptake inhibitors, oral contraceptives, and anti-retroviral drugs are particularly impacted.

A more unbiased appreciation of the uncommon incidence of adverse herb drug interactions and herb effects comes from review of the few systematic reviews of clinical trials that have been properly performed. These studies repetitively show a low incidence of side effects of the herbal preparations studied, with those side effects being almost invariably minor in severity and scope.

Other studies have been conducted to specifically quantify the actual incidence of adverse events and herb-drug interactions when herbal therapies are used in every day clinical settings alongside conventional medications. In one study, 194 patients receiving herbs from several different clinics were surveyed. A total of 20 patients reported 32 adverse events associated with Chinese herbal medicine over the 4-week period, none of them serious. The most commonly reported adverse events were diarrhea, fatigue, and nausea. Associations were not verified, just reported. Contrary to what would be expected, however, if the adverse events were
genuine, they declined in incidence as herb use continued, suggesting the adverse events were not genuine, but merely coincidental and ascribed initially to the herbs out of an initial sense of distrust.

Virtually all other surveys of this nature have uncovered similarly low rates and severity of adverse events. Another study surveyed all practitioners using acupuncture and Chinese herbs in Australia, regardless of profession or status (lay or licensed). One adverse event was recorded for every 633 acupuncture and herbal consultations combined. While low in general, the rate of adverse events was twice as high for MDs as for practitioners who prescribed according to traditional medical models, lending support to the continued use of these medical systems even as scientific data on the biomedical effects of herbal medicine accrues.

A third study compared the predicted versus actual incidence of adverse herb-drug interactions in 800 consecutive patients seen at 6 outpatient clinics. 122 used both herbal medicines and pharmaceuticals, and of these, only 12 reported adverse events. This low incidence was in contrast to the 85 adverse reactions speculated in the medical literature. Ironically, 8 of these 12 reactions were the result of herbs working too well, namely the lowering of blood sugar by Prickly Pear cactus in diabetics co-managed with insulin. Lowering of insulin dose eliminates the reaction.

Clinical Trials in Humans

For the most part, efficacy has been confirmed wherever systematic reviews of clinical trials of herbal medicine have looked for it. Some of the most recent systematic review results of potential interest to veterinarians are summarized below.

Pain

Avocado soybean unsaponifiables (ASU) were shown to have promising results in two studies and further studies would be desirable to verify efficacy. Saponification is the process of making soap from oil and lye. Unsaponifiables are small portions, less than 1%, of oil that is left over and cannot be made into soap. ASU is a mixture of one part avocado oil unsaponifiable to two parts soybean oil unsaponifiable. It is also called A1S2 and Piasclédine®. ASU was developed in France and first studied for its effect on osteoarthritis in the early 1990’s. Single studies of other interventions, a willow bark preparation (Reumalex), topical capsaicin and tipi tea, were inconclusive.

Patients in RCTs of ASU reported increased activity, reduced pain, and reduced need for pain medication. Taking higher doses of ASU did not improve outcomes. Reduction of cytokines was noted. ASU also contains a component that is associated with increased cartilage synthesis.

Even disc disease is amenable to herbal therapy. Chinese herbal medicine in humans has been shown to be effective for the management of chronic neck pain due to severe degenerative disc disease.

Gastrointestinal Disorders

Curcumin has recently been evaluated for the management of inflammatory bowel disease in people. Normally, systematic reviews are cautious in their wording, so the enthusiasm in this review is notable. The anti-inflammatory activity of curcumin stem from its comprehensive anti-inflammatory effects, including inhibition of cyclooxygenases 1, 2 (COX-1, COX-2); lipoxygenase (LOX); TNF-alpha; interferon gamma (IFN-gamma); inducible nitric oxide synthase (iNOS); and the transcriptional nuclear factor kappa B (NF-kappaB). It also is a strong anti-oxidant.

Clinical studies demonstrated a “striking suppression of induced IBD colitis and changes in cytokine profiles, from the pro-inflammatory Th1 to the anti-inflammatory Th2 type”. In clinical trials, patients with quiescent IBD given curcumin (360 mg/dose) 3 or 4 times/day for three months experienced a significantly reduced rate of clinical relapse. Randomized controlled clinical investigations in large cohorts of patients are still needed to fully evaluate its clinical potential.

Irritable bowel syndrome is a ubiquitous human complaint. While more studies are needed of higher quality to confirm efficacy, clinical trials showed the ability of herbal medicine to relieve many of its most common symptoms, including abdominal pain, diarrhea, and constipation.

Renal Disease

One of the surprising arenas where herbal medicine has been shown to be beneficial is in the treatment of renal failure and nephrotic syndrome in humans. Veterinarians have experienced success in renal failure using Rehmannia and Bupleurum roots; the latter has significant anti-inflammatory effects and has been helpful in glomerulonephritis. What is especially interesting about the human studies is that the improvements in renal inflammation were achieved with Astragalus (Huang Qi), which is a powerful immune stimulant.
Hyperthyroidism

Hyperthyroidism is another arena where herbal medicine has shown benefit. Veterinarians have been using Chinese herbal medicine to stabilize, reverse, and prevent feline hyperthyroidism for several years. RCTs in humans suggest its efficacy enough to warrant its use as a therapeutic adjunct. Better designed studies are needed.

Diabetes Mellitus

Another endocrinopathy for which herbal medicine has been shown to be effective is diabetes mellitus. Studies of these herbs are becoming increasingly important as the incidence of diabetes rises, although RCTs are surprisingly uncommon. Much of the justification for supplement use comes from bench studies, which are numerous. The most commonly used and researched medicines include Bitter Melon (Momordica charantia), Fenugreek (Trigonella foenum graecum), Gymnema Sylvestre, Ivy Gourd (Coccinia indica), Nopal or Prickly Pear Cactus (Opuntia streptacantha), Ginseng, Aloe Vera, Russian Tarragon (Artemisia dracunculus), and Garlic (Allium sativum). While data is encouraging, lack of clinical trials means it is premature to actively recommend them for diabetes. There is sufficient evidence, however, to conclude that Chinese herbs are sufficient to normalize blood glucose in pre-diabetic states of impaired glucose tolerance and impaired fasting blood glucose.

Epilepsy

Tian Ma (Gastrodia) has been used for decades by veterinarians as a component of protocols for the treatment of idiopathic epilepsy in dogs. Research attention has also been extended to the symbiotic fungus Armillaria mellea that permits Tian Ma, an orchid, to survive. Antiepileptic properties have been identified no benefits of herbs studied. Studies of the treatment of epilepsy in dogs reportedly demonstrated the AED effects of vanillin, an isolate of Tian Ma. Reviews conclude Tian Ma and Armillaria hold promise as cost-effective and less toxic alternatives to standard AEDs, and as an inspiration to future drug development.

Infections

In human medicine, there is a temptation to over-prescribe antibiotics to appease patient demand and prevent bacterial upper respiratory infections. Because of the tendency toward antibiotic abuse, interest has turned towards the use herbal antimicrobials as a way of minimizing the development of bacterial resistance to unnecessarily prescribed antibiotic drugs. Systematic reviews have focused on the use of the leaves of Andrographis paniculata, an antimicrobial herb that is also being increasingly used for the treatment of Lyme’s disease in dogs. Andrographis was shown superior to placebo in the treatment of clinical cases of upper respiratory infection. There is also preliminary evidence it is effective to prevent infection. Adverse events reported following administration of A. paniculata were generally mild and infrequent, making it a serious candidate for solving the problem of antibiotic overuse in respiratory infection.

Another gap in the anti-infective arsenal is the lack of safe effective antiviral drugs. Plants are prone to viral infection and manufacture several compounds that afford them protection. These have been recently reviewed and studied. Many hundreds of herbal preparations with antiviral activity were identified, yet extracts from only 11 species met the inclusion criteria of this review and were tested in clinical trials. Only four of these 26 trials reported no benefit from the herbal product under study. Despite this encouraging study, it is interesting to note that no benefits are yet able to be claimed for the use of Chinese herbs in bronchitis or the common cold.

Lastly, there are lingering concerns about the potential for particularly pathogenic species like Salmonella to become resistant to our antibiotics arsenal. In one study, routine culture and sensitivity testing showed efficacy of Schizandra fruit extract against 16 different strains and 6 serotypes of Salmonella. Methanolic extracts were the most potent.

Next, the in vivo antibacterial activity of Schizandra fruit was examined for S. Typhimurium infection in mice. Mice were initially infected with S. Typhimurium, and then administered with herbal extract. The extract was found to markedly reduce mortality and the numbers of viable S. Typhimurium recovered from feces. Clinical signs and histological damages were rarely observed in the treated mice, whereas the untreated controls showed clinical signs such as lethargy and histological evidence of damage to the kidney, liver, intestine, and spleen.

Skin Disease

While one of the most important applications of herbs in small animal medicine is the treatment allergic skin disease, an evidence-based review of pharmacotherapy of skin disease in general identified no benefits of herbs studied. Studies of the treatment of herbal therapy for skin disease in dogs are prone to error, however, unless they span a period of several months, since improvements often appear only gradually. In addition, any systematic review should include a comparison of side effects and the permanence of improvements in final assessments of safety and efficacy.

Studies of herbal therapy in human skin disease have been more encouraging. The herbs studied were supported by a clinical lore stemming from many centuries of use in Asia and Europe. The paper reviewed the scientific evidence supporting the use of some of these therapies in skin disorders, together with precautions and recommendations regarding their prescription.
Cancer

Use of herbs in cancer warrants particular discussion, due to the frequent sweeping generalization by oncologists that the antioxidant effects of herbs and supplements will interfere with conventional treatment efficacy, and should thus not be used in patients receiving chemotherapy or radiation therapy. This generalization has not been substantiated in multiple studies. While some potential for interference exists on a case-by-case basis, many specific herbal and dietary supplement strategies interact synergistically with conventional therapies. Interdictions should not be sweeping, but more nuanced. Mechanisms generating synergistic effects are multiple, but some of the most important include the blocking one or more targets of the signal transduction pathway; increasing the bioavailability of chemotherapeutic drugs; and the stabilization of chemotherapeutic drugs.

Negative Studies

Some studies found no evidence for particular herbal strategies to work. Many times these are products heavily promoted by the lay public but some, ironically, are those extensively used by even conventional veterinarians. Negative studies are not necessarily ‘proof positive’ that a herb doesn’t work, but when repeated using many versions of the herb, or when they are systematic reviews of many studies, they make it more likely.

Essiac

One such systematic review is for Essiac, a herbal formula commonly advocated for the treatment of cancer. It is not as popular a strategy amongst clinicians, however, in part because its constituents are not particularly known for their anti-cancer effects when used individually. A review of several trials, most of which were low quality, failed to support Essiac’s anti-cancer claims. Weak evidence from preclinical, animal, and laboratory data warranted a discussion in the paper regarding Essiac’s use for cancer, but the results of even these studies are inconclusive. Some of the blame for the lack of results may lie in variability of ingredients among different versions of the product. Although the ‘lore’ of the formula’s development is that it is a combination of herbs traditionally used by the North American Ojibway population for the treatment of cancer, and passed along to Canadian nurse Rene Caisse (Essiac backwards), the formula has shown substantial variability in its constituents since then. Since there is substantial product variability, safety data for all its forms is lacking.

Weight Loss

Another popular lay treatment, this time for weight gain, is Citrus aurantium. Attention turned to the plant (citrus peel) following the removal of Ephedra from the market as a weight loss supplement. Since Citrus aurantium, contains some of the same synephrine alkaloids as Ephedra, it was hoped it would prove a safe alternative to Ephedra in appetite suppression and weight loss. Only 1 eligible randomized placebo controlled trial was found for the product, which followed 20 patients for 6 weeks. The RCT demonstrated no statistically significant benefit for weight loss.

Milk Thistle

Some other negative studies may surprise veterinarians. Several have looked at the use of milk thistle for chronic active hepatitis in humans. Despite a surprising lack of research evidence in humans, the plant is increasingly marketed even by conventional pharmaceutical manufacturers for the treatment of liver disease, and is in common usage by conventional veterinarians. One study at the University of Calgary specifically examined its efficacy in chronic active (viral) hepatitis in humans. No antiviral activity was noted, and its active ingredient, Silymarin, resulted in a decrease in serum transaminases compared with baseline in only four of seven studies, and compared with placebo in only one. Since active ingredients may not be as effective as whole herb extracts, due to the inherent loss of synergies between primary and secondary constituents, it is prudent to also examine the efficacy of the whole herb extract in treating liver disease. Another study looked for evidence of milk thistle efficacy by examining its impact on mortality, histological findings on liver biopsy specimens, serum aminotransferase and albumin levels, and prothrombin times in patients with liver disease. The only statistically significant difference was a greater reduction in alanine aminotransferase levels among patients with chronic liver disease assigned to milk thistle (-9 IU/L, 95% CI: -18 to -1 IU/L; P = 0.05). This reduction was of negligible clinical importance and no longer statistically significant after limiting analyses to studies of longer duration or of higher quality. The frequency of adverse effects was low and, in clinical trials, indistinguishable from placebo.

Equivalence to Drug Therapy

In contrast to these negative studies, there are also positive studies where herbs have performed as well as conventional medical treatment, but with a reduced risk of adverse events.

Low Back Pain

One such study was in the treatment of low back pain. White Willow bark and Devil’s Claw both relieved acute and chronic low back pain equivalent to Vioxx in six week studies. In addition, Capsicum and an over-the-counter homeopathic preparation both exceeded benefits of placebo, but were not compared with conventional treatment. It’s worth noting that these therapies were shown to not be effective in the treatment of pain from osteoarthritis (see ‘Pain’, above).
Vioxx equivalency was established in high quality trials evaluating a total of over 1500 patients. Vioxx has since been withdrawn from the market following its implication in many cardiac deaths.

**Nausea and Vomiting**

Ginger has a long history in many medical traditions of use in the treatment and prevention of nausea and vomiting. A systematic review of RCTs validated this effect. Three were of post-operative nausea and vomiting. Two of these trials suggested that Ginger was superior to placebo and equivalent to metoclopramide in efficacy. One trial each validated its efficacy in the treatment of nausea due to seasickness, morning sickness, and chemotherapy. The diversity of indications stems from Ginger being a centrally and locally acting anti-emetic, as well as an anti-inflammatory, anti-oxidant, and smooth muscle anti-spasmodic.

**Depression and Anxiety**

Mention has already been made (see ‘Herb Drug Interactions’, above) of the efficacy and safety of Hypericum (St John's Wort) in the treatment of depression. Another plant, Kava (Piper methysticum) has been shown to be equivalent to the efficacy of commonly used drugs in the treatment of mild to moderate anxiety. Unlike with many of these pharmaceuticals, however, withdrawal symptoms are not observed with Kava use. Kava is a social and ceremonial herb from the South Pacific. It is available in the west as an over-the-counter preparation. Its biological effects, due to a mixture of compounds called kavalactones, are reported to include sedative, anxiolytic, antistress, analgesic, local anaesthetic, anticonvulsant and neuroprotective properties. The pharmacological properties of kava are postulated to include blockade of voltage-gated sodium ion channels, enhanced ligand binding to gamma-aminobutyric acid (GABA) type A receptors, diminished excitatory neurotransmitter release due to calcium ion channel blockade, reduced neuronal reuptake of noradrenaline (norepinephrine), reversible inhibition of monoamine oxidase B and suppression of the synthesis of the eicosanoid thromboxane A(2), which antagonises GABA(A) receptor function.

Adverse events from properly manufactured Kava are negligible. Extracts improperly made from toxic (and normally discarded) stem peelings by European pharmaceutical companies resulted, however, in several cases severe liver toxicity in Europe and the US. The European drug company was attempting capitalize on the growing popularity of Kava and seized the opportunity to purchase discarded leavings of the plant cheaply, unaware there are known to be toxic. Properly manufactured Kava rhizome extract remains safe and has returned to at least the US market place. It has a clinical reputation of effectiveness in the treatment of thunderstorm phobia in dogs.

**Herbal Medicine in Agriculture**

A surprising arena of research that is validating the effectiveness of herbal medicines in a wide variety of animal species is the examination of their value in animal agriculture. Interest in herbal medicine in the livestock industry has grown with the rising demand for certified organic milk, eggs and meat. Organic farming regulations often preclude the use of pharmaceutical agents, even though husbandry conditions are still often intensive. Herbal medicines have been found to be able to replace pharmaceuticals in these circumstances by promoting growth; preventing and treating infection; and eradicating mastitis. They are also of particular interest to developing nations where access to pharmaceuticals is sometimes inconsistent.

This economic incentive has stimulated many studies of herbal efficacy in cattle and poultry in particular. Such studies are often high quality and highly reliable due to:

- The use of large sample sizes
- The ability to ethically establish test and control groups
- The large number of metrics (e.g. growth rate, organ weight, etc.) that can be ethically obtained to help ascertain or disprove a herb's physiological and medical impact
- The ability to do challenge testing of resistance to infectious diseases
- The short life spans of the models, allowing more rapid completion of studies
- The freedom from philosophical bias created by an emphasis on an economically significant impact
- The simultaneous assessment of herb safety

As in human medicine, benefits have largely been shown wherever they have been looked for.

**Mastitis**

Due to the growing demand for organic milk, the treatment of mastitis using herbal medicine has become an important area of research. Interestingly, herbal preparations have not only had similar benefits to drugs in limiting infection, but have added immune stimulating effects that pharmaceuticals do not typically achieve.

As an example, Ocimum sanctum (Holy Basil), an Ayurvedic herb, was administered by intra-mammary infusion to assess its ability to resolve bovine subclinical mastitis. The preparation was shown to reduce the TBC and increased neutrophil and lymphocyte counts while enhancing their phagocytic activity and phagocytic index. The lysosomal enzyme content of the milk polymorphonuclear cells (PMNs) was also enhanced significantly in animals treated with the extract.
Similar studies have been performed using other plant species. The efficacy of a hydro-methanolic extract of Azadirachta indica (Neem, a plant antimicrobial) was studied for bovine clinical mastitis (CM). Treatment significantly decreased the SCC, TBC, and milk neutrophil percent and significantly enhanced milk lymphocyte percent, H(2)O(2) and O(2) (-) production by milk cells. In addition, the immune stimulating cytokines IL-2 and IFN-gamma were expressed in normal healthy cows, and diseased cows after A. indica treatment, whereas both cytokines were not expressed in cows treated with antibiotic only, and in cows receiving no treatment at all. In short, the study pointed to the herb possessing significant anti inflammatory, antibacterial and immuno-modulatory potential in the treatment of mastitis.

**Poultry Science**

Studies in poultry science have focused on increasing resistance to common parasites and infections; and to improving calcium metabolism and bone strength in laying hens.

The effects of herbs on osteoporosis and calcium metabolism were investigated by administering Gu Shu Kang (GSK; literally, Strengthen Bone Combination) to end-of-lay hens. One thousand birds were divided into treatment and control groups. While preserving bone integrity, GSK also significantly increased the egg laying rate and decreased the percentage of cracked eggs. The serum calcium, phosphate, and alkaline phosphatase were decreased in the GSK-treated group compared with the control group, whereas bone characteristics were significantly improved. The authors concluded GSK can improve egg production and prevent bone loss by inhibiting bone turnover.

Several studies have investigated herbal extracts as primary and adjunct methods of treatment of Eimeria infection in poultry. Immune polysaccharides from various mushroom species and Astragalus (Huang Qi) have been particularly studied. Dosing regimens were notably modest, yet significantly higher levels of antibodies specific to Eimeria were noted in treated birds versus controls within two to three weeks.

The same authors next explored the possibility of whether these immune stimulating extracts could be used as an alternative to virginiamycin in the growth promotion of broilers. Growth promotion exceeded that seen from VRG until day 21, when the herbal extract had an effect equivalent to drug therapy.

Lastly, the team explored the ability of the same extracts to treat Eimeria infection in comparison to the effects of live oocyst vaccination. The extracts did not perform as well as the vaccine in preventing loss of body condition and weight loss. Weight gains were higher in the herbally treated group than untreated controls, however. The most impressive weight gains were seen in the populations given both the herbal extract and the vaccine, suggesting that the immune stimulating effects of the extracts heightened the immune response to the vaccine, creating a synergistic effect.

**Conclusions**

Reverse publication bias has been diligently avoided in this review of the most rigorously designed clinical trials of herbal extracts in human and animal medicine. Despite this, it is quite clear that herbal medicine efficacy is not confined to particular species or conditions. There are vastly more studies finding clear evidence of efficacy than those that do not. Dog and cat studies are notably lacking, however, in the medical literature. It is important that impressions of herbal efficacy in dogs and cats be subjected to scrutiny through well designed randomized clinical trials. On balance, it is clear that the weight of evidence in support of herbal medicine efficacy is much higher and study conclusions much less equivocal than for other modalities like acupuncture which, ironically, has been embraced by veterinarians as ‘more scientific’.

One oft-quoted concern by veterinarians is the risk of adverse events and adverse herb-drug interactions following use of herbal medicine. Studies of the actual rate of incidence suggest this risk of significant adverse events is very low, despite an abundance of speculative literature to the contrary. It is hoped that the well documented bias of the mainstream medical literature against herbal medicine will disappear in the years to come, and the normally high editorial standards of these journals more consistently applied. Likewise it is hoped that foreign journals continue to increase their rigor with which they review articles submitted for publication in order to avoid a historical bias towards printing any favourable studies.
APPENDIX

While veterinarians await high quality small animal research, the best sources of information on how to integrate herbs into small animal medicine are numerous formal training programs, as well as consultation with acknowledged experts on a case-by-case basis. Some major resources include:

**College of Integrative Veterinary Therapies**
- Leading provider worldwide of distance learning programs in alternative medicine for veterinarians
- Distance-learning courses in herbal medicine
- Introductory and certification levels available
- In-person training sessions and individual tutors are provided as part of certification training
- Courses count toward internationally recognized post-graduate degrees
- Major provider of education to North American and Australian veterinarians in herbal medicine
- Chief provider of education in Chinese veterinary herbal medicine to SE Asia, including China, Malaysia, Taiwan, and Thailand
- [www.civtedu.org](http://www.civtedu.org)

**Natural Path Herb Company**
- Supplier of high quality organic herbal formulations, exclusively by veterinarians, for veterinarians
- Brand and modality independent case-by-case advice to maximize efficacy while minimizing risk
- Forum to facilitate exchange of information and networking
- On-line consulting tools
- Becoming the VIN of alternative medicine by serving as a location where clinical experience can be shared
- [www.nphc.ca](http://www.nphc.ca)

**A Time to Heal**
- In-person training programs in veterinary herbal medicine
- Both beginner and advanced programs available
- [www.atimetohealherbs.com](http://www.atimetohealherbs.com)

**Veterinary Botanical Medical Association**
- [www.vbma.org](http://www.vbma.org)
- Active discussion list for a variety of topics related to veterinary herbal medicine
- Regular webinars
- Networking and fellowship
- Sponsors tracks at major veterinary conferences including NAVC and the AHVMA

**American Holistic Veterinary Medical Association**
- [www.ahvma.org](http://www.ahvma.org)
- Active discussion list for a variety of holistic modalities, including herbal medicine
- Annual conference opportunity for in-person training
References Cited


11 Arch Fam Med. 2000 Nov-Dec;9(10):1071-8

12 Altern Ther Health Med. 2007 Mar-Apr(13(2):30-5


