SPEAKER SCIENTIFIC PRESENTATIONS 2012

DRY LABS – Wednesday, July 11, 2012

Dr. Brian Beale, DVM, Diplomate ACVS
Cranial Cruciate Ligament Stabilization Using Novel Extracapsular Stabilization Technique (Dry Lab)
Tears of the cranial cruciate ligament are the most common orthopedic injury seen in dogs and cats. Extracapsular repair techniques are an effective method of stabilizing the cruciate-deficient stifle. This comprehensive hands-on laboratory will discuss the indications and theory of properly positioned prosthetic cranial cruciate ligaments using new implant systems, the latest design in suture anchors and knotless ACL repair. The advantages of these techniques are increased strength of repair, ease of application, and affordability. Meniscal diagnosis and treatment will also be addressed.

Mrs. Maria Cecere, AHT, CCRP
Therapy Lasers in Veterinary Medicine Workshop
This combination lecture and interactive lab is an introduction to the use of a Class IV therapy laser in small animal practice. New and prospective users will learn how diode lasers operate and how Class IV laser light interacts with tissue to reduce pain, reduce inflammation, and accelerate healing. Participants will learn to operate a Class IV therapy laser and can practice performing everyday laser therapy techniques.

Dr. Laurie Dunbar, DVM, CCRP
Therapy Lasers in Veterinary Medicine Workshop
This combination lecture and interactive lab is an introduction to the use of a Class IV therapy laser in small animal practice. New and prospective users will learn how diode lasers operate and how Class IV laser light interacts with tissue to reduce pain, reduce inflammation, and accelerate healing. Participants will learn to operate a Class IV therapy laser and can practice performing everyday laser therapy techniques.

Dr. Alicia Karas, MS, DVM, Diplomate ACVA
Video Laboratory on the Assessment of Pain in Dogs and Cats (morning only drylab)
Pain assessment in our practice is something we all want to do well. There is no gold standard measurement of pain, but most experts agree: studying behavior is the best way to assess pain in animals. Therefore, training yourself and caregivers to observe and communicate your findings is essential. The goal of this laboratory is to teach attendees ways in which we can assess pain, and to differentiate pain from anxiety and dysphoria, using video clips of canine and feline patients. We will also incorporate an interactive discussion of the ways in which you can skillfully reduce negative experiences for your patients with acute pain by blending pharmacologic therapy with physical, environmental, and emotional support. Technicians and veterinarians are welcome.
A SUCCESSFUL CAREER, A BALANCED LIFE – Wednesday, July 11, 2012

Dr. Jane Shaw, DVM, PhD

Four Core Skills of Highly Effective Practitioners

Not available at time of printing.
From Paternalism to Partnership

Introduction
Veterinarian-Client-Patient Relationship
Jane R. Shaw, DVM, PhD

Introduction
To set the stage for the conversations that follow in this book, it is helpful to take an in-depth look at the veterinarian-client-patient relationship. This relationship serves as the foundation for all that we are trying to achieve in veterinary medicine, including satisfying the client, caring for the animal and promoting professional fulfillment. The dynamics of the veterinarian-client-patient relationship are complex with multiple dimensions to take into consideration.

As you read on...
Reflect on what approach meets your style. Often, we recognize a pattern that suits us most, is our dominant style and is our default pattern during times of stress. We are in our comfort zone in interacting with clients in this manner.
Take time to identify the relationship style preference of your clients. The overall goal is to demonstrate elasticity in your client communication, so that you can tailor your approach to meet the client’s needs to enhance clinical outcomes. Frequently, communication challenges result from a mismatch in communication styles. Expanding your repertoire will enable you to meet the needs of a diverse clientele.
As an initial assessment, ask yourself “Who is doing the talking?” This is a simple litmus test for assessing your communication style. “Are you doing all the talking?” or “Are you creating space for the client to share their story and take an active role in the conversation?” “How much time do you spend listening to the client during the clinical interview?”

A Paradigm Shift
Recent societal changes have caused a paradigm shift in the veterinarian-client-patient relationship. Over the past decade, societal changes have caused substantial transformation in the veterinary profession. One of the major changes is the increasing recognition of the relationships that people may have with their companion animals. When asked about their relationship with their pets, 85% of pet owners reported that they viewed their pets as family members. In conjunction with this, there is a growing recognition that provision of veterinary services in a manner that acknowledges the human-animal bond will lead to better outcomes for veterinary practices and their patients. Appreciating the impact of animal companionship on the health and well-being of humans creates a new dimension in public health. Veterinary professional’s responsibilities have expanded to include the mental health and well-being of their clients, as well as their clients’ pets.

With the advent of the internet, today’s veterinary professionals are faced with educated clients armed with questions and greater expectations. Veterinarian’s responsibilities for addressing questions and providing client education are increased. In an increasingly litigious
society, consumers are not forgiving of unprofessional services. Most complaints to regulatory bodies are related to poor communication and deficient interpersonal skills, with breakdowns in communication being a major cause of client dissatisfaction.

An adaptive response is integral to successfully addressing these societal and professional changes. Given growing client expectations, the strong attachment between people and their pets and increasing consumer knowledge, demands a shift in communication style from the traditional paternalistic approach to a collaborative partnership. Many clients are no longer content with taking a passive role in their animal’s healthcare and want to take an active role in decision-making on their pet’s behalf.

**Veterinarian-Client-Patient Relationship Styles**

The relationship dynamic between veterinarian and client is based on 3 criteria:

Who sets the agenda for the appointment (ie, the veterinarian, the veterinarian and client in negotiation, or the client).

Importance placed on the client’s values (ie, the veterinary team assumes that the client’s values are the same as the veterinarian’s, the veterinary team explores the client’s values with the client, or the veterinary team does not explore the client’s values).

Functional role of the veterinary professional (ie, guardian, advisor, or consultant).

**Paternalism**

On the basis of these criteria, three veterinarian-client-patient relationship styles have been described. At one end of the relationship spectrum lies paternalism, characterized as a relationship in which the veterinary professional sets the agenda for the appointment, assumes that the client’s values are the same as the veterinarian’s, and takes on the role of a guardian. Traditionally, paternalism is the most common approach to medical visits. Shaw et al reported in veterinary medicine that 58% of all visits were paternalistic and specifically, in 85% of problem visits veterinarians use a paternalistic approach. The topic of conversation is primarily biomedical in nature, focusing on the medical condition, diagnosis, treatment and prognosis.

In a paternalistic relationship, the veterinary team does most of the talking and the client plays a passive role. This approach is often referred to as the “data dump” and symbolized by a “shot-put.” Throwing a shot-put is unidirectional, the intent is on the delivery, the information to be delivered is large in mass and it is challenging to receive the message. Intuitively it seems like this directive approach enhances efficiency and promotes time management. The challenge is that the agenda and subsequent diagnostic or treatment plan may not be shared between the veterinarian and client, compromising the ability to reach agreement, move forward and achieve full compliance. This could result in a roadblock, taking steps backward to recover and regain client understanding, commitment and trust.

**Consumerism**

At the opposite end of the spectrum lies consumerism, which is characterized by a reversal of the traditional power relationship between veterinarian and client: the client sets the agenda for the appointment; the veterinary team does not explore the client’s values; and the
veterinary team plays the role of a technical consultant, providing information and services on the basis of the client’s demands. The consumerist approach was not reported in veterinary visits and seems to be a infrequent approach. While the paternalism model has been criticized for ignoring the client’s perspective, the consumerism model errs in limiting the role of the veterinary team. The challenge in this situation is to engage with the client as a working partner and to build trust with the veterinary team to reach an agreement between the client and the veterinarian’s agenda.

**Partnership**

Between these 2 extremes is relationship-centered care, which represents a balance of power between veterinarian and client and is based on mutuality. In the relationship-centered model, the relationship between veterinarian and client is characterized by negotiation between partners, resulting in creation of a joint venture, with the veterinarian taking on the role of advisor or counselor. Respect for the client’s perspective and interests and recognition of the role the animal plays in the life of the client are incorporated into all aspects of care. Shaw et al reported that in 42 percent of all veterinary visits were characterized as relationship-centered and specifically in 69% of wellness visits veterinarians used a relationship-centered approach.

The conversation content of relationship-centered visits is broad including biomedical topics, lifestyle discussion of the pet’s daily living activities (e.g. exercise regimen, environment, travel, diet and sleeping habits) and social interactions (eg. personality or temperament, behavior, human-animal interaction and animal-animal interactions).

In addition, a relationship-centered approach encompasses building rapport, establishing a partnership, and encouraging client participation in the animal’s care, all of which have the potential to enhance outcomes of veterinary care. This collaborative relationship is symbolized by a Frisbee. In playing Frisbee, the interaction is reciprocal, the intent is on dialogue, the delivery is airy, light and free, small pieces of information are delivered at a time and the deliverer and receiver adjust their message to stay on target. Intuitively, it seems like this facilitative approach takes more time, however it was found that relationship-centered care appointments were shorter in length due to achieving common ground between the veterinary team and client.

Communication style has implications for the veterinary team, client and patient outcomes based on research on physician-patient communication that reported a positive relationship between aspects of relationship-centered care and patient satisfaction, physician satisfaction, patient health outcomes and a reduction in malpractice risk. Specifically, the following principles of relationship-centered care are associated with significant outcomes:

- Broadening the explanatory perspective of disease beyond the biomedical to include lifestyle and social factors is related to expanding the field of inquiry and improved diagnostic reasoning and accuracy.
- Building a strong relationship is associated with increased accuracy of data gathering, patient satisfaction and physician satisfaction.
Encouraging participation, negotiation and shared decision-making promotes patient satisfaction\textsuperscript{9,10,11}, adherence\textsuperscript{14} and improved health\textsuperscript{15}.

**Shared Decision Making**

Shared decision making is a key component of relationship-centered care. There is two-way exchange between the veterinarian and client, identifying preferences and working towards consensus. An interactive approach (eg. Frisbee) is promoted in giving information, in contrast to direct transmission (eg. shot-put).\textsuperscript{6} With a direct transmission approach, the sender assumes that his or her responsibilities are complete once the message has been formulated and sent, whereas with an interactive approach, the interaction is considered complete only if the sender receives feedback about how the message was interpreted, whether it was understood, and what impact it had on the receiver.

Silverman et al\textsuperscript{6} recommend using a “chunk and check” method (eg. Frisbee) when giving information to avoid giving a one-sided speech and providing a large amount of information all at once (eg. shot-put). The aim of this technique is to increase recall, understanding, and commitment to plans. It consists of giving information in small pieces (ie, chunks), followed by checking for understanding before proceeding further (ie, check). In this manner, the information-giving process is responsive to the client’s needs and provides an opportunity for the client to participate in the conversation, provide feedback or ask for clarification.

Taking the client’s perspective into account and establishing mutual understanding and agreement encourages the client to fully participate in the discussion and commit to the diagnostic or treatment plan. This entails encouraging the client to contribute to the conversation (eg. check) (“What questions do you have?”), picking up on client cues (“You seem a little hesitant about surgery.”), asking for client suggestions (“What options have you and your husband discussed?”), and checking for the client’s understanding (“What will be most difficult for you?”). Use open-ended inquiry to explore the client’s perspective (“How do you feel Max is doing since the surgery?”), ascertain the client’s thoughts (“What do you attribute to his good progress?”) and assess the client’s starting point (“What do you know about the risks of arthritis?”). Extrapolating from medical communication outcomes-based studies, obtaining the client’s expectations, thoughts, feelings and fears about the pet’s health or illness enhances client participation in the appointment, with the potential to increase client satisfaction and adherence to veterinary recommendations.\textsuperscript{16}

**Conclusion**

In revisiting the questions posed at the beginning of this chapter, which relationship style reflects your approach, what is your client’s style and who does the talking in your visits. Given the answers to these questions what steps would you like to take to expand your repertoire to meet the needs of your client. Flexibility in your approach is instrumental in meeting the diverse preferences of your clients. It seems appropriate to incorporate both paternalism and partnership in your toolbox and to interchange your pattern to meet that of your client. Matching the relationship between the veterinarian and client enhances the potential of achieving significant clinical outcomes, including enhancing client satisfaction, improving
patient health and as a result, professional fulfillment.

References


Succession Planning, Preparing Your Practice for Sale and Value Drivers

Introduction
One of the biggest steps a veterinarian will take is the move to practice or business ownership. Succession or transition planning is a process highlighted by a series of events. By definition, succession planning for a practice is the transfer of both the physical assets and management of an existing practice from one practitioner, or group of practitioners, to another. The process of complete succession planning involves a number of phases, such as initiating, negotiating, and closing a practice sale. The negotiation phase is the period during which the parties agree upon the terms of sale of the practice. The inheritance phase happens quickly and is the formal transfer of a business that occurs at closing. The actual succession phase is the cultural shift, whereby a new owner or owners assume control and impart their leadership, views, and cultural philosophy. Finally, the withdrawal phase is the departure of the former owner or owners; this may happen coincidentally with the inheritance phase, during the succession phase, or after the succession phase.

The Process
The process of succession planning can take years to complete, or it may happen very quickly, depending on the life stage status of the parties involved and their level of motivation and commitment to move the process along. No matter how long the process takes, smooth succession planning is important for a number of reasons. It maintains the continuity of the practice and the goodwill—generally where the most value lies in a purchase. For both buyer and seller, the transition into and out of practice facilitates a key step in their career planning. According to a Scotiabank survey conducted in 2001, over 50% of companion animal practice owners already are or will be at retirement age in the next 10 years. As professionals, we move through a series of life stages when our actions with respect to acquisition versus divestiture of businesses complement our particular life stage, as shown below in Figure 1. A significant group of practice owners, on or near the leading edge of the baby boomer generation, are moving into the pre-retirement stage.
Figure 1 – Financial life stages.

On the surface, it often appears that buying a practice is about legal documents, practice values, debt, and control. In fact, buying an existing practice (which is more common than establishing a new practice) is more about establishing and changing a relationship, letting go, and altering life roles.

The Issues at Hand: The ICEBERG

J. Fast, Centre for Family Business, Waterloo
Figure 2 – The various issues involved in buying a practice, using an “iceberg” analogy to illustrate their individual “weight.”

The inherent personality of an owner will affect his or her approachability when it comes to selling a practice. A very controlling, micromanaging owner may never be able to let go of the hospital, whereas an owner who enjoys seeing the practice transition to the next generation will take the lead in driving the process.

Buying into a practice or completely buying out an existing owner or owners has certain advantages. For example, a client base already exists, with a management structure in place and immediate cash flow available. Often some transition support is available from existing owner(s), experienced staff personnel are in place, and the practice is an established entity with proven systems. A marketing infrastructure is set, and a presence in the community is already known, thereby reducing the risk of failure. Depending on the amount of money being borrowed for the buy-in, debt financiers or banks often prefer existing businesses, as they have proven cash flow and capacity to handle debt. Buy-ins carry their own form of risk, however. If you have never worked in the practice, and you are unfamiliar with the culture, standards, and philosophy, you may have difficulty adjusting to or changing the culture without investing a considerable amount of time in the process and in managing staff changes.

A credible reason should explain why the business is for sale. Is there an upcoming retirement, or is the business in trouble because of the health or death of the owner? Is the area already saturated? Is the facility not able to deliver the services? Is the business poorly run and having cash-management problems? Prior to buying into or buying out an existing business, it often helps to create your own vision or profile of the practice you want, taking into account the location, style of practice, price, and timing.

The Details
The planning and executing of a buy-in or complete buy-out should follow an orderly process. The ultimate legal transfer of a practice will proceed through a number of steps. First are the data gathering and initial discussions, and then the confirmation of main objectives, primary agreement items, and any remaining details of the agreement. Lastly, the agreements are formalized into legal language, followed by closure. Assuming both the vendor (i.e., seller) and buyer (i.e., purchaser) are motivated and considered to be compatible, they will proceed in parallel with the creation of a purchase and sale agreement to further detail the terms of their partnership agreement if the transaction is a buy-in, or they will proceed with the assessment of value and commence negotiating on the terms of the sale.

Financing an existing practice almost always requires a fair market value assessment or practice evaluation. Unless a purchaser has close to two dollars for every one borrowed, a debt financier is going to request some justification of the value and expect assurance that the practice is able to generate enough cash flow to support the purchaser’s living costs and debt repayments. A practice evaluation can be commissioned by vendor and purchaser jointly, by one party or the other, or by each separately. Certainly a jointly agreed-upon assessment reduces the risk of
variance or the need for extensive, further discussions on the price. It is highly advisable to utilize an appraiser who has some background in the veterinary industry and is well-versed in evaluating veterinary practices. The inherent risk factors evident in a veterinary practice are not as obvious to an inexperienced assessor who has not had exposure to the industry, and this introduces the risk of the assessor arriving at a final value that is considerably different from the actual transactions and price points in the marketplace.

A practice appraisal is much more than a document to facilitate a purchase price. An evaluation will provide input into a new purchaser’s adjusted cost base for a share sale and the subsequent calculation of capital gains when part or all of the practice is sold. The appraisal may be required in the case of an estate sale or for the owner’s own estate planning and wealth management. It will be required in the case of a marital dissolution and may be required when a practice owner is seeking to borrow additional capital as security. Practice evaluations created and updated over time can be vehicles to monitor the owner’s return on investment. An appraisal can also help identify what areas of the practice need additional resources or time to positively affect the profit and future value of the business.

Once an appraisal price is agreed upon, it may be subject to adjustments based on whether the sale is an asset or share-based sale. Prior to arriving at a final price, advice from an accountant and/or lawyer on the tax consequences and structure of the deal is recommended. Accountants and lawyers and other consultants (in addition to the appraiser) form a key part of the primary resource base for buying a practice. These individuals can be facilitators who often aid in streamlining the process, reducing unnecessary costs and time, and bringing an informed perspective to expedite the process. While you are still the ultimate decision-maker, a facilitator can provide a different perspective.

A vendor always retains the right to ask more than an assessed price for a practice. The purchaser must then decide to accept the vendor’s asking price, negotiate on the price, or walk away from the deal. Psychology, desire, and motivation will always play a role on both sides of the transaction in these circumstances. People will overpay when they are highly motivated to possess a specific asset or business in a specific area, and vendors with an urgent sense of timing to divest themselves from the business may sell under an assessed price, while those with multiple offers and who are in no rush to sell can wait and/or bid up a purchase price. It is often difficult to practice emotional detachment in such situations, but decisions should not be rushed and should be based on sound information and a solid financial perspective.

While there are a number of items to negotiate and agree upon, three are critical: the practice purchase price, the occupancy status, and the role of the outgoing owner. The facility will be either leased or owned. If the facility is leased, a purchaser should ensure a long-term lease is secured, with predictability in current and future rental costs. If the facility is owned by the practice owner and not being purchased along with the practice, a lease should be arranged, and an option for purchase should be created at a future date if that is an opportunity.

It is important for outgoing owners to recognize what the value drivers are in practice. In small business they are considered to be:
- Management depth: some dedication individual to provide responsiveness, proactivity, HR systems support
- Customer diversity and magnitude: mostly an analytic function conveyed through number of active clients per vet, profit centre distribution, client turnover, client retention rates etc.
- Owner involvement: is the owner the vet the mgr., or both? Is the owner passive or a decision maker? Is the owner part of the vision and change?
- Competitive presence: also an analytic function. Considers the number of active households per full time vet in the primary catchment area.
- Client retention: relates directly to customer satisfaction.
- Human capital: captured by staff turnover, systems mgt. approach, reward system, credentials of staff, tasks they are involved in
- Operating efficiencies: include comparing the practice to benchmarks in revenue distribution and expenses to drive a healthy profit
- Recurring revenue: value comes from upward stable trend, repeat business and expanding activity
- Professional sense/image. For a practice to maximize value it needs to have an organized marketing plan including branding, facility tangibles, training and a commitment to education among others.

An expansion of these concepts will be provided during the lecture.

Additional Reading
Building the Successful Veterinary Practice, Volume 1,2,3. Catanzaro T. Iowa State University Press; 1998.
Synopsis
Indications for bone marrow aspiration and core biopsy will be briefly reviewed, followed by a discussion focusing on the techniques to obtain quality samples in general practice. The discussion will include selection of biopsy needles, sternal and costochondral bone marrow aspiration, and core biopsy using power drivers.

Indications
Bone marrow (BM) aspiration biopsy ± core biopsy should be considered with the following disorders1-3:

1) Low peripheral blood cell counts without new cell production in one or more cell lines.
   a) Non-regenerative anemia (see lecture 3).
   b) Thrombocytopenia without shift platelets. BM biopsy is not routinely recommended in cases of suspected immune-mediated thrombocytopenia (see lecture 5) or disseminated intravascular coagulation.
   c) Neutropenia without bands. BM biopsy is not routinely performed with predictable myelosuppression resulting from cytotoxic anticancer therapy.

2) Unexplained increased peripheral blood cell counts or atypical cells in circulation. The purpose of BM biopsy is to help rule-in our rule-out leukemia.

3) Hyperproteinemia/monoclonal gammopathy. The purpose of BM biopsy is to rule-in or rule-out multiple myeloma.

4) Staging of cancer. BM biopsy is extensively used in the staging of specific solid tumors in humans, but is not routinely used in dogs and cats for this purpose. BM biopsy may be considered in the staging of mast cell tumors, but is not routinely used in the staging of lymphoma.

5) Unexplained fever, weight loss, hypercalcemia. The purpose of BM biopsy is to detect occult neoplasia, infections, and inflammation.

6) Hemolytic anemia in breeds at risk for histiocytic sarcoma. BM biopsy is recommended in cases of regenerative anemia in Bernese mountain dogs and flat-coated retrievers, and should be considered in Golden retrievers, Rottweilers, Labrador retrievers and other breeds if there is any evidence that the disease is not “typical” immune-mediated hemolytic anemia (see lecture 5).
BM aspiration yields a sample that is usually examined cytologically (BM clot biopsies may be examined histologically). BM aspirates may also be analyzed by immunologic tests, PCR, flow cytometry, tissue culture and other advanced techniques to further characterize cell populations and detect infectious organisms; some of these techniques are only available in research laboratories. BM core biopsy yields a sample that is examined histologically (cytologic preparations may also be made). Core biopsies may also be analyzed by immunologic and other special tests further characterize cell populations and detect infectious organisms. Core biopsy should always be performed if aspiration is unsuccessful or yields a sample of low cellularity. Routine core biopsy may be considered, as it allows for examination of BM architecture (hematopoiesis is not normally evenly distributed throughout the BM), identifies myelofibrosis, may help finalize equivocal aspiration results, and is superior for detection of neoplasia and inflammation. Core biopsy is least likely to yield clinically relevant additional information when aspiration biopsy yields a hypercellular marrow.

**Limitations**

1) Except for identifying neoplastic cells, myelofibrosis, and infectious organisms, BM biopsy does not usually elucidate the mechanism of cell hypoplasia. For example, BM biopsy usually cannot distinguish between myelosuppression due to a toxic insult (e.g. due to autumn crocus poisoning) and immune-mediated pancytopenia (“aplastic anemia”).

2) In some cases serial BM biopsy is needed to trend changes as with serial CBCs.

3) A highly regenerative BM may mimic leukemia, as it is difficult to distinguish proliferating normal immature BM cells from abnormal immature BM cells on cell morphology (appearance) alone.

4) The value of routine biopsy in lymphoma and mast cell tumour is limited as results often do not change treatment plans.

**Complications**

Complications of BM biopsy are uncommon, but include:

1) Hematoma formation at biopsy site in thrombocytopenic animals. This is usually minor and thrombocytopenia and coagulopathy are not contraindications to BM biopsy. Digital pressure for 0.5-2 minutes is normally applied to a biopsy site, and longer pressure may be considered.

2) Infection. Cellulitis at the biopsy site and bacteremia/sepsis are rarely problems if aseptic technique is practiced, even in the presence of neutropenia. Febrile neutropenic animals are typically already receiving antibiotics prior to biopsy. Prophylactic antibiotics should be considered in non-febrile neutropenic animals. Cefazolin, 30 mg/kg IV, is the
author’s default choice of antibiotic if the neutrophil count is < 1.0 x 10⁹/L.

3) Broken needle remaining in tissue. This is a rare complication and is most likely to occur with old dull reusable needles.

4) Fracture of the biopsy site. This is a rare complication that is most likely to occur when performing a BM core biopsy of the ilium with too large a needle.

**Contraindications**

Septic or neoplastic tissue over a biopsy site. This is rarely a problem as there are numerous potential biopsy sites.

**Biopsy sites**

The most commonly used sites are the proximal humerus, proximal femur, iliac crest, sternum and costochondral junction.⁴⁻⁸ The latter two sites are not suitable for core biopsy. Most hematopoietic disorders are widespread in distribution and all sites will be representative. Occasionally samples from different sites are discordant. Two or more sites may be biopsied in an effort to improve diagnostic utility, but the value of doing so is not known. If a BM sample does not fit the hematologic picture, then repeat biopsy at another site is recommended. Focal lesions identified by imaging should also be biopsied. Flat bones (ilium, sternum, rib) contain active hematopoietic tissue throughout the marrow cavity in the adult and aspirates are less hemodiluted compared to those from long bones (humerus and femur), where hematopoiesis is restricted to the ends of the bones and adjacent to the cortices. The marrow cavity of flat bones, however, is narrow compared to that of long bones. The proximal femur contains more abundant active hematopoietic tissue than the proximal humerus. Prior to growth plate closure in the long bones, active hematopoietic tissue is located below the epiphyseal plates. Referring to a skeleton while performing a BM biopsy may be beneficial in orientation and identification of landmarks. Biopsy of the proximal humerus and sternum is easier to perform than biopsy of the ilium in both dogs and cats.

1) Iliac crest: Advantages – unlikely to cause joint damage; core biopsies may be less prone to fragmentation. Disadvantages – difficult to palpate in obese animals; narrow bone which is easy to slip off during needle insertion. Positioning – sternal recumbency. Landmarks – identify both iliac crests lateral to the lumbosacral spinous processes. The iliac crests may seem to be surprisingly close to the spine in obese animals. This is the author’s preferred site for core biopsy in immature animals in order to avoid damage to epiphyseal plates of long bones.

a) Dorsal approach. Palpate caudally along an iliac crest. Any part of the crest is suitable for aspiration. The aspiration needle is directed dorsoventrally perpendicular to the long axis of the ilium and “walked” or “tapped” on the crest to identify a region where the needle will not slip off when advanced. In medium-to-large dogs, the same direction is used for core biopsy, and the preferred site for core needle placement is at the caudalmost palpable part of the crest, as the ilium is thicker there. This will also avoid
the growth plate in the iliac crest (and damage to this epiphysis is not likely to be problematic). Moving the needle more caudally increases risk for sciatic nerve damage.
b) Dorsomedial approach. Beginning on the dorsomedial surface of the ilium, the coring needle is directed in a ventrolateral direction across the ilium. The biopsy path may traverse the sacroiliac junction. This is the author’s preferred technique for core biopsy in cats and toy breed dogs using standard 13ga needles.
c) Lateral approach. The iliac crest is palpated as before, but the needle is directed from the side rather than from the top – this may result in a better aspiration sample in cats. A core biopsy may also be obtained by directing the needle lateromedially across the thicker areas of the ilium. Depth of needle advancement should be measured to minimize the risk of vertebral body penetration.

2) Trochanteric fossa of the proximal femur: Advantages – once the needle is notched in the trochanteric fossa, it is easy to penetrate the cortex with little risk of slipping; less risk for lameness than with the proximal humerus. Disadvantages – more overlying soft tissue than other sites; biopsy may damage epiphyseal plate of greater trochanter. Positioning – lateral recumbency. Landmarks - Identify the trochanter major of the femur and ischium (rotating the femur outwards will displace your thumb when placed between these two landmarks). The leg is held in adduction to minimize risk of damaging the sciatic nerve, the needle is inserted just medial to the trochanter major and is advanced parallel to the long axis of the femur along the trochanter major into the trochanteric fossa.

3) Greater tubercle of the humeral head: Advantages – easy to identify, even in obese animals, because of minimal soft tissue coverage. Disadvantages – risk for joint penetration; risk for lameness due to excessive torque or pressure on the shoulder joint; biopsy may damage epiphyseal plate. Positioning – lateral recumbency. Landmarks – in mature animals, flex the shoulder joint and identify the acromion of the scapula and greater tubercle of the humerus. Palpate a flat depression on the cranioventral aspect of the greater tubercle and advance the needle caudoventrally at a 45-60° angle. It is important to have an assistant provide counter pressure to the elbow to avoid overstretched injury to ligaments. The needle may also be directed medially from the lateral aspect of the greater tubercle. Another approach is to rotate the humerus outward and approach the humeral head cranially at a perpendicular angle. These latter two approaches are preferred in immature animals in an effort to sample below the epiphysis.

Sternum: Advantages – easy site to identify, even in obese animals; soft bone which is easy to penetrate; less painful. Disadvantages – not suitable for core biopsy; risk (minimal) for pneumothorax. Positioning – sternal recumbency (preferred) with head held up (as for jugular venipuncture) ± legs hanging over edge of table, lateral recumbency, or dorsal recumbency. Landmarks – palpate sternum as obvious protuberance below thoracic inlet. Insert the needle into the manubrium at the most convenient angle varying from directed caudally along the long axis of the dog to
directed dorsally perpendicular to the long axis. The second sternebra may also be aspirated. The risk for pneumothorax with perpendicular aspiration of the sternebrae with the dog in dorsal recumbency may be higher.

Costrocondral junction: Advantages and disadvantages – as for the sternum. Positioning – lateral recumbency. Landmarks – Usually the 7-9th rib, directing needle dorsomedially into the junction along the long axis of the rib. Aspiration of the rib at the junction of the top and middle third has also been described, but site identification is more difficult and risk for pneumothorax may be higher.

**Patient restraint**

BM aspiration and core biopsy have been performed on awake or lightly sedated dogs using local anesthesia, but heavy sedation or general anesthesia is recommended, especially when first learning the procedures, because they are painful. Pain upon BM aspiration is mediated by endosteal nerve endings and is not eliminated well by systemic analgesics, and is not blocked by periosteal infiltration of a local anesthetic agent. Aspiration is typically painful: awake animals may vocalize and sedated animals may move; even under general anesthesia animals may lighten or heart rates increase when aspiration is performed. A clinical impression is that aspiration of the sternum and costrocondral junction in dogs using hypodermic or spinal needles (without local anesthesia) is not as painful as aspiration of other sites. A recent study in normal dogs demonstrated that sternal aspiration was less painful than iliac aspiration, but that sternal aspiration was still mild-to-moderately painful.7 In the author’s practice reaction of dogs to sternal aspiration has varied from none-to-vocalization, with most dogs showing minimal-to-mild reaction. For biopsy sites other than the sternum or costochronral junction, if the animal is not anesthetized, 1-3 mL of local anesthetic agent is used. The skin and SC tissue are infiltrated using a 22-25 gauge needle; the needle is then directed against the periosteum, and local anesthetic agent is infiltrated there – resistance to injection should be felt if the needle is in the correct location. One disadvantage of the use of local anesthesia is that it makes landmarks more difficult to identify by palpation.

Anesthesia is recommended for bone marrow biopsy in cats and toy-breed dogs (unless otherwise contraindicated) because of their small size (making procedures more difficult) and more problematic manual restraint. The sedative or anesthetic protocol chosen should be based upon patient considerations; different protocols will not affect sample quality.

**Patient preparation**

A 1-3 inch² area is clipped over the biopsy site and surgically prepared. Adjacent hair may be matted down with alcohol to prevent entry into the area. A small slit drape (without clamps) and/or adhesive drape is placed over the site. A drape is not used when aspirating the sternum with the dog in sternal recumbency.
The operator washes hands and dons sterile gloves. The procedure is not typically performed in an operating room nor does the operator typically wear a gown, cap or surgical mask, although consideration to the latter should be given with severely neutropenic patients. The operator should have a nonsterile assistant.

**Bone marrow biopsy needles and their use**

Several reusable and disposable needles are available with different diameters, lengths and designs, and product design and availability are evolving. Sharper needles are less painful on insertion. *Reusable needles* are made entirely of surgical steel and may be re-sharpened. They are more expensive than disposable needles, but may last for many years. They may become bent during a biopsy, although the bend may often be adequately corrected to permit future use. *Disposable needles* have a steel needle and plastic hub/handle. Although designed for single use, they usually remain sufficiently sharp for several procedures. All needles re-used in the author’s practice are sterilized with the STERRAD® system (Advanced Sterilization Products Division of Ethicon). Re-usable needles and most disposable needles may also be autoclaved. There is some evidence that autoclaving may slightly dull needles.

BM needles are designed for aspiration of the sternum, aspiration or core biopsy of the iliac crest, or intraosseous fluid therapy in humans, but many of the needles are suitable for use as biopsy instruments in dogs and cats.

1) Bone marrow aspiration needles

These typically consist of: a bevel-ended thick-walled cannula (outer needle), to facilitate penetration of cortical bone; a locking-stylet, to prevent blockage of the needle by soft-tissue or bone, which is flush with the tip of the needle and may include a handle; and a hub which accepts a standard regular or Luer-lock syringe, to permit aspiration.

a) Reusable aspiration needles

These have an overall narrow hub/handle for finger-grip, and a flat head which allows the use of an orthopedic mallet to help insert the needle. Choices include:

i) Rosenthal needle – available in even gauge sizes from 2 – 22ga x 1 – 2.5in length, depending on gauge. Commonly used sizes in dogs are 16-18ga x 1 in.

ii) Osgood needle – available in 16ga x 1 & 5/16in, 18ga x 1in, and 19ga x 0.5in. The overall small size of 19ga Osgood needles make them very suitable for use in cats and toy breed dogs.

iii) Klima and Sahla needles – available in 14, 16, 18ga x 1 or 2in, with a collar to adjust depth of penetration.

b) Disposable aspiration needles include:

i) Illinois sternal needle (CardinalHealth) – available in 15 & 18ga with a collar to adjust maximal depth of penetration from 0.5 – 1.5in. The hub/handle of the needle is similar in design to that of the reusable needles, allowing a finger grip.

ii) Several aspiration needles from different manufacturers have a T-palm grip (similar to
core biopsy needles), which allows for more insertion pressure, but makes the needles more unwieldy for use in cats and toy breed dogs. Size varies from 8 – 18ga, and length from 2 – 4in, with a collar to adjust depth of penetration.

iii) EZ-IO® Intraosseous Infusion system (Vidacare) needles are available in 15ga x 15mm (5/8in), 25mm (1 in), and 45mm (1¾ in). These needles have a trocar-like point rather than a bevel point, which facilitates insertion, and the needle has a crown/trephine-like tip which facilitates infusion of fluids and aspiration. The needles may be inserted either manually using a finger-grip on the needle hub or using a ball-like palm grip, or very rapidly with use of a drill (G3 Power Driver). The small size, design, and insertion options make them very suitable for use in cats and dogs. The author prefers to insert the needles manually. The needles may be reused multiple times and retain sharpness.

iv) The OnControl™ biopsy system (Vidacare) includes a similar 25mm (1in) needle (as well as 68 and 90mm needles) intended for BM aspiration which is inserted using a power driver that operates at a lower speed than the EZ-IO® driver. Its use has been reported in cats, and was found to be easier to use than a 15ga Illinois needle. The needles may be reused multiple times and retain sharpness.

v) The Bone Injection Gun (B.I.G., WaisMed Ltd.) intraosseous fluid system may be used to place a 15ga needle to a depth of 2.5cm or an 18ga needle to a depth of 0.5 – 1.5 cm, which may then be used for bone marrow aspiration. The preferred sites for needle placement are the proximal medial tibia and proximal humerus. The author has no experience with this system.

vi) Standard 18 – 22 ga x 1 - 1.5in hypodermic needles. These may be used to aspirate the sternum and costo-chondral junction. The bone in these areas is softer; the narrower gauge needles are less likely to become plugged.

vii) Spinal needles, 18 – 22 ga x 1 – 2.5in. These may be used if a longer needle is needed to aspirate the sternum because of overlying adipose tissue; the stylet also stiffens the needle and reduces the chance of blockage. The cannulae of hypodermic and spinal needles have thinner walls than do bone marrow needles and are more likely to bend, but have been used in human pediatrics and may be sufficiently robust to use in the iliac crests, proximal humerus and proximal femur of immature dogs and cats because the bones are softer.

2) Aspiration procedure
The use of sterile 5-10% EDTA is recommended to reduce clotting and to give the operator more time for sample collection and preparation. This may be obtained by swirling 0.5mL sterile saline in a 7mL purple-top tube. It may also be obtained from a laboratory supply company or compounding pharmacy. The EDTA is aspirated into a 12mL syringe. The stylet is removed from the bone marrow needle, and the syringe containing EDTA is attached. A small amount of EDTA is injected through the cannula, leaving about 0.05 – 0.2mL in the syringe. Alternatively the bone marrow aspirate may be immediately transferred into a purple-top tube after collection, although this does not reduce the risk for clotting during the aspiration. Other anticoagulants may affect cell morphology, although ACD has been used successfully. Another alternative to reduce clotting after collection is to inject the aspirate into a Petri dish sitting on ice.
A 1mm stab incision using a #11 scalpel blade should be considered, especially if using a reusable needle. The skin is immobilized over the site with one hand while the needle is advanced with clockwise-counterclockwise rotation through the cortex while minimizing play in the needle. (A reusable needle may be inserted more easily with less play into the iliac crest by gentle tapping it in with an orthopedic mallet.) Occasionally a lessening of resistance is appreciated when the needle enters the marrow cavity of the humerus or femur. A properly placed needle will feel solidly embedded; this sensation is not as pronounced in the sternum. If a hypodermic needle is used, it is attached to the syringe and advanced directly without rotation into the sternum or costochondral junction.

The stylet is removed and the needle tested again for solid placement. If it is not solid the stylet is replaced and the needle re-advanced. The 12mL collecting syringe is attached. One hand should hold the syringe barrel (not the needle) while the other hand pulls on the plunger. A Luer-lock syringe helps maintain aspiration vacuum. If a non-Luer lock syringe is used, the hand on the syringe barrel should push it firmly into the needle hub during aspiration to prevent disconnection. The plunger is sharply withdrawn 1-3 times and held at maximum aspiration volume. Further repeated aspiration movement is not recommended as this does not help retrieve a sample and heats the syringe barrel, promoting clotting. The initial aspirate fraction is probably the most diagnostic. If the desire is strictly to make slides for cytology smears, aspiration into 0.05 mL EDTA should be stopped once the 0.4 - 0.5mL mark is reached. Collection of additional volume increases the risk of hemodilution of marrow particles. If the desire is to collect a larger volume for additional assays, then up to 3mL is collected into 0.2mL EDTA and the sample is promptly transferred to a purple-top blood tube. If no anticoagulant is used, aspiration should be stopped as soon as blood is seen to be entering the syringe. If there is no yield, the stylet is replaced, the needle advanced, and aspiration repeated. If the needle is believed to be deeply imbedded, then an attempt is made to withdraw the needle with slow rotation while maintaining negative pressure. The needle should not be advanced in the ilium, femur or humerus without the stylet. If there is still no yield, the needle is withdrawn and reinserted. If three attempts are negative, another site is used.

3) Sample handling
Communication with the laboratory for preferred slide preparation is recommended. In the author’s practice, a generous drop of EDTA-anticoagulated marrow is placed on a slide, allowed to sit for several seconds, and the slide is tipped to allow blood to run off onto absorbent paper while marrow particles stick to the glass. Excessive blood may also be wicked away with absorbent paper. A spreader slide is then used to make a smear similar to a blood smear. The procedure is repeated, but the spreader slide is placed on top of the drop at a 90° angle, and then drawn along the bottom slide to make a squash smear. At least 10 smears are made. An alternative method for slide preparation is to inject the aspirate into a Petri dish, and visually remove marrow particles (which have the appearance of small irregular greyish particles which tend to
adhere to the bottom of the dish when tilted) with a transfer pipette, tuberculin syringe or microhematocrit tube. These are placed onto a slide, blood is wicked away and squash smears are prepared. The presence of fat globules confirms that the sample is of marrow origin, but rapid staining and examination of one slide at point-of-care is recommended to confirm an adequate sample. Necessary staining time is usually twice that for a blood smear. The remaining slides are submitted unstained to a laboratory.

4) Bone marrow core biopsy needles
These are also available as reusable or disposable instruments. A disadvantage is that most needles are too large for easy use in cats and small dogs. The most common contemporary needle sizes are 11ga x 4in (human adult), which is easiest to use in large dogs, and 13ga x 2-3.5in (human pediatric), which may be used in medium-to-large dogs, and with increasing difficulty in smaller dogs and cats. Older smaller gauge coring needles are still available. The prevailing opinion is that the bigger the biopsy the better, both with respect to gauge and length. The minimally-acceptable biopsy length in an adult human is 2 cm. Larger-gauge biopsies markedly increase marrow volume, which is important for detecting metastasis (and hence the introduction of 8ga needles), but is not that applicable to dogs and cats. Smaller biopsies in dogs and cats are probably actually capturing a greater proportion of a patient’s marrow than are typical biopsies in humans, but a genuine concern is that smaller gauge biopsies are more fragile and prone to fragmentation and other artefacts, and may be less representative.

Choices for procuring samples for histology include:
a) Open surgical biopsy using saw-toothed (eg. Michele) trephines or partial rib resection. These procedures are more traumatic and are out-dated by newer needles for closed biopsy.
b) Saw-toothed microtrephine needles (e.g. Ackermans 12ga x 4in, Turkels 12 & 14ga x 0.75 in, Gardner 13ga x 1.3 in) designed for BM biopsy are still available. With an Ackermans or Turkels needle, an aspiration-like needle is inserted through the cortex into the bone marrow. The stylet is removed, and a saw-tooth trephine is then inserted through the cannula and extended with rotation beyond the tip. With a Gardner needle, the cannula itself has a trephine tip and is advanced without the stylet once the cortex is penetrated. A disadvantage of these needles is that there is no specific mechanism to retain the core within the cannula upon withdrawal from the marrow.
c) Modified Vim-Silverman biopsy needles were once extensively used. A needle similar to an aspiration needle is inserted through the cortex into the bone marrow. The stylet is removed, and a split stylet is then inserted through the cannula and extended beyond the tip, thereby entrapping bone marrow. The cannula is advanced over the stylet, both are sharply twisted clockwise, and then removed. The 14ga x 2 in Conrad-Crosby needle (with a 17ga stylet) is available for this method.
d) Most currently used core biopsy needles are of the Jamshidi-type. These classically consist of a tapered bevel-ended cannula, a bevel-ended locking-stylet which extends ≈2 mm beyond the tip of the cannula, and a hub to accept a standard regular or Luer-lock syringe. The purpose of the stylet is to penetrate the cortex and provide a “pilot
hole” so that the cannula may be advanced through the cortex. The purpose of the taper (which narrows the cannula by two gauge units) is to help retain the BM core. The handle allows a palm-grip.

5) Biopsy procedure with a Jamshidi-type needle
A 2-3 mm stab incision is made using a #11 scalpel blade. Sterile sponges are used to swab blood. The skin is immobilized over the site with one hand while the needle is advanced through the skin and SC tissue until the cortex is encountered. Using moderate pressure, the needle is advanced into and through the cortex with clockwise-counter-clockwise rotation. The index finger of the operating hand may be placed on the cannula shaft to minimize play in the needle. Occasionally a lessening of resistance is appreciated when the needle enters the marrow cavity. A properly placed needle will feel solidly embedded. The stylet is removed and the cannula tested again for solid placement. If it is not solid the stylet is replaced and the needle re-advanced. Once the cannula is solidly embedded, a cap is attached to the hub and the needle is advanced with rotation as far as possible. Except occasionally in young animals, it is not possible to penetrate the opposite cortex without a stylet in place. Further advancement of the needle is variably indicated by visual judgement of depth, increased pressure and lack of needle advancement, and painful reaction or increased heart rate under anesthesia (from stimulation of the endosteum). The extrusion probe may also be placed down the needle to judge the size of the core. Although the cannula is tapered, this is not relied upon to retain the BM core. The cannula is carefully rotationally withdrawn 1-2 mm, and re-advanced at a slightly different angle, being careful not to bend the cannula. As the cannula is carefully and slowly withdrawn, withdrawal should be stopped two to three times and rotational play applied to the needle. These manoeuvres are performed in an effort to crack off the core within the cannula. Once withdrawn, a probe placed through a hand protector is used to extrude the specimen retrograde out the top of the needle. If no specimen is present, the procedure may be repeated at the same site (usually using the same cortical hole) up to two more times before using another site.

i) Because standard Jamshidi-type needles do not always capture a biopsy, innovations to further help retain the core include the use of a constricting coil within the needle (SNARECOIL™ [Ranfac Corporation]), the passing of a split stylet down the needle (Core-Lock™ [Worldwide Medical Technologies], T-Lok™ [Angiotech Interventional]), and the passing of a Jamshidi® marrow acquisition cradle down a T-handle Jamshidi® needle (CardinalHealth). These needles also have modified stylet and cannula tips which facilitate needle insertion advancement. They are all available in 13 ga, but are more expensive than regular Jamshidi-style needles.

ii) The OnControl™ biopsy system uses a needle with a marrow capture thread inside a non-tapered cannula to retain the core. The cannula has a trephine tip and is advanced with a power driver. The needle is 11 ga, but the biopsies are thicker than those obtained with other 11 ga needles that taper. Preliminary experience in large dogs is that the needle reliably captures good quality specimens. The use of the OnControl™ core biopsy needle in cats has also been reported.5

iii) A 25 mm EZ-IO® needle may be used to obtain 15 ga core biopsies from the humerus
of small dogs and cats. The assembled needle is manually inserted into the bone marrow. The stylet is removed, and a second hub, from which the stylet has been cut, is attached to the cannula, which is then rapidly advanced into the BM with the power driver. Alternatively the cannula may be manually advanced with or without a needleless hub. The needle is removed manually (a syringe may be attached) using clockwise action during withdrawal. Although the needle does not have a specific core entrapping mechanism as it is not designed for biopsy, this approach is similar to the use of a microtrephine needle, and 15ga biopsies were reliably obtained. Play may be intentionally introduced during cannula withdrawal (ie. standard Jamshidi-style technique) in an effort to pinch the crown tips of the cannula shut thereby providing a marrow capture mechanism. This intentional damaging of the needle, however, precludes re-use. Biopsies of the ilium with this needle are more difficult to obtain and of lower quality than humeral biopsies. Preliminary experience suggests this needle may also be used for femoral biopsy. The OnControl™ 15ga x 25mm aspiration needle may similarly be used to obtain a core biopsy.

When the core is extruded from the cannula with a probe, it is examined for quality. Based loosely on human pediatric criteria, a good biopsy is unfragmented and >0.5 cm in cats and small dogs, >1 cm in medium size dogs, and >1.5 cm in large dogs. Bone marrow will have a grossly visible trabecular pattern, while cortical bone is smooth and white. It is common to capture a small piece of cortical bone in addition to marrow. Normal marrow is red. Marrow with erythroid hyperplasia/red cell leukemia is darker red. Marrow with myeloid/lymphoid leukemia is cream white. Hypoplastic marrow is pale pink-to-white. The specimen is extruded into formalin or other fixative and submitted for processing. Davidson’s (Hartmann’s) fixative results in more rapid fixation, and may stabilize smaller biopsies, but is not commercially available. Contacting a histopathology laboratory to procure this fixative is recommended if 15ga biopsy is planned. 15ga biopsies may also benefit from being wrapped in specimen paper and placed in a cassette prior to transport. These supplies are also available from histopathology laboratories.

Any core biopsy needle may also be used for BM aspiration. If the same site is used, core biopsy should be performed before aspiration as prior aspiration may cause the core to appear hypoplastic. The author prefers aspiration of one site and core biopsy of a separate site. If only one biopsy is desired, the core may be gently rolled on a glass slide to yield a cytologic specimen prior to placing into formalin. If aspiration of a site is successful but core biopsy is not, another aspirate may be obtained without anticoagulant and allowed to clot. In a technique used in human neonatal medicine, a 19ga x 0.5in Osgood needle is inserted into the marrow. Once the stylet is removed, the needle is advanced another 2-3 mm. A 3mL syringe is attached, and aspiration applied until blood first appears in the hub. When the clot is firm it is placed into formalin for histological examination of bone marrow particles.

**Patient after care:** Digital pressure is applied over the biopsy site up to two minutes in
an effort to minimize cortical and subcutaneous hematoma formation. The incision is not routinely sutured. Appropriate opioid or NSAID analgesics are given on the day of biopsy and as deemed necessary on subsequent days based on assessment of the patient and biopsy site. If a biopsy is uncomplicated, analgesics are not usually deemed necessary after 48 hours.

Summary
In the author’s practice currently:
1) Preferred BM aspiration in dogs is from the sternum using a 20ga hypodermic or spinal needle, with the dog sedated.
2) Preferred BM aspiration in mature cats is from the humerus using a 15ga x 15mm EZ-IO® or 15ga x 25mm OnControl™ needle, and in immature cats from the lateral aspect of the ilium using a 19ga x 0.5in Osgood needle, with the cat anesthetized.
3) Core biopsy is always considered, and is always obtained if aspiration results are equivocal, suggest a hypocellular marrow, or are non-diagnostic.
4) Core biopsy is performed using anesthesia.
5) Preferred BM core biopsy in mature dogs is from the humerus using an 11ga OnControl™ needle in medium-to-large dogs, 13ga SNARECOIL™ or T-handle Jamshidi® in small dogs, and 15ga EZ-IO® needle in toy breeds.
6) Preferred BM core biopsy in immature dogs is from the ilium using a 13ga SNARECOIL™ or T-handle Jamshidi® needle in small-to-large dogs, and a 15ga EZ-IO® needle in toy breeds.
7) Preferred BM core biopsy in mature cats is from the humerus using a 15ga EZ-IO® needle.
8) Preferred BM core biopsy in immature cats is the trochanteric fossa using a 15ga EZ-IO® needle.
9) If a biopsy is non-diagnostic it is repeated to a maximum of 3 times at any site.
10) Animals are given opioid or NSAID analgesics for 48 hours post-biopsy.

References
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Regenerative Anemia in Cats
Dr. Anthony Abrams-Ogg, DVM, DVSC, DACVIM (SAIM)

Synopsis
Detection of regeneration; etiology, diagnostic evaluation, and treatment of hemotropic mycoplasma infections and other causes of hemolytic anemias; and etiology, evaluation and treatment of hemorrhagic anemias, in cats, will be discussed using illustrative cases.

Overview
Anemia is defined as a reduction in Hct, RBC count, or Hb. Hb is the best measurement because Hct is affected by RBC size and RBC counts are less precise than Hb measurements, but microHct (“PVC tube”) is the most commonly used value because of ease of measurement in veterinary clinics. Anemia is a common problem in cats, in part because of the reduced erythroid mass and short RBC lifespan of cats (73 days\(^1\)) vs. compared to dogs (104 days for most breeds\(^2\)). This is compensated for by the lower affinity of feline Hb for oxygen, promoting the release of oxygen to needy tissues, thereby allowing cats to better tolerate anemia.

Anemia may be classified pathophysiologically as being due to hemolysis, hemorrhage, [erythroid] hypoplasia, or hemodilution (4 H’s). Hemolytic and hemorrhagic anemias are typically regenerative, while hypoplastic anemia is, by definition, nonregenerative. Anemia may also be classified by duration as acute or chronic, with a spectrum in between the two. Acute anemias may be caused by either hemolysis or hemorrhage. Chronic anemias may be caused by hemolysis, hemorrhage or hypoplasia. Chronic nonregenerative anemias in the cat are discussed in Lecture 3. Hemodilution will also cause a drop in Hct, Hb, and RBCs, but does not cause a true anemia in that there is no decrease in red cell mass. Dilutional pseudoanemia may occur with crystalloid and colloid fluid therapy, as well as physiologic states that cause plasma volume expansion. Anesthesia and sedation may also cause a drop in Hct, Hb, and RBCs\(^3\), which is likely secondary to fluid shifts.

Regeneration\(^4,5\)
Following an episode of RBC loss, regeneration begins as erythroid hyperplasia in the bone marrow (BM). In the last stage of maturation in the marrow, metarubricytes express their nuclei, becoming reticulocytes. The latter are normally held in the BM for 1-2 days before release. Once released, reticulocytes are in their aggregate form, and remain so for about 1 day. Aggregate reticulocytes have larger clusters (aggregates) of endoplasmic reticulum when stained with new methylene blue (NMB). These are roughly equivalent to the polychromatophils seen with a routine hematology (e.g. Wright’s) stain, and what are typically reported when a reticulocyte count is performed for a cat. The intensity of regeneration is usually less than for a dog. After a day the reticulocytes continue to mature into punctate reticulocytes that contain small clusters of endoplasmic reticulum (resulting in a punctate appearance), which are detectable 5 days after RBC loss. These reticulocytes mature into a normal RBC over several weeks.
An increase in aggregate reticulocytes is best to document regeneration in the presence of a moderate-to-severe anemia, but a short episode of regeneration may be missed. An increase in punctate reticulocytes is best to document low-grade regeneration, but there are no counting standards.

Nucleated red cells (nRBCs) are normally released from the BM in small numbers. An increase in nRBCs occurs with: a) regeneration, b) numerous disorders affecting bone marrow stroma, c) erythremic myelosis and d) in extra-medullary hematopoeisis. The key question to ask is whether or not the presence of nRBCs is appropriate for the degree of reticulocytosis. If it is not, then a cause of inappropriate rubricytosis should be sought.

Guidelines to judge adequacy of peak regeneration (4 – 7 days) following an episode of acute RBC loss in the cat are:

<table>
<thead>
<tr>
<th>Degree of anemia</th>
<th>PCV(%) Polychromasia (/oil field)</th>
<th>Reticulocytes (/μL), (x 10⁹/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ref interval</td>
<td>28 – 49</td>
<td>&lt; 40,000</td>
</tr>
<tr>
<td>Mild</td>
<td>21 – 27</td>
<td>40,000</td>
</tr>
<tr>
<td>Moderate</td>
<td>12 – 20</td>
<td>50,000</td>
</tr>
<tr>
<td>Marked</td>
<td>≤ 11</td>
<td>100,000</td>
</tr>
</tbody>
</table>

Peak punctuate reticulocyte numbers are seen at 10 days post-RBC loss.

**Clinical Signs of Anemia**

Signs of anemia are related to its severity and duration. Cats with moderate-to-marked acute anemia will be weak and inappetent. Many nonregenerative anemias are chronic and cats may be minimally weak and inappetent because of mechanisms that alleviate tissue hypoxia to compensate for decreased Hb. These mechanisms are hemodynamic and non-hemodynamic. The main non-hemodynamic mechanism in dogs (increased DPG synthesis) is not present in cats. The main hemodynamic compensatory mechanism is increased cardiac output, due to decreased afterload, increased preload, increased contractility, and increased heart rate. Decreased afterload is due to lower blood viscosity and nitric oxide-mediated vasodilation, because Hb, which is low, is a potent binder of nitric oxide. Increased preload is due to salt and water retention, due to low systemic vascular resistance. Anemic cats may be at increased risk for acute and chronic volume overload (which causes eccentric cardiac hypertrophy) compared to other species, and this may reflect more exuberant hemodynamic compensation because of no mechanism to decrease Hb affinity to oxygen. Sympathetic nervous system activation causes increased heart rate and contractility. In acutely anemic cats, these changes are manifested as tachycardia and increased pulse strength. As anemia becomes more chronic, heart rate and pulse strength normalize. Acute and chronic anemia may cause pica characterized by eating cat litter and licking concrete or ceramics.

**Causes of Regenerative Anemia (Hemolysis/Hemorrhage - DAMNPIT Scheme):**

**A. Hemolytic Anemias**
1. Degenerative – None

2. Anomalous – Hereditary RBC Defects

Increased osmotic fragility (IOF) and pyruvate kinase (PK) deficiency have been described in Somali, Abbysinian, Siamese (IOF only) and DSH cats. IOF is believed to be due to an inherited RBC membrane defect. In PK deficiency, RBCs lack ATP production, resulting in hemolysis. Both disorders may be characterized by chronic regenerative anemia and/or recurrent hemolytic crises (± mild icterus) first appearing in young cats. Cats with PK deficiency may also be asymptomatic. Other findings include hyperglobulinemia, splenomegaly (IOF > PK deficiency), and hepatic and splenic hemosiderosis. Of importance for ruling-out IMHA, Coomb’s tests and agglutination after RBC washing with saline should be negative. Cats with IOF also have persistent macrocytosis even in the absence of regeneration. IOF is demonstrated by exposing RBCs to hypotonic saline solutions; osmotic fragility is normal in PK deficiency. The PK deficiency allele is highly distributed in Somali and Abbysinians in North America, Europe and Australia. A genetic test is available. Treatment involves avoiding stress (which may precipitate hemolytic crises), and transfusion if necessary (although this arguably may increase risk for iron overload); prednisone and splenectomy may be beneficial. Severe poikilocytosis associated with severe anemia and regeneration has also been seen in DSH cats.

3. Metabolic – Hypophosphatemia

Hypophosphatemia may occur during treatment of DKA - phosphorus is shifted out of cells during acidosis and insulin deficiency, and lost in the urine because of polyuria, resulting in a phosphorus depletion. When insulin is given and acidosis corrected, phosphorus is shifted back into cells. The result is insufficient phosphorus for RBC ATP production, which is needed to maintain membrane integrity (via Na-K ATPase). This is most likely to occur on the 2nd or 3rd day of treatment of DKA. Supplementation with phosphate (using KPO4), 0.01 – 0.03 mmol/kg/h for 6 hours, is recommended if a serum phosphorus level of < 0.32 mmol/L is measured or anticipated. Occasionally higher doses, up to 0.12 mmol/kg/h for 24 hours, are needed. This requires the use of NaPO4 to avoid hyperkalemia, which is substantially more expensive and not as readily available.

Hypophosphatemia may also occur as a result of severe malnutrition, typically associated with liver disease. It is most likely to occur during a “refeeding syndrome” when nutritional support is started. Total body phosphorus is low, insulin release is stimulated by feeding, and hypophosphatemia results. Hemolysis is uncommon, but monitoring serum phosphorus is recommended for the first 3 days after starting enteral feeding. Hypophosphatemia has also been reported as a complication of renal transplantation.

4. Neoplasia – Disseminated Histiocytic Sarcoma

Disseminated histiocytic sarcoma (malignant histiocytes) is a rare cancer of macrophages, presumed to be arising from the BM, spleen or liver. Hemophagocytosis may occur, where extravascular hemolysis by malignant macrophages causes a
regenerative anemia (nonregenerative anemia may also occur). Concurrent thrombocytopenia is likely. Historical signs are nonspecific. Hepatosplenomegaly is likely to be present. As with hemophagocytic histiocytic sarcoma in dogs, the initial clinical and hematologic picture may be identical to idiopathic IMHA. The presence of thrombocytopenia, splenic masses on ultrasound, or poor response to immunosuppression should prompt fine needle aspiration of the spleen. Treatment of HS is poorly defined, but treatment with prednisone, lomustine, or liposomal clodronate is suggested.

5. Physical – None
6a. Infectious – Hemoplasmas, ehrlichiosis, babesiosis, FeLV
There have been exciting major developments in the understanding of the classification, PCR-based diagnosis, genetic sequencing and immunogenetics of the hemoplasmas, but the fundamental clinical picture of acute clinical Mycoplasma haemofelis infection has not changed. The mode of transmission is unknown, but outdoor male cats are at increased risk. It probably remains the most common cause of acute hemolytic anemia in cats in North America. The clinical signs are those of subacute, acute or peracute anemia; less consistent signs are fever, jaundice and splenomegaly. A CBC typically reveals a regenerative anemia. Occasionally the anemia is preregenerative, and may be nonregenerative if there is concurrent FeLV infection. Hemolysis is extravascular, agglutination may be seen, and a Coomb’s test may be positive. The diagnosis historically relied on visualizing the organism on a blood smear - this is still the only diagnostic test available in an emergency situation, and may be the only test within owner financial constraints. The best blood sample to demonstrate the organism is a fresh sample from the marginal ear vein. Organisms may fall off the cells over time in a EDTA sample. The organisms reside on the RBC surface and cocci, ring, and rod forms may be seen, sometimes in short chains. They should not be confused with Howell-Jolly bodies (bigger, denser, within RBC), stain precipitate (refractile and also present off RBC), crenation (may be refractile), basophilic stippling (delicate, within RBC), Heinz bodies (larger, refractile, cell-edge or extruding from membrane) or punctate reticulocytes. Because infected cells are rapidly cleared from the circulation the organism may be difficult to see, and PCR tests have been shown to be much more sensitive. If a cat is presented with hemolytic anemia, and there is confident organism identification, then a PCR confirmatory test is not mandatory. PCR is most useful if organisms are not seen or if there is uncertainty concerning their identification. The risk for false-positives is present with all tests, but the risk is particularly high with PCR tests precisely because they are more sensitive and therefore at risk for errors due to contamination, and should only be ordered from a laboratory with proven strict quality control and a good track record. Thorough cytologic examination of a blood smear should not be omitted just because a PCR test is being ordered. A sample for PCR should be drawn before antibiotics are given, but treatment should be started while awaiting results. An FeLV test should be obtained (it is part of the minimum data base in feline hemolytic anemia). If serum chemistries are obtained, pre-renal azotemia and elevated liver enzymes (due to hypoxic injury to the liver) may be seen. Effective antibiotics
include doxycycline (10 mg/kg or 50 mg/cat), enrofloxacin (5 mg/kg), or marbofloxacin (2.75 mg/kg). All doses are given PO once daily for 14 - 28 days. (Treatment up to 8 weeks with a goal to achieve a negative PCR has been suggested.) The author prefers to use doxycycline or marbofloxacin. Azithromycin is not a good choice. Prednisone/prednisolone (2 mg/kg once to twice daily) has often been recommended, but it has been reported than RBC membrane antibodies are probably not involved in hemolysis, which puts corticosteroid use into question. If the organism is not seen in a cat with moderate-to-severe acute hemolytic anemia, then a corticosteroid is recommended pending confirmation by PCR, and then may be discontinued. If a cat at risk for infection has only a mild anemia, then antibiotics alone should be considered. Supportive care and transfusion should be given as necessary. The prognosis is good-to-excellent. Infection may not be eliminated; reactivation may occur with stress or immunosuppression, but the risk appears to be low.

_Candidatus_ Mycoplasma haemominutum and _candidatus_ Mycoplasma turicensis infections are actually more common in some surveys than _M. haemofelis_. _M_. haemominutum does not cause acute hemolytic anemia on its own, but may make _M. haemofelis_ infection worse. It may also increase the risk for myeloproliferative diseases in FeLV-positive cats. _M_. haemominutum is about half the size of _M. haemofelis_ and is more difficult to detect on a blood smear. _M_. turicensis may cause anemia, but does not appear to be as pathogenic both with respect to causing anemia and severity of anemia. The organism has never been seen and can only be diagnosed by PCR. If a cat with acute hemolytic anemia is negative cytologically and by PCR for _M. haemofelis_, but positive for _M_. haemominutum, there are several possibilities: 1) the cat has IMHA and the hemoplasma is an underlying cause or is coincidental; 2) the cat has another cause of hemolysis and the hemoplasma is coincidental; 3) lab error. If the cat is positive for _M_. turicensis, it is also possible that this is the cause of anemia. Other causes of hemolysis and other clinical signs should be investigated, but treatment with doxycycline or a fluoroquinolone should be started.

_Cytauxzoon felis_ is a tick-borne protozoan parasite best characterized in the southeastern and southcentral USA, where bobcats and other wild felidae are natural hosts. Cases have also been described in Brazil, and similar organisms exist in southern Europe and Africa. Originally reported as a uniformly fatal disease, surviving and asymptomatic carrier cats have now been reported. The clinical disease is very rapid, and characterized by fever (which may progress to hypothermia), pallor, jaundice, splenomegaly and dyspnea. The anemia itself at presentation may not initially be severe (Hct between 20-30%); leukopenia and thrombocytopenia are common. Diagnosis is by identification of the organisms on a blood smear – coccal, “signet ring” and “safety pin” (bipolar) morphology have been described. Parasite numbers and staining are variable, and may be confused with _M. haemofelis_. Repeat blood smears, and splenic and bone marrow aspirates help to identify the organism. Confirmatory PCR tests are available. Early supportive care improves survival. The benefit of drug therapy is not clear, but imidocarb dipropionate (5 mg IM, with atropine 30 minutes prior to treatment) is most
often recommended. Surviving cats have usually also received doxycycline or a fluoroquinolone, and these should be given in the event the organism visualized is _M. haemofelis_.

Babesiosis\textsuperscript{16,17} is an emerging disease in cats, caused by different _Babesia_ spp. It has been reported in domestic cats mostly in South Africa, but also in India, Israel, Spain and Portugal. Babesiosis has been identified in wild cats in Florida, and it may be a matter of time until it is recognized in domestic cats in North America. Clinical disease ranges from an asymptomatic carrier state to acute hemolytic anemia. Diagnosis is by identifying the organisms (piroplasms), as well as immunologic and PCR techniques. Only the former should currently be considered when evaluating a cat with hemolytic anemia in North America.

FeLV increases the risk for hemolytic anemia due to _H. haemofelis_, and IMHA is also seen in cats with FeLV-associated lymphoma. However, there are cases of hemolytic anemia that have been seen in FeLV-positive cats that are PCR-negative for hemoplasmas and do not have neoplasia. The mechanism for FeLV-associated IMHA is not clear. The treatment is as for idiopathic primary IMHA, which is paradoxical in that it involves immunosuppression of an imunosuppressed cat. Initial treatment with prednisone (2 mg/kg/day) alone is recommended. Cyclosporine has been anecdotally associated with clinical decompensation.

**6b. Immune – Autoimmune (IMHA)\textsuperscript{18,19} and Alloimmune Hemolytic Anemia**

IMHA in dogs is most often characterized by an acute anemia and strong regenerative response. Regeneration is stronger than hemorrhagic anemia because iron is not lost and rapidly available for reuse. Consistent with the overall lower prevalence of immune-mediated diseases in cats than in dogs, IMHA is less common in cats. Compared to dogs, nonregenerative forms of IMHA are more common (see Lecture 3), fulminant disease and intravascular hemolysis are rare, and icterus is less common (reflecting the more subacute nature of hemolysis). Diagnosis of IMHA is based on: a) acute-to-subacute anemia, b) ruling-out hemorrhage, and c) identifying agglutination. Cats are more likely to form rouleaux than dogs, so caution must be taken in identifying agglutination (see Lecture 5). (Note: EDTA-induced agglutination may rarely occur, and should be ruled-out by re-examining the blood in citrate if a cat is not anemic.) A Coomb’s test is usually positive in regenerative IMHA (and becomes less often positive the “deeper” IMHA becomes – see Lecture 3). Increased osmotic fragility may also be used to support the diagnosis, but has not been routinely performed. Spherocytes are more difficult to identify in cats compared to dogs, and, also given less intense regeneration, anisocytosis (and RDW) are less pronounced compared to dogs. Secondary IMHA is considered to be more common than primary, although there appears to be increasing recognition of primary idiopathic IMHA. Regardless, investigation for underlying diseases is recommended. A minimum investigation includes probing for history of drug therapy (especially antithyroid drugs), FeLV/FIV tests, and PCR for hemoplasmas. More extended investigation includes abdominal ultrasound examination, fine needle aspiration of the
spleen (which may be enlarged in cats with IMHA as with dogs) and other lesions, thoracic radiographs, bone marrow biopsy (especially if nonregenerative), ANA, and testing for infectious diseases for which the cat is considered at risk. Treatment is as for IMHA in the dog, except that routine thromboprophyaxis is not given (see Lecture 5). Thromboembolic disease appears to be rare in cats with IMHA, which is perhaps unexpected because cats have more reactive platelets and faster clotting times than dogs, and likely reflects the less acute nature of the disease in cats. The need for immunosuppression beyond corticosteroids alone in cats is controversial as it is in the dog. Most cats have initially responded to corticosteroids alone, but there is strong anecdotal evidence that the chronic nonregenerative forms need more intense immunosuppression. The author prefers to start with prednisone alone if there is strong regeneration and/or an acute history, and use more intense treatment the “deeper” and more chronic the problem is (see Lecture 3). However, if there is no response within several days to 2 weeks, depending upon how aggressive the disease appears to be, the addition of cyclophosphamide (50 mg/cat PO q2weeks), chlorambucil (2 mg/cat PO q2-3 days), mycophenylate mofetil (10 mg/kg, PO, q12h) or cyclosporine (5 mg/kg PO q12h) should be considered. Routine doxycycline or marbofloxacin is also recommended while awaiting hemoplasma PCR, or if cost restraints preclude theses tests. The prognosis is better than with IMHA in dogs, with a long-term survival rate >75%, perhaps reflecting the less acute nature of the disease in cats. A recurrence of 30% has been reported, therefore the possibility of long-term therapy should be discussed with owners, and is definitely recommended following relapse. Cats may become diabetic and/or cushingoid with long-term steroid use necessitating the use of other immunosuppressive drugs.

Alloimmune hemolytic anemias result from blood-type incompatibilities. Transfusion reactions occur because cats have naturally occurring anti-RBC alloantibodies. The severity of a reaction is related to antibody titer and type (IgM being worse than IgG). In the unlikely event that a type A cat is given type B or AB blood, weak (probably IgG) anti-B titers will result in subacute extravascular hemolysis (delayed transfusion reaction/premature RBC loss), which is manifested as the transfusion not lasting as long as expected. If a type B cat is given type A blood, a typically high IgM anti-B antibody titer will result in peracute intravascular hemolysis, of which the salient finding is severe hypotension. Hemoglobinemia/uria may not be seen because the reaction occurs after very few RBCs are transfused. Some type B cats have lower IgM titers and IgG antibodies, resulting in a spectrum of less severe reactions of intravascular and extravascular hemolysis. Blood-typing and cross-matching is easy to perform in cats, so A-B transfusion reactions should not occur. However, another blood group, Mik, has been reported, where natural antibodies may result in an acute hemolytic reaction. In addition, other crossmatch incompatibilities have been seen and some type A cats that have received multiple transfusions have developed delayed hemolysis, suggesting sensitization to other blood groups. Ideally all cats should be crossmatched as well as blood-typed prior to transfusion.

Neonatal isoerythrolysis occurs when type A kittens are born to a type B queen (from
mating with a type A tom). Anti-A antibodies in the queen’s colostrum will attack the kittens type A RBCs resulting in acute death, fading, transient weakness, or delayed tail-tip necrosis. This can be prevented by removing all kittens (or blood-typing and removing type A kittens) from the queen and hand-rearing for 24 hours if the blood type of the tom is type A or not known. In the event of a first time presentation of a queen and fading kittens, part of the investigation should be blood-typing of the queen and kittens. Fading kittens should be transfused.

7. Toxic – Heinz Body Hemolytic Anemia
Each molecule of cat Hb contains up to 8 sulfhydryl groups (whereas most other species have 4 or less). This increases susceptibility to oxidative injury, which turn promotes Heinz body formation (aggregates of denatured hemoglobin). These are best seen with NMB. Heinz bodies make red cells less deformable. In most species, as these red cells pass through the spleen the presence of Heinz bodies results in extravascular hemolysis – as the red cells squeeze through splenic sinusoids they come into contact with splenic macrophages, which phagocytose the red cells and/or the Heinz body, resulting in hemolysis and/or spherocytosis. Feline spleens are not sinusoidal, and are inefficient at removing Heinz-body containing red cells from the circulation, accounting for the ability of cats to be predisposed to Heinz body formation without constant hemolytic anemia. Nonetheless, excessive Heinz body formation can lead to premature RBC loss, although the mechanisms for this occurrence are not fully understood. Toxic causes of Heinz body formation include benzocaine (Cetacaine), n-propyl disulfide (onions), propylene glycol (previously common in semi-moist foods), DL-methionine (high dose), methylene blue (high dose), cytotoxic drugs, propofol (repetitive administration), vitamin K injection, and acetaminophen. In the latter acute poisoning results in brown mucous membranes and edema of the face and paws, but hemolysis may subsequently result in pale mucous membranes and then jaundice. Increased Heinz body formation has also been noted in diabetes, hyperthyroidism, and lymphoma, but do not typically result in overt hemolytic anemia. Except for acetaminophen poisoning, regenerative anemia should not be attributed to Heinz body-induced hemolysis until other causes have been ruled-out.

8. Trauma – None

B. Hemorrhagic Anemias
Depending on severity, an episode of bleeding may result in sudden death, depression with preregenerative anemia, or presentation after the episode with regenerative anemia. Hemorrhage should be considered in any regenerative anemia without evidence of agglutination (and negative Coomb’s test), and the cat closely examined and imaged for evidence of trauma, and intrathoracic and abdominal hemorrhage, and tested for hemostatic defects.

1. Degenerative – None

2. Anomalous
Inherited platelet function defects, hemophilia and vitamin K responsive coagulopathy in the Devon Rex have been reported (see Lecture 4). Peliosis hepatis may result in spontaneous hemoabdomen.

3. Metabolic
Hepatic lipidosis, EPI, and severe IBD may cause vitamin K deficiency. The liver is friable with lipidosis and is more likely to bleed post-biopsy.

4. Neoplasia
Hepatic or splenic lymphoma, mast cell tumor and hemangiosarcoma (rare) may cause spontaneous hemoabdomen.

5. Physical – None

6a. Infectious
Severe thrombocytopenia sufficient to cause hemorrhage has been seen with pancytopenia due to FeLV, FIV and histoplasmosis.

6b. Immune/inflammatory
Immune-mediated thrombocytopenia is similar to the disease in dogs, although much less common. It may also be a feature of immune-mediated pancytopenia (see Lecture 3). A circulating anticoagulant may rarely cause a bleeding tendency (see Lecture 4). Hepatic amyloidosis and hepatic necrosis may result in spontaneous hemoabdomen.

7. Toxic
Cats are not as frequently poisoned with vitamin-K antagonist rodenticides as are dogs, but poisoning does occur, and may be secondary to ingesting a poisoned rodent. The clinical picture is similar to dogs, and presenting clinical signs include unexplained depression, re-bleeding from wounds, internal bleeding, and widespread cutaneous hemorrhages. Hemostasis should be assessed if bleeding is excessive for any injuries. Treatment is as for dogs. Toxins and drugs may cause pancytopenia (see Lecture 3). Snake envenomation may cause bleeding.

8. Trauma
A cat presented with anemia should be examined for external signs of trauma such as shorn nails, road burns, soiled fur, dried nasal bleeding, blood in the ear canals and oral bruising. Cats suffering motor vehicle and other major trauma are most likely to present with pneumothorax, signs due to head trauma, and fractures. Anemia, if present, is preregenerative. Occasionally major intrabdominal bleeding from trauma occurs, and if a cat is presented several days later, a regenerative anemia may be present.

References
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Non-regenerative Anemia in Cats
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Synopsis
Etiology, diagnostic evaluation, and treatment of non-regenerative anemia in cats will be discussed using illustrative cases. The discussion will focus on anemia that is not due to retroviral infection or chronic kidney disease.

Overview
Causes of nonregenerative anemia may be categorized by a mnemonic using the letter D: Detection, Delay, Destruction, Deficiency, Deep-Development-Dam, Diversion, Displacement, Depression, Dilution and Drugs.1 The categories are not mutually exclusive and anemia may be multifactorial in etiology. A pathophysiologic mechanism common to many of the causes is defective development of erythroid cells in the BM, which is reflected in a BM (BM) biopsy quantitatively as altered absolute and relative numbers of developing RBCs, and qualitatively as altered appearance of these cells (dysplasia).2 Cats with chronic anemia may develop reversible cardiomegaly and heart failure, multifocal retinal bleeding, and pica.

1. Detection of regeneration
Regeneration is discussed in Lecture 2. It may not be detected because of clerical or laboratory errors. Polychromasia is not as intense using in-house quick stains, and experience is needed for accurate counting of reticulocytes using new methylene blue stain.3

Mild regeneration may be difficult to detect when there is only low-grade hemolysis or hemorrhage, especially with a chronic problem because of adaptation to anemia. Counting of polychromatophils is less precise than reticulocyte counting, and also mildly underestimates regeneration, so the latter is recommended to confirm poor regeneration. Increases in MCV and RDW are supportive of regeneration. It is also possible that a cat is not presented during an episode of acute anemia, and that the anemia is diagnosed after regeneration has abated. An increase in punctate reticulocytes will help identify a previous or ongoing regenerative response. BM biopsy (see Lecture 1) may be necessary to examine for erythroid hyperplasia.

Many “nonregenerative” anemias are actually minimally regenerative, where a regenerative response is present, but is inappropriate for the degree of anemia. If it is not recognized, then complicating factors limiting erythropoiesis and contributing to the anemia may be overlooked. Guidelines to judge appropriateness of peak regeneration following an episode of acute red cell loss are given in Lecture 2. As anemia becomes more chronic and regeneration becomes less intense, it becomes more difficult to judge the adequacy of regeneration.

2. Delay in regeneration (preregenerative anemia)
A cat with acute anemia may be presented before regeneration can be seen on a CBC. Because acute anemia is due to hemorrhage or hemolysis, other clinical signs will often help differentiate an acute preregenerative anemia from a chronic nonregenerative one. Although a cat with a chronic nonregenerative anemia may present for acute clinical signs when the Hct falls below a critical level, most cats will have some chronic signs, unlike acute anemia, where peracute signs prompt rapid presentation by the owner. Causes in the cat where acute anemia itself is responsible for the presenting clinical signs include hemotropic mycoplasma infection and cytauxzoonosis. Less common causes include internal hemorrhage due to vitamin-k antagonist poisoning, and hemoabdomen due to bleeding tumors or peliosis hepatis.

Distinguishing acute pre-regenerative, from chronic non-regenerative, anemia, becomes more difficult as the anemia becomes less severe. Fortunately the clinical urgency also becomes less, and serial CBCs may be examined to observe for regeneration.

3. Destruction of bone marrow

Cytotoxic drugs and toxins
Cytotoxic drugs used to treat neoplasia and immune-mediated diseases kill mitotically active cells, thereby causing transient injury to BM progenitor cells. The result is predictable, dose-dependent, myeloid, megakaryocytic, and erythroid hypoplasia in the BN, and pancytopenia in the peripheral blood. The changes in peripheral blood are a reflection of mature cell lifespan: neutropenia appears prior to, and is more severe than, thrombocytopenia, which appears prior to, and is more severe than, anemia. Cats with cytotoxic BM suppression will be presented for lethargy and fever due to infection secondary to neutropenia. Anemia due to chemotherapy is usually mild in dogs, but cats may develop more severe anemia because of shorter RBC lifespan. The anemia is initially normocytic normochromic, but later some macrocytosis and anisocytosis may develop because of regeneration.

Unexplained transient BM suppression sometimes occurs - it is assumed that the BM received an unidentified toxic insult. If a BM biopsy is obtained during BM recovery, an exuberant response of progenitor cells, which occurs before the increase in mature BM and peripheral blood cells, may be mistaken for leukemia. Early myeloid hyperplasia is characterized by proliferation of normal blast cells, while myeloid leukemia is characterized by proliferation of mutant blast cells. Distinguishing the two is difficult, and serial CBCs and BM biopsies may be necessary to confirm a diagnosis.

Infectious diseases
Feline parvovirus damages intestinal crypt cells with resulting diarrhea and sepsis. The virus is also cytopathic to BM progenitor cells and may cause dysplasia. The salient hematologic findings are neutropenia and lymphopenia, but anemia may also occur, due to blood loss, malnutrition, and erythroid infection by the virus.
4. Deficiency of nutrients required for erythropoiesis

**Iron deficiency and other causes of microcytic hypochromic anemia**

Iron deficiency suppresses regeneration. This occurs in kittens consuming an all-milk diet, contributing to the normal transient drop in Hct seen before weaning. Iron deficiency in a mature cat is due to chronic blood loss. An important cause is chronic gastrointestinal bleeding due to a tumor. Diagnosis may be delayed because vomiting and diarrhea are often not present in cats with gastrointestinal tumors, and non-specific signs predominate. Anemia is initially regenerative, but regeneration then wanes and becomes inappropriately low. Iron-deficiency anemia is classically microcytic hypochromic, but in cats microcytosis is more difficult to detect and hypochromasia is often not present. Also, RBCs swell during storage in EDTA, so MCV should be measured on the day of collection. Microcytosis develops because metarubribytes undergo an additional cell division while awaiting completion of Hb synthesis. MCV is an average value, and the initial drop in MCV is within normal range as the percentage of microcytes increases. There is a concurrent increase in RDW because of microcytosis and polychromasia. The MCV continues to decrease to below reference range, at which point MCH is usually low. The last red cell index to drop is MCHC. Poikilocytosis and thrombocytosis are common findings. Iron deficiency is further supported by documenting low to low-normal total serum iron, and high-normal to elevated transferrin (total iron binding capacity) with low saturation. Serum iron and transferrin are measurable in most reference laboratories but the broad reference ranges for cats may make interpretation difficult. Serum ferritin is more reliable, and should be low-normal to low, but must be measured by a validated species-specific assay. BM biopsy will reveal erythroid hyperplasia, with expansion of rubribytes and metarubribytes, and often megakaryocytic hyperplasia. Cats do not normally store iron in the BM. Treatment involves correcting the underlying disorder, correcting the anemia, and correcting the iron deficiency. Transfusion, in addition to ameliorating anemia, will provide an immediately available source of iron to the BM. Following transfusion, improved regeneration will be seen in several days as the hyperplastic erythropoietic tissue thrives on the influx of iron. Iron supplementation should then be given as for rHuEPO treatment in chronic renal failure.

Portosystemic shunts may also cause microcytosis and hypochromia due to defects in iron metabolism, although changes are not as marked in cats as in dogs. Cats are usually presented for clinical signs of liver dysfunction and not anemia. Sideroblastic anemia is characterized by iron deposits in mature red cells in blood and in nucleated red cells in BM. It is a result of a defect in Hb synthesis and has been seen in cats with myelodysplastic syndrome (MDS).

**Folate and cobalamin deficiency**

Metabolism of folate and cobalamin are intricately related, and deficiency of one or both may cause megaloblastic anemia by interfering with DNA synthesis. Naturally-occurring megaloblastic anemia in cats responding to folate and cobalamin
supplementation has been reported infrequently. The anemia is normocytic-to-macrocytic. Serum levels of folate and/or cobalamin may be reduced with various gastrointestinal, pancreatic and hepatic disorders, and in hyperthyroidism. Anemia or macrocytosis are not common. Presumptive cobalamin deficiency resulting from a genetic disorder of intestinal malabsorption has been reported. Idiopathic BM failure and serous atrophy causing pancytopenia was associated with prolonged anorexia in 4 cats; cobalamin/folate malnutrition may have been contributory.

5. Development Dam Deep in the bone marrow

Nonregenerative immune-mediated hemolytic anemia
Immune-mediated hemolytic anemia (IMHA), with peripheral destruction of RBCs and strong regeneration, is uncommon in cats. However, the immune attack on erythropoiesis may occur “deeper” in the BM instead of, or in addition to, the peripheral blood. This is the more frequent form of IMHA in cats. The attack may occur at several levels of erythropoiesis and act as a “dam” in the BM preventing maturation.

At the first level, an attack on reticulocytes in the blood results in lack of regeneration as seen on the CBC but evidence of synchronous erythroid hyperplasia on BM biopsy. At the second level, an attack on later erythroid cells causes a maturation arrest. In some cases the production of immature erythroid cells behind the “immunologic dam” is so strong that it may be mistaken for erythroid leukemia. In other cases of ineffective erythropoiesis, erythroid production is seen to taper into the later stages on BM biopsy. In both cases there may be marked erythroid dysplasia. At the third level, the attack occurs on the earliest committed erythroid progenitor cells, and a BM biopsy reveals reduction or absence of all stages of erythropoiesis, and the diagnosis is erythroid hypoplasia or pure red cell aplasia. At the fourth level, the attack occurs on the pluripotent hematopoietic stem cells, and a CBC reveals pancytopenia and a BM biopsy reveals hypoplasia or aplasia of all cell lines (“aplastic anemia”). In all the above the anemia is normocytic normochromic. Serum iron and transferrin levels may be elevated. There is no pathognomonic finding on BM biopsy. BM plasma cell hyperplasia or lymphocytosis support the diagnosis, but its absence does not rule it out. IMHA at the BM level is presumptively diagnosed 1) by ruling-out other causes of erythroid hypoplasia, 2) supportive BM biopsy findings, and 3) response to immunosuppression (lack of response does not rule-out the diagnosis). The diagnosis is also supported by other concurrent immune-mediated disorders, positive ANA and Coomb’s tests, increased globulins, and spherocytes. Young cats are at increased risk. Cases often need more immunosuppression than provided by glucocorticoids alone, and the author prefers to initiate treatment with prednisone, cyclophosphamide or chlorambucil, and cyclosporine at standard doses. Because the role of hemotropic mycoplasma is not known, testing and treatment with doxycycline or marbofloxacin is recommended. When measured serum EPO levels have been elevated as expected, therefore routine rHuEPO therapy is not recommended. At least 70% of cats are expected to recover.
**Feline Leukemia Virus infection**

FeLV is not cytopathic but infection of erythroid cells alters their development causing a normocytic normochromic or macrocytic anemia (pure red cell hypoplasia).\(^\text{15}\) FeLV-positive cats may also develop anemia secondary to hematropic mycoplasma infections, hematopoietic and non-hematopoietic neoplasia, and possibly IMHA. Endogenous EPO levels are elevated and in principle rHuEPO therapy should not help, but there are anecdotal reports of benefit.

**Porphyria**

Erythropoietic porphyria is a rare genetic defect in heme synthesis in which heme precursors accumulate in cells and body fluids, resulting in anemia and discolored teeth.\(^\text{16}\)

6. **Diversion of hematopoietic cells**

**Acute myeloid (myelogenous) leukemia (AML)**

“Myeloid” leukemia refers to neoplasia arising from any hematopoietic cell in the BM. (The term “myeloid” in the context of leukemia is used to distinguish such leukemias from lymphoid ones, while in the context of normal hematopoiesis “myeloid” refers to granulocyte and monocyte cells as distinct from erythroid cells.) Hematopoiesis is diverted into production of neoplastic cells, decreasing normal cell production. Any AML may cause anemia, but the most profound tend to be with leukemias arising from erythropoiesis, i.e. erythroleukemia and erythremic myelosis.\(^\text{17}\) Cats are presented for inappetence and lethargy. The main physical examination finding in a cat with erythroid leukemia is pale mucous membranes. Petechiation may be present if there is concurrent thrombocytopenia. Fever may be present, especially if the cat is neutropenic. Leukemic cell infiltration may cause splenomegalay, hepatomegaly and lymphadenopathy, but the latter is not typically as marked as with lymphoma.

A CBC may reveal many combinations of leukemic cells and low normal cell counts. It is important not to misinterpret the presence of nucleated red blood cells (rubricytes and metarubricytes) as a sign of regeneration. BM biopsy is needed for diagnosis if only anemia ± other cytopenias are present. If leukemic cells are present in the blood, BM biopsy is not strictly necessary but may further characterize the disorder. BM biopsy of a cat with erythremic myelosis will reveal marked erythroid hyperplasia with maturation arrest and erythroid dysplasia. Following on the previous discussion of nonregenerative IMHA, it is difficult to distinguish malignant from non-malignant causes of erythroid hyperplasia with dyserythropoiesis. How may AML, which carries a poor prognosis, be distinguished from similar appearing nonregenerative IMHA, which carries a relatively good prognosis? First, a positive test for FeLV supports a diagnosis of AML. Second, the greater the percentage of blast cells in the BM the greater the likelihood of AML, but there is no specific cut-off value. Third, the greater the presence of abnormal cells on the CBC, the greater the likelihood of leukemia. Fourth, aspiration of enlarged spleen, liver or lymph nodes may reveal neoplastic cell infiltration. Fifth, concurrent immune-
mediated disorders, and positive ANA or Coomb’s tests support nonregenerative IMHA. Finally, FeLV-negative AML is uncommon in cats. Because it is difficult to confirm a diagnosis of AML, and cats with this diagnosis are often euthanized, the clinician is strongly encouraged to treat for IMHA in an FeLV-negative cat.

**Myelodysplastic syndrome (MDS)**
MDS is characterized by low blood cell counts but normal-to-increased BM cell counts, myelodysplasia, and a risk of progression to AML. It may also be caused by FeLV. The key problem is an abnormal clone of progenitor cells that may suppress, displace and progressively replace normal marrow cells. The hematologic and clinical pictures are highly variable, and it may be even more difficult to distinguish MDS from a nonregenerative IMHA. There are also other causes of dysplastic changes in the marrow, and work-up should include investigation for infections, drugs, toxins, nutritional deficiencies and non-AML neoplasia. Treatment of MDS has historically relied on transfusions. If MDS is a differential diagnosis based on CBC and BM biopsy, but the cat is FeLV-negative, then as with AML, immunosuppression and observation of response is the most practical course.

7. **Displacement of erythroid tissue (myelophthisis)**
Erythroid cells may be crowded out by metastatic cancer cells, lymphoblasts, granulomatous inflammation (e.g. histoplasmosis), myelofibrosis, or osteopetrosis. As with other causes of generalized BM injury, neutropenia and thrombocytopenia may be more responsible for clinical signs than anemia. BM biopsy is diagnostic and usually lymphoid leukemia does not pose the same diagnostic dilemma as do AML and MDS.

Myelofibrosis is a non-specific finding: it may be idiopathic, the result of FeLV infection, chronic inflammation in the BM (including autoimmunity – it is a common finding in nonregenerative IMHA), secondary to a primary leukemia, or in some cases a primary tumor of BM stromal cells. Neutrophil and platelet numbers are often normal, and moderate-to-severe nonregenerative anemia is the salient finding. The anemia is typically normocytic normochromic. Idiopathic myelofibrosis is usually treated immunosuppression. Osteopetrosis may be a feature of FeLV subgroup C induced BM failure.

8. **Depression by disease (“anemia of chronic disease”)**
Non-hematologic diseases depress erythropoiesis by various mechanisms. Diseases include infections, non-septic inflammation, neoplasia, liver diseases and chronic kidney disease. Some mechanisms of anemia are shared by the disorders, but there are enough differences in mechanisms and in onset of anemia that render “anemia of chronic disease” too broad a term.

**Anemia of inflammatory disease (AID) and cancer-associated anemia**
Anemia of inflammatory disease (AID) is caused by complex derangements of cytokines, which decrease EPO production and function, iron metabolism, and RBC lifespan. A key
event is iron sequestration, which is believed to be an adaptive mechanism that makes it unavailable to infecting micro-organisms. These mechanisms are present in acute inflammation and Hb levels start decreasing in several days. In contrast to dogs, anemia may develop within 1 - 2 weeks in cats and become severe, in part because of the shorter red cell lifespan. The anemia is normocytic and normochromic. Serum iron should be low-normal to low, but in contrast to iron deficiency, AID is supported by documenting low-normal to decreased transferrin, and high-normal to elevated ferritin, although these changes are not always present. BM biopsy should reveal normal-to-mildly depressed erythropoiesis.

Cancer-related anemia is common in humans and is considered to be common in animals, but is not well-documented. The mechanisms of AID are involved in cancer-related anemia, and iron-sequestration in paraneoplastic AID may be protective. Correcting anemia with transfusion or rHuEPO improves quality of life, but the effect on tumor response, tumor progression, and overall patient survival in humans is not clear. The current recommendation in cats is to treat anemia in the oncology patient using the same transfusion triggers as with patients with non-neoplastic disorders. Treatment with rHuEPO has also been used for this purpose; It is not known if the risk for antibody formation is the same as with chronic kidney disease.

**Feline immunodeficiency virus (FIV)**

Anemia was present in 36% of FIV-positive cats in one case series. Some of the anemias are due to secondary diseases including hemotropic mycoplasma infections and neoplasia, but anemia may occur without these. Anemia is likely due to alteration of the marrow inductive microenvironment and regulatory T-cells. Treatment with rHuEPO therapy may be beneficial, and with a low risk of anti-EPO antibody formation.

**Chronic liver and kidney diseases**

Mechanisms of anemia in liver diseases include AID, malnutrition, reduced RBC lifespan, and bleeding due to coagulopathy and hepatic rupture. Excluding a major bleed, the anemia is mild-to-moderate normocytic normochromic. Anemia in chronic renal failure is progressive and due to EPO deficiency (important mechanism), decreased RBC lifespan, and uremic bleeding. Benefits of EPO go beyond the correction of anemia, but side-effects may occur including aggravation of hypertension and red cell aplasia due to antibody formation. The risk for the latter is less with darbropoetin than erythropoietin. Currently the prognosis for acceptable correction of anemia is about 65%.

**Critical illness**

Anemia is common in critically ill cats because of repetitive blood sampling, surgery, AID, decreased red cell lifespan, and nutritional deficiencies. Transfusion has been associated with worse outcomes in humans in some situations, therefore rHuEPO is being used with increasing frequency. The current recommendation in cats is to transfuse using the same triggers as with anemia due to other causes, ie. based on clinical status.
9. Dilution (pseudoanemia)

Fluid therapy
Hct will decrease with fluid therapy, but is not usually a concern in anemic cats as red cell mass is not affected; tissue oxygenation is likely to improve in anemic dehydrated cats because of improved circulation. The main concern is volume overload.

Pregnancy
Plasma volume and erythropoiesis increase during pregnancy, but the degree of plasma volume expansion is relatively greater than the increase in red cell mass, therefore Hct decreases. In a study of six cats, mean Hct one day after breeding was 36%. At term Hct was 28%, and mild increases in aggregate and punctuate reticulocytes were reported. Congestive heart failure due to volume expansion has been reported in pregnancy.

Anesthesia
See Lecture 2.

10. Drugs
Drugs may cause an idiosyncratic nonregenerative anemia, either by immunologic or toxic mechanisms - this has been seen rarely in cats with cephalosporins and TMS-sulfa. There is a known risk for BM suppression from griseofulvin, especially in FIV-positive cats, and antithyroid drugs may cause anemia, thrombocytopenia or neutropenia. Chloramphenicol causes reversible erythroid suppression by inhibiting Hb synthesis, and rHuEPO may result in pure red cell aplasia with a risk of 20-25%.

References & Suggested Reading:
Point-of-Care Evaluation of Hemostasis
Dr. Anthony Abrams-Ogg, DVM, DVSC, DACVIM (SAIM)

Synopsis
Traditional and novel methods to evaluate primary hemostasis, secondary hemostasis, and fibrinolysis in a general practice and emergency clinic will be discussed, focusing on both technical aspects and interpretation of results. The classical and cell-based models of hemostasis will be briefly reviewed.

Overview
The cell-based\(^1\) and traditional cascade models of hemostasis are reviewed elsewhere in this conference. The cell-based model gives a better understanding of the integration of the various steps of hemostasis, but the traditional model remains the most useful for daily clinical work. The traditional model divides hemostasis into primary and secondary hemostasis. Primary hemostasis refers to the interactions between the blood vessel wall, platelets and vWf. Primary hemostatic defects may be inherited or acquired. Classically primary hemostatic defects are characterized by petechiae, ecchymoses, and mucosal bleeding at multiple sites, as well as excessive and prolonged bleeding after injury, although this characterization is influenced by thrombocytopenia, which is the most common defect. Secondary hemostasis consists of the coagulation factor cascades that ultimately result in stabilizing a platelet plug with fibrin. The coagulation factors are: I (fibrinogen), II (prothrombin), III (tissue factor or platelet phospholipid), IV (calcium), V, (there is no factor VI), VII, VIII, IX, X, XI, XII (Hageman Factor), and XIII. These factors are named more in order of discovery and naming than functional order. They classically belong to the intrinsic, extrinsic, and common systems. Bleeding due to coagulopathies may be localized or widespread. Hemarthrosis, pericardial bleeding, hemothorax, hemoabdomen and subcutaneous hematomas may occur, which are not typical of primary hemostatic defects. Excessive bleeding may also be delayed after injury, and rebleeding may occur, especially with defects in the intrinsic system. In the classical view of hemostasis this occurs because bleeding is initially controlled by primary hemostasis, but the platelet plug is not converted into a firm clot. In the cell-based model, this occurs because the initiation phase initially controls bleeding, but the thrombin burst of the propagation phase does not occur. Depending on severity, vitamin-K antagonist poisoning may be characterized by either immediate or delayed bleeding after injury.

I. Disorders of primary hemostasis
A) Disorders of blood vessels
Hereditary hemorrhagic telangiectasia is a rare disorder in humans that may cause widespread cutaneous petechial-like lesions and mucosal bleeding. It has not been reported in dogs or cats, although focal congenital telangiectasias have been reported, and Welsh Corgis may be affected by renal telangiectasia causing hematuria. Hepatic telangiectasia may occur in older dogs and cats and is asymptomatic. However, a similar condition, peliosis hepatis, is characterized by blood-filled cysts, that may rupture
spontaneously and cause hemoabdomen. It has been associated with *Bartonella henselae* in humans and in a dog but not in cats. Acquired cutaneous telangiectasia resulted in erythema in a dog.

Cutaneous asthenia (Ehlers-Danlos syndrome) refers to several hereditary collagen defects that cause hyperelastic skin and easy wounding. They may be associated with ecchymoses, subcutaneous hematomas, and excessive bleeding in humans, but such bleeding has not been a prominent feature of the disorders in dogs and cats. Acquired vascular fragility may occur with hyperadrenocorticism in dogs, characterized by ecchymoses. Hyperadrenocorticism in cats causes easy wounding, but bleeding is usually minimal.

Vasculitis may result in focal or generalized erythema, edema, ecchymoses, and ulcers. There are numerous causes.

Presumptive diagnosis of vascular disorders relies on clinical, laboratory and imaging findings typical of the associated disorder. Definitive diagnosis is by skin biopsy or biopsy of other affected organs; there is no specific point-of-care test. Hemostasis would normally be evaluated before biopsy, and results would be normal unless there was an associated abnormality (e.g. ITP and vasculitis; vasculitis, thrombocytopenia, and thrombocytopenia causing bleeding in ehrlichiosis).

**B) Thrombocytopenia**

Hereditary asymptomatic thrombocytopenia with macroplatelets occurs in Cavalier King Charles spaniels, greyhounds often have asymptomatic mild thrombocytopenia, and thrombocytopenia also occurs in the Polish ogar dog.

Symptomatic thrombocytopenia is usually acquired in dogs and cats. The most common mechanisms are reduced platelet production (megakaryocytic hypoplasia), increased consumption (DIC), and increased destruction (ITP, dogs >> cats), which may be due to a number of disorders, especially neoplastic, infectious, autoimmune or toxic causes. Platelet loss during marked bleeding is not a common mechanism for clinically relevant thrombocytopenia, but may occur following massive volume replacement with fluids or platelet-poor blood products. Platelet sequestration due to splenomegaly is uncommon.

Thrombocytopenia may cause spontaneous, immediate, excessive and prolonged bleeding, often at multiple sites. Clinical signs of thrombocytopenia include cutaneous bleeding (petechiae and ecchymoses), mucosal bleeding (epistaxis, petechiae, melena, hematuria), neurologic signs (CNS bleeding) and ocular hemorrhages. The risk for bleeding is inversely proportional to platelet count and is nominally logarithmic (Table 1).

**Severity and risk for bleeding with thrombocytopenia**

<table>
<thead>
<tr>
<th>Definition</th>
<th>Platelet Count</th>
<th>Risk for Bleeding</th>
</tr>
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Grade 1  100 x 10^9/L–lower ref.  Not increased
Grade 2  50 – 99 x 10^9/L  Increased surgical bleeding
Grade 3  25 – 49 x 10^9/L  Microscopic spontaneous bleeding
Grade 4  < 25 x 10^9/L  Spontaneous clinical bleeding – mild risk
      < 10 x 10^9/L  Spontaneous clinical bleeding – moderate risk
      < 5 x 10^9/L  Spontaneous clinical bleeding – marked risk

These figures are guidelines based on megakaryocytic hypoplasia. Bleeding at any platelet count is worse if there is concurrent sepsis, coagulopathy (e.g. DIC), vWD, platelet function defect, or vasculitis. Dogs with ITP and cats typically bleed less than expected at a given platelet count.

Point-of-care tests for thrombocytopenia include routine blood smears and in-house hematology analyzers. Platelet counts vary with the counting method used and may not be directly comparable, and enumeration is more imprecise at low values.

Thrombocytopenia as reported by a hematology analyzer should always be confirmed by microscopic examination of a blood smear, and a platelet count should be disregarded if there is an error message. The feather edge should be examined for platelet clumps, usually a result of difficult venipuncture (and rarely induced by EDTA). The platelet count should be estimated from the red cell monolayer where about 50% of cells are touching - each platelet per oil immersion field represents 15,000–25,000/µL.

Mean platelet volume and platelet distribution width are both inversely related to platelet count and usually do not help in differentiating the causes of thrombocytopenia. Similarly, platelet morphology changes are non-specific, and the diagnosis of cause is based on clinical and other laboratory findings, imaging and biopsies for neoplasia and liver disease, testing for infectious diseases, and bone marrow biopsy. Cavalier King Charles Spaniels may be affected by pathologic thrombocytopenia, in which case the clinical presentation is similar to other dogs.

Plateletcrit (analogous to hematocrit) reflects platelet mass; a QBC analyzer calculates platelet number from platelet mass and thus may be the best method to confirm pathologic thrombocytopenia in this breed.

Treatment of thrombocytopenia includes addressing the primary cause, gentle handling, transfusion, thrombopoietic drugs and prothrombic drugs (See Lecture 5). Platelet transfusion is a short-term emergency measure. Blood products include: 1) fresh whole blood, platelet-rich plasma, or fresh-frozen plasma (which contain platelet particles), 10 - 20 mL/kg; 2) 1 unit/10-30kg fresh, cryopreserved or lyophilized platelet concentrate or cryoprecipitate, where 1 canine unit refers to product derived from a 450 mL unit of whole blood; 3) platelet concentrates produced by apheresis. Transfusion should be given if critical bleeding is occurring, and prophylactic transfusion may be considered with platelet counts < 5–10 x 10^9/L. Transfusions may be needed q1-3 days if severe thrombocytopenia persists and economic constraints and limited blood bank resources usually preclude long-term transfusion support. Transfusion has the greatest utility when thrombocytopenia is due to reduced production and rapid resolution is
anticipated. It is less useful in DIC, and least useful, but not always useless, in ITP (See Lecture 5). Fresh whole blood is normally procured in-house. The other blood products are variably available from commercial blood banks.

**C) Thrombocytopenia**

Hereditary platelet function defects may be diagnosed at all ages, but often first appear when excessive bleeding occurs with loss of deciduous teeth. Platelet counts are usually normal. Defects have been identified in the Bassett hound, Landseer, Finnish Spitz, otter hound, Great Pyrenees, grey collie (associated with cyclic neutropenia), American cocker spaniels, boxers, German shepherd dogs, and mixed-breed dogs.  

Chediak-Higashi syndrome is a defect in Persian cats, which also have dilute smoke-blue coat color and yellow-green irises. Hereditary platelet function defects are rare, and the most likely one encountered is perhaps Basset hound thrombopathy.

The most clinically relevant naturally acquired platelet function defect is probably with ehrlichiosis in dogs. Acute ehrlichiosis may cause mild-to-moderate thrombocytopenia, but bleeding characteristic of primary hemostatic defects such as epistaxis occurs at platelet counts that would not typically result in bleeding. Platelet function defects also occur in leishmaniasis. Platelet function defects have been variably demonstrated in uremia in dogs, but the contribution of thrombocytopenia to bleeding from oral and gastrointestinal ulcers, and to bleeding in hypertensive retinopathy is not known. The author encountered one dog in renal failure where severe bilateral hyphema was attributed to thrombocytopenia and hypertension. Platelet function defects are also common with monoclonal gammopathies from multiple myeloma, and probably contribute to the epistaxis that may be seen with this disorder. Compared to ITP, immune-mediated thrombocytopenia is rare.

Numerous drugs are reported to affect platelet function in humans, but dog and cat platelets are less sensitive. Specifically, commonly used antibiotics that affect platelet function in humans do not do so in dogs (and probably not in cats either). Although acepromazine has been reported to affect platelet function in normal dogs, this was not confirmed in recent studies. Depending on dose and other factors, NSAIDS that affect COX-1 may inhibit platelet function in dogs and cats, as does clopidogrel, and these effects are used for thromboprophylaxis.

For a long-time platelet function testing was restricted to platelet aggregometry in specialized laboratories. A number of point-of-care instruments for platelet function testing are now available. These include the PFA-100®, TEG® Platelet Mapping, and Impact-R™, but are currently cost-prohibitive to most practices and samples must be fresh which limits submission to a reference laboratory. Plateletworks® is another point-of-care test that holds promise for use in general clinical practice as it uses a Coulter-counter technology. As of now, the only routinely available platelet function test to most veterinarians is bleeding time, which should only be performed after ruling-
out thrombocytopenia. Various methods have been used – the current standard test is buccal mucosal bleeding time (BMBT). The test is operator-dependent.

**Buccal Mucosal Bleeding Time Procedure:**
1) Lateral recumbency; cats must be sedated or anesthetized. 
2) Strip of gauze around maxilla, folding-up upper lip, causing moderate engorgement; 
3) Position Surgicutt* (ITC) device gently against upper lip mucosa and push trigger. (If not available, make an incision 5 mm long x 1 mm deep with a #11 scalpel blade); 
4) Note start time; 
5) Blot blood with blotting paper 1-3 mm below incision (do not directly touch incision); 
6) Note time when bleeding stops. 
7) Normal BMBT is = 3 min in cats and ≈ 4 min in dogs.

**D) Von Willebrand Disease (vWD)**\(^{9,14,15}\)
This is the most common hereditary blood disorder of dogs and has been identified in many breeds. Different genotypes and inheritance patterns have been identified and the phenotype (risk for bleeding) is variable. The clinical hallmark of the disorder in bleeders is immediate, excessive, and prolonged bleeding from oral, cutaneous and deeper wounds. Mucosal bleeding may occur, manifested by oral bleeding (most common), hematuria, epistaxis, and excessive bleeding in estrus and post-partum, but melena is not common. Ecchymoses and occasionally hematomas may occur, presumably due to excessive bleeding post blunt trauma, but petechiae are not typical. The lower risk for severe spontaneous bleeding, petechiation and melena are features distinguishing vWD from platelet disorders. Some of these differences occur because other proteins are also involved in platelet adhesion.

Von Willebrand factor is a variable-sized glycoprotein comprised of identical subunits held together by disulfide bonds. Circulating vWF varies from dimers to multimers >38 monomers. The larger multimers are more functional. Canine vWD is classified as Type 1, 2 or 3 based on plasma vWF concentration and multimer size. Type 1 is characterized by variably low vWF concentration, but normal multimer distribution, resulting in mild-to-moderate bleeding tendency. It is the most common form affecting many breeds, including Doberman Pinschers. Type 2 vWD is characterized by variably low vWF concentration, as well as reduction in larger multimers, resulting in moderate-to-severe bleeding tendency. Type 2 vWD affects German shorthaired Pointers and German Wirehaired Pointers. Type 3 vWD disease is characterized by complete absence of vWF resulting in a severe bleeding tendency. Type 3 vWD has been identified in Chesapeake Bay Retrievers, Scottish Terriers, Shetland sheepdogs, Dutch Kooikers and sporadically in other breeds. Generally a single type of vWD is seen in a given breed, although there are exceptions – for example both Type 1 and 3 vWD have been seen in Shetland sheepdogs.

The standard test for vWD is measurement of plasma vWF concentration at a reference laboratory. This is done by immunologic tests hence is reported as antigen (vWF:Ag). It is
reported as a percentage of the concentration found in pooled plasma from normal dogs, which is given a value of 100%. Given that vWF:Ag is variable in normal dogs, and a dog with very mild vWF deficiency could have contributed to the plasma pool, it is easy to understand why there is a grey zone between abnormal and normal. Plasma vWF:Ag shows variation in individual animals and may be affected by other physiologic and pathologic states. For example, vWF:Ag tends to rise during gestation and exercise. These changes are most likely to affect assigning a vWD positive or negative status to dogs with values in or close to the grey zone between normal and abnormal values. The vWF:Ag is a quantitative measure – it does not measure vWF function, which is dependent on multimer size. However, if multimer distribution is normal, the lower the vWF:Ag the lower the vWF function and the greater the risk for bleeding. Multimer distribution may be determined in some specialty hematology laboratories by Western blot, and may also assay vWF function by collagen binding (vWF:CBA). Specialty laboratories may also offer PFA-100© testing, where vWD may increase closure time. The most commonly used point-of-care test is BMBT. A questionnaire was also recently reported investigating history of bleeding episodes. The questionnaire is not sufficiently sensitive to act as a screening test for vWD for dogs in general, but positive responses increase the likelihood of vWD; the predictive value of the questionnaire for vWD when a dog is presented for a bleeding episode is unknown.

Testing a dog for vWD may be with intent to: 1) identify the cause in a dog presented for clinical bleeding; 2) screen for risk for bleeding prior to surgery; or 3) screen for a known genetic mutation prior to breeding. In a dog with abnormal bleeding due to vWD, the likely hemostatic results will be prolonged BMBT, normal platelet count, normal ACT, normal-to-slightly prolonged aPTT, normal PT, normal fibrinogen/TT, and decreased vWF:Ag and vWF:CBA. In an emergency situation with limited testing resources, prolonged BMBT, normal platelet estimate, and normal whole blood clotting time or ACT in a dog with signs of a primary hemostatic defect will have a high positive predictive value for vWD. Assuming a normal BMBT of <4 min, dogs with Type 1 vWD have BMBT > 5-6 min, and dogs with Type 2 and 3 vWD have BMBT > 12 min.

With respect to screening a dog for risk of hemorrhage before surgery, neither BMBT, vWF:Ag or history can be used to directly predict risk. This probably reflects, at least in part, operator variation in BMBT, surgical procedures, and judgment of excessive bleeding. Predicting hemorrhage is most difficult for dogs with mild-to-moderate Type 1 vWD. Dogs have been judged to be bleeding excessively with normal BMBT, and dogs with low vWF:Ag and prolonged BMBT have also been judged to not have excessive bleeding. However, this does not mean the tests are useless. Overall the longer the BMBT and the lower the vWF:Ag, the more likely there is to be excessive bleeding and these tests will be abnormal in dogs with moderate-to-severe vWD, i.e. dogs that are at increased risk for bleeding. Genetic testing for a number of vWD genotypes is also commercially available (VetGen). A finding of an abnormal genotype will not predict risk for bleeding in Type 1 vWD, whereas a dog with a normal genotype would not have abnormal bleeding due to vWD.
Testing for vWD genotype and removing positive dogs from a breeding program has proven to be a valuable tool to reduce prevalence of the disorder in a breed.\textsuperscript{16} Measurement of vWF:Ag is not sufficiently sensitive for screening as some carriers will fall in the grey zone between normal and abnormal. However, data correlating genotype and phenotype are not complete, and measurement of vWF:Ag in dogs that have had a genetic test is encouraged.

Prophylactic treatment or treatment of a bleeding episode includes desmopressin and transfusion. Desmopressin is given at a dose of 1 \(\mu g/kg\) SC 30 min before surgery to raise plasma vWF concentration. This has resulted in improved vWF function, decreased BMBT and PFA-100 closure times in dogs with Type 1 vWD.\textsuperscript{17-19} Transfusion of vWF may be given via fresh whole blood (10 – 20 mL/kg), fresh-frozen plasma (10 mL/kg), fresh platelet-rich plasma (10 mL/kg) or cryoprecipitate (1 unit/10kg). Fresh-whole blood or fresh-frozen plasma are the most readily available products. Canine vWF is labile, but studies at the author’s institution have shown it is stable in plasma at room temperature for 24 hours, so deterioration during transfusion is not a concern. Transfusions may need to be repeated every 6 – 12 hours to maintain adequate vWF concentrations. Repetitive transfusions with fresh whole blood may create volume overload and polycythemia. Although of unproven additional benefit, at the author’s institution donors are given desmopressin to boost vWF levels in fresh-frozen plasma and cryoprecipitate.

Acquired von Willebrand Syndrome is a well-documented but rare disorder in humans. It may be a complication of autoimmunity, hematologic neoplasia, hypothyroidism, and increased shear forces with cardiovascular implants. The disorder has been reported as a complication of angyostrongylosis in a dog.\textsuperscript{20} It has not been documented as a complication of hypothyroidism in dogs.\textsuperscript{21} Mitral valve insufficiency may cause a lowering of vWF concentration and function, but not to the point of causing a bleeding tendency.\textsuperscript{22}

II. Disorders of secondary hemostasis

Extrinsic System (Tissue Factor Pathway)
Tissue factor (formerly tissue factor III) is released from damaged tissue into the bloodstream. (It is called the extrinsic system as tissue factor is extrinsic to the blood.) Tissue factor activates Factor VII, which then activates the common system. The extrinsic system is the most important for the initiation of coagulation (mnemonic “lucky 7”).

Intrinsic System (Contact Activation Pathway)
Factor XII is activated by contact with a negatively charged surface, and then Factors XI, IX, and VIII are sequentially activated, followed by activation of the common system (discount store mnemonic “It’s not $12 it’s $11.98”). The intrinsic system is most
important for sustaining coagulation once initiated.

**Common System**
This is termed the common system as it is the terminal pathway for both the extrinsic and intrinsic systems. Factor X and Factor V activate Factor II (mnemonic X = V times II), which converts Factor I into Fibrin. The fibrin clot is stabilized by Factor XIII.

**Fibrinolytic System**
*Tissue plasminogen activator* (tPA), urokinase plasminogen activator, and *Factor XII* (Hageman factor) convert plasminogen into plasmin, which breaks down the fibrin clot, releasing fibrin degradation products (FDPs) and D-dimers.

**A) Routine coagulation tests – whole blood clotting assays**
The first test of coagulation was observing for whole blood clotting in a tube, and this may still be used if nothing else is available. For the Lee-White clotting time, 0.5 - 1 mL of fresh blood is put in a tube and incubated in a 37 °C water bath/heating block, axilla or hand, and inverted every 30 sec to observe for a clot. With a minimally traumatic venipuncture there is minimal tissue factor contamination and coagulation is initiated by contact of Factor XII with the wall of the tube. It is thus an assessment of the intrinsic and common systems. The main problems with the test are the time it takes and the broad reference interval (5 – 15 min for glass tubes in humans) which makes it relatively insensitive. Values have never been reported for dogs and cats, but most samples in glass tubes clot < 5 min and in plastic < 6 min. A Lee-White clotting time > 10 min in a dog or cat is likely to represent a coagulopathy.

A modification of the Lee-White clotting time is the activated clotting or coagulation time (ACT). This is a very useful point-of-care test of coagulation. It was first introduced in 1966 for the rapid diagnosis of hemophilia A, but is now used in human medicine predominantly for the monitoring of heparin therapy during cardiopulmonary bypass. In veterinary medicine it is used mostly as a screening test for coagulopathy. For this assay a special tube is used which contains silica-based particulate matter to activate Factor XII (the mechanism of activation is not well-understood). Similar to Lee-White time, it is an assessment of the intrinsic and common systems, but the activator accelerates the clotting time and gives a more predictable interval. For many years the test was performed in dogs and cats using the glass Vacutainer ACT tube by Becton Dickinson, which contained diatomaceous earth (aka siliceous earth, Celite, celite), but it is no longer available. Two mL of blood was added to the tube, which was incubated at 37 °C in a water bath or heating block for 60 sec, and then examined every 5-10 sec by tipping the tube to the side. The ACT was recorded as the time where a clot was first noticed; a solid clot normally formed within another 10 – 20 sec. Axillary incubation instead of heating block/water bath incubation was validated. In addition 0.5 - 1 mL was frequently used in cats and small dogs. The test is operator-dependent, and various reference intervals were reported, but in general the ACT for dogs is < 2 min and for cats < 1.5 min. Subtle changes in ACT and subclinical disorders in coagulation could be
missed by a clinic not having established its own reference interval, but any major bleeding tendency will result in substantial prolongation of ACT.

Alternative tubes for this visual testing method that are now available include: 1) The AcTube™ (Vetlab Supply), which contain celite; and 2) the SCAT-ACT tube (Haematologic Technologies), which contain kaolin. Results are similar to those with the Vacutainer ACT, but they have not been formally evaluated in dogs and cats, and the results between the two activators are probably not identical (they are not in humans). As above, these differences are probably not relevant for screening for a major bleeding tendency. However, a practice is encouraged to develop its own reference interval and keep a log of values in sick animals. In addition to the tubes designed for visual reading, there are ACT tubes designed for reading by instruments. These include: 1) Hemochron® (celite activator); 2) Hepcon® (kaolin activator); and 3) Actalyke®, which has celite tubes, kaolin tubes, and MAX-ACT tubes, which contain celite, kaolin and glass beads designed to maximally activate Factor XII. The Actalyke® system contains a small magnet - once a clot forms the magnet can no longer turn, and this is the end point. All the instruments have been used in research, but reference intervals for clinical use have not been reported. Clot formation in the tubes for these systems can also all be read visually, but results have only been reported for the MAX-ACT.24 Using a 0.5 mL sample incubated in a 37 °C water-bath with the end-point being solid clot formation, ACT in dogs was 55 – 80 sec and in cats 55 – 85 sec.2 Current studies at the author’s institution are comparing axillary incubation (temperature ~36 °C), 37 °C water-bath, and Actalyke incubation set at 38.4 °C. Results reveal essentially the same values as previously reported for 37 °C, but longer values for axillary and 38.4 °C Actalyke® incubation. The close correlation between axillary and 38.4 °C Actalyke® values has been maintained over a range of abnormal values. The working reference interval for axillary incubation in normal dogs is 70 – 105 sec using an end-point of first clot formation, and in normal cats < 60 sec – 90 sec using an end-point of solid clot formation. First clot formation may be difficult to judge with the small volume, therefore the author recommends recording both first clot and solid clot end-points.

In addition to the tube-based ACT, two veterinary point-of-care instruments, the SCA2000 (Synbiotics) and VetScan i-STAT 1 (Abaxis), measure ACT on small samples. With the SCA2000 blood is mixed with celite and LED optical detectors recognize decreased movement of the forming clot. Another instrument, Hemochron® Signature Elite uses the same principle. With the i-STAT the blood is mixed with celite, but the end-point is not conversion of fibrinogen to fibrin by thrombin, but rather conversion of another molecule by thrombin, which is then detected electrochemically.

All the above methodologies of ACT are valid, but results with different systems cannot be directly compared. A prolonged ACT should not in-itself be taken as a transfusion trigger for fresh-frozen plasma. Prolongation of the ACT must be interpreted in the context of clinical findings and other test results. The ACT may be prolonged due to: 1)
Factor deficiency; 2) DIC; 3) inflammation; 4) acquired anticoagulants; 5) pharmacologic anticoagulants; and 6) severe thrombocytopenia. These will be considered in turn:

1) Factor deficiency. Vitamin-K deficiency or antagonism (poisoning) results in decreased production of Factors II, VII, IX and X. The prolongation of clotting times is dependent on the half-life of the Factors. Factor VII has the shortest half-life, therefore PT is affected first, followed by APTT. The ACT is affected after APTT, probably because it is a less precise test. However, ACT is usually prolonged if an animal is spontaneously bleeding from vitamin K deficiency/antagonism. It is not as sensitive as PT for monitoring response to treatment or for detecting early vitamin K deficiency. The ACT will also be markedly prolonged in animals clinically bleeding from deficiencies of Factor VIII (Hemophilia A), Factor IX (Hemophilia B), and Factor XI (Hemophilia C). The test is not sufficiently sensitive to detect carriers. As Factor XII is the initiating step of the intrinsic system, deficiency of this Factor (Hageman trait) results in markedly prolonged ACT. This trait is seen occasionally in cats and rarely in dogs. The animals do not have a spontaneous bleeding tendency, as coagulation is initiated via the extrinsic system, and Factor XII is not needed to sustain coagulation as explained by the cell-based model. However, excessive gastrointestinal bleeding may have been contributed to by Factor XII deficiency in one dog and one cat (in the author’s practice). Humans with Factor XII deficiency are at increased risk for thrombosis because Factor XII is an activator of plasminogen. The ACT will also be prolonged in prekallikrein deficiency, a rare inherited disorder reported in dogs, which is also not associated with a bleeding tendency.

2) DIC. The ACT may be prolonged in DIC because of factor consumption and possibly Factor inhibition. In one study the ACT was the most sensitive of the routine coagulation tests for DIC in dogs. Unfortunately ACT has not been included in many studies examining tests for DIC. Some of the other more sensitive tests include platelet count and D-dimers. D-dimers are typically measured in reference laboratories but point-of-care kits exist. Blood smears may also be examined for schistocytes, but these are often not present.

3) Inflammation. Inflammation promotes coagulation through three main processes: 1) activation of both the extrinsic and intrinsic pathways; 2) reduced natural anticoagulants such as antithrombin; and 3) inhibition of fibrinolysis. Furthermore, there is evidence that the more prolonged (and progressive prolongation of) the ACT, the worse is the underlying inflammatory disorder as well as the prognosis. Cases have been trended with respect to ACT, demonstrating normalization as the disorders resolved. It is counterintuitive that ACT is prolonged in animals in a procoagulant inflammatory state. It is possible that clot formation in ACT is normally sufficiently rapid that it is not sensitive enough to detect a procoagulant state; however, once the ACT begins to prolong, this reflects a consumptive coagulopathy secondary to thrombosis (DIC). Most animals with prolonged ACT secondary to inflammation have values under 4-5 min and do not have a spontaneous bleeding tendency. Whether prolongation of ACT is a direct effect of inflammation and/or represents a progression from a procoagulant state to a
consumptive coagulopathy (DIC) is not known. A persistently elevated ACT that cannot be explained by anticoagulants, vitamin K antagonism, inherited Factor deficiency or underlying neoplasm causing DIC should prompt investigation for an inflammatory disorder.

4) Acquired anticoagulants. See discussion in aPTT below.

5) Pharmacologic anticoagulants. The main use of ACT in humans is for monitoring heparinization. The ACT may be used for this purpose as well in animals, but unfortunately optimal target ACT values for specific situations are not known. A general recommendation when treating thromboembolic disease is to target an ACT of 1.5 – 2x baseline value.

6) Severe thrombocytopenia. Platelet phospholipid is needed for coagulation, therefore there is concern that severe thrombocytopenia may prolong ACT. This is poorly documented, but there is some evidence that platelet counts < 10 x 10⁹/L may prolong ACT in humans.²⁹

Neither Lee-White clotting time nor ACT are sufficiently sensitive to detect most procoagulant states, therefore more sophisticated point-of-care whole blood clotting assays are employed. These include Thrombelastography® (TEG®), rotational Thromboelastometry® (ROTEM®), and Sonoclot® analyzers, which are all based on the principle of recording reduced movements of a probe as a clot forms. One point in a tracing would correspond to ACT, and the Sonoclot specifically generates an ACT value. These instruments are cost-prohibitive to most practices, but their use in research will continue to shed light on the interrelationships between coagulation and inflammation and help in interpretation of ACT.

B) Routine Coagulation tests – PT, aPTT, fibrinogen/TT
These were traditionally plasma-based tests to allow freezing and shipment to a reference laboratory. A blood sample is collected in a citrate anticoagulant (blue-top) tube. It is important to not over or underfill the tube, which is best accomplished by letting the tube’s vacuum draw the proper amount. Recently the use of EDTA anticoagulated samples was reported.³⁰ Point-of-care units (e.g. SCA2000, Coag Dx™ Analyzer [IDEXX], VetScan Vspro [Abaxis]) use citrated or fresh whole blood, and may be used to determine PT and aPTT. For the assays, the citrated sample is recalcified and a thromboplastin (a chemical that initiates a clotting cascade) is added, and the time to clot formation is determined using various methods. The clotting times vary with methodology, and it is important to use the laboratory’s/instrument’s reference interval. The PT and aPTT are classically used to detect risk for bleeding. However, a specific value in itself does not imply a given risk for bleeding, and as with ACT, the result must be interpreted in context of the disease which is creating the abnormality. All laboratory tests may give erroneous results, but coagulation tests are particularly prone to pre-analytic error, mostly due to difficult blood collection, especially in cats.
Shortened clotting times may indicate increased thrombotic risk, but they are not sensitive in this regard.

Prothrombin time (PT) assesses the extrinsic and common system. It was the first specific coagulation test to be developed. Its main clinical use is in detecting vitamin K deficiency or antagonism, and in this context the longer the PT the greater the risk for bleeding. If PT is not prolonged, then a vitamin K problem is not contributing to a bleeding tendency. The Thrombotest is a commercially available modified PT. It is also known as a PIVKA (Proteins Induced by Vitamin K Absence/Antagonism) test in the veterinary literature, but it is not clear if it is actually measuring the nonfunctional clotting factors. It is more sensitive than a routine PT for detecting a vitamin K disorder. It is not necessary for the diagnosis of vitamin K antagonist poisoning in the bleeding animal, but is useful in identifying subclinical vitamin K deficiency and early poisoning. The PT may also be prolonged in liver failure and DIC, and by circulating anticoagulants (not reported in dogs or cats). Hereditary Factor VII deficiency has been described in beagles and Alaskan Klee Kai dog; PT is prolonged in dogs with bleeding but not in carriers.31

Activated partial thromboplastin time was developed after PT. Similar to ACT, it evaluates the intrinsic and common pathways. Similar to ACT, an activator (e.g. celite, kaolin) is used to activate Factor XII. Phospholipid is also added as there is no platelet phospholipid in the plasma samples, which is essential for the clotting process. The “partial thromboplastin” refers to the absence of tissue factor. Any process that prolongs ACT may also prolong aPTT, but the two tests do not often directly correlate well with each other, reflecting different sensitivities and precision under different circumstances. The aPTT is not sufficiently sensitive to detect carriers of hemophilia A, B or C. As with other hereditary coagulopathies, specific factor quantification through a reference laboratory is needed to characterize the abnormality and detect carriers. An aPTT is more likely to be prolonged in chronic hepatitis than PT.32 Prolongation of the aPTT (and ACT) due to acquired circulating anticoagulants is uncommonly seen.33 These are autoantibodies against a coagulation factor (e.g. Factor VIII) or phospholipid. This can be investigated by mixing patient plasma 50:50 with normal plasma and repeating the aPTT. As aPTT will not be prolonged until there is < 25% Factor function, the aPTT should normalize if it is prolonged due to Factor deficiency. In contrast, because an inhibitor in the patient is likely to antagonize Factors in normal plasma, the aPTT will not normalize if such an anticoagulant is present.

Thrombin time measures the time for thrombin added to a plasma sample to convert fibrinogen into a fibrin clot. It is primarily used to measure fibrinogen concentration (other methods are also used). The test will be prolonged if fibrinogen is low (e.g. advanced DIC) or abnormal (rare), and with heparin. Thrombin levels are decreased with vitamin K antagonist poisoning, but thrombin time is not affected as exogenous thrombin is added to the sample to run the test.
Summary of point-of-care tests of hemostasis

Vascular disorder – none (except ruling-out other causes)
Thrombocytopenia – blood smear, in-house hematology analyzer
Thrombocytopenia – BMBT, Plateletworks®
Von Willebrands disease – BMBT, history questionnaire
Coagulopathy – ACT, in-house PT/aPTT instruments
Prothrombotic inflammatory state – ACT, in-house PT/aPTT instruments
Excessive fibrinolysis (DIC) – D-dimer, schistocytes
Active thrombosis – D-dimer

References
Standard-of-Care for Treatment of Immune-Mediated Hemolytic Anemia and Immune-Mediated Thrombocytopenia in Dogs  
Dr. Anthony Abrams-Ogg, DVM, DVSC, DACVIM (SAIM)

Synopsis  
Current understanding of prognostic factors, immunosuppressive therapy, thromboprophylaxis, transfusion, and supportive care of dogs with IMHA and/or ITP will be discussed.

Overview  
In the last decade there have been insights into the hemostatic and inflammatory changes associated with immune-mediated hemolytic anemia (IMHA), but despite some modifications of therapy, the mortality rate remains unacceptably high. The average mortality rate reported during first hospitalization or immediate follow-up period from referral hospitals had been > 50% for more than two decades. In 2005, a large retrospective study reported a significantly lower mortality rate (< 30%) with a protocol based on azathioprine and low-dose acetylsalicylic acid (ASA), in addition to standard glucocorticoid therapy. This protocol was rapidly widely adopted, although subsequent reports have not always had the same success. Current common standard-of-care for acute/fulminant IMHA includes immunosuppressive and anti-inflammatory therapy with glucocorticoids ± azathioprine ± cyclosporine; ± thromboprophylaxis with ASA, clopidogrel, or heparin; transfusion to correct severe anemia; gastroprotection; and supportive care. Standard-of-care for immune-mediated thrombocytopenia (ITP) includes similar immunosuppression, transfusion, and gastroprotection ± treatment with vincristine. The mortality rate for ITP is lower than with IMHA (<10% in two recent studies), but cases refractory to standard-of-care persist. Standard-of-care management of IMHA and ITP will be reviewed, with a recognition that some of the trend to intensify therapy is born out of frustration and a need to do more, but is not of proven benefit. Other potentially beneficial treatments exist, but many are potentially prohibitively expensive and/or of limited availability.

Etiology and Pathophysiology  
IMHA results from antibodies binding to red cells. Hemolysis is most often extravascular, where red cells are destroyed by the mononuclear phagocyte system, in particular in the spleen. Red cell autoantibodies may also cause complement-mediated intravascular hemolysis. The period of acute hemolysis is associated with a systemic inflammatory response. IMHA may be primary, or secondary to another disorder or treatment. When a cause cannot be found, IMHA is assumed to be primary, which is the most frequent diagnosis. There are two broad forms of IMHA: the subacute to chronic form, where there is a history of slowly progressive inappetence and exercise intolerance, and initial treatment is on an out-patient basis using prednisone; and the acute-to-fulminant form, which is the focus of this review.
The cause of death in IMHA may be euthanasia due to cost of therapy, refractory anemia, organ dysfunction presumed secondary to thromboembolism, or hemorrhage secondary to disseminated intravascular coagulation (DIC). Natural death is presumed due to thromboembolism. The pathogenesis of thromboembolism is multifactorial.\(^6\) Thrombocytopenia may occur from concurrent ITP (Evan’s syndrome) or DIC.

ITP similarly results from antibodies binding to platelets, is either primary or secondary, and has a spectrum from acute to chronic. It is assumed that most platelet destruction is extravascular. Overall dogs with ITP are not as sick as dogs with IMHA, perhaps because there is less of an inflammatory response. The causes of death in ITP include euthanasia due to cost of therapy, refractory thrombocytopenia, or severe hemorrhage. Natural death is due to hemorrhage, and, if not present, then likely arrhythmias or thromboembolism. Severe hemorrhage is usually gastrointestinal, but central nervous system bleeding may also occur. There is evidence that humans and dogs with ITP are prothrombotic,\(^8\) but the risk for thromboembolism is lower, presumably because of thrombocytopenia itself and possibly because ITP may be a less inflammatory state than IMHA.

**Diagnostic Plan**

Signalment - Middle-aged spayed female dogs are at increased risk, although IMHA and ITP may occur in young and old dogs of either sex. Various breeds have been overrepresented in different studies, but American Cocker Spaniels have emerged as a breed at risk for both disorders.

History - Dogs with IMHA have: 1) acute onset of depression, inappetence, weakness, and exercise intolerance due to acute anemia and acute phase response; and 2) variable vomiting and diarrhea, presumed due to gastrointestinal ischemia and/or pancreatitis. Additionally, 3) owners may report discoloured urine, due to bilirubinuria or hemoglobinuria. Underlying/concurrent disorders may cause additional signs. Dogs with ITP have: 1) variable depression, etc, due to anemia; and 2) variable vomiting and diarrhea. 3) Owners may report/present dogs for hematuria, melena, petechiae and ecchymoses, epistaxis, or other bleeding.

Physical Examination - For dogs with IMHA: 1) pale mucous membranes; 2) icterus is common; 3) tachycardia and prominent pulses; a heart murmur due to anemia may be present; 4) polypnea and, occasionally, dyspnea; 5) variable abdominal pain and fever; 6) hepatosplenomegaly; 7) occasional lymphadenopathy; 8) petechiae and ecchymoses may be evident if there is concurrent ITP. For dogs with ITP: Cutaneous and mucosal petechiae and ecchymoses are the hallmarks. Ocular hemorrhages may also be present. Physical exam findings of with ITP are otherwise similar to those of IMHA except for icterus.

**Laboratory Evaluation/Diagnostic Imaging**
The goals of the work-up of the dog with suspected IMHA and/or ITP are to: 1) confirm the diagnosis; 2) identify any underlying causes of autoimmunity; 3) identify concurrent disorders of autoimmunity or disorders which may impact therapy; and 4) identify initial prognostic factors. These goals justify a routine CBC, serum chemistry profile and urinalysis. Blood samples of dogs with IMHA should be handled gently because of increased red cell fragility.

Confirming the diagnosis of IMHA - The main disorders that may present with a similar clinical picture as acute IMHA are zinc and onion poisoning, and red cell infections (hemotropic *Mycoplasma, Babesia*). Hemotropic *Mycoplasma* infection is rare without a history of splenectomy. Other non-immune causes of hemolysis include hypophosphatemia and hereditary red cell enzyme deficiencies (Springer Spaniels, Basenjis and black Miniature Poodles). The diagnosis of IMHA is most commonly made on the basis of a CBC demonstrating a regenerative anemia and spherocytosis and/or agglutination and/or ghost red cells, and absence of hemoparasites. Regeneration should be present after 3-4 days. Occasionally the anemia is non-regenerative because of destruction of immature red cells in the bone marrow.

To detect agglutination, gently mix equal volumes of EDTA blood and saline in a tube to minimize rouleaux formation, and observe for gross agglutination (blood is flocculent). If agglutination is not seen, place several drops of the mixture on a glass slide and gently rock the slide back and forth to observe for agglutination within 1 minute. (After several minutes rouleaux formation will increase on a glass slide as the sample dessicates.) It is impossible to distinguish rouleaux and agglutination macroscopically. In all cases place a coverslip on the slide and examine the wet-mount microscopically, especially if the diagnosis of IMHA is based on the presence of agglutination. Preparing a wet-mount will also facilitate detection of microscopic agglutination. The common practice of simply observing a drop of whole blood on a glass slide for flocculence and dispersion of potential rouleaux by adding saline is discouraged. Rouleaux microscopically appear as “stacks of coins” and agglutination appears as “clusters of grapes”. If antibodies are not present in a sufficiently high titer to cause agglutination, a lower titer is usually detectable with a Coomb’s test or flow cytometry.

Identify underlying causes of autoimmunity - Review history for drug therapy (presumptive cause). Thoracic radiographs and abdominal radiographs or ultrasound are used to identify underlying foci of inflammation or neoplasia, and problem-based/risk-factor specific testing rather than routine imaging (especially abdominal ultrasound examination) is strongly advocated. Imaging is expensive and financial resources are often better directed towards therapy. The same principle applies for screening for infectious and parasitic diseases. Bone marrow biopsy should be performed to rule-out histiocytic sarcoma in Bernese mountain dogs and flat-coated retrievers.

Identify concurrent disorders of autoimmunity, unrelated disorders which may impact therapy, and complications of IMHA - Concurrent autoimmune disorders include glomerulonephritis (proteinuria), polyarthritis (lameness, swollen joints), and
dermatitis/vasculitis (skin lesions). These are not common but abnormalities would prompt additional work-up. Animals with IMHA may develop subcutaneous edema with or without pleural effusion and ascites during therapy. This is not a sign of vasculitis, but rather reflects water retention triggered by acute anemia, possibly altered endothelial permeability, and overhydration. Older animals are at increased risk for concurrent unrelated disorders. IMHA may cause pancreatitis, which may increase amylase, lipase, and PLI, but pancreatitis has not been identified as a prognostic factor. Azathioprine may also cause pancreatitis; if this is the putative cause, then the drug should be discontinued. Acute dyspnea with unremarkable thoracic radiographs is suggestive of pulmonary thromboembolism.

Identify initial prognostic factors - Negative laboratory prognostic indicators of variable strength include: 1) Degree of leukocytosis – a neutrophilia with or without a left shift is common, due to stress, bone marrow stimulation and tissue necrosis. The higher the neutrophil count the more likely microthrombi are present. 2) Degree of thrombocytopenia, reflecting Evan’s syndrome or DIC; 3) Decreased albumin – perhaps related to acute phase response or water retention; 4) Degree of bilirubinemia, reflecting severity of hemolysis and other factors – this is the negative prognostic factor most consistently (but not invariably) identified; 5) Degree of elevation in ALT, reflecting hypoxic/ischemic necrosis of the liver; 6) Degree of elevation in urea, possibly reflect dehydration, gastrointestinal bleeding, or negative nitrogen balance; 7) Intravascular hemolysis; 8) Prolonged clotting times. Increased activated clotting time (ACT), aPTT, and less often PT, are presumed to be prolonged due to DIC. Thrombelastography (TEG®, a whole blood assessment of hemostasis) also reveals that relatively hypocooagulable dogs have a worse prognosis.10,11 Severity of anemia at presentation does not appear to be a prognostic factor.

The principles of work-up are similar for ITP. The diagnosis of ITP is ultimately based on exclusion of other causes of thrombocytopenia. Platelet-bound antibody tests are not widely available or specific; a negative test likely rules-out ITP. The diagnosis is usually made in a dog with thrombocytopenic bleeding that does not have evidence of bone marrow failure or of a disorder causing DIC. Bone marrow biopsy is not necessary if shift platelets are present and/or red cell, neutrophil and monocyte production are normal. Bone marrow biopsy classically reveals megakaryocytic hyperplasia (as may DIC), but cases with initial normal-to-low megakaryocyte counts may occur, and there is evidence of impaired platelet production in humans.12 In the author’s opinion, megakaryocyte hypoplasia does not imply a worse prognosis for recovery, but does indicate that recovery may take several more days. (Similarly non-regenerative forms of IMHA do not carry a worse prognosis if time is not an issue.)

**Treatment**
Therapeutic goals in IMHA and ITP are: 1) reduce hemolysis and/or platelet destruction; 2) reduce autoantibody production; 3) reduce inflammation; 4) prevent/treat thromboembolism and DIC; 5) correct anemia-hypoxia; 6) prevent hemorrhage; 7)
prevent/treat gastrointestinal ulceration; 8) provide supportive care. An understandable although unproven prevailing principle guiding clinician behavior is that the more negative the prognostic indicators are, the more aggressive the therapy should be. There are a large number of case series reported with different treatments and conflicting results, but little firm evidence. It is emphasized that the following describes, more than justifies, clinician behavior.

**Immunosuppressive/anti-inflammatory/antiphagocytosis therapy**

*Glucocorticoids* address all of these and remain standard-of-care. They reduce Fc-mediated phagocytosis of antibody-coated red cells and platelets by splenic macrophages, reduce complement fixation, reduce antibody production, and are anti-inflammatory. They are given at standard “immunosuppressive doses” and there are little data to promote the benefit of one protocol over another. In the author’s practice, dexamethasone (0.25 – 0.5 mg/kg q24h) is usually given to animals that have an intravenous catheter, and prednisone (2 mg/kg q24h) is used for oral treatment (minimal initial treatment period 2 weeks). Glucocorticoids make dogs relatively prothrombotic, but whether this aggravates the preexisting prothrombotic state of IMHA is not known. The apparent benefits of glucocorticoids exceed any potential risk for aggravating thromboembolism.

*Cytotoxic immunosuppressive drugs* are given primarily to reduce antibody production and inflammation, and are not believed to immediately affect phagacytosis. Historically they were used only when glucocorticoids had failed, but gradually became first-line therapy in IMHA in an effort to improve outcome. Azathioprine (a purine antimetabolite) and cyclophosphamide (a nitrogen mustard alkylating agent) are both potent immunosuppressive drugs and have been used the most as they are the oldest and least expensive. Cyclophosphamide was more popular for a period because azathioprine was believed to have a lag-effect, but the former now has fallen out of favor because of some evidence for unimproved or even worsening outcome, while azathioprine has been associated with improved outcome in two large case series. Currently azathioprine is used routinely for IMHA in the author’s practice. It also has the advantage of being given at the same dose (2 mg/kg PO q24h) and being tapered in a similar fashion to prednisone. A potential side-effect of cytotoxic drugs is myelosuppression, reducing regenerative erythropoiesis and thrombopoiesis. Dogs have variable sensitivity to azathioprine with respect to this side-effect, which may be due to variation in absorption or metabolism - it is important therefore to monitor CBCs a minimum of every 2 weeks (ideally weekly) to examine neutrophil counts as well as platelet counts. No attempt has been made to correlate mild myelosuppression (suggesting a therapeutic serum level?) to clinical response. Because of tablet strength, azathioprine must be compounded into a liquid suspension for small dogs. Vomiting may preclude initial administration - azathioprine may be given intravenously, although the injectable drug is substantially more expensive and experience with this route is limited. An initial IV dose of at least half the oral dose is suggested (ie. 0.5 -1 mg/kg q24-48 hours).
Other cytotoxic immunosuppressive agents (in increasing order of cost) include chlorambucil (a nitrogen mustard alkylating agent), mycophenolate (a purine antimetabolite), and leflunomide (a pyrimidine antimetabolite). The apparent lack of benefit of cyclophosphamide gives little support to the use of chlorambucil. The use of the other drugs at this time is generally reserved for dogs with refractory IMHA or ITP or dogs not tolerating other drugs, but some clinicians do use them as initial therapy for dogs with poor prognosis IMHA because of encouraging preliminary results.

_Cyclosporine_ (a non-myelosuppressive calcineurin antagonist) was first used in dogs as an immunosuppressive agent for organ transplantation, but as familiarity increased, and cost decreased somewhat, it ushered its way into therapy for a variety of immune-mediated and inflammatory disorders. It has a rapid onset of action but highly variable oral bioavailability. Currently the tendency in the author’s practice is to use cyclosporine immediately in dogs with IMHA with multiple poor prognostic indicators, and it is also given early consideration in cases of ITP with severe bleeding. It is not recommended to use cyclosporine as a “substitute” for azathioprine. Cyclosporine is used in transplantation at an initial dose of 10 mg/kg q12h with a target trough level of 400-600 ng/mL. There are anecdotes of using the drug in clinical practice at an initial dose of 5 mg/kg q24h and not monitoring drug levels. Clinical responses in some conditions (e.g. perianal fistulas, atopic skin disease), and laboratory markers of immunosuppression, have been associated with low doses and/or drug levels.\(^\text{15}\) Acknowledging that the optimal drug level for treating IMHA is unknown (assuming it is a beneficial drug), and that cyclosporine has highly variable bioavailability and biological effect for a given drug level, in the author’s practice cyclosporine is typically started at a dose of 3-5 mg/kg q12h PO or IV targeting a blood level of 350 - 500 ng/mL. The effect of variable pharmacokinetics and pharmacodynamics is not unique to cyclosporine – this occurs with azathioprine, and indeed for corticosteroids as well. Being a newer drug however, it reinforces the point that clinicians should not attribute therapeutic failure to a drug without considering that flexible dosing between patients is necessary. Preliminary findings are that cyclosporine does not appear to make dogs more thrombotic.\(^\text{16}\)

_Human intravenous immunoglobulin (IVIG)_ (0.5 g/kg over 6-12 hours) is believed to block Fc-mediated phagocytosis, and in this way is similar to this effect of glucocorticoids. It also has other immunosuppressive properties. The drug is expensive and has not been shown to improve survival in IMHA, but does appear to be useful in ITP. It is generally considered if a dog is refractory to conventional treatment, but in one trial was used as initial therapy.\(^\text{5}\) Human IVIG may be prothrombotic and proinflammatory. It is not available for veterinary use in Canada.

_Vincristine_ has been used to treat ITP for several decades. The use remains somewhat controversial, as does the mechanism of action, but one trial demonstrated benefit in dogs.\(^\text{18}\) Given the relatively low cost, ease of administration and minimal side-effects at “ITP doses”, this drug is routinely given within the first 48 hours of admission in the
author’s practice, at a dose of 0.02 mg/kg for dogs <15 kg and 0.5 mg/m² for dogs >15 kg. If a mechanism of action is reduction of macrophage reduction, then it may also be beneficial in IMHA, but has not been evaluated for this purpose.

**Vinca-loaded platelets** are an extension of vincristine therapy. One-hundred mL of platelet-rich plasma (containing about 250 x 10⁹ platelets/L) are incubated with 3 mg vincristine sulfate for 1 hour at 37°C using constant agitation. The platelet-rich plasma is then centrifuged to prepare a platelet concentrate, and the supernatant plasma is discarded. The platelets are resuspended into a total of 35 mL saline and transfused over 30-60 minutes. The platelets act as “Trojan horse” carriers to deliver a higher dose of the drug to macrophages. Vincristine-loaded platelet therapy appears to have induced remission of disease in several dogs with ITP in the author’s practice, and there is evidence of benefit in ITP and IMHA in humans, but they are impractical to prepare in most clinics.

**Danazol** is a modified androgen that is reportedly beneficial in human and canine IMHA and ITP; part of the benefit is believed to by modifying phagocytosis. Although there appears to have been clear benefit in individual cases, its use is generally restricted to dogs refractory or failing other treatments. It may be hepatotoxic.

**Liposomal clodronate** infusion is another treatment to selectively target macrophages and reduce phagocytosis. Initial results in IMHA are encouraging and no adverse affects have been noted.

**Splenectomy** is a commonly used second line treatment in humans with IMHA, but has not been as commonly used in dogs, especially during first hospitalization. This is presumably because of reluctance to perform surgery on critically ill dogs. However, a small case series demonstrated that splenectomy could be performed under these circumstances, with the potential for improved outcome.

**Antithrombotic therapy**
Thromboembolism is common in IMHA and a significant cause of death. It is currently a major focus of clinical investigation with respect to pathophysiology and therapy, perhaps in part because the limits of conventional immunosuppression have been reached. While it is clear that IMHA is associated with a procoagulant state, a seemingly paradoxical finding is that animals with relative hypocoagulability (as demonstrated by TEG®, thrombocytopenia, and prolonged aPTT and ACT) have a worse prognosis. The assumption is that these dogs have a consumptive coagulopathy from DIC. It seems rational to treat hypercoagulable dogs with antithrombotic drugs, however in two recent studies routine thromboprophylaxis was not given with no apparent effect on outcome. What is even more problematic is the question as to whether antithrombotic therapy given to hypocoagulable dogs should be less aggressive (because they have prolonged clotting times) or more aggressive (because disseminated microthrombosis is the cause of the prolonged clotting times).
Unfractionated heparin has been the oldest antithrombotic drug used in IMHA. It blocks the coagulation cascade at several levels. Dose recommendations have ranged from micro-doses of < 100 U/kg SC q12h to 200-300 U/kg SC q6h to constant-rate-infusions, and the drug has unpredictable bioavailability and effect. Heparin fell out of favor because of lack of evidence of improved survival (which is not perhaps surprising given how it has been used), and increasing use of antiplatelet therapy with ASA and then with clopidogrel. Heparin therapy was traditionally monitored by aPTT or ACT, with a target to prolong the values, which is problematic because the baseline values vary widely. With the advent of measuring heparin levels and factor Xa activity, it has become possible to better evaluate effects of heparin therapy and individually adjust heparin dose. Results clearly show that the clinician cannot rely even on a standard dose of 300 U/kg SC to achieve antithrombotic effect. However, a recent trial demonstrated that individualizing heparin therapy based on factor Xa activity could reduce mortality. Heparin also has anti-inflammatory effects which could contribute to therapeutic benefit. Unfortunately the ability to provide individual patient monitoring is currently not widely available and such monitoring increases expense. Non-individualized heparin therapy is not recommended.

Low-molecular weight heparins (enoxaparin [Lovenox], dalteparin [Fragmin]) have more predictable bioavailability and may reduce the need for individual dose adjustment. Unfortunately the drugs are expensive and the effective doses for antithrombosis in IMHA are currently unknown.

(Ultra)Low-dose ASA, 0.5 mg/kg PO q24h, has been established as a standard treatment for IMHA, but there is actually little evidence that this dose affects platelet function in normal dogs. If the apparent improved outcome of dogs with IMHA treated with glucocorticoids, azathioprine and ASA is real, and if ASA is in part responsible for this outcome, then it may be that ASA affects platelet function of dogs with IMHA (where platelets are activated), or it may be that its benefits are anti-inflammatory. Another large study reported a similar survival rate using glucocorticoids and azathioprine without ASA. ASA is routinely used in the author’s practice at a dose of 0.5 – 1.0 mg/kg PO q24h.

Clopidogrel (Plavex, given at a dose of 2 mg/kg PO q24h) inhibits platelet aggregation via a platelet ADP receptor subtype, resulting in blocking the glycoprotein IIb/IIIa complex which is the receptor for fibrinogen and von Willebrand factor. In a preliminary study, there were no differences between low-dose ASA, clopidogrel and the two drugs together; interestingly, overall survival was 79%.

Where does this leave the clinician presented with a dog with IMHA? Until further results are available, given that dogs with IMHA are at risk for thrombosis, and that thromboprophylaxis does not seem to be detrimental, given the difficulty of providing accurate unfractionated heparin therapy in most practices, either low-dose ASA (1
mg/kg), clopidogrel, or enoxaparin at the above doses is recommended. Clinicians using ASA at a dose of 1 mg/kg have not reported an increased risk for gastroduodenal ulceration. Some clinicians in the author’s practice prescribe both ASA and clopidogrel to dogs with multiple negative prognostic indicators (e.g. high bilirubin, intravascular hemolysis, prolonged ACT or normocoagulable TEG").

Dogs with ITP are likely also prothrombotic and thromboembolism has occurred. However, given that thromboembolic events are infrequently recognized, and that thrombocytopenia is likely self-treating the risk, antithrombotic therapy is not recommended. However, during recovery platelet counts may exceed 1000 x 10^9/L, at which time thromboprophylaxis with ASA or clopidogrel at the above doses is recommended until platelet counts normalize.

**Anemia/hypoxia**
Concerns have always been expressed that transfusion of dogs with IMHA will “add fuel to the fire”, and this concern is not unwarranted. However, severe anemia must be addressed, and transfusion is recommended for animals showing signs of hypoxia, as evidenced by weakness, increased ALT, and polypnea. Transfusion should be given to a target PCV of 20-22%, which will address critical tissue hypoxia. Red cells are prothrombic, and transfusing to a higher PCV may increase the risk of thromboembolism, in addition to suppressing the normal regenerative response. Ideally a transfusion should be withheld until at least 1 hour after glucocorticoid therapy, by which time there is theoretically onset of macrophage inhibition. Because dogs are in a fluid-retentive state and the tendency is towards excessive volume expansion, the use of packed red cells is recommended. Anti-red cell antibodies may interfere with blood-typing and cross-matching, but in the author’s practice cross-matching has been performed successfully in an effort to identity the most compatible donor. However, most practices do not have ready access to multiple donors or complete cross-matching, therefore the recommendation is to transfuse, at least for the first transfusion, with a DEA 1.1 negative donor. Oxyglobin is no longer available. The number of blood transfusion is a negative prognostic factor for IMHA, but it is not known if this reflects severity of the disease or the transfusions themselves.

Dogs with ITP are anemic because of hemorrhage, therefore whole blood transfusion is acceptable. Because these dogs are at less risk for thromboembolism, and because bleeding time is directly correlated to PCV, transfusion to a higher PCV of 30-35 is recommended.

**Hemorrhage**
The risk for hemorrhage in ITP is directly related to platelet count. Dogs with platelet counts > 20 x 10^9/L are unlikely to have critical hemorrhage, and even dogs with platelet counts < 5 x 10^9/L bleed less than expected, likely because most platelets are young. However, some dogs are not initially producing platelets, and fatal bleeding may occur. General measures to reduce risk for bleeding include keeping dogs quiet and avoiding
overexuberant fluid therapy which will lower PCV, platelet concentration and coagulation factor concentration.

Transfusion. As previously noted, while transfusion to dogs with ITP is given primarily with intent to address anemia, another advantage is that it will reduce bleeding. Platelet transfusion has long been assumed to be useless in ITP because of rapid platelet destruction. However, platelet transfusion is of documented benefit in humans with ITP, and has been life-saving in several cases in the author’s practice. Unfortunately, fresh platelet transfusion is resource-intensive with respect to donors, equipment and personnel, and is not available to most practices. Lyophilized platelets are commercially available, have a good shelf-life, and there are ongoing clinical trials evaluating their efficacy. They should be considered in the face of life-threatening thrombocytopenic bleeding due to ITP. Platelet alloimmunization will occur rapidly with repetitive transfusions and can be minimized by changing donors. Alloimmunization can be abrogated by cyclosporine, and its use is recommended if multiple transfusions are anticipated. Prednisone and cyclophosphamide, and presumably other cytotoxic drugs, will not abrogate alloimmunization.

Thrombopoietic drugs. Because ITP is primarily a disorder of platelet destruction, this has been the focus of therapy. However, there is also evidence of impaired platelet production (thrombopoietin levels are low in some human cases), and stimulating platelet production is an emerging therapy. Currently there is no practical drug for this purpose in dogs (unless this is one of the mechanisms of vincristine) except perhaps for melatonin (3 mg PO q12 hr <20 kg, 6 mg PO q12 hr >20 kg). Lithium carbonate is not recommended. It is inexpensive, but would probably have a minimal effect, drug levels must be monitored, and there are potentially serious side-effects. Recombinant human Interleukin-11 (Neumega) has been anecdotally beneficial in some human cases, but is very expensive and there is a theoretical concern for platelet antibody formation in dogs. There is minimal information on the newer thrombopoietic agents, romiplostin and eltrombopag, in dogs, but they are expensive and are unlikely to be effective.

Procoagulant drugs. Aminocaproic acid and tranexamic acid are antifibrinolytic lysine analogs that have been beneficial in controlling bleeding in ITP in humans. The only reported use of aminocaproic acid in dogs is for degenerative myelopathy. The dose used was about 12.5-15 mg/kg PO, which is approximately 10% of the human dose. Doses ranging from 10-100 mg/kg PO, IV have been used anecdotally in dogs for hemostatic benefit, though not specifically for ITP. The dose of tranexamic acid is 10-15 mg/kg SC, IM or slow IV (rapid IV causes vomiting), but it has not been specifically evaluated in ITP. The drugs should be considered if there is life-threatening critical bleeding or hyphema. Aprotinin is an antifibrinolytic serine protease inhibitor that also has anti-inflammatory properties, but there is even less experience with it as a hemostatic drugs in dogs. Recombinant human FVIIa has been useful in selected human cases of ITP, but the drug is expensive, of limited availability, and likely to cause antibody formation. Procoagulant drugs should not be given if there is any possibility of
DIC.

Gastroprotectants
Dogs with IMHA are assumed, but not proven, to be at risk for gastroduodenal ulceration secondary to gastrointestinal ischemia and glucocorticoid therapy. Although not firmly evidence-based, it is common to treat dogs with H2 blockers and/or sulcralfate, and there is no evidence that these treatments do harm. Similarly, dogs with ITP are frequently given gastroprotectants because they have melena – this is likely due to thrombocytopenia, but ulceration cannot usually be ruled out.

Supportive care
Critically ill dogs require an intravenous catheter, although this does increase the risk for thromboembolism. The risk is higher with jugular catheters, which should be avoided if possible. Thromboprophylaxis is recommended if a dog is catheterized. While acknowledging the need for fluid therapy for prothrombotic dogs, the tendency is to overhydrate, and fluid therapy should be tapered. Aggressive fluid therapy should be avoided in dogs with ITP. Dogs that are not eating should be given nutritional support.

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Diagnostic Evaluation and Treatment of Lymphoma in Dogs and Cats
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Synopsis
Laboratory and imaging work-up for the purposes of diagnosis, diagnostic staging and classification; prognostic factors; and treatment options for lymphoma, in dogs and cats, will be discussed.

Overview
Malignant lymphoma (aka lymphoma, lymphosarcoma, LSA) is a very heterogeneous cancer of lymphocytes (“lymphomas” would be a better term) that may present as a systemic and/or local disease in many different forms. Various classification and grading schemes for LSA have been used in the past and are evolving, in part in an effort to guide therapy and provide prognosis. Currently, cell-type (grade) in dogs is classified according to the WHO scheme (which differs from WHO clinical stage). Some clinical forms of LSA are most commonly due to a specific cell-type (e.g. multicentric LSA in the dog is most often a diffuse large B-cell LSA), but different cell-types may result in similar clinical presentations (multicentric LSA may also be due to a T-cell LSA). In an intentional oversimplication, LSA in both dogs and cats may be considered as aggressive or indolent. Although there has been progress in characterizing LSA in dogs and cats and in identifying prognostic factors, this has actually had little impact within major subtypes of LSA to date on tailoring treatment to individual animals. For this reason, unless an animal is included in a research study or clinical trial which provides financial support, there is little point in exhausting owner financial resources in extensive work-up and it is preferable to save these resources for treatment. However, the clinical staging and characterizing of LSA is important in order to advance understanding and treatment of the disease. In addition, LSAs in animals, and in particular dogs, are good biomedical models for non-hodgkins lymphoma in humans, and as such studies evaluating LSA in dogs and cats are often occurring. It is worthwhile to investigate if an academic or private referral practice is participating in such a study to help offset costs of diagnostic work-up and treatment. In many instances case-management may be shared between the regular veterinarian and the referral practice.

Etiology
Although there is some epidemiologic association between environmental pollution and LSA in dogs, and some suggestion of a role for viruses and immunodeficiency, LSA in the dog is considered to be a spontaneously arising neoplasm. The increased prevalence in certain breeds (e.g. golden retrievers) indicates that there is a genetic component. In cats, FeLV is oncogenic, and immunosuppression by FIV, and possibly by drugs, increases risk. In cats there is an epidemiologic association with tobacco smoke but not environmental pollution. The proportion of FeLV-negative LSA is increasing in cats, likely because of cats being more confined to indoors, living longer and/or being vaccinated.

Clinical signs
LSA may cause nominally any sign and is often a differential diagnosis for many chief
complaints. Clinical signs of lymphoma may be due to local disease (e.g. lymphadenopathy, uveitis, nasal discharge, diarrhea), organ dysfunction (e.g. liver failure, kidney failure, hypoadrenocorticism), infection (secondary to immunosuppression or neutropenia), and paraneoplastic syndromes, including hypercalcemia (PU/PD), cachexia, non-septic fever, cancer-related anemia, and secondary ITP.

**Diagnosis**

As with all cancer, definitive diagnosis rests on biopsy. Fine needle biopsy procures a specimen for cytology. For lymph node biopsy, the recommendation is to insert and redirect a 22ga needle in the tissue rather than to aspirate. The tissue is expressed onto a slide and a smear made similarly to a blood smear. Because the most common LSAs in dogs are rapidly proliferating high-grade cell types with diffuse distribution within a node, the diagnosis may often be made on the basis of clinical signs and cytology alone\(^1\) - there are not many disorders that cause marked rapidly-developing painless generalized lymphadenopathy in dogs. The cytologic hallmark of high-grade nodal LSA is a homogeneous monomorphic population of cells, while normal and hyperplastic lymph nodes demonstrate heterogeneous populations. (This is the opposite to many other cancers, where normal tissue is more monomorphic and cancer is characterized by variation in appearance.) In some cases of LSA there is a mixture of malignant and normal cells, in which case the diagnosis rests to some extent on the percentage of immature cells. For this reason, earlier in the disease process an LSA-bearing node may be interpreted as being hyperplastic. In this case either repeat fine needle biopsy in several weeks should be planned, or a sample for histology obtained. Unlike with many cancers, delaying diagnosis in this scenario may not have a major impact on treatment outcome. If a surgical biopsy is performed as the first-line biopsy, then touch preparations on a slide should always be made prior to placing the specimen in formalin in the event that cytology will be desired (as cytology and histology provide complimentary information). If the diagnosis is questionable, then excisional or incisional lymph node biopsy should be performed. Histology of a high-grade LSA will reveal effacement of normal lymph node architecture and frequently extracapsular invasion. Histology is usually necessary for the diagnosis of the less common small cell LSAs, lower-grade LSAs and follicular LSAs in dogs. The diagnosis of nodal LSA in cats is often not as straightforward as in dogs. In particular, atypical lymphadenopathy may resemble LSA in cats,\(^6\) and the author encourages lymph node biopsy in cats unless there is firm evidence of LSA in another organ.

Extra-nodal LSAs are also diagnosed on the basis of clinical signs and biopsy. Mediastinal LSA (causing dyspnea) may usually be diagnosed on cytology following thoracocentesis. Alimentary LSA (causing vomiting and/or diarrhea and/or weight loss and/or iron-deficiency anemia) may be diagnosed on ultrasound-guided fine-needle aspiration of thickened intestines, endoscopic biopsy, or surgical biopsy. Touch preparations of gastrointestinal biopsies may provide a rapid cytologic diagnosis. Hepatic (non-specific signs ± hepatomegaly ± jaundice) and renal LSA (non-specific signs, renomegaly ± PU/PD) may be diagnosed on the basis of fine needle biopsy, but occasionally LSA cells do not exfoliate from these organs and Tru-Cut\(^*\) or other larger biopsies are required. Splenic LSA
may usually be diagnosed with fine needle aspiration. Nasal (nasal discharge) and LSA invading bone (lameness) may usually be diagnosed on both cytology and histology. CNS signs may be a feature of both primary CNS lymphoma, in which diagnosis relies on cytology of CSF (± MRI), or of multicentric LSA, in which case the diagnosis of CNS involvement is usually presumed, but may be confirmed by MRI and/or CSF collection. Similarly ocular signs may be a feature of both primary ocular lymphoma, in which diagnosis relies on cytology, or of multicentric LSA where ocular involvement is presumed. Epitheliotropic cutaneous LSA may require multiple biopsies over time to achieve a diagnosis.

Rarely, biopsy is not feasible and in this situation, in the author’s opinion, response to treatment is acceptable for presumptive diagnosis following clear communication with the owner. Examples in the author’s practice have included: 1) Myocardial LSA (infiltrative cardiomyopathy) in dogs and cats with strong evidence of the diagnosis based on echocardiography. 2) Acute hypercalcemia where routine work-up including and imaging and bone marrow biopsy did not identify the disease. As the dogs were sick from hypercalcemia, treatment was started while awaiting measurement of PTH and PTH-rp levels. Asparaginase was used as the treatment as corticosteroids may lower Ca blood levels independent of anti-LSA effect. Prompt resolution of hypercalcemia is strong evidence for LSA. (An alternative approach is to treat with a bisphosphonate while awaiting PTH level). 3) Two cases of imaging-confirmed marked bilateral renomegalay in cats which received corticosteroids prior to referral. Renomegaly and renal failure promptly resolved; these cats were treated with multiagent chemotherapy rather than risk relapse and resistance to chemotherapy.

The PARR (PCR for Antigen Receptor Rearrangement) test may be used to support diagnosis of LSA.\(^7\)\(^-\)\(^10\) It is considered to be overall >90% specific in both dogs and cats, and approximately 75% sensitive and 65% sensitive for LSA in dogs and cats, respectively. In one report sensitivity was only 67% for GI LSA in dogs whereas sensitivity was 79% for cats.\(^8\)\(^9\) One cause of false positive results are Rickettsial diseases, which may also cause lymphadenopathy and a variety of signs. The PARR tests are most useful for supporting the diagnosis of indolent small cell LSAs.

Flow cytometry is used to characterize the immunophenotype of LSA (as B-cell, T-cell, NK-cell and subtypes thereof)\(^1\), and is not used to diagnose LSA. Samples for flow cytometry are obtained by 3-4 aspirations from the tissue and rinsing the needle and syringe with a special buffer solution (if available from the laboratory) or saline to retrieve as many cells as possible, after which the sample should be refrigerated and analyzed within 24hr.

**Work-Up – clinical staging and identification of prognostic indicators**

The purpose of work-up of the cancer patient is to stage the disease (i.e. what is the distribution of the disease in the body) as necessary to optimize therapy based on predicted behaviour of the cancer, identify prognostic indicators, and identify concurrent illnesses that may affect treatment and response. Clinical staging is achieved
based on history, physical exam findings, laboratory work, diagnostic imaging and biopsies. It may not provide specific clinically relevant or prognostic information for an individual patient with LSA and is most useful in comparing results of studies for groups of patients.

For dogs, LSA has been classified by its anatomic site as: A) Generalized, B) Alimentary (GI) C) Thymic (anterior mediastinal), D) Skin, E) Leukemia, and F) Other sites, eg. CNS. The term “Generalized” for site A is better termed “Nodal”, where the assumption is that the malignant clone has arisen in a peripheral lymph node, but the cancer may progress to extra-nodal sites. Although most nodal LSA in dogs is high-grade and generalized, indolent non-generalized nodal LSA does occur.

The WHO organization classification of nodal LSA in dogs is: Stage 1: Single node; Stage 2: multiple nodes on one side of the diaphragm; Stage 3: multicentric; Stage 4: multicentric + liver and/or spleen; and Stage 5: multicentric + bone marrow, gastrointestinal tract or other extra-nodal sites. Unfortunately stage 4 and 5 has also been used to include primary extra-nodal LSA, many of which carry a worse prognosis, including primary GI, hepatic, and splenic LSA and acute lymphoid leukemia. If the WHO classification is restricted to primarily nodal LSA, clinical staging has little impact on treatment. First, stage 1 and 2 LSA with high-grade cell types (the most common) will progress to stage 3 and higher (multicentric), and the dog is going to be treated with chemotherapy regardless of stage. Secondly, classification of LSA as stage 1-5 is dependent upon how aggressively one looks for involvement of other nodes and organs (“stage migration”). For example, based on endoscopic airway washes dogs may have lung involvement which are not detected radiographically, and PARR testing will detect LSA cells in the blood not seen cytologically. Dogs are also classified as substage a (feeling well) and substage b (ill).

For LSA in extra-nodal sites (B-F) the assumption is that the malignant clone has primarily arisen in that tissue (and hence is referred to as primary), but may go on to involve regional nodes or spread to other tissues. The crucial clinical question is whether or not LSA is confined to that anatomic site. If it is, and if it is amenable to local treatment (radiation therapy or surgery), then local treatment or multimodality treatment may be a better choice than chemotherapy alone. Local radiation therapy can provide a much larger local “cytotoxic hit” to cancer cells than can chemotherapy, as the latter is more restricted by systemic side-effects. Unfortunately in many cases it is not known if the cancer is restricted to a site, and there is always the risk with LSA that it may generalize even if the disease can only be detected at one site. The decision whether or not to use multimodality therapy must be decided on an individual case by case basis in consultation with the owner.

LSA in cats is similarly classified by anatomic site and whether the behaviour of the LSA is aggressive or indolent. Multicentric nodal LSA, characterized by peripheral lymphadenopathy, frequently involves the liver and/or spleen. Anterior mediastinal LSA is most often localized but may involve nodes and other sites. These forms are often associated with FeLV infection. Aggressive and indolent primary GI LSAs are well-recognized. Aggressive GI LSA may affect various sites in the GI tract and mesenteric
lymph nodes, and may extend into the liver, spleen or kidneys. Extra-abdominal sites (e.g. lung, bone marrow) may also be involved. Cell-type is usually large B-cell (lymphoblastic), and infrequently NK or T-cell (LGL - large granular lymphocyte LSA). Indolent lymphoma has a chronic history where the main DDx is IBD, and the cell-type is small T-cell (lymphocytic). All are infrequently associated with FeLV infection.

Routine blood work (CBC and serum biochemistry profile, and FeLV/FIV testing in cats) and diagnostic imaging will have often been performed in the work-up of an animal in the process of making the diagnosis of LSA. Unless dictated by participation in a trial, the minimum database should be defined by clinical signs and intended therapy. In an animal with extra-nodal LSA an exhaustive imaging and biopsy investigation of other tissues is often not warranted if the treatment is chemotherapy which will treat LSA at all sites.

The minimum database for a dog with confirmed multicentric LSA where chemotherapy is planned is a CBC, profile, and urinalysis. These are obtained primarily to establish baseline hematologic parameters which may be affected by chemotherapy, and to evaluate liver and kidney function which may affect doses of some drugs. Some clinicians also recommend measuring LDH and uric acid in an effort to better identify animals at risk for tumor lysis syndrome, especially if phosphorus is elevated. Thoracic radiographs, abdominal radiographs and abdominal ultrasound examinations should be offered, especially in older animals, in that they may detect concurrent problems (e.g. splenic hemangiosarcoma), but they are not mandatory in that they otherwise have minimal predictive value on outcome. However, diagnostic imaging is recommended if indicated by clinical signs. Thoracic radiographs are recommended in dogs that are dyspneic and/or febrile to rule-out concurrent pneumonia (LSA does not typically cause alveolar change/air bronchograms/lung lobe consolidation). Similarly, abdominal ultrasound examination is recommended in dogs that have abdominal pain and/or fever to rule-out pancreatitis or septic peritonitis; ultrasound is also useful to confirm that diarrhea is due to GI involvement. The need for BM aspiration or core biopsy is controversial, but is not routinely performed in the author’s practice. Just as very careful extended scrutiny of peripheral blood smears will often reveal malignant lymphocytes in the blood, so sufficiently detailed and repetitive BM examination will likely reveal BM involvement. The degree of BM involvement (or other organs for that matter) is perhaps more important than whether or not it is involved, but this has not been well-evaluated. BM biopsy should be offered, as occasionally the lack of a baseline hampers interpretation of future blood changes during therapy. The most important role for BM biopsy is in the diagnosis of acute lymphoblastic leukemia in an animal with pancytopenia when blood lymphocyte numbers are not elevated above the reference interval (although lymphocyte numbers are typically inappropriately high within the reference interval for a sick animal).

**Prognostic indicators**

Prognostic factors may be examined both with respect to rate of attaining remission and
disease-free interval (DFI, which is calculated from the first day of treatment.) Factors consistently identified as negative prognostic indicators for dogs with multicentric LSA include WHO substage b, immunophenotype, hypercalcemia, and prior use of corticosteroids. Factors with inconsistent negative predictive values include thrombocytopenia, anemia and diarrhea. Substage b is defined in humans as 10% weight loss, fever and night sweating. A precise definition in dogs and cats is lacking. Substage a is typically reserved for dogs with no constitutional signs, but that is dependent on history and owner observation. Substage b is typically ascribed to dogs based on lethargy, inappetence, weight loss, fever or PU/PD, but importance of degree of systemic signs in substage b has not been evaluated. Aggressive T-cell LSA typically carries a worse prognosis than B-cell LSA both for attaining remission and DFI, as does hypercalcemia which reflects an aggressive T-cell immunophenotype. Prior use of corticosteroids is most detrimental if a dog attains a complete (CR) or partial remission (PR), and then multiagent therapy is started when progressive disease is noted. For the purposes of clinical trials, the prior use of corticosteroids for <1 week prior to more complete therapy is believed not to influence outcome. Attaining a rapid CR (i.e. within 1 week after the first treatment) is the main positive prognostic indicator. Animals that achieve PR may enjoy as good a quality of life and have survival times as long as many dogs who attain a CR. However, it is highly unlikely that any dog that does not attain a prompt remission will survive into the cohort with DFI > 2 years.

It is emphasized that these prognostic indicators are based on percentages for populations of dogs and that it is difficult to give a prognosis for the individual animal. The extreme examples that illustrate this from the author’s practice are: 1) a dog with WHO Stage IIIa LSA (based on imaging and BM biopsy) that achieved a prompt and complete remission (the owners reported the lymph nodes were shrinking the day of treatment), but relapsed at 3 months; 2) a dog with WHO Stage Vb LSA with hypercalcemia that had a DFI > 5 years; and 3) a dog who had previously partially responded to corticosteroids and was presented with progressive disease that had a DFI > 5 years. Ultimately the only way to know how an animal will respond is to treat that animal. Initiating chemotherapy is not signing a contract and an owner may withdraw their animal at any time if they are not satisfied with response, side-effects or accumulating costs.

Treatment

The prevailing principle is to treat an aggressive LSA aggressively and a non-aggressive LSA non-aggressively. Canine high-grade multicentric LSA is best treated with a multiagent protocol while indolent GI LSA in cats is best treated with only corticosteroids and chlorambucil. While immunotherapy and other treatments continue to be investigated, chemotherapy, radiation therapy, and surgery, in that order, remain the cornerstones of treatment in addition to detailed attention to supportive care. Remission is defined clinically as normalization of lymph nodes and other tissues and resolution of clinical signs. It is not standard to perform a lymph node biopsy, or biopsy of other tissues, and such biopsies may be difficult to interpret. Remission is not cure in most
cases and malignant lymphocytes remain (“minimal residual disease”).

Currently standard-of-care of care chemotherapy typically uses a CHOP-based protocol consisting of an induction phase and abbreviated maintenance phase. CHOP refers to cyclophosphamide, hydroxydaunorubicin (doxorubicin), Oncovin (vincristine) and prednisone. Drugs are given weekly for the first 2 - 3 months and then every two weeks for the next 3 - 4 months. For dogs with multicentric LSA, there have been many variations with respect to exact dosing schedules, with out any clear advantages of one over another. Assuming a typical distribution of LSA cell-types and breeds, the CR rate is \( \approx 80 – 90\% \), 1-year DFI is \( \approx 60\% \), and 2-year DFI is \( \approx 20\text{-}25\% \). Besides concurrent disease, once an animal is in remission the only factor than predicts ongoing remission is remission itself, and many animals in remission at 2-year will go on to be effectively cured of their disease. The CHOP protocol in use in the author’s practice is the UW-25 protocol, with cyclophosphamide given orally. Asparaginase is usually reserved for relapse therapy and ALL. Most dogs tolerate the protocol well and owner satisfaction is high. At least 50% of dogs will have at least one delay during treatment because of neutropenia. Most GI side-effects may be controlled with appropriate anti-emetic and symptomatic therapy. The rate of hospitalization for treatment-related disorders is <5% and treatment-related mortality is <1%. Most animals relapse with nodal LSA, Repeat fine needle biopsy is recommended. However, lymph node hyperplasia seen on fine needle cytology in an animal suspected to be relapse based on lymph node enlargement without another explanation is, in the author’s opinion, usually sufficient evidence to diagnose relapse. Numerous relapse protocols have been reported. The default first-line treatment is to repeat induction, with the rate of second CR and DFI lower than the first, although the occasional dog has had a better response on the second course of treatment.

As the acronym indicates, the main difference between CHOP protocols and the COP-based protocols is the use of doxorubicin, for which the evidence is strong that both CR and DFI are superior. Similarly CHOP is superior with respect to CR and DFI than the use of doxorubicin as a single-agent. However, the rate of CR for COP and single-agent doxorubicin is 60-70%, and some dogs so treated have enjoyed DFI > 3 years. Other less-expensive treatment options for owners who elect not to treat their dogs with the above protocols include lomustine, prednisone and cyclophosphamide. Following chemotherapy, CR may be consolidated by: 1) sequential half-body irradiation; or 2) high-dose chemotherapy and/or total body irradiation + autologous or allogeneic bone marrow or peripheral blood hematopoietic stem cell transplantation. These treatments are variably available by referral. In the author’s opinion these treatments likely increase the likelihood that a dog with CR after induction chemotherapy will achieve a long-term DFI, but data are not firm and the increment in percentage of dogs achieving long-term DFI is not known.

CHOP and COP-based protocols are both accepted standard-of-care for cats with aggressive LSA. Depending on how CR and PR are judged, \( \approx 1/3 \) to 2/3 of cats with
aggressive GI LSA achieve a CR, with other cats achieving PR with overall response rates up to \( \approx 75 - 90\% \), and one-year survival \( \approx 20 - 40\% \). Some of these cats will become long-term survivors. Surgery is reserved for cats with intestinal obstruction. For cats with mediastinal or multicentric LSA, CR up to 90% and 1-year survival up to \( \approx 55\% \) have been reported. As with dogs, regardless of anatomic location, usually only cats that achieve CR with induction chemotherapy have the possibility of having a long-term DFI. FeLV and FIV status do not affect initial response to therapy. They do negatively affect long-term survival, mostly because of the occurrence of other FeLV and FIV-related disorders. Cats with indolent GI LSA treated with prednisone and chlorambucil have a response rate (CR + PR) > 95% and median duration of response of 16-26%. As with other forms of LSA, cats that achieve CR have a better long-term prognosis than cats that achieve PR. Some cats with solitary nodal LSA have been cured by surgical excision alone. If work-up fails to identify LSA at another site, then surgery without subsequent chemotherapy may be considered, especially for Hodgkin’s-like lymphoma. Most cats tolerate chemotherapy for lymphoma well and, as with dogs, owner satisfaction is high. Cats are not as susceptible to neutropenia-associated sepsis. The most common side-effect is inappetence, which may respond to appetite-stimulants. There is an increasing role for primary and adjunctive radiation therapy in the treatment of regional nodal, anterior mediastinal, and abdominal LSA in cats.

References
Gastric dilation volvulus (GDV) is a very important disease process in small animal veterinary medicine. It has many facets to explore and this has made it a popular topic for research. Treatment for the acute syndrome has improved over the past 20 years and one important indicator of this has been a progressive decrease in reported mortality rates for dogs with GDV from 28.6% in the late 1990’s and early 2000’s to the most recent paper looking at this statistic reporting a 10% mortality rate. (Broome 2003, Glickman 2000, Glickman 1998, Brockman 1995, Mackenzie 2010, Evans 2010).

Prior to the recognition that immediate surgical intervention improved outcome in these patients reported mortality rates for conservative management were higher. For an initial episode, 33% survived with deflation of the stomach and treatment for shock, 71% of these dogs experienced recurrence within 1 year, 56% within 3 months. 81% of dogs treated conservatively died within 1 year. (Eggertsdóttir 1995) There are few diseases processes with as much advancement and success within such a short period of time! The research that has been done has greatly improved our understanding of which animals are at risk, how we can prevent GDV and specifics of assessing morbidity and mortality associated with the acute GDV such as improved recognition of preoperative conditions which affect outcome following surgery, and improvements in post-operative care.

**Risk Factors Associated with GDV in Dogs**

Two important studies by Lawrence Glickman were published in 2000, which evaluated large numbers of dogs to try to determine what the true breed related risk factors and dietary risk factors are for dogs developing GDV. Prior to these papers there were a number of theories but this research lead to some new knowledge, which allows us to better predict dogs at risk.

In a study of 1,914 dogs of 11 different breeds in North America lifetime risk of GDV was shown to be 24% for large breed dogs and 21% for giant breed dogs assuming a average life span of 10 years for large breed dogs and 8 years for giant breed dogs. Given a mortality rate for GDV of approximately 30% this would mean that 7% of these dogs would die due to GDV. Great Danes however had a much higher lifetime risk of 42.4% meaning a mortality rate of 12.6%. (Glickman 2000)

Other characteristics of these dogs, which increased their risk of developing GDV include conformation, temperament and familiar history of GDV in the line. Conformation and familial history would be genetically linked traits, which would warrant discussion with the breeder. Temperament however was an unexpected finding. The owners described
affected dogs as having a nervous or “unhappy” personality. Dogs described as happy had a 78% reduced risk of GDV. Risk of GDV was increased by 257% in fearful versus non-fearful dogs. (Glickman 2000) The exact mechanism for these differences is unknown but are important when making decisions which may put these dogs in a more stressful situation such as hospitalization or boarding. Prophylactic gastropexy would be an important topic to discuss with owners.

Diet has also been implicated in the development of GDV in dogs but no studies have conclusively implicated diet in the development of GDV. A prospective study of 1,637 dogs including ii different breeds showed that increased age of the dog, having a first degree relative with a history of GDV, having a faster speed of eating and eating from a raised food dish all increased the life time risk of GDV. Of the GDV cases in giant breed dogs, 52% of them were fed from a raised food dish as compared to 20% of the large breed dogs. (Glickman 2000) Management of lifestyle then becomes important in these dogs to decrease the risk of GDV.

**Chronic Gastric Instability**

When we consider GDV we think about the acute gastric dilation and volvulus syndrome. Although rarely reported in the literature, there appears to be a subset of dogs that are believed to have chronic intermittent volvulus. These dogs tend to be of the same breeds as dogs who develop the acute syndrome. Their clinical signs are inability to gain weight and or weight loss, eructation and flatulence, chronic vomiting, lethargy and abdominal pain. The period of time for clinical signs can range from a weeks to years and dogs range in age from 11 months to 12 years. (Paris 2011)

Diagnosis in these dogs was achieved using a combination of imaging techniques. Plain abdominal radiographs may identify abnormal orientation of the gastric axis with a radiographic diagnosis of incomplete gastric volvulus. In a few cases a gastrointestinal barium series demonstrated delayed gastric emptying suggesting a partial pyloric obstruction. Ultrasound examination may show evidence of reduced gastric motility and displacement from a normal orientation. Endoscopy, which is commonly used in human medicine, can also be valuable in making a diagnosis of chronic gastric volvulus. Suspicious findings would be difficulty passing the endoscope through the cardia and abnormal positioning of the normal gastric landmarks. (Paris 2011)

It is important to note that this condition can be intermittent and although you may have supporting evidence though your diagnostic work-up, when you explore the abdomen the stomach may be in a normal orientation. This does not mean that you have made an incorrect diagnosis. Biopsies of the stomach, duodenum, jejunum and ileum are also be obtained in these cases to ensure a second problem is not complicating the patient’s GI problems. Once the abdominal exploratory is complete a right incisional gastropexy is performed prior to routine closure of the abdomen.
In the majority of reported cases, gastropexy has resulted in resolution of clinical signs and weight gain. In 2 dogs, concurrent megaesophagus was present prior to surgery. In one of these cases, the megaesophagus resolved, in the other case the megaesophagus likely contributed to development of aspiration pneumonia leading to euthanasia. This illustrates the importance of evaluation of the entire digestive tract prior to surgery to ensure the client is well informed of potential complications.

Factors Affecting Morbidity and Mortality in Dogs with Acute Gastric Dilation-Volvulus

Although we have seen evidence of improvements in survival for dogs treated surgically for GDV, there are factors which, when present, can negatively impact morbidity and mortality. Several of these conditions have been described in the literature and in general these factors reflect dogs, which have likely been compromised for relatively longer periods of time. Dogs with presence of clinical signs relating to GDV for longer than 6 hours have a significantly higher risk of death. These dogs also often have a higher risk of gastric necrosis leading to the requirement for partial gastrectomy. The relationship between lengths of time the stomach is compromised and gastric necrosis has been shown both experimentally and in clinical cases. (Beck 2006) Gastric decompression appears to be one of the most important aspects of early management, as this improves blood flow to both the stomach wall and return to the cranial half of the body. Partial gastrectomy due to compromise of the gastric wall is associated with increased risk of post-operative complications such as sepsis, peritonitis, arrhythmias, and disseminated intravascular coagulation (DIC). In older studies these conditions were linked to significantly higher rates of death, however in the study by Beck, this was not the case. This is likely due to the fact that we are anticipating these problems and can recognize and treat these issues at an earlier stage. (Beck 2006) The anatomic location of gastric necrosis is clinically significant. When necrosis is at the cardia, mortality rate is much higher, 40%, likely due to the difficulty in resecting this portion of the stomach and the lower esophageal sphincter. (Beck 2006)

Serum lactate has become a very popular test in trying to determine severity of gastric necrosis in dogs with GDV. Reported significance of plasma lactate values in predicting gastric necrosis have varied. It seems to be consistent that the higher the initial plasma lactate value, the higher the risk of gastric necrosis. Plasma lactate values greater than 9.0 mmol/L seem to be associated with a much higher risk of death, as high as 47% in one study. (Zacher 2010) For dogs with high initial lactate concentrations predictions about final outcome are more guarded than with lower initial concentrations but a more important factors appears to be what happens to the plasma lactate values after surgery. Drops in lactate are positive prognostic indicators for survival. The numbers reported vary from levels less than 6 mmol/L to changes greater than 4 mmol/L (Zacher 2010) to decrease by greater than 50% within 12 hours of the initial value as all being indicators of survival (Green 2011).

Is There a Relationship between the Spleen and GDV?
The question about a relationship between GDV and splenic torsion has been asked for some time. The breeds of dogs developing both of these conditions are the same so the thought that these conditions may be related was reasonable. Also, given the fact that in almost all GDV cases, the spleen in very large and engorged, giving the appearance of a possible torsion, this may lead some veterinarians to make the assumption that the spleen is also torsed and remove the spleen during the GDV surgery. Despite these consistencies between the two conditions, there has been no research to prove that splenic torsion leads to GDV, that GDV leads to splenic torsion or that removal of the spleen increases patient risk of developing GDV. (Goldhammer 2010) The fact that these two life-threatening conditions occur in the same breeds of dogs, however, is a good justification to perform a prophylactic gastropexy when the abdomen is already open for celiotomy, as long as the patient is stable.

Management of dogs presenting with GDV

Clinical findings
- Dogs presenting with GDV are generally presumptively diagnosed based on history, signalment and physical exam findings.
- Clinical signs generally include abdominal distension and abdominal discomfort, retching which is often non-productive, restlessness, hypersalivation and sometimes increased effort for breathing.
- Physical exam findings are commonly related to abdominal distension and shock. The cranial abdomen may be large, firm and painful. Vital signs often reflect compensatory hypovolemic shock and include increased heart rate, respiratory rate and capillary refill time. Animals presenting in a decompensated state have pale mucous membranes, bradycardia, decreased mentation and hypothermia.
- Splenomegaly mat also be palpable, most commonly due to splenic congestion.

Diagnostics
- A right lateral abdominal radiograph is the projection most commonly used to allow visualization of the classic shape of the stomach in GDV cases. The stomach is seen with the pylorus dorsal and cranial, air trapped within the stomach and a fold of soft tissue which separates the pylorus form the body of the stomach in the classic “double bubble” shape.
- Laboratory findings are non-specific and relate to the secondary processes occurring in the patient. Increased packed cell volume and total serum protein are common. If the patient is decompensating, changes consistent with disseminated intravascular coagulation may include decreased platelet counts. Hypotension can lead to decreased renal function and elevations in urea and creatinine.

Early intervention and treatment
• Fluid therapy to address hypotension secondary to hypovolemic shock.
  o Two large bore IV catheters in the font limbs or jugular veins.

<table>
<thead>
<tr>
<th>Fluid Type</th>
<th>Rate</th>
<th>Special Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Crystalloids: LRS, 0.9% NaCl, plasmalyte</td>
<td>90 ml/kg/hr</td>
<td>Can give in ¼ bolus and evaluate changes in vitals</td>
</tr>
<tr>
<td>Colloids</td>
<td>20 ml/kg/day</td>
<td>Can give a 5 ml/kg bolus Use concurrently with crystalloids</td>
</tr>
<tr>
<td>Hetastarch</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pentastarch</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertonic saline</td>
<td>6 ml/kg</td>
<td>Can give for rapid vascular reexpansion BUT must follow with crystalloids</td>
</tr>
</tbody>
</table>

• Gastric decompression to prevent further cardiovascular collapse.
  o Placement of an orogastric tube
    ▪ Measure from the tip of the nose to the xiphoid
    ▪ Place tape around the orogastric tube at the length measured
    ▪ Place a two inch role of tape in the mouth
    ▪ Lubricate the tube and pass the tube through the roll of tape into the oral cavity
    ▪ Feed the tube into the esophagus.
    ▪ Depending on the mentation of the patient, passing the orogastric tube may require mild sedation
  o Trochar
    ▪ Clip and prepare the skin over the point of maximum distention overlying the stomach.
    ▪ Using a long, large bore, over the needle catheter (12 – 14 gauge 6 inch long large animal catheters work well) pierce of the skin and advance the catheter into the stomach.
    ▪ When the catheter is within the lumen of the stomach, remove the needle and allow the air to escape the stomach.

• Oxygen therapy
  o Flow by oxygen therapy improves oxygen saturation of hemoglobin and overall tissue oxygenation.

• Pain management
  o Use of narcotic pain management is warranted in GDV patients

Surgical Intervention
• Reposition the stomach
  o The stomach will generally be twisted with the omentum being pulled over the ventral aspect of the stomach as it is lying in the abdomen.
If the stomach is still large, a catheter can be used to decompress or an orogastric tube can be placed. Once the stomach is decompressed, if standing on the right hand side of the dog, grasp the pylorus (which is usually located dorsal and cranial to the body of the stomach, near the esophageal sphincter) with the right hand and place the palm of the left hand on the body of the stomach. Pull the pylorus toward the right side of the abdomen while pushing the body towards the left side of the abdomen. The stomach should twist back to a normal position in 95% of cases.

- Evaluate the stomach for evidence of gastric necrosis
- Explore the entire abdomen for other abnormalities, foreign bodies, etc...
- Reevaluate the stomach for evidence of gastric necrosis
- If gastric necrosis is present, resect the affected area.
- Perform an incisional gastropexy.
- Close the abdominal wall routinely.

Post-operative Care
- Fluid therapy – crystalloids and correction of any electrolyte abnormalities.
- Treat cardiac arrhythmias if present and resulting in clinical decline such as with ventricular tachycardia.
- Pain management with narcotic drugs.
- Treatment of post-operative ileus with a metoclopramide constant rate infusion at 1-2 mg/kg/day
- Feeding 12-24 hours after surgery
- Close monitoring for signs relating to gastric compromise, gastric rupture/perforation, sepsis, disseminated intravascular coagulation, aspiration pneumonia, hypotension, bloating, and gastric ulcers.

**Prophylactic Gastropexy**

Given that there are no current medical management protocols, which effectively prevent GDV, prophylactic gastropexy seems to be very attractive options for owners of dogs in one of the high-risk breeds. Owners who had the experience of having a pet have a GDV and either survive or succumb to the incident are very likely to request prophylactic gastropexy for subsequent dogs and currently prophylactic gastropexy is the only effective method of preventing GDV in dogs. Method vary from open approach at the time of OHE, castration or another abdominal procedure, to laparoscopic assisted gastropexy to laparoscopic gastropexy. (Ward 2003, Rivier 2011, Glickman 2000) The most common technique used for prophylactic gastropexy is the incisional gastropexy. This technique is simple, quick and can be performed without an assistant. The incisional gastropexy also provides a secure adhesion to the body wall. (Wacker 1998)
References:


Surgery of the Small Intestine
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Surgery of the small intestine is commonly performed in small animal practice for the purposes of both diagnostics and therapeutics. Although surgery involving the gastrointestinal (GI) tract is common and the results are generally favorable, there are many factors to consider when deciding if surgery is the correct choice and what the potential risks of intestinal surgery are for each individual patient. Looking at all of these factors allows the owner to make an informed decision and the veterinarian and his or her team to plan appropriately for the procedure and complications that may arise.

Intestinal Biopsy:

Indications for intestinal biopsy include chronic disease, localized to the small intestine such as vomiting, weight loss, hypoprotienemia, and diarrhea or when non-diagnostic endoscopic biopsies have been obtained. Benefits to open surgical biopsy include the ability to obtain full thickness samples from the small intestine in several different locations, the ability to feel and visualize the small intestine to identify any obvious gross abnormalities and the ability to inspect the other abdominal organs for signs of disease and obtain samples if necessary. Downsides to open biopsy are increased discomfort for the patient and increased cost for the owner as well as increased risk of complications following the procedure.

Serious complications associated with intestinal biopsy occur in as many as 12% of patients (Shales), the most important complication being enteric wound breakdown. Pre-operative factors that can help predict increased risk for dehiscence are not consistent in the literature but some larger studies suggest that animals with hypoprotienemia are at a higher risk of problems with healing and therefore a higher risk of dehiscence. (Grimes) One suspected risk factor has always been GI neoplasia. A recent study looked at the incidence of post-operative dehiscence in 70 cats with alimentary lymphoma. Fifty-three of these cases had enterotomy biopsy performed and 31 had a resection and anastomosis either concurrent or alone. The important finding was that no cases of enteric wound breakdown were noted. (Smith)

Intestinal Biopsy Technique:

- After complete abdominal exploration and identification of any abnormalities, one section from the duodenum, jejunum and ileum are isolated for biopsy.
- Each selected biopsy site is isolated with moist laparotomy sponges and the lumen is occluded with Doyen forceps or with an assistant’s fingers.
- The biopsy is obtained by creating two parallel incisions, approximately 1 cm long and 1 mm apart, using a new 11 blade for each biopsy. Each biopsy is
placed in separate formalin containers. The ends are then connected with the blade and the biopsy sample is handed to the technician.

- The incision is closed side to side, incorporating the submucosa but avoiding the mucosa, with 4/0 PDS or another monofilament absorbable suture on a taper swaged needle.
- If the diameter of the lumen is small, the biopsy site may be closed end to end to decrease the risk of compromising the diameter of the lumen.
- The sutures are placed approximately 1 mm apart and 3-4 mm from the cut edge along the width of the small intestine.
- If you choose to leak test the biopsy sites, use a 25g needle, occlude the site on either end with fingers and inject a small volume of saline which can then be compressed gently to check for leakage through the sutures.
- Once all biopsies have been obtained from the GI tract, the sites are lavaged with sterile saline. All persons then change gloves and clean instruments are used to close the body wall.

Post-operatively these patients are managed for pain with a narcotic and sometimes an NSAID. I do not put these animals on antibiotics past the surgical prophylactic antibiotics used during surgery. Animals are fed small frequent meals as soon as they will eat. They are kept in hospital for 2-3 days to ensure they are eating and that there are no signs of dehiscence such as increase in temperature, loss of appetite or leakage from the abdominal incision. Enteric breakdown generally occurs within 3-5 days following biopsy procedures and thus the animals should be monitored closely during this time frame.

**Intestinal Foreign Bodies:**

This is one of the most common indications for surgery of the small intestine in companion animals. Foreign bodies generally fall into one of several categories:

- Complete obstruction
- Partial obstruction
- Linear foreign bodies

Diagnosis can be difficult in many of these cases as the classic radiographic signs of obstruction and visible foreign bodies to allow a definitive diagnosis are not always evident. Thankfully there are many other techniques to assist in making the decision as to whether surgery is indicated in a particular patient. A number of studies have evaluated the use of ultrasonography as compared to traditional radiography and have found that ultrasound can be very helpful, especially in cases of linear foreign bodies. (Tyrrell) Ultrasound also has the advantage of being able to detect small amounts of free fluid or air in the abdomen and assist in gaining samples for cytology prior to surgery. An upper GI series can also be very helpful in gaining information regarding intestinal obstruction. For patients in which an intestinal perforation is suspected, the use of
barium sulfate is contraindicated and iohexal is a good substitute. (Williams)

Several prognostic indicators for successful outcome in dogs and cats with intestinal foreign bodies have been identified. Longer duration of clinical signs, presence of septic peritonitis, hypoproteinemia and hypotension are all considered negative prognostic indicators. (Ralphs, Grimes, Hayes) Discrete foreign bodies have a higher survival rate of 94% versus linear foreign bodies, with survival rates decreasing to 80% in dogs, and 63% in cats. (Hayes)

There are two main surgical techniques for extraction of intestinal foreign bodies. The ideal situation is the use of enterotomy in the healthy intestine, along the antimesenteric border and distal to the location of the foreign body. Approach and closure are as described for intestinal biopsy with the exception one incision is used rather than two. This requires that the intestinal wall overlying the foreign body is still viable, which can sometimes be difficult to evaluate. If the viability of the intestinal wall is questionable, a resection and anastomosis (R&A) must be performed. The more complicated R&A does lead to increased risk of enteric wound breakdown. Necessity for R&A also generally indicates a longer period of time in which the animal has been obstructed or the presence of a linear foreign body which has compromised the mesenteric border. Signs of intestinal compromise include adhesions of the omentum to the intestinal wall; black, dark purple or green discoloration of the intestinal wall; thinning of the intestinal wall overlying the foreign body and evidence of a perforation of the intestinal wall. In the case of a linear foreign body, evidence of disruption of the mesenteric blood supply would also be an indication for resection and anastomosis.

When performing an intestinal resection and anastomosis the risk of technical error is greater. You are working with a circular incision rather than a straight incision and often the lumen size of the two bowel ends to be anastomosed are not equal due to the fact that the oral end has been distended. The following suggestions for technique will help decrease the risk of technical error which would compound any other patient factors that may increase the risk of post-operative morbidity and mortality.

- Always make the smaller end larger; never make the larger end smaller. This can be achieved by making a small incision in the anti-mesenteric border on the distal end, as this is generally the smaller side, or by incising the smaller side on an angle. This allows for a closer fit between the two ends and less chance of leakage.
- Sutures can be either simple interrupted or simple continuous. If choosing simple continuous, placing two lines halfway around rather than one line completely around the circumference will decrease the risk of a purse-string effect and stricture of the anastomosis site.
- Always start your sutures on the mesenteric border and place 2-3 sutures in this location first. This site is the most difficult to visualize and this is the most common site for technical error and leakage leading to dehiscence. You can hold
the intestinal lumen open and ensure that you see the suture passing through the submucosal layer.

- Take the next bites on the antimesenteric border and then fill the remaining sites with sutures halfway between the previous sutures.
- Sutures bites should be 1-2mm apart and 3-4 mm from the cut edge.
- If you choose to leak test your surgical closure, use a small gauge needle (25) and remember that you do not need to distend the intestine with saline, just a small volume to allow you to massage the intestinal closure is all that is required and is closest to what the intestinal closure will be required to do once the animal is eating. Digesta in the small intestine is normally a liquid.

Recovery in an uncomplicated case is usually routine. Post-operatively, however, most of these animals do require intensive care as most of these patients have some degree of compromise.

- IV fluids to replace loss as well as maintain hydration are indicated. Crystalloids are generally sufficient
- In an uncomplicated case of intestinal foreign body removal, dogs and cats should eat almost immediately and have no signs of vomiting or fever. Enteric nutrition is a key factor in gut health and promoting healing in these cases and I feed these patients as soon as they will eat.
- If motility was questionable during surgery, I will put these patients on a metoclopramide constant rate infusion (CRI) at 1-2mg/kg/day until they are eating with no vomiting. You do not need a CRI pump to run a constant rate infusion. This can be achieved with a burette or even a regular bag of fluids.

Recovery and management of complicated cases such as those with pre-operative peritonitis and hypoprotienemia are much more labor intensive and require care in an ICU or 24 hours facility to ensure that problems are addressed as they arise. Discussion regarding care of these cases will be covered in the seminar on Acute Abdomen.

Animals requiring extensive resection of small intestine that is greater than or equal to 50% can still have good outcome with minimal to no clinical signs of short bowel syndrome. Preservation of the ileocolic valve is suggested to improve outcome as this helps prevent bacterial overgrowth from the colonic flora. The ileum is also considered to have the most potential for adaptation within the GI tract. (Gorman)

Indications that enteric wound breakdown is occurring may be difficult to assess. This generally occurs within 3-5 days after surgery, however, depending on the site that is dehiscing (i.e. an ileal dehiscence would cause more severe and acute clinical signs due to increased numbers and virulence of bacteria in the distal GI tract), clinical signs may develop much sooner. If you suspect that your intestinal surgical site is leaking, taking your patient back to surgery is indicated, even if you do not have the cytological evidence to confirm your suspicions. Early intervention always provides the best chance
for a positive outcome!

**Intussusception:**

Intussusception is generally a problem causing signs of gastrointestinal obstruction in young animals affected by viral or parasitic gastrointestinal disease. When this cause for intestinal obstruction occurs in older animals, suspicion of an intestinal mass should be moved to first on the differential list.

Diagnostics for intussusception are the same as for any other GI obstruction. The use of ultrasound is often valuable as the classic “target sign” is diagnostic for intussusception. Additional diagnostics in these cases which are important are parvovirus tests and a fecal float to help determine the underlying cause for the intestinal hyper motility, as surgery to relieve the obstruction without treatment of the primary problem will likely lead to reoccurrence.

Surgery in the case of intussusception involves a great deal of decision-making based on intra-operative findings and whether of not a primary cause has been diagnosed.

- If the intussusception can be easily reduced, this should be done. At the same time, viability of the affected intestine should be evaluated and monitoring for recurrence during surgery at the same or at another site should be performed.
- If the intussusception cannot be reduced, a resection and anastomosis should be performed. The affected segment should be submitted for histopathology, especially in the case of an older animal or an animal in which underlying cause has not been identified.
- If the small intestine continues to intussuscept, a bowel plication is indicated.
  - Use small monofilament suture, i.e. monocryl or biosyn, which will not maintain tensile strength for a long period of time. You need the sutures in place only long enough to get the primary problem under control.
  - The sutures should be placed just through the serosa and muscularis
  - The intestine should be placed in long, gently curving lines, side by side, to prevent kinking and minimize risk of foreign body obstruction should the animal be an indiscriminate eater.

Post-operatively, these patients are monitored and treated as for other GI surgery cases with the exception of directed treatment towards the primary cause of the intussusception if the cause if known. If the animal is young, treatment for GI parasites is indicated even if a positive fecal was not obtained.

**Serosal patching:**

Serosal patching is a technique that can be used if there is evidence that the small intestine had been previously breached and peritonitis is a concern. This technique uses
an adjacent loop of small intestine to protect an enterotomy site or a resection and anastomosis site from the inflammation occurring in the abdominal cavity. The loop of intestine is sutured to the affected intestine on both sides of the suture line using 4/0 or 5/0 monofilament absorbable suture such as monocryl. Serosal patching does not take the place of good surgical technique and will not “save” an enterotomy performed in unhealthy tissue.

**Diagnostic plan for determining if dehiscence is occurring:**

**Physical examination**
- Cardiovascular perfusion – HR, CRT, MM colour and hydration, peripheral pulses
- Respiratory system – tachypnea, dyspnea
- Abdominal palpation – Pain, which quadrant? Abdominal Fluid? Does palpation make the animal nauseous?

**Laboratory Evaluation**
- CBC and Chemistry panel are ideal but in critically surgical patients QUATS are adequate for quick evaluation and the results of the CBC and Chemistry panel can be used as baseline information for evaluating improvement or decline post-treatment.
  - Degree of dehydration – increased PVC, increased total protein, increased BUN
  - Sepsis – Decreased glucose, increased lactate, decreased platelets, decreased protein
  - DIC – decreased platelets, increased lactate

**Radiology**
- Can be difficult to interpret given that free air will be expected following abdominal surgery. Large quantities of fluid that completely obscure the abdominal organs should not be expected.
  - Loss of abdominal detail – free fluid or blood in the abdomen, due to septic peritonitis, bleeding, ascites, uroabdomen...
  - Abdominal Ultrasound
    - Very useful to find pockets or fluid for sampling and analysis.
    - If you do not have an ultrasound machine, blind abdominocentesis or diagnostic peritoneal lavage is indicated to determine the etiology of the fluid.

**Abdominocentesis**
• This is very important in trying to identify the etiology of the acute abdomen if abdominal effusion is suspected. This is easy to do and does not require an ultrasound if you do not have one available.
  
  o Check 4 quadrants
  
  o Evaluate the fluid collected for quantity and quality of nucleated cells and evidence of bacteria both intra- and extra-cellular
  
  o False negatives are common
    ▪ Generally an animal must have 5-25 ml/kg of free fluid in the abdomen to allow collection by abdominocentesis
  
  o Diagnostic Peritoneal Lavage (DPL)
    ▪ Place an IV catheter into the abdominal cavity
    ▪ Instill 20ml/kg of warmed saline
    ▪ Rock the patient back and forth gently for a few minutes
    ▪ Aspirate the abdomen as with a routine abdominocentesis
    ▪ Generally the volume of fluid instilled will be much greater than the volume retrieved
    ▪ Can do direct smears form this technique but performing a cytospin will allow for concentration of any cells present.
  
  o False negatives can be present with both direct abdominocentesis and if the breach in the GI tract happened less than 4-6 hours before the test was performed.
  
  o Fluid analysis
    ▪ Should see less than 2-5 % RBC’s
    ▪ WBC’s should not be greater than 500/ul
    ▪ 1000 – 2000 indicates inflammation
    ▪ Greater than 2000 indicates peritonitis
    ▪ Degenerate WBC’s indicates septic peritonitis, unless the animal has had recent abdominal surgery.
    ▪ Intracellular bacteria indicates septic peritonitis

Specific Tests and Diagnosis for Abdominal Fluid Samples as compared to Peripheral Blood Samples

<table>
<thead>
<tr>
<th>Specific Tests</th>
<th>Indication</th>
<th>Expected Result</th>
<th>Diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Creatinine / Potassium</td>
<td>Azotemia, trauma</td>
<td>Creatinine ↑ 2X Potassium ↑ 1.4X</td>
<td>Uroabdomen</td>
</tr>
<tr>
<td>Lipase Amylase</td>
<td>Suspect pancreatitis</td>
<td>↑ lipase and amylase</td>
<td>Pancreatitis</td>
</tr>
<tr>
<td>Bilirubin</td>
<td>Trauma, bile pigmentation</td>
<td>Fluid greater than serum</td>
<td>Bile peritonitis</td>
</tr>
<tr>
<td>Bacterial culture</td>
<td>Intracellular bacteria, sepsis</td>
<td>Positive culture / positive gram stain</td>
<td>Septic peritonitis (E. coli most common)</td>
</tr>
<tr>
<td>Glucose</td>
<td>Suspect septic peritonitis</td>
<td>Blood to glucose difference &gt; 20mg/dl</td>
<td>Septic Peritonitis</td>
</tr>
<tr>
<td>---------</td>
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<td>---------------------------------------</td>
<td>-------------------</td>
</tr>
<tr>
<td>Lactate</td>
<td>Suspect septic peritonitis</td>
<td>Peritoneal fluid lactate concentration &gt;2.5 mmol/L</td>
<td>95% accuracy for septic peritonitis in dogs only</td>
</tr>
</tbody>
</table>

What are the goals in surgery?
- Complete abdominal exploratory, even if we know what the problem is,
- Fluid and or tissue samples for analysis, culture and sensitivity and or histopathology. Never leave empty handed!!!
- Omental patching for incisions into hollow organs. Serosal patching may be beneficial if peritonitis is present. Serosal patching will not make up for poor surgical technique but may help protect your surgical site.
- Feeding tube? Is this animal likely to eat after surgery, if not, now is the time to place a feeding tube for assisted enteral feeding. A jejunostomy tube would be an appropriate choice if you need to bypass the proximal GI tract, i.e. pancreatic abscess. A gastrostomy tube may be appropriate for a cat with a picky appetite and recovering from trauma and a ruptured urinary tract. An esophagostomy tube would also be appropriate in a case in which the upper GI tract did not need to be bypassed.
- Abdominal drains may be appropriate depending on the case. How do we decide if a drain is appropriate?

**To Drain or Not to Drain?**

<table>
<thead>
<tr>
<th>Technique</th>
<th>Indication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary Abdominal Closure</td>
<td>Source of contamination found and eliminated</td>
</tr>
<tr>
<td>Open Peritoneal Drainage</td>
<td>Cannot eliminate contamination or widespread contamination</td>
</tr>
<tr>
<td>Closed Suction Drains</td>
<td>Drain specific sites of contamination i.e. pancreatic abscess</td>
</tr>
<tr>
<td>Penrose Drains</td>
<td>Drain specific sites of contamination within an abdominal bandage</td>
</tr>
</tbody>
</table>

Post-operative care:

Monitoring
- Cardiovascular function
• Heart rate and rhythm
  ▪ Tachycardia – Is the cause pain, hypotension, anemia?
• ECG – Arrhythmias? Why? Is the cause anemia, pain, hypoxia, reperfusion?
  ▪ Pulse strength and deficits if no ECG on site
• Blood Pressure
  ▪ Doppler – want to maintain a mean arterial pressure greater than 90.
    • Less than 90? Hypovolemic? Septic? DIC? Inaccurate reading?
  ▪ Central venous pressure - Want 0-5 cm of water
    • Central line and manometer
    • Adequate fluid therapy?

• Respiratory function
  o Low oxygen saturation
    ▪ Pain? Aspiration? Atelectasis? Anemia?

• Renal Function
  o Monitor urine output in these critical cases
  o Indwelling urinary catheter
    ▪ Minimum of 1ml/kg/hr
      • If the patient is not producing urine what is the problem?
        o Hypotension
        o Obstruction
        o Sepsis and multiple organ dysfunction
        o Hypovolemia
        o Catheter is obstructed?

• Hypothermia
  o Common in critically ill patients, shock, DIC, post-operative
    ▪ Warm water blankets and warm water bottles
    ▪ Warm fluids
  o If you suspect or have confirmed sepsis and or DIC these patients require extremely close monitoring
    o Platelet counts – if low, careful with blood collection, monitor for improvement
    o Glucose – supplement if decreased
    o Activated clotting time, monitor for increases vs decreases
    o Urine output – keep a close eye on ins and outs, once dehydration has been corrected, urine output should match input.
Monitor white blood cells, increases or dramatic decreases are both important prognostic indicators.

- Plasma when needed in cases of sepsis
- Appropriate IV antibiotics based ultimately on culture and sensitivity

- Analgesia – These animals are painful and pain can have a negative impact on overall recovery and systemic functions. Pain can cause:
  - VPC’s
  - Hypoxia if the patient is not ventilating effectively
  - Hypertension
  - Restlessness
  - Anorexia

- Treat pain with narcotics. Constant rate infusions work very nicely because the pain control is constant and titration is quick to allow the desired pain relief without over-sedation.

- Be very careful with NSAID administration if renal or liver function is in question or if dehydration has not been corrected.

**Take Home Message**

Incidence of complications with small intestinal surgery is higher than we may think

**BUT**

Watching the patients and listening to your intuition if you suspect something is not correct, will save lives! Good surgical technique and identifying potential issues with healing is the key to minimizing incidence of problems with GI surgery. When the client and veterinary team works together success happens!!

**References:**


Hospitalized dogs and cats often do not meet their daily energy requirements due to a variety of factors. For some this problem is behavioral. Animals with anxiety, or fear are often unwilling to eat when taken out of their home environment. For others the problem is secondary to the disease process for which they are hospitalized, with these animals being unwilling to eat because they are not feeling well or unable to keep food down due to mechanical obstruction or other problems such as megaesophagus or pancreatitis. For yet another group they may physically be unable eat due to trauma such as a jaw fracture or recent surgery. One thing that all of these groups have in common is that they all have basal daily energy requirements which, if not met, will adversely affect recovery, wound healing, immune function and overall prognosis. (Michael 2006)

Feeding tubes are not to be taken as the first line of treatment for an animal who is just unwilling to eat in the hospital but for cases in which negative caloric intake is impacting overall health and ability to recover from injury or disease, they can significantly impact when and if an animal will be discharged from hospital. In a study of 455 dogs and 50 cats, energy intake was positively associated with hospital discharge. (Brunetto, 2010) Enterally delivered nutrition (EN) is generally preferred over parenteral nutrition due to evidence from human clinical trials and animal models that enteral nutrition may preserve gut integrity, minimize bacterial translocation, improve the immune response, and attenuate the release of inflammatory mediators. (Campbell, 2010) Enterocytes undergo atrophy within several days in the absence of direct trophic stimuli within the intestinal lumen so if the gut works, use it!! (Mazzaferro, 2001)

Methods of Delivering Enteral Nutrition:

- **Coaxed feeding:** This is the most common type of assisted enteral feeding used in veterinary settings. Warmed canned food is a common choice and for patients who are just nervous and have a normal gastrointestinal tract, this can work well. This type of feeding however is only appropriate for up to 2 days as the ability to provide adequate caloric requirements and the tendency for some animals, especially cats, to become adverse to this type of intervention can actually exacerbate the problem. Other potential complications such as aspiration pneumonia can also be encountered. (Davidson, 2011)

- **Orogastric intubation:** This type of force-feeding is generally reserved for neonates, as older animals will usually not tolerate placement of an orogastric
tube. Aspiration is the greatest potential complication of orogastric tube feeding.

- **Nasoesophageal (NE) tubes**: This type of feeding tube is placed through the nares into the esophagus. Indications for placement of a nasoesophageal tube include severe debilitation in which anesthesia for placement of a larger tube would be a concern or for short term feeding for less than 1-2 weeks. (Davidson 2011, Han, 2004) Nasoesophageal tubes are contraindicated in animals with loss of gag reflex, abnormal esophageal motility, coma or any condition increasing the risk of aspiration pneumonia. Potential complications include epistaxis, sneezing, displacement of the tube prior to intended removal, and aspiration pneumonia if the tube becomes displaced into the oropharynx. Due to the small size of tube which can be placed in the nasal cavity, liquid diets such as Clinicare are recommended. These diets can be fed as a constant rate infusion or by intermittent feedings. (Campbell, 2010)

- **Nasogastric (NG) tubes**: These feeding tubes are placed through the nares in to the gastric lumen. Feeding with these tubes can also be via bolus or contrast infusion. (Klaus 2009). The indications, complications and contraindications are similar to NE tubes.

- **Nasojejunal tubes**: This feeding tube is placed from the nares though to the jejunum via fluoroscopic guidance. In animals with gastric disease or persistent vomiting due to other illness, jejunal feedings offer the advantage of bypassing the stomach and feeding directly into the jejunum, thus allowing for the provision of EN support in the patient population for whom gastric feeding is contraindicated or poorly tolerated. (Beal 2011, Papa 2009) The most common complication of nasojejunal tubes is migration orally which occurred in 27% of patients in one study. However, the migration in most of these patients was into the distal duodenum and ability to continue assisted enteral nutrition was not compromised. (Beal 2011)

- **Esophagostomy Tubes**: These feeding tubes are placed directly into the esophagus though the left side of the neck and extend from the proximal esophagus to just proximal to the esophageal sphincter. Indications for esophagostomy tube placement include anorexia, hyporrhexia, facial or pharyngeal trauma, mucositis, oral or pharyngeal neoplasia, stomatitis and surgery of the face or oropharynx. Contraindications include aspiration pneumonitis, comatose patients, esophagitis, esophageal motility disorders, lack of gag reflex, megaesophagus and excessive vomiting. Placement is relatively easy and does not require special equipment. Potential complications include cellulitis/infection at placement site, improper placement (i.e., into mediastinum or stomach), reflux esophagitis, displacement oral with excessive vomiting, esophageal stricture and jugular vein or carotid artery puncture. (Mazzaferro,
These tubes can remain in place for weeks to months and removal can be done safely at any time after insertion by simply pulling the tube and allowing the stoma to heal by second intention.

- **Gastrostomy Tubes:** These tubes are placed directly into the gastric lumen, exiting the body wall on the left side. Gastrostomy tubes can be placed in several different manners from surgical placement at the time of an exploratory celiotomy to percutaneous placement either endoscopic assisted or non-endoscopic assisted. Indications for gastrostomy tube placement include anorexia, anticipation of anorexia or inability to eat. A gastrostomy tube is preferred over esophagostomy tubes or NE tubes in clinical cases in which the oral cavity, pharynx and or esophagus must be bypassed due to injury, disease, obstruction or surgery. Contraindications to a gastrostomy tube would include primary gastric disease or persistent vomiting. In animals with an abnormally functioning esophagus or abnormal mentation, these tubes should be used with caution as these patients are at a higher risk for aspiration pneumonia if they are vomiting or regurgitating. A major advantage to gastrostomy tubes is the ability to feed blenderized food since the lumen of these tubes can be quite large. These tubes can also remain in place for months if necessary and owners find using the tubes relatively easy. Potential complications of gastrostomy tubes are not common but range from tube site irritation to vomiting of food if fed too rapidly or too frequently to most seriously, peritonitis should the feeding tube become dislodged and be redirected into the abdominal cavity. Once placed a gastrostomy tube should be left in place for at least 7-10 days to allow the stoma to seal and reduce the risk of leakage form the gastric lumen into the abdominal cavity. To remove gastrostomy tubes, the mushroom tip can either be cut or left to pass through the small intestines, or it can be elongated and pulled through the stoma. The stoma will heal by second intention within 24-48 hours.

- **Enterostomy Tubes:** These tubes are placed directly into the distal duodenum or proximal jejunum through the body wall. Techniques for placement include open surgical approach, laparoscopic assisted placement or placement through and existing gastrostomy tube. (Hewitt 2004) Indications for Enterostomy tube placement include inability to feed orad to the small intestine due to disease, trauma or surgical intervention. Enterostomy tubes also have a smaller risk of gastric reflux and vomiting so may be useful in animals with esophageal dysfunction. Obstruction of the small intestine distal to the tube termination is the only contraindication to enterostomy tube placement. (Davidson 2011, Yagil-Kelmer 2006) Complications include premature removal, leakage around the tube, cellulitis, tube occlusion, jejunal obstruction, and intestinal perforation leading to peritonitis. (Yagil-Kelmer 2006) Reported overall complication rates are as high as 46%, however most of these complications were minor issues such as cellulitis around the tube site. Serious complications such as peritonitis are rare. Feeding can be started within hours after surgery and the tube can remain
in place for weeks to months. Tube removal can be performed as early as 5 days post placement if the tube is not needed or the animal begins to eat without problems. When removed the stoma is left to heal by second intention.

**Techniques for Feeding Tube Placement**

Nasojejunal tubes and PEG tubes will not be discussed as these techniques require advanced equipment which most private practices will not have in house.

**Nasoesophageal and Nasogastric Tubes:**

**Equipment Required for Placement of NE and NG Tubes**

- 0.5% proparacaine hydrochloride ophthalmic solution and a water soluble lubricant
- nylon suture material or skin staples or cyanoacrylate glue
- 3.5 – 5.0 French red rubber catheter or 3.5 – 8.0 French Kendall Argyle feeding tube with radiopaque marker
- Elizabethan collar

**Technique for Placement of NE and NG tubes**

- If necessary light sedation should be administered prior to tube placement. Reversible drugs are ideal if possible, depending on the medical situation of the animal.
- The nares should be numbed using 2-3 drops of 0.5% proparacaine.
- For NE tubes the distance from the nares to the 9th rib should be measured and marked; for NG tubes, the distance from the nares to the 13th rib is marked. When using marked Kendall Argyle feeding tubes this can be translated to the markings on the tube. For red rubber catheters, the tube can be marked with a marker or tape.
- The tube tip should be lubricated and directed into the nares in a caudoventral medial direction and advanced to the desired distance.
- Once advanced the tube can be sutured into place next to the nares and on top of the head between the eyes or glued in these positions
- A lateral thoracic radiograph should be taken post placement to ensure proper positioning. Argyle feeding tubes are preferable as they have a radiopaque marker.
- After positioning has been confirmed an Elizabethan collar should be placed to prevent dislodgement of the tube by pawing or rubbing of the nose.
- These tubes can be used immediately or after recovery from sedation if sedation was necessary.
Esophagostomy Tubes:

Equipment Required for Placement of Esophagostomy Tubes

- Long curved forceps (i.e. curved Carmalt)
- Long straight forceps (i.e. Carmalt)
- #10 scalpel blade
- 5-10 French red rubber, polyvinyl chloride or polyurethane tube depending on the size of the animal
- Nylon suture material on a cutting needle
- Light bandage material – non-adherent layer (i.e. Telfa), cast padding, roll gauze and Vet Wrap.
- Catheter adaptor
- Injection cap

Technique for Manual Placement of Esophagostomy Tubes

- The animal is anesthetized and positioned in right lateral recumbency.
- The left side of the neck should be clipped and the skin surgically prepped from the ramus of the mandible to the thoracic inlet from dorsal midline to ventral midline.
- Drapes or huck towels should be positioned to protect the site.
- The tube length should be measured from the mid cervical esophagus to the 9th rib and marked.
- A large curved forceps, size will depend on the size of the animal, is placed though the oral cavity into the cervical esophagus with the tips pointed laterally to allow palpation of the tips in an avascular location dorsal to the jugular vein.
- A scalpel blade is used to make a small stab incision though the skin, subcutaneous tissue and the esophageal wall to allow the tips of the instrument to protrude through the skin.
- The tip of the tube is grasped securely with the forceps and pulled into the esophagus and out through the oral cavity, leaving the proximal end of the tube still protruding through the cervical skin incision.
- The distal tip of the tube is then grasped securely with long straight forceps or fingers and redirected through the oral cavity and into the cervical esophagus through the oropharynx. You may need to pull the proximal end out of the cervical incision a few centimeters to allow the tube to be straightened.
- Once the tube has been directed in the appropriate position it can be inserted to the premarked position.
- The tube is secured in place with a purse string suture and a finger trap to prevent migration. The tube exit site is covered with a non-adherent dressing and a light bandage around the neck.
• The tube can be capped with a catheter adapter inserted into the tube and then an injection cap.
• A lateral thoracic radiograph is then taken to ensure correct placement of the esophagostomy tube.
• The tube exit site should be visually inspected daily for signs of cellulitis and the tube and exit site should be cleaned with chlorhexidine daily.
• The tube can be used as soon as the animal has recovered from anesthesia.

**Gastrostomy Tubes:**

**Equipment Required for Surgical Placement of Gastrostomy Tubes:**

• Mushroom tip catheters of various sizes depending on the size of the animal. (Foley catheters are not appropriate for gastrostomy tubes, as the gastric acid will weaken the bulb, risking deflation of the bulb and displacement into the abdomen.)
• Routine surgical pack and drapes
• Nylon on a cutting needle
• Catheter adaptor
• Injection cap

**Technique for Surgical Placement of Gastrostomy Tubes via Ventral Midline Celiotomy**

• Routine open ventral midline celiotomy with complete abdominal exploratory should be performed.
• Gastrostomy tube placement should be the final procedure prior to the closing the abdominal wall.
• 2/0 or 0 absorbable suture is used to make a full thickness purse string in the left body wall.
• A stab incision is made in the middle of the purse string and the tip of the mushroom catheter is introduced into the stomach lumen.
• The purse string is tightened.
• A stab incision is made in the left body wall just caudal to the 13th rib.
• The left body of the stomach is pulled to the left body wall and stay sutures are placed from the stomach wall to the body wall. I generally place 4 sutures using 2/0 monofilament absorbable suture.
• If desired the gastropexy site can be encircled with omentum.
• The abdominal wall is closed routinely and the tube is secured to the outside of the abdomen with a purse string and finger trap suture.
• The tube should be capped with a catheter adaptor or clamped with a tube clamp.
• The tube exit site is covered with a non-adherent dressing and stockinet can be used to cover the tube and keep it from dragging.
• An Elizabethan collar should be placed on the animal to help prevent inadvertent removal.
• Visually inspect the tube site daily for signs of cellulitis and clean the tube and site with chlorhexidine daily.
• Feeding can be instituted within 24 hours of tube placement.

Enterostomy Tubes:

Equipment Required for Surgical Placement of Enterostomy Tubes:

• 8 French argyle feeding tube or red rubber tube with the blind end removed
• Routine surgical pack and drapes.
• Nylon on a cutting needle
• Catheter adaptor
• Injection cap

Technique for Surgical Placement of Enterostomy Tubes via Ventral Midline Celiotomy

• Perform a routine open ventral midline celiotomy with complete abdominal exploratory.
• Enterostomy tube placement should be the final procedure prior to the closing the abdominal wall.
• A segment of distal duodenum or proximal jejunum is isolated.
• A stab incision is made in the right body wall at a site chosen for the enteropexy over a Kelly forceps directed from the peritoneal surface to the skin.
• The feeding tube is grasped with the forceps and pulled from the skin into the abdominal cavity.
• A 1.5 – 2.0 cm antimesenteric incision is made at the isolated segment of small intestine through the serosa and muscularis, leaving the mucosa intact.
• A small incision, just large enough to pass the catheter through, is made at the aboral end of the intestinal incision and the catheter is passed into the intestinal lumen and advanced 20 to 40 cm, depending on the size of the animal.
• The serosa and muscularis are closed with 4/0 monofilament absorbable suture in a simple interrupted pattern and a purse string suture is placed around the tube at the orad end of the incision.
• The intestinal wall is secured to the abdominal wall using stay sutures.
• The abdominal wall is closed routinely and the tube is secured to the outside of the abdomen with a purse string and finger trap suture.
• The tube is then capped with a catheter adaptor or clamped with a tube clamp.
• The tube exit site is covered with a non-adherent dressing and stockinet is used to cover the tube and keep it from dragging.
• An Elizabethan collar is placed on the animal to help prevent inadvertent removal.
- Visually inspect the tube site daily for signs of cellulitis and clean the tube and site with chlorhexidine daily.
- Feeding can be instituted within 24 hours of tube placement.


**Tube Feeding:**

**Diet**

The type of diet chosen will depend on the size of the feeding tube that is positioned, the condition of the animal and the nutritional needs of that animal. Liquid diets are best suited to small diameter tubes like NE, NG and enterostomy tubes. Blended diets can be used in larger diameter tubes such as esophagostomy and gastrostomy tubes. Diets can be administered as a continuous infusion, which is more common with smaller tubes and liquid diets, or as bolus feedings. As long as nutritional requirements are being met, either rate of feeding is acceptable. A general rule is that the diet chosen should have maximal energy density so the volumes fed can be kept small. Water can be added to diets to improve viscosity for transfer through the tubes. This volume should be monitored and kept in mind if the animal is also on intravenous fluids so as not to fluid overload.

**Starting Enteral Feedings**

The energy requirement for an animal is based on a calculation of resting energy requirement:

\[
\text{RER in kcal/day} = 70 \times (\text{body weight in kg})^{0.75}
\]

This is a good starting point for calculating energy requirements and using daily body weights and blood work can allow regular adjustment of food administration.

When beginning assisted feeding in an animal that has not been eating for a period of days, it is best to start on day 1 with 1/3 of the calculated requirement and increase this amount by 1/3 over a three-day period until the total calculated requirement is being administered.

For constant rate infusion of liquid diets, the residual volume should be checked every 8 hours and if the volume removed from the tube is greater than the volume administered over a 2-hour period, feeding should be discontinued for 2 hours and resumed at a rate 25% less than the pervious rate.

For intermittent bolus feedings, initial volumes recommended are 3 to 5 mls/kg q2-4hr gradually increasing the volume over a three-day period until the total calculated
requirement is being administered. Most dogs and cats can ultimately be fed 22 – 30 ml/kg per feeding. The amount fed can also be calculated using the 1/3 requirement on day 1 and dividing that volume into 4-6 feedings. Gastric tubes allow aspiration of gastric contents and if more than 1/2 of the volume fed is aspirated, the volume should be returned to the stomach and the feeding skipped until the next scheduled time. If this is a constant problem, motility enhancers such as metoclopramide can be considered at a rate of 1-2mg/kg/day as a constant rate infusion providing documentation that there is no obstruction of the gastrointestinal tract aboral to the tube.

If animals are experiencing diarrhea secondary to feedings, the volume and or frequency of feedings can be decreased until the diarrhea resolves, which will generally occur within a few days, unless an underlying problem is present.

Unless otherwise indicated, offering food by mouth should still be continued daily and once adequate intake is voluntary; the feeding tube can be removed.

Diet and feeding recommendations adapted from Davidson 2012.

References:


Michel KE, Higgins C. Investigation of the percentage of prescribed enteral nutrition actually delivered to hospitalized companion animals. *J Vet Emerg Crit Care.* 2006;16(2)(S1):S2-S6


Otitis refers to inflammation of the ear and may include not only the external ear canal in otitis externa, but may also involve the middle ear in otitis media, and the ear pinnae as well. Otitis externa is the most common ear disease in the dog and cat. The reported incidence is between 10 to 20% in the dog and 2 to 10% in the cat. Otitis externa is one of the most common reasons for animals to be referred to dermatology specialists, and is a very common clinical problem managed by general practitioners as well. It is important to be able to recognize normal otic anatomy to be able to diagnose otic disease.

Prior to examination of the animal, it is important to obtain a complete and thorough history from the owner. Even though this can be a time-consuming step, it is invaluable for a complete assessment of the animal and for insight into the primary cause of the otitis. A dermatologic history form can be mailed to the client prior to the appointment, or it can be filled out when the client arrives. Questions to include:

- Onset of the otitis, unilateral or bilateral
- Seasonal, non-seasonal, or seasonally non-seasonal
- Ears pruritic or painful
- Current and previous treatment(s) used for the otitis as well as outcome, side effects, drug reactions
- Any hearing loss or vestibular signs - be careful to distinguish “head tilt” from “holding head to one side”
- Previous steroid administration
- Other dermatologic concerns: pruritus, alopecia, “rash”
- Current and previous diets and treats
- Current treatments for other concurrent diseases or preventive treatments (flea control, heartworm prevention)
- Any others in home with skin problems

One should also inquire about the clinical signs that prompted the owner to seek veterinary care. Common clinical signs associated with otitis externa include:

- Head shaking
- Scratching and rubbing the ears
- Pinnaal alopecia
- Excoriations
- Odor
- Pain
• Hearing loss
• Behavioral changes

The next step is to perform a general examination as well as a dermatologic examination. In some cases, a neurological examination may be needed if one suspects the animal to have concurrent otitis media or otitis interna. If otitis media is present as well, the animal may exhibit signs such as facial nerve paralysis or Horner’s syndrome. However, remember that the most common clinical sign of otitis media is recurrent otitis externa. Head tilting, circling, and nystagmus may indicate otitis interna.

The otic examination is best done as the last part of the examination. The condition of the pinnae should be noted, including the ear margins, looking for erythema, scaling or crusting. Next, palpate the vertical, then the horizontal ear canals down to where they attach to the skull. Are they firm or calcified? Is one side more affected than the other? Once this is completed, begin the otoscopic examination with the otoscope resting gently in the intertragic incisure, gently moving down the canal and evaluate the vertical then horizontal ear canals for erythema, exudate, hyperplasia, stenosis, and ulcers. Once into the horizontal ear canal, evaluate the tympanic membrane.

EXTERNAL EAR CANAL

The external ear is composed of two elastic cartilages: the annular and auricular cartilage. The auricular cartilage expands to form the pinna. The pinna is a mobile structure designed to localize and collect sound waves and transmit them to the tympanic membrane. The auricular cartilage of the pinna becomes funnel shaped at the opening of the external ear canal.

The opening of the external ear canal is bounded by the helix (the free, slightly folded margin of cartilage at the base of the pinna) rostrally, the tragus laterally, and the antitragus caudally. The antitragus is a thin, elongated piece of cartilage caudal to the tragus, and separated from it by the intertragic incisure. This anatomical region is the area in which I will insert the otoscopic cone or otoendoscope into the ear canal for the otoscopic examination. The vertical ear canal runs for about 1 inch, extending ventrally and slightly rostrally before taking a medial turn and forming the horizontal ear canal. There is a prominent cartilaginous ridge (“Noxon’s Ridge”) that separates the vertical and horizontal ear canals and when the ear is in its normal position, makes otic examination of the horizontal ear canal difficult without elevating this ridge by grasping the ear pinna and lifting the ear.

The horizontal ear canal is composed of auricular and annular cartilage. The auricular cartilage rolls as it forms a tube. A separate cartilaginous band, the annular cartilage fits within the base of this tube. The annular cartilage overlaps with the osseous external acoustic meatus and articulates via ligamentous tissue, giving the external ear canal flexibility.

In most breeds of dogs, hairs are present in the external ear canal, decreasing in number from distal to proximal. A very few fine hairs are found at the entrance of the cartilaginous external acoustic meatus. I find these hairs are a useful landmark when flushing an ear to locate the tympanic membrane.

Cerumen is an emulsion that coats the ear canal. It is composed of desquamated
The self-cleaning function of the external ear canal is primarily achieved by a process called epithelial migration. The epithelium in the ear canal grows outward from the tympanic membrane toward the opening of the external ear canal. These epithelial cells carry debris with them as well. When the anatomy of the epithelium of the ear canal is altered, or when the rate of epithelial movement is slowed due to age, debris accumulates in the ear canal or on the tympanic membrane. This condition is termed “failure of epithelial migration”. Wax and keratin accumulate to form either soft wax plugs or ceruminoliths. Removal of these usually requires the patient to be under general anesthesia or heavily sedated. Soft wax plugs are usually easy to remove by flushing with a ceruminolytic agent and saline. However, ceruminoliths may be more difficult to remove, requiring additional soaking time with a ceruminolytic agent as well as the use of grasping forceps. After the removal of a ceruminolith, the tympanic membrane may appear abnormal, or may even have small tears in it. These small tears heal rapidly. If the tympanic membrane is torn while removing the ceruminolith, it is important to flush the ear with sterile saline to remove the ceruminolytic agent from the ear canal.

THE MIDDLE EAR

The middle ear consists of an air-filled tympanic cavity, three auditory ossicles, and the tympanic membrane. The tympanic membrane is located at a 45-degree angle in relation to the central axis of the horizontal part of the external ear canal. The tympanic membrane is a semitransparent membrane that separates the external ear canal from the middle ear, is thin in the center and thicker at the periphery, and is divided into two sections, the small upper pars flaccida and the larger lower pars tensa. The pars flaccida is the pink, small, loosely attached region forming the upper quadrant of the tympanic membrane that contains small blood vessels. In most dogs, grossly the pars flaccida is flat, while on occasion, in other dogs, this structure bulges into the external ear canal. This bulging pars flaccida can be present in the ears of normal dogs as well as in ears of dogs with otitis externa. Since no differences can be found histologically between a bulging pars flaccida and a flat pars flaccida, it appears unlikely that there is a structural difference causing the pars flaccida to bulge. There may be increased pressure in the middle ear of dogs with a bulging pars flaccida.

In the Cavalier King Charles spaniel, however, it does appear that a bulging pars flaccida is indicative of a middle ear disease, specifically primary secretory otitis media. Primary secretory otitis media (PSOM) or “glue ear” is a disease described almost exclusively in the Cavalier King Charles spaniel (CKCS). Dogs with this condition may
exhibit head and neck pain, “air” scratching, neurological signs (facial paralysis, head tilt, vestibular signs), and hearing loss. In a retrospective review of 61 cases of PSOM by Stern-Bertholtz et. al., the diagnosis was made based on visualization of a bulging opaque tympanic membrane with an operating microscope and the finding of an accumulation of mucus in the middle ear after myringotomy. No additional tests were used to evaluate the dogs for otitis media.

Since the publication of that study, the author has prospectively evaluated otoscopy, tympanic bulla ultrasonography, pneumotoscopy, and impedance audiometry for the diagnosis of PSOM in 60 CKCS dogs utilizing computed tomography (CT) as the gold standard for diagnosis. The following are the conclusions made based on the results of the study. PSOM is common disease in the CKCS. It is uncommon to be associated with infection and rare to be associated with bulla changes. Clinical signs may resolve post-myringotomy and middle ear flush, albeit temporarily. Lack of mucus removed from middle ear does not equate to lack of disease. A large bulging pars flaccida indicates PSOM in the CKCS and there is no need for further testing; however, the majority of CKCS with PSOM had a flat pars flaccida. Tympanic bulla ultrasonography, pneumotoscopy, and tympanometry had poor specificity and sensitivity for diagnosis of PSOM, therefore, a CT scan is the best test if the pars flaccida is flat.

Currently, the cause of PSOM is unknown; however, it has been speculated to be due to a dysfunction of the Eustachian tube. Hayes et. al. evaluated the relationship between nasopharyngeal conformation and otitis media with effusion (OME) in CKCS. Based on the results of their study, there appeared to be an association between OME and the brachycephalic conformation; those CKCS with bilateral OME had a significantly greater thickness of the soft palate and reduced cross-sectional area of the nasopharynx compared to CKCS with unilateral OME and those without OME. The study demonstrates an association between changes in the nasopharyngeal soft tissues and the incidence of OME, but cannot predict the nature of this relationship.

Current treatment of PSOM is removal of the mucus via a deep ear flushing of the middle ear. Culture and cytology of the mucoid exudate is usually negative; however, is still recommended. In the above retrospective study, various forms of medical management were used post-flushing, however, a number of CKCS did require repeated middle ear flushes to remove the mucus from the middle ear. This is not necessarily unexpected, since the cause of this disease has yet to be identified. The CKCS may develop an infectious otitis externa post-flushing.

Tympanostomy tubes have been used in human medicine to provide continual tympanic cavity ventilation and pressure equalization for the treatment of otitis media with effusion. Two studies have been published utilizing tympanostomy tubes as an alternative treatment to myringotomy and middle ear flushes in CKCS with PSOM. In both studies the insertion of the tympanostomy tube provided relief of the presenting clinical signs for a maximum of 8 months. The CKCS in the first study by Cox et. al. had unilateral otitis media with effusion that was first treated surgically with a lateral wall resection, prior to the insertion of the tympanostomy tube. It is uncertain as to which author of the first study inserted the tympanostomy tube; however an ENT surgeon was
the one to actually perform the procedure in the second study (personal communication) by Corfield et al. Tympanostomy tube insertion may be an alternative to repeated myringotomy and middle ear flushes for the treatment of PSOM, but requires specialized equipment (e.g. operating microscope) and training to perform the procedure.

The pars tensa, the thin, tough, gray structure with radiating strands, occupies the remainder of the membrane. The pars tensa is attached to the osseous ring of the external acoustic meatus. The manubrium of the malleus attaches to the medial surface of the tympanic membrane. The outline of the manubrium of the malleus, the stria mallearis, may be visualized when the tympanic membrane is viewed externally.

The tympanic cavity consists of a small epitympanic recess and a large ventral bulla. The tympanic bulla proper is adjacent to the tympanic membrane. In the dog, there is an incomplete bony septum or tympanic bulla ridge (“Rosychuk’s Ridge”), which allows communication between the tympanic bulla proper and the ventral tympanic bulla. On the medial wall of the tympanic cavity, there is a bony eminence, the promontory, which houses the cochlea, and lies opposite the tympanic membrane. The cochlear (round) window is located on the caudolateral portion of the promontory and is covered by a thin membrane. The vestibular (oval) window lies on the dorsolateral surface of the promontory immediately adjacent to the pars flaccida and is covered by a thin diaphragm. The footplate of the stapes is attached to the diaphragm over the vestibular window. When flushing the middle ear, one must be very careful to avoid damaging the promontory or the round window, to avoid damaging the inner ear.

The middle ear cavity of the cat is different than the dog, and is divided by a septum into two separate tympanic cavities. In the small dorsolateral compartment lie the auditory ossicles, the osteum of the auditory tube, and the tympanic membrane. The larger ventromedial compartment is the air-filled tympanic bulla. In order to remove exudate or a mass from the ventromedial compartment of the bulla, the bony septum would need to be perforated to gain entry. Rough handling of the bony septum may result in damage to the postganglionic sympathetic nerves. The nerves, which are visible submucosally as fine strands over the cochlear promontory, should be avoided during surgical removal of the septum in the cat.

The auditory tube is a short canal that extends from the nasopharynx to the rostral portion of the tympanic cavity proper. It exits on the ventral aspect of the skull immediately rostral to the bulla and is protected ventrally by the sharp pointed muscular process of the temporal bone. The lateral wall is about 8 mm long and is nearly twice the length of the medial wall. The tube is oval in diameter and at its greatest diameter is 1.5 mm. It functions to equalize pressure on both sides of the tympanic membrane. The auditory tube is divided into three portions: cartilaginous (proximal and opens into the nasopharynx), junctional (part of tube at which the cartilaginous and osseous portions connect) and the osseous portion (distal and opens into the anterior middle ear). The osseous portion of the auditory tube is patent at all times while the cartilaginous portion is closed at rest and opens during swallowing. The entrance to the auditory tube is obscured behind the soft palate, midway between the caudal aspect of the nares and the caudal border of the soft palate. Based on contrast-
enhanced computed tomographic imaging, the auditory tube originates from the rostral, dorsomedial aspect of the bulla and exits the dorsolateral aspect of the nasopharynx just caudal to the hamulus process of the pterygoid bone.

The three auditory ossicles, the malleus, incus and stapes, are the bones that transmit and amplify air vibrations from the tympanic membrane to the inner ear. The malleus is attached to the tympanic membrane, the petrous temporal bone, and the incus. The incus is suspended between the malleus and the stapes. The footplate (base) of the stapes is attached to the vestibular (oval) window, which is in direct contact with the perilymph fluid. The vestibular window is approximately 18 to 20 times smaller in area than the tympanic membrane. When performing a myringotomy, the incision should be made into the caudoventral portion of the pars tensa to avoid damaging the ossicles.

Normal canine tympanic membranes experimentally ruptured have been demonstrated to regenerate by day 14, with complete healing occurring between 21 to 35 days. Histology confirmed that the tympanic membranes healed, but were thicker than the control tympanic membrane that had not been ruptured. In the cat, the average number of days to heal the tympanic membrane is dependant upon the size of the perforations, with those that involve 25% of the tympanic membrane requiring an average of 25 days, while a complete rupture required an average of 61 days for complete healing. Histologically, repair was underway as early as 12 hours.

**THE INNER EAR**

The inner ear is housed in a bony labyrinth in the petrous portion of the temporal bone. The bony labyrinth consists of a perilymphatic chamber (vestibule), three semicircular canals (each with an ampulla), and a spiral cochlea. The perilymphatic space of the vestibule encloses the utricle, saccule, and the base of the cochlear duct. The vestibule is an irregular oval space that communicates with the cochlea rostrally and the semicircular canals caudally. The lateral wall of the vestibule has two windows, the vestibular (oval) window and the cochlear (round) window.

The bony labyrinth surrounds the membranous labyrinth. The membranous labyrinth contains the sensory organs that control hearing and balance. There are three functionally related parts of the inner ear. The first is the semicircular ducts, containing hair cells that detect acceleration of the endolymph caused by rotation of the head. The second is the utricle and saccule, containing hair cells with a membrane, the macula, which responds to linear acceleration of the head and its static position. The third part is the membranous cochlea, which is the auditory portion of the labyrinth, containing the hair cells involved in hearing, in the organ of Corti.

The membranous cochlea follows the shape of the bony cochlea. The membranous cochlea has three ducts, the scala vestibuli, the scala media or cochlear duct, and the scala tympani. These ducts are created by two membranes. The basilar membrane separates the scala tympani from the cochlear duct, while Reissner’s membrane separates the scala vestibule from the cochlear duct. The scala tympani and scala vestibule communicate at the apex of the cochlea at the helicotrema. The membranous cochlea is a fluid-filled system. The scala tympani and scala vestibuli are filled with perilymph, which is high in sodium and low in potassium and has a 0 mV
electrical charge, while the cochlear duct is filled with endolymph, which is high in potassium and low in sodium and has a +80 mV electrical charge.

The stria vascularis is a highly vascular structure on the lateral wall of the cochlear duct and produces endolymph. The stria vascularis plays a critical role in maintaining the ionic composition of the endolymph and the endocochlear potential by replenishing potassium in the cochlear duct. In breeds such as the Australian cattle dog and Dalmatian, congenital hereditary hearing loss results from the degeneration of the blood supply to the stria vascularis. The nerve cells of the cochlea subsequently die and permanent hearing loss results.

The basilar membrane runs the length of the cochlea and is wider at the apex than at the base and its fibers are stiffer at the base than at the apex. Resting on the basilar membrane is the organ of Corti, the end organ of hearing, which runs the length of the cochlear duct. The sensory cells of the organ of Corti are the inner and outer hair cells. The inner hair cells are the actual receptor for hearing and transmit information to the auditory nerve while the outer hair cells play an important role in adjusting the tuning and sensitivity of the inner hair cells. Stereocilia are located on the top of the hair cells and when the stereocilia are deflected, pores open allowing potassium ions to flow into the cell causing depolarization of the hair cell and transmission of sound-evoked information to the auditory nerve. The hair cells are tuned along the length of the basilar membrane, and those hair cells that are located at the basal part of the cochlea respond to high-pitched sounds while those at the apex respond to low-pitched sounds. The auditory nerve carries electrical impulses to the brainstem.

In summary, the pinna and external ear canal collect sound waves, which then cause vibrations of the tympanic membrane. These vibrations are transmitted through the ossicles to the vestibular window. These vibrations are then transferred from the vestibular window to the perilymph of the scala vestibuli. Within the cochlear duct the sound waves propagated along the scala vestibuli produce bending of the hair cell stereocilia of the organ of Corti, depolarizing the cells. From there, synaptic connections between hair cells and spiral ganglion neurons result in transmission of information through the auditory nerve.

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Predisposing factors facilitate the inflammation by permitting the external ear canal microenvironment to be altered allowing pathogenic or opportunistic bacteria to become established.

**Ear Canal Conformation**

The conformation of the ear canal may predispose an animal to otitis externa. Small, stenotic ear canals, seen in breeds such as the Chinese Shar-Pei, allow otic secretions to build up in the external ear canal and inhibit proper cleaning of the ear. The excess secretions provide a medium for growth of pathogenic bacteria. Treatment should be aimed at performing maintenance ear cleaning with a cleaning/drying agent. Hair removal should be performed in dogs prone to otitis externa; however, routine hair removal in dogs lacking ear disease is not recommended. Frequent hair plucking may lead to follicular trauma and inflammation of the canal.

Long, pendulous pinna may restrict free airflow within the ear canal. Dog breeds with pendulous ears were shown to be at higher risk for developing otitis externa. Originally, the difference was thought to be due to variations in temperature or humidity; however, none were found. It may be more a factor of the breed (cocker spaniel, poodle) of dog rather than the conformation of the pinna or whether there is excessive hair in the ear canal. Although breeds with pendulous pinna may be over-represented in the breeds presented with otitis externa, not all breeds with pendulous pinna are affected and some breeds with erect ears (German Shepherd dog) appear to be prone to otitis externa.

**Climatic Variations**

Chronic abnormally high moisture content of the ear canal can lead to maceration of the stratum corneum and impairment of barrier function, predisposing to microbial colonization and infection. The most common cause of a moist external ear canal is frequent swimming or bathing. “Swimmer’s ear” is managed with the use of cleaning/drying agents applied after swimming.

**Treatment Errors**

Inappropriate treatment and treatment errors may predispose an animal to otitis externa. Mechanical trauma of the canal during cleaning with cotton swabs may lead to swelling and erosions of the epithelium, predisposing to secondary microbial infections.
In addition, the use of cotton swabs for ear cleaning causes maceration and subsequent ulceration of the ear canal, and they push exudate further down the ear canal, which may result in rupture of the tympanic membrane. Instead of using a cotton swab for cleaning of the ear, otic cleaners are used to soften the exudate and allow its removal.

Irritant antiseptic solutions can cause maceration of the tissue of the ear canal. This is especially true of “home-made” cleaners that contain isopropyl alcohol and hydrogen peroxide.

Improper antibiotic usage, which results in the destruction of the normal microflora, may lead to opportunistic infections. Combination products are indicated for treatment of acute otitis externa, for short-term use in the ear with a mixed infection and inflammation. However, the continued use of these combination products may result in the development of resistant gram-negative infections. Treatment should be as specific as possible, based on cytologic and/or culture results. Continual use of a combination product should be avoided.

**Primary Causes of Otitis Externa**

Primary causes of otitis externa are conditions or disorders that initiate the inflammatory process within the ear canal. The epithelium of the external ear canal is an extension of the rest of the skin on the body; thus most cases of otitis externa are associated with an underlying dermatologic condition.

**Otodectes cynotis**

*Otodectes cynotis* is the most common parasite causing 50% of the otitis externa cases in cats and 5 to 10% in dogs. This figure is probably underestimated for dogs since it is easy to miss small numbers of mites in chronically inflamed ears. The mite most commonly affects animals less than one year of age. The mite causes direct irritation of the ceruminous glands and results in a hypersensitivity reaction to the mites’ salivary antigens. The life cycle is 3 to 4 weeks and is completed entirely on the host. The mites appear to irritate the ceruminous glands resulting in a thick, dark brown, ceruminous exudate that provides favorable growth medium for *Malassezia pachydermatis*. Ear mites are most commonly found in the external ear canal, but may survive on the skin surface. Secondary bacterial infections are commonly seen. Diagnosis is made by otoscopic examination or microscopic examination of the exudate. Treatment consists of removal of the ceruminous exudate with a ceruminolytic agent and specific topical or systemic miticidal therapy.

**Other Parasites**

*Demodex canis* in the dog and *Demodex cati* in the cat rarely causes ceruminous otitis externa without generalized skin disease. Treatment usually requires topical or systemic therapy.
**Foreign Bodies**

Foreign bodies may be a primary cause of otitis externa. Plant material (foxtails), dirt, sand, dried medicaments, cross-lodged hairs may cause an acute, unilateral, painful otitis externa. Diagnosis is made by performing a complete otic examination. Primary danger of foreign bodies is perforation of the tympanic membrane with development of otitis media. Removal of the foreign body usually requires sedation or general anesthesia.

**Hypersensitivity**

Allergic diseases are the most common causes of persistent bilateral otitis externa in the dog. Atopic dermatitis causes a bilateral pruritic otitis externa in at least 50% of cases. Otitis externa may be the only clinical sign in 10% of atopic dogs. The otitis is usually manifested by head shaking and ear scratching. In the early stages of the disease pinnal lesions will be present but the ear canals will appear normal or only slightly inflamed. Later a more severe atopic otitis externa develops with secondary bacterial or yeast infections. Although atopic otitis externa is usually present bilaterally, one ear may appear worse if the infection is localized to that side. In dogs with atopic dermatitis only affecting the ears, cleaning/drying agents followed by topical glucocorticoids may be all that is necessary to control the inflammation. However, if this fails or the dog has generalized pruritus, then treatment options include antihistamines, fatty acids, systemic glucocorticoids, immunotherapy, and/or cyclosporine.

Cutaneous adverse food reaction (CAFR) causes a bilateral pruritic otitis externa in up to 88% of cases. Otitis may be the only clinical sign in 25% in dogs with CAFR. Animals present with similar history and clinical signs to those seen with atopic dermatitis. CAFR should be considered a top differential for otitis externa in a young dog (< 1 year of age) and older dogs without a previous history of otic or skin problems. Intense pruritus limited to the face and head with ear involvement is a common clinical presentation in the cat and is seen occasionally in the dog. Diagnosis involves feeding of a novel protein diet or hydrolyzed diet for at least 8 weeks. Management consists of dietary restriction.

Contact allergy is a rare cause of dermatitis and otitis in small animals. It may affect the non-haired portion on the inside of the pinna and the external ear canal along with other non-haired contact areas of the body. Contact allergic otitis externa is usually iatrogenic. Topical medications such as neomycin or propylene glycol may result in sensitization and a subsequent contact allergic otitis. This should be suspected when a case of otitis fails to respond or worsens after appropriate topical otic medication is administered. Treatment is discontinuation of the topical otic product that induced the reaction and utilizing otic products that do not contain the same ingredients as the offending otic product.
Juvenile Cellulitis

Juvenile cellulitis is a vesiculopustular disease of puppies between 3 and 16 weeks of age. Etiology is unknown, but may be related to a hypersensitivity reaction or viral disease. Dachshunds, Golden Retrievers, and Pointers are over-represented. These animals present with head and facial lesions, including purulent otitis, blepharitis, and regional lymphadenopathy. Diagnosis is made based on rule out of other diseases that mimic this disease and confirmed via biopsy. Immunosuppressive doses of glucocorticoids are used to manage this disease.

Disorders of Keratinization

Disorders of keratinization produce a primary ceruminous otitis externa. Primary idiopathic seborrhea and hypothyroidism may be associated with a ceruminous otitis externa. Ceruminolytic agents are needed to control the excessive cerumen production. In some cases, topical glucocorticoids may be needed to control otic inflammation, as well as topical or systemic antimicrobial agents to control secondary infections.

Autoimmune Diseases

Autoimmune diseases may be associated with pinnal disease that may extend into the external ear canal. Pemphigus foliaceus and discoid lupus erythematosus are the two most common immune-mediated diseases in the dog. Other cutaneous lesions are usually present on physical examination. Autoimmune diseases should be considered whenever primary lesions are present on the inner pinna and within the external ear canal. Treatment and prognosis are dependant on the specific autoimmune disease.

Ear Tumors and Polyps

Ear tumors and polyps cause obstruction of the external ear canal, preventing the removal of normal secretions. All masses should be biopsied for diagnosis. The most common tumor of the ear canal is a ceruminal gland neoplasm. These tumors are more common in dogs than in cats but the feline otic tumors have a greater tendency to be malignant.

Feline nasopharyngeal polyps may originate from the pharyngeal mucosa, the Eustachian tube, and the middle ear. Their etiology is unknown, but may be congenital, viral, or bacterial in origin. They result in a purulent or ceruminous otitis externa with possible dysphagia and sinusitis.
PERPETUATING CAUSES OF EAR DISEASE
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In dogs with chronic otitis externa, one or more perpetuating factor will be present. These perpetuating factors include progressive pathologic changes, otitis media, and bacterial and yeast infections. These factors sustain and aggravate the inflammatory process and prevent the resolution or worsen an already present otitis externa.

Progressive Pathological Changes
Once the acute inflammatory stage has been initiated by any one of the primary causes, there appears to be a common course for the development of chronic otitis externa, leading to progressive, pathologic changes of the ear canal. During the acute stage, the ear canal becomes edematous and erythematous. Since the canal is surrounded by cartilage, this swelling causes constriction of the canal lumen resulting in pain from nerve entrapment. As the inflammation progresses, the dermis becomes infiltrated with a mixed population of lymphocytes, polymorphonuclear cells, and mast cells. As the otitis progresses, in some dog breeds, apocrine glands dilate and become hyperplastic, leading to excessive cerumen production.

Continued inflammation can lead to permanent microanatomical changes of the ear canal. Calcification of the auditory cartilage and fibrosis of the dermis and subcutis lead to additional thickening of the ear canal. These changes can lead to total occlusion of the ear canal which may impede or reverse normal epithelial migration. This may result in the migration of stratified epithelium into the middle ear causing the formation of keratin plugs. Another possible consequence of canal occlusion is the formation of an aural cholesteatoma. An aural cholesteatoma forms from a pocket of tympanic membrane that bulges into the middle ear and subsequently fills with keratin produced by the stratified epithelium of the tympanic membrane. When this pocket completely fills the middle ear cavity, it is referred to as a false middle ear.

Middle Ear Anatomy
The middle ear begins at the tympanic membrane, which separates the horizontal ear canal from the middle ear. The tympanic membrane is semitransparent and is thin in the center and thicker at the periphery. The normal tympanic membrane is concave. It is divided into two sections, the small upper pars flaccida and the larger lower pars tensa. The pars flaccida is the pink, small, loosely attached region forming the upper quadrant of the tympanic membrane, which contains small blood vessels. The pars tensa occupies the remainder of the membrane. It is a thin, tough, pearl gray structure with radiating strands. The pars tensa is attached firmly to the surrounding bone by a fibrocartilaginous ring. This fibrocartilaginous ring, known as the annulus
fibrocartilaginous, is attached to the osseous ring of the external acoustic meatus by fibrous tissue. The manubrium of the malleus attaches to the medial surface of the tympanic membrane. The outline of the manubrium of the malleus, the stria mallearis, may be visualized when the tympanic membrane is viewed externally.

The tympanic cavity comprises the majority of the middle ear and is divided into three portions. The epitympanic recess is the smallest of the three areas. It is occupied by the head of the malleus and the incus at their articulation. The three auditory ossicles, the malleus, incus, and stapes, are the bones that transmit and amplify air vibrations from the tympanic membrane to the inner ear. The footplate (base) of the stapes is attached to the vestibular (oval) window, which is in direct contact with the perilymph fluid. The tympanic cavity proper is adjacent to the tympanic membrane. On the medial wall of the tympanic cavity proper, there is a bony eminence, the promontory, which houses the cochlea, and lies opposite the tympanic membrane. The cochlear (round) window is located on the caudolateral portion of the promontory. The auditory tube (Eustachian tube) connects the nasopharynx to the rostral portion of the tympanic cavity proper. The largest region of the tympanic cavity is the ventral portion, which is contained within the tympanic bulla. Other important structures adjacent to the middle ear include the sympathetic nerve, facial nerve, vagus nerve, and the carotid and lingual arteries.

**Otitis Media**

Otitis media is an important perpetuating cause of recurrent otitis externa. Infectious otitis media occurs as a direct extension from an existing otitis externa through a ruptured tympanic membrane. Less common routes of infection include extension through the Eustachian tube or hematogenous dissemination. The reported incidence of infectious otitis media in dogs with acute otitis externa is 16%, while in dogs with chronic, recurrent otitis externa; the reported incidence is 50-88.9%. In one study evaluating dogs with chronic otitis externa, 80 (88.9%) ears had concurrent otitis media and the tympanic membrane was intact in 73% of the ears. Therefore, an intact tympanic membrane does not rule out otitis media.

**Bacteria/Yeast**

Bacterial and yeast infections are another perpetuating factor and are considered secondary to a primary cause. The most commonly isolated organisms from the horizontal ear canal in dogs with chronic otitis externa are *Staphylococcus pseudintermedius*, yeast, and *Pseudomonas* spp. Similar pathogens are isolated from the middle ears of dogs with concurrent otitis media.

**Noninfectious Causes of Otitis Media**

There are noninfectious causes of otitis media. Radiographic signs of middle ear disease may be associated with congenital palatine defects (secondary cleft palate). Neoplasia and polyps may result in otitis media. Additional non-infectious causes of otitis media include trauma, foreign bodies and primary secretory otitis media (PSOM).
Clinical Signs of Otitis Media

Usually the only clinical sign of infectious otitis media are those seen with otitis externa. These include discharge from the external ear canal, pawing or rubbing the affected ear, head shaking, and pain. Neurological signs of otitis media include facial nerve paralysis and Horner’s syndrome. Otitis media may lead to otitis interna. Clinical signs of otitis interna include horizontal nystagmus, asymmetric ataxia; and head tilt, circling, falling, or rolling toward the affected side.

Management of Otitis Media

Management of chronic, recurrent otitis externa requires diagnosis and control of the predisposing factors, primary causes, and perpetuating factors. In addition, in-hospital cleaning the ear canals and middle ear, topical glucocorticoids, topical antimicrobials, systemic glucocorticoids, and/or systemic antimicrobials may be necessary. Since permanent changes in the microanatomy of the ear canal are present, life-long ear cleaning and drying agents may be required to prevent relapse.

In some cases of chronic, recurrent otitis externa, surgical intervention may be warranted. Surgery is indicated when there is inadequate response of the external ear infection to medical management (oral antimicrobials, oral glucocorticoids, topical antimicrobials, topical glucocorticoids) due to poor owner compliance or the presence of a resistant organism, otitis media fails to respond to myringotomy, ear canal flushing, and medical management; or progressive pathologic changes of the ear (calcification, proliferation, and stenosis) have resulted in permanent occlusion of the ear canal.
OTIC CYTOLOGY
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Otic cytology is an important diagnostic procedure used for determining what, if any, infective organisms are present in the ear, as well as the types of inflammatory cells, in animals presented with the complaint of otitis externa.

Utility of cytology
Unfortunately, there is no reliable correlation between the nature of otic discharge and a specific organism. In some instances, the exudate is only wax. Therefore, it is important to sample the exudate in the ear for cytologic evaluation in all animals suspected of otitis externa. The technique is quick and simple, and allows one to begin treatment immediately based on the results obtained. It is important to perform cytologic evaluation of otic exudate at the initial examination as well as each recheck, in order to monitor the response to the therapy. In addition, cytology is the best diagnostic technique for identification of yeast organisms.

Sampling collection and preparation
After the ears have been examined otoscopically, exudate from the horizontal ear canal is collected by inserting a cotton-tipped applicator into the canal. Samples should be collected from both ear canals. The swab samples are rolled onto a glass microscope slide. If a frosted edge slide is used, roll the sample from the left ear next to the frosted edge, and the sample from the right ear on the opposite side of the slide. By doing this, only one slide is required for both ears. The next step is to heat fix the slide. Performing this step will prevent a waxy sample from being washed away during staining. The sample is now ready to be stained. The most commonly used stains are Romanowsky-type stains (Wright’s stain, Giemsa stain, Diff-Quik), since they are very rapid and easy to perform, compared to the more cumbersome and time-consuming Gram stain. The disadvantage of these stains is that all bacteria stain blue and the gram-staining properties cannot be determined. After staining, the slides should be rinsed with cool water and allowed to dry. Drying may be expedited with the use of a handheld dryer.

Viewing and evaluation
Once the slide is dry, the cytologic smear should be viewed beginning with a low power magnification and then advancing to a higher magnification. The slide is first scanned with the low power lens (10x objective; 100x magnification) to find keratinized squamous epithelial cells or inflammatory cells. This is where infective organisms would be if they are present. Once these are located, I will go directly to oil immersion (100x objective; 1000x magnification) to evaluate the cells and any infective organisms. The
smears should be evaluated for the number and morphology of bacteria, the number of yeast, the number and type of leukocyte, the presence of excessive cerumen, the presence of excessive keratinized squamous epithelial cells, and the presence of neoplastic cells. I will routinely evaluate 10 fields under oil immersion and estimate the number of organisms and leukocytes per those 10 fields. If the mean number of bacterial or yeast organisms is \( \geq 4 \), then the organisms should be considered pathogens. For consistency, a grading system of the numbers of organisms and leukocytes counted should be developed.

**Cytologic appearance of the of the normal ear canal compared to the otitic ear**

Cerumen coats the external ear canal and is composed of secretions from the sebaceous glands, ceruminous glands as well as sloughed keratinized squamous epithelial cells. Cerumen is clear and usually does not stain due to its high lipid content. Ears that are inflamed will have a lower lipid content resulting cytologically in a stained smear that is bluer than a smear from a normal ear. Keratinized squamous epithelial cells are usually anucleate and stain blue/purple.

A recent study evaluated numbers of inflammatory cells, epithelial cells, bacteria and yeast organisms cytologically from ear swab samples from normal dogs and cats and compared these to the results obtained from dogs and cats with otitis externa. The slides were scanned at low power (100x) to locate inflammatory cells or keratinized squamous epithelial cells where infectious organisms were expected to be found. Ten fields were counted under high-power field (400x) and the mean number of cells and/or organisms per sample was calculated. Inflammatory cells were absent in all the samples from the normal ears. Inflammatory cells were abundant in affected ears with purulent otitis but were absent in 36% of the samples obtained from affected ears with ceruminous exudate. It is interesting to note that the authors did not find any significant difference in the mean numbers of keratinized squamous epithelial cells between the normal and affected ears. It is believed that in cases of chronic otitis externa and diseases with increased epithelial turnover rate (keratinization disorders) or atopic dermatitis, there would be increased numbers of keratinized squamous epithelial cells. One explanation is that the selection of the fields for counting was based on finding keratinized squamous epithelial cells, which may have resulted in a higher density of these cells from the dogs with normal ears.

The authors next evaluated yeast and bacterial organisms from the swab samples of the normal and affected dogs and cats. *Malassezia pachydermatis* is identified morphologically as a broad-based, budding yeast, typically “peanut-shaped”. Bacterial organisms are identified morphologically as cocci and rods. Ten selected fields were counted under high-power field (400x) and the mean number of organisms per sample was calculated. In dogs and cats with clinical signs of otitis externa, the mean number of *Malassezia* organisms was significantly higher than from the dogs and cats with normal ears. Only coccoid bacteria were identified in swab samples from normal ears. In the dogs and cats with clinical signs of otitis externa, the mean counts of bacteria were significantly higher than from the dogs and cats with normal ears. According to the results of this study, mean counts of yeast and bacterial organisms per
high-power dry field (400x) from otic swabs in the dog of ≥ 5 and ≥ 25, respectively, are to be considered abnormally increased, while mean counts of yeast and bacterial organisms per high-power dry field (400x) from otic swabs in the cat of ≥ 12 and ≥ 15, respectively, are to be considered abnormally increased.

It would be unusual to find cytologic evidence of neoplasia in the otic exudate from an ear with a mass. Cytology would be used to identify any secondary infectious organisms or inflammatory cells.

Cytologic appearance of artifacts
Artifacts may be present cytologically and be mistaken for infectious organisms. One such artifact is melanin granules. Melanin granules may be present in the keratinized squamous epithelial cells and should not be confused with rod bacteria. The melanin granules will stain brown, while rod bacteria will stain blue/black.

Precipitates on the slide stain blue/black and may be mistaken for coccoid bacteria. They differ from bacteria, since they form large, indistinguishable clumps while coccoid bacteria are very round and uniform in size. This artifact is due to inadequate stain filtration. This can be avoided by periodically filtering the stain and replenishing the buffer solution.

Treatment based on cytologic evaluation
Treatment should begin based on the results obtained cytologically. If there is only cerumen and keratinized squamous epithelial cells present, an ear cleaning solution should be dispensed. If infectious organisms are identified, treatment for the specific organism(s) should be started. If rod bacteria are found cytologically, a culture and susceptibility should be performed to determine the type of bacteria present. The most common rod bacteria from chronically infected ears is *Pseudomonas aeruginosa* which can be difficult to treat, since it may be resistant to numerous antibiotics. While awaiting culture results, assume the infection to be *P. aeruginosa*, and begin treatment accordingly. In addition, primary underlying diseases such as primary idiopathic seborrhea, atopic dermatitis, food allergy, endocrinopathies, and otodectes should be considered in cases of chronic, recurrent otitis externa.
DIAGNOSTIC PROCEDURES AND TESTS FOR EAR DISEASE
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The diagnosis of otitis is based on history, physical examination, and otic diagnostics. Tests used for the diagnosis of otitis externa and otitis media include video otoscopy, cytology, and bacterial culture and susceptibility testing (C/S), while radiography, pneumotoscopy, impedance audiometry, positive contrast canalography, endoscopy, and myringotomy are used for the diagnosis of otitis media. Information obtained in the history includes the duration of the otitis, the age of onset, frequency of recurrence, and previous treatments and response. A complete physical examination is performed including evaluation of the oral cavity and nasopharyngeal region. A dermatological examination is important to determine the underlying primary disease. A neurological examination is performed to evaluate for signs suggestive of otitis media/interna, which include facial nerve paralysis, nystagmus, keratoconjunctivitis sicca (KCS), vestibular disease and deafness. The pinnae are examined for alopecia, excoriation, and crusts. After the history has been obtained and the physical examination is completed, an otic examination is performed with a hand-held otoscope or video otoscope. An otic examination of the ear canal allows visual assessment of the vertical ear canal, horizontal ear canal, and tympanic membrane. In some cases, examination of the external ear canal and tympanic membrane may require sedation or general anesthesia. Furthermore, a complete otic examination at the first visit may not be possible if the ear canal is stenotic, hyperplastic, edematous, or ulcerated with secondary hemorrhage. In addition, ears with significant amounts of debris and exudate may need to be flushed prior to examination. Therefore, prior to ear flushing or examination, if the ears are ulcerated, hyperplastic or stenotic, 2 to 3 weeks of topical and/or systemic glucocorticoids are needed.

Video Otoscopy
The use of video otoscopy has grown rapidly in small animal veterinary medicine. Video otoscopy is performed using an otoendoscope, camera, light source, and monitor. A video otoscope is used to evaluate patients’ ears when awake, sedated, or under general anesthesia. In severely stenotic ear canals, a complete examination may not be possible. When the video otoscope is used in the examination room, the client is able to visualize the ear and participate in the diagnosis of the ear disease. The vertical and horizontal ear canal and tympanic membrane are brightly illuminated and magnified, allowing greater visualization of these structures. While the patient is sedated or anesthetized, utilizing the opening on the otoendoscope, the ears can be flushed; foreign objects, debris, or parasites may be retrieved with the grasping forceps, biopsies obtained with biopsy forceps, and myringotomy performed with a catheter. With an attachable dual-port adapter, suction and saline may be used simultaneously to
completely clean the ear. Another option for visualization of the ear canal and tympanic membrane in the anesthetized patient is endoscopy utilizing a rigid endoscope.

**Otic Cytology**

The most underused but one of the most important otic diagnostic tests is cytology. Cytology is a rapid and inexpensive procedure indicated in all cases of otitis. It is the best method for detection of yeast organisms. It should be performed at the initial examination and on all subsequent reevaluations. After collecting the sample from the ear canal, the swab is rolled onto a glass slide, heat fixed and stained with a modified Wright’s stain (Diff-Quik) or gram stain. Under scanning power (100X), keratinocytes or inflammatory cells should be located. Once those are found, immersion oil is applied to the slide and the slide is examined under oil immersion power (1000X). The number and type of bacteria, yeast, and inflammatory cells should be counted and recorded.

**Bacterial Culture and Susceptibility Testing (C/S)**

Bacterial C/S of otic exudates is not routinely performed at each visit. It is reserved for chronic recurrent or unresponsive cases of otitis externa or when cytology reveals numerous rod bacteria. In addition, if otitis media is suspected, then swabs for bacterial C/S should be performed from both the horizontal ear canal and middle ear while the patient is under anesthesia.

**Additional Diagnostic Tests**

The epithelium of the external ear canal is an extension of the rest of the skin on the body; thus most cases of otitis externa are associated with an underlying dermatologic condition. Additional diagnostic testing may include skin scrapings, thyroid evaluation, allergy testing (intradermal testing and/or serum allergy testing for inhaled allergens; food elimination trials), ear canal biopsy, hemogram, and serum biochemistry profile. The decision to perform these tests will depend on the findings on the physical examination.

**Radiographic Imaging**

Plain radiographs are performed to visualize the bony integrity of the tympanic bulla and soft tissue changes in the external auditory canal, middle ear, and inner ear. They are also used as a prognostic indicator for the success of medical management of otitis media. With significant radiographic changes, such as sclerosis or lysis, surgical intervention may be required for resolution. The radiographic views include the dorsoventral, rostroventral-caudodorsal open mouth (best view for evaluation of the tympanic bulla), and the right and left lateral obliques. The overall accuracy of computed tomography (CT) and radiographs for the diagnosis of otitis media are similar, however, CT appears to be a more sensitive indicator of otitis media. With CT, it is
important to use an extended viewing scale with a window of 2000 or 4000 CT numbers with a low slice thickness to reduce the possibility of increased bulla wall thickness when the tympanic bulla is fluid filled. Otherwise this artifact may be interpreted incorrectly as bulla osteitis. Radiographic abnormalities on CT or plain radiographs suggestive of otitis media/interna include thickening of the bulla, lysis of the bulla or petrous temporal bone, opacity in the bulla, or sclerosis and proliferation of the petrous temporal bone. Magnetic resonance imaging (MRI) has also been used to assess the middle ear. Material in the affected bulla appears isointense compared to the cerebral cortex on T1-weighted images and hyperintense on T2-weighted images. For CT, MRI and plain radiographs, normal findings do not rule out otitis media.

Positive Contrast Canalography

Positive contrast canalography evaluates the patency of the tympanic membrane by infusing positive contrast medium into the ear canals of anaesthetized dogs. With the dog in lateral recumbency, the non-dependent ear canal is filled with radiographic contrast medium (iohexol or Urografin). The contrast medium is diluted with an equal volume of sterile saline prior to filling the ear canal. The ear canal is massaged along the full extent of the vertical and horizontal canals to aid the distribution of the medium and a cotton ball is used to plug the vertical canal. Pre- and post- contrast bullae radiographs are performed. In one study, this technique detected 14% of ears with a ruptured tympanic membrane that were identified otoscopically to be intact. False-negative results occurred since contrast did not enter the bulla in 42% of the ears which demonstrated tympanic membrane rupture otoscopically. This may have been due either to the inaccuracy of otoscopy or to the passage of contrast being impeded by inflammatory tissue within the bulla. A subsequent study performed canalography on 222 ear canals in 111 dogs. This procedure identified the tympanic membrane in 200 (90%) ears. Due to the severe stenosis in 22 (10%) ears (18 canals in pugs and 4 in Pekingese), the tympanic membrane could not be identified. Three ears had contrast medium detected in the tympanic bulla, which had been identified otoscopically to have intact tympanic membranes. This procedure may aid in the identification of tympanic membrane rupture, however, lack of contrast medium in the tympanic bulla does not rule out a ruptured tympanic membrane.

Bulla Ultrasonography

Recently, a procedure for imaging the canine tympanic bulla, external ear canal, and adjacent structures using ultrasound equipment has been described. The sonographic appearance of these structures in cadavers and live dogs with no evidence of otitis was documented. Water was introduced into the tympanic bullae of the cadavers and the presence of the fluid in the tympanic bullae could be differentiated from gas with the ultrasound equipment. The fluid-filled tympanic bulla was visible as an oval shaped, anechoic region while the gas filled tympanic bulla created a reverberation artifact causing an acoustic shadow obscuring deeper areas. In another
study, ultrasonography was compared to radiography for the detection of fluid in the tympanic bullae. The tympanic bullae were filled with either air or sterile saline prior to the ultrasonographic and radiographic study. Ultrasonography was more sensitive and specific than radiography for the detection of fluid in the tympanic bullae of the cadavers.

**Brain-stem Auditory Evoked Response**

There are two general types of deafness recognized in the dog: conductive and sensorineural. Conductive hearing loss occurs when the transmission or transduction of a sound is compromised in the external ear canal or middle ear. Conductive hearing loss can occur when the external ear canal is occluded by exudates in the ear canal or stenosis and hyperplasia of the ear canal, when the tympanic membrane is ruptured, when the tympanic membrane becomes stiff, if the ossicular chain is broken or becomes stiff or if there is fluid, exudates, or mucus in the middle ear. Sensorineural hearing loss occurs when the physics or hydrodynamics of the inner ear are altered or when there is an abnormality of the receptor cells of the cochlea or any part of the auditory pathway. Causes of sensorineural deafness include hereditary deafness, congenital deafness, cochlear degeneration due to viruses or ototoxic drugs, and neural degeneration due to senility.

Hearing in animals can be evaluated using electrodiagnostic procedures that assess the integrity of the peripheral and central auditory components. These tests are noninvasive and do not require conscious cooperation of the animal. One such electrodiagnostic procedure is the brainstem auditory evoked response (BAER). The brainstem auditory evoked response can be defined as a far-field recording of the neuroelectrical activity of the auditory nerve (part of CN VIII) and brainstem auditory pathways that occur over the first 10 milliseconds (ms) after a sound stimulus has been delivered to the ear. The BAER is unaffected by level of arousal or a wide range of pharmacologic agents, including general anesthesia. The intensity of the click is most commonly calibrated in decibels.

To record the BAER, the animal is placed in sternal recumbency with the head slightly elevated. Tubal inserts are positioned in the ear canals so that the tips are not occluded. Subdermal needle electrodes are placed at the vertex of the head (non-inverting electrode) and just rostral to the base of each ear with the inverting electrode inserted at the base of the test ear, and the ground or reference electrode inserted at the base of the contralateral ear. The overall morphologic features of the canine BAER are similar to other animals and humans. In mature, clinically normal dogs, the BAER is characterized by six to seven vertex-positive waves, which are labeled with Roman numerals.

The actual neural generators of the BAER are only partially known at this time. Wave I of the BAER is generated by the auditory (cochlear) nerve activity. Wave II appears to be generated by the ipsilateral cochlear nucleus and the unmyelinated central terminals of the cochlear nerve. The generator of wave III is thought to be the superior olivary complex of the ipsilateral and/ or contralateral brain stem. The
generators of wave V are thought to be the ipsilateral and/or contralateral inferior colliculus. Generators of waves IV, VI and VII have not been clearly defined. The BAER has been used as a site-of-lesion tool for evaluating conductive hearing loss, sensory lesions in the inner ear, neural lesions of the auditory nerve and brain stem, estimating hearing threshold, and as a screening tool for inherited/pigment-associated deafness.
TREATMENT OF OTITIS EXTERNA AND OTITIS MEDIA
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Once an otic infection has been diagnosed, treatment may include topical as well as systemic therapy. In the majority of the cases of infectious otitis externa, topical therapy alone is sufficient. In those ears with severe infections, or those that have long-standing chronic otitis externa, the addition of a systemic antimicrobial agent may be required, to clear the infection that is present in the ear tissue as well as in the lumen of the ear canal. For those dogs with infectious otitis externa and otitis media, topical and systemic therapy is usually required.

Topical Otic Therapy

Topical therapies may be divided into the following categories: ceruminolytic agents, ear cleaning and drying agents, topical glucocorticoids, and topical antimicrobial agents. It is important to stock your veterinary clinic with at least one product from each category, since they have different indications and uses.

Ceruminolytic agents
Ceruminolytic agents are normally used for in-hospital ear cleaning, but on rare occasion can be dispensed for at-home ear cleaning. They work by surfactant, detergent or bubbling activity. Water miscible preparations may contain dioctyl sodium sulfosuccinate (DSS) or propylene glycol. Oils, such as squalene or mineral oil are effective, however messy, ceruminolytic agents. Products containing urea peroxide in addition to DSS are very potent and recommended for in-hospital usage only. Most of these products are contraindicated with a ruptured tympanic membrane, since many are ototoxic. Ceruminolytics that contain DSS, carbamate peroxide, and triethanolamine have been shown to be ototoxic when infused into the tympanic bulla of dogs, while squalene appears to be safe.

Ear cleaning/drying agents
Once the ear has been cleaned in-hospital, or for routine ear cleaning at home, it is important to dispense a cleaning and drying agent. Most contain an acid with or without alcohol. They are used to clean the ear, as well as keep it dry to discourage the over growth of bacterial and yeast organisms. The frequency of their use depends on the chronicity of the otitis externa and the severity of the infection. The goal is to begin treatment with the most frequent application (i.e. daily) and over time, decrease to a maintenance frequency (i.e. weekly). In some cases, daily treatment with an ear cleaning and drying agent may be effective for resolution of bacterial and Malassezia
Otic infections.

**Topical antiseptics**

Topical antiseptics such as acetic acid and chlorhexidine have been used in cases of otitis. Acetic acid works by acidification and other mechanisms as an antimicrobial agent. A 2% solution is effective *in vitro* against *Pseudomonas*.

Chlorhexidine has broad spectrum activity against many gram-positive and gram-negative bacteria and fungi; however, *Pseudomonas* may be resistant. Chlorhexidine is ototoxic in humans and therefore should be used with caution in animals ears with ruptured tympanic membranes. However, a study done in normal greyhounds with experimentally ruptured tympanic membranes treated twice a day for 21 days with a topical application of 0.2% chlorhexidine failed to induce any clinical vestibular or brainstem auditory evoked potential changes.

**Indication for specific topical therapy**

In most cases of otitis, specific topical therapy is indicated. The topical otic preparations usually contain various combinations of glucocorticoids, antibiotics, and/or antifungals in a vehicle base. For dry lesions, an oil or ointment base product is used to help moisturize the skin of the ear, while in moist lesions a solution or lotion is recommended. Selection of the active ingredient needed in the product should be based on cytology and culture and susceptibility (C/S) results. It is important to remember that C/S results indicate the plasma level of an antimicrobial agent. The advantage of topical therapy is that you can achieve 100 to 1000 times the plasma level of the antimicrobial agent by administering it topically. The patient’s progress while on these medications should be monitored cytologically at each re-evaluation and the topical therapy adjusted accordingly.

None of the commercially available otic topical treatments or the extra-label otic preparations are labeled for use with a non-intact tympanic membrane. However, most all of these products have been used to treat otic infections in dogs with otitis media. Always warn the owner of the possibility of neurological signs of ototoxicity while administering topical medications when the tympanic membrane is not intact. The otic topicals that I will not use in the ear with a non-intact tympanic membrane are those in an ointment base.

**Glucocorticoids**

Glucocorticoids are antipruritic, anti-inflammatory, and antiproliferative. During the acute stage of otitis, the ear canal becomes edematous and erythematous. As the inflammation progresses, the dermis becomes infiltrated with a mixed population of cells. Apocrine glands dilate and become hyperplastic, which leads to excessive cerumen production. Therefore, glucocorticoids are beneficial in decreasing the pain, pruritus, stenosis, and edema associated with otitis. In addition, they are effective in decreasing sebaceous and apocrine secretions. They are usually in combination with other agents but may be beneficial when used alone in allergic cases of otitis and some ceruminous otitis cases. It is important to use the lowest potency glucocorticoid at the
lowest frequency needed to control the otitis to prevent iatrogenic hyperadrenocorticism.

**Topical antibiotic agents**
Topical aminoglycosides such as neomycin and gentamicin have good activity against gram-positive and gram-negative otic pathogens. Topical aminoglycosides are ototoxic in humans, and therefore should be used with caution in animals ears with ruptured tympanic membranes. However, a study done in normal greyhounds with experimentally ruptured tympanic membranes treated twice a day for 21 days with a topical application of gentamicin sulfate (7 drops of 3 mg/ml) in buffered aqueous solution failed to induce clinical vestibular or brainstem auditory evoked potential changes. Gentamicin and neomycin are available in many combination products, some which contain an antifungal and glucocorticoid.

Polymyxin has excellent *in vitro* activity against *Pseudomonas*. Polymyxin is inactivated in purulent debris so the ear needs to be kept clean during treatment.

**Topical antifungal agents**
Antifungal agents are used in cases of otitis caused by *Malassezia* or *Candida*. Ingredients that are active against yeast include nystatin, miconazole, and clotrimazole.

**Systemic Otic Therapy**

**Systemic antibiotics**
Systemic antimicrobial therapy for infectious otitis externa and otitis media is controversial. In dogs with end-stage otitis externa and concurrent otitis media, bacterial organisms may be isolated from the exudate in the lumen of the vertical ear canal and middle ear cavity as well as from the tissue from these sites. Therefore, most agree that systemic antibiotics (based on culture and susceptibility testing) are indicated in patients with otitis media, patients with severe proliferative chronic otitis externa, patients with ulcerative otitis externa, patients where inflammatory cells are seen cytologically (indicating deeper skin involvement) and in patients where owners cannot administer topical therapy. The selection of systemic antimicrobial agent must be made based on C/S from the external ear (for otitis externa) and middle ear (for otitis media). However, therapy may be initiated based on cytologic results while awaiting the C/S.

**Systemic antifungals**
Indications for systemic antifungal agents are similar to those above for bacterial infections and include patients with yeast otitis media, patients with severe yeast otitis externa, or in patients where owners cannot administer topical therapy. Neither pulse-dose or daily-dose itraconazole alone significantly decreased yeast organisms identified cytologically from otic exudate in dogs with yeast otitis externa, suggesting that otic yeast infections may require topical therapy in addition to systemic therapy for resolution. Both ketoconazole (5 mg/kg q 24 hr) and itraconazole (Sporanox 5 mg/kg PO q 24 hr or pulse-dosed 2 days on and 5 days off) have been used in dogs.
Systemic glucocorticoids

Systemic glucocorticoids are used to decrease stenosis, edema, and hyperplasia of the vertical and horizontal ear canal to allow a complete otic examination as well as allow proper cleaning of the ear. They are also indicated in cases of allergic otitis externa. Initially, prednisolone at 0.5-1 mg/kg SID orally may be needed, followed by a low-dose, alternate day dosing schedule. As with topical glucocorticoids, the lowest dose needed should be administered to prevent the occurrence of side effects. In older patients, or those with concurrent diseases, it may be necessary to perform bloodwork prior to using glucocorticoids.

Overtreatment

In some instances, too vigorous topical therapy may result in maceration of the lining of the ear canal. Clinically, this appears as a large accumulation of white ceruminous debris in the ear canal. Cytologically, there is no infection, only desquamated epithelial cells. Treatment is directed at discontinuation or reduction of the frequency of topical medications.

Specific Otic Therapy

Treatment of acute bacterial and yeast otitis externa

For a first or second time acute otitis externa case, therapy is empirical and based on results from cytologic examination of otic exudate. If rods, coci, and yeast are found, a combination product is indicated. These products contain an antifungal and antibacterial for the infection in addition to a glucocorticoid for the erythema, stenosis, and pain. If cytology reveals only bacteria or yeast, choose a single agent antimicrobial with or without a glucocorticoid, based on the otic examination. The topical products should be used twice daily. Another option is to treat these infections with an ear cleaning and drying agent twice a day. Re-evaluation (including an otic examination and cytology) in 2 weeks is necessary to monitor response to therapy and to determine future therapy.

Treatment of chronic recurrent otitis externa

In cases of a recurrent or unresponsive otitis, cytologic examination of otic exudates should be performed along with bacterial C/S. Oral antibiotics may be indicated and should be based on C/S results. Empirical topical therapy may be started initially based on cytology while waiting on C/S results, and modified once the results have been obtained. Chronic pathologic changes, such as stenosis, hyperplasia, or fibrosis, may be present. These ears then may require frequent ear cleansings to remove excessive otic secretions and topical steroids to decrease inflammation and stop the progression of the pathologic changes. If there is no response to this approach, additional diagnostics should be performed to determine the presence of concurrent otitis media. This work-up may include general anesthesia, bulla radiographs, external
ear flush, evaluation of the tympanic membrane, cytology and C/S of otic exudate from the middle ear and a middle ear flush. Oral and topical therapy should be based on results from cytology and C/S. Re-evaluations should be scheduled every 3-4 weeks, until the infection is under control.

**Monitoring**

Re-evaluations include an otoscopic and/or video otoscopic evaluation to monitor response to therapy. Cytology of otic exudate is performed at each re-evaluation, while bacterial C/S is performed if the infection worsens or is non-responsive to therapy. Topical and/or systemic medications are modified based on these results.

If otitis media is present, otoscopic evaluation includes monitoring healing of the tympanic membrane. In some instances, repeat ear flushing under general anesthesia may be required to keep the ear canal clean to monitor healing of the tympanic membrane.

Surgery is necessary if middle ear polyps, neoplasia, foreign bodies, cholesteatoma, or osteomyelitis of the tympanic bulla is present. In addition, if there is inadequate response of the middle ear infection to otic flushing, myringotomy, and medical management, or if the otitis media is recurrent, surgical intervention may be necessary for resolution.
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ASCITES IN SMALL ANIMALS
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Abstract
Fluid accumulation in the peritoneal cavity (ascites) is a nonspecific abnormality in dogs and cats. It can occur as a result of intra-abdominal diseases, cardiovascular diseases such as right-sided congestive heart failure, or systemic disturbances. The first half of this presentation reviews the common causes of ascites, the practical ways of differentiating between causes of ascites to establish the most accurate diagnosis possible, and treatment modalities for managing patients with ascites.

Introduction
The accumulation of free fluid is only one of many causes of abdominal enlargement. When it is detected, it generally indicates a serious underlying problem warranting treatment and carrying a widely variable prognosis- some animals with ascites have disorders that can be corrected and cured, whereas in others the ascitic fluid accumulation represents an advanced stage of irreversible illness. In order to properly assess a patient suspected of having ascites, it is important to rule out “impostors” that may mimic ascites. Once ascites is confirmed, the cause must be sought so that the best treatment and prognosis can be provided.

Differential diagnosis for abdominal enlargement
- Organomegaly (abdominal palpation and/or radiographs may help elucidate)
- Obesity (physical examination +/- radiographs to elucidate)
- Pregnancy (abdominal palpation reveals multiple fetuses; radiographs may confirm)
- Overeating (abdominal palpation reveals massively enlarged stomach; radiographs may confirm)
- Hyperadrenocorticism/Cushing’s disease (history, physical exam to differentiate)

The most reliable diagnostic test for confirming ascites, especially with very small amounts, is abdominal ultrasonography.

Causes of ascites
<table>
<thead>
<tr>
<th>Disorder</th>
<th>Mechanism</th>
<th>Type of ascites</th>
</tr>
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<tbody>
<tr>
<td>Chronic hepatopathy</td>
<td>Portal hypertension</td>
<td>Modified</td>
</tr>
<tr>
<td></td>
<td>Transudate/pure transudate-</td>
<td>depends on lesion</td>
</tr>
<tr>
<td>Congestive heart failure</td>
<td>Intrahepatic +/- portal hypertension Modified</td>
<td></td>
</tr>
</tbody>
</table>
Hypoalbuminemia | Loss of intravascular oncotic pressure | Pure
--- | --- | ---
Transudate
Hemorrhage (anticoagulant, Hemoabdomen/hemorrhage intoxication, trauma, neoplasia)
Infectious (bacterial, viral, sterile or septic parasitic)
Neoplasia
Penetrating wounds
Pancreatitis
Urinary tract rupture

Steps in assessing the patient suspected of having ascites
The patient’s signalment and history provide an enormous amount of useful information for determining the cause of ascites. For example, chronic liver diseases often affect certain breeds disproportionately, including the doberman and cocker spaniel. Similarly, certain breeds are more commonly affected with disorders that cause right-sided congestive heart failure, which include dilated cardiomyopathy (doberman, great Dane, Irish wolfhound, retriever breeds, cocker spaniel, boxer), pericardial effusion (retriever breeds, rottweiler, greyhound, German shepherd, brachycephalic breeds) and others.

The medical history may provide an immediate and important clue regarding the cause of ascites—trauma (causing hemoabdomen or uroabdomen), witnessed anticoagulant ingestion, penetrating wounds, and similar events may be reported by the client. The owner’s description of other clinical signs also is important. Dyspnea or coughing may raise the likelihood of congestive heart failure, or chronic vomiting, diarrhea, and weight loss may raise the possibility of hypoalbuminemia due to gastrointestinal protein loss. A large part of the useful information in a patient with ascites is obtained from the client. Nevertheless, it is important to avoid overestimating a client’s perception; the “evil” neighbour suspected of poisoning a pet—or such similar misconceptions—have routinely been blamed for hemoabdomen that is actually caused by a nonmalicious etiology (e.g., ruptured abdominal mass). Therefore, the client must be allowed, and encouraged, to provide information, but the clinician must also avoid accepting it all at face value.

A complete clinical exam also is a cornerstone of assessment of patients with ascites. Concurrent abnormalities in addition to ascites are common, and these often offer a clue regarding underlying cause. Examples include heart murmurs, a gallop sound, dyspnea, decreased breath sounds on auscultation (pleural effusion), and jugular vein distension, any of which raises the suspicion of right-sided congestive heart failure; icterus, which suggests chronic hepatopathy; peripheral edema, which suggests hypoalbuminemia (or rarely—and almost exclusively in large breed dogs—right-sided...
congestive heart failure); petechiae or ecchymoses on the skin or mucous membranes, epistaxis, hyphema, and other sources of overt bleeding in cases of anticoagulant ingestion; overt injuries and skin lacerations/stab wounds in cases of penetrating abdominal trauma; and systemic, cutaneous, ocular, or other signs of systemic infection in cases of feline infectious peritonitis, *Mesocestoides* infection, and other infectious causes.

Routine laboratory testing (hematology/complete blood count, serum biochemistry panel, urinalysis) are helpful in many cases of ascites. Relevant abnormalities may include hypoalbuminemia (diagnostic test of choice for hypoalbuminemia-induced ascites) and possibly concurrent hypoglobulinemia if protein-losing enteropathy (or proteinuria +/- azotemia if protein-losing nephropathy) is present; elevations in liver enzymes and/or bilirubin and/or hypoalbuminemia and/or hypocholesterolemia with chronic hepatopathies; neutrophilia with left shift (possibly degenerative) with systemic infection and/or penetrating trauma; and azotemia and hyperkalemia with uroabdomen.

Advanced and specific evaluation of ascites involves two processes: imaging, and sampling. The abdominal imaging technique of choice is ultrasonography. It identifies the presence of ascites but also allows visualization of the abdominal organs, which is compromised or even impossible in abdominal radiography. Paradoxically, the yield of ultrasound may be compromised when there is a large volume of ascites because extreme ascites makes it difficult to identify the point of origin of masses and confuses other interpretations. Ultrasonography also allows the identification of areas for sampling of ascetic fluid via centesis. Abdominocentesis is the minimally-invasive diagnostic procedure of choice in patients with ascites. Samples are typically withdrawn with a sterile 22-gauge needle and submitted for fluid analysis (including cytology), aerobic and anaerobic bacterial culture if appropriate, and additional tests as needed based on the case. For example, an animal suspected of having uroabdomen should have the creatinine level of the ascitic fluid evaluated; a creatinine concentration that is higher in ascites than in blood is diagnostic of uroabdomen.

The fluid type is important, since it can narrow the differential diagnosis list, as shown in the list of causes of ascites, above. Ascitic fluid types commonly recognized include:

- **Pure transudate.** Total protein content of ascites < 2.5 mg/dl; generally poorly cellular or acellular
- **Modified transudate.** Total protein content of ascites = 2.5 - 5 mg/dl; no evidence of inflammation
- **Sterile exudate.** Total protein content >4 mg/dl. Evidence of inflammation without infection (e.g., neutrophils)
- **Septic exudate.** Total protein content > 4 mg/dl. Evidence of infection (e.g., intracellular bacteria, plant material)
- **Hemorrhage.** Hematocrit of ascites approximates hematocrit of patient’s whole blood
• Urine. Creatinine & potassium concentrations of ascites exceed their counterparts in the patient’s serum.

Treatment
Ascites is generally a manifestation of a problem, rather than a problem itself. Therefore, treatment is aimed overall at correcting the problem that it causing the ascites.

A major exception is the recurrent, intractable formation of serous ascites caused by irreversible heart disease or liver disease. These modified or pure transudates may accumulate to the point of causing “tense ascites”, a situation of extreme peritoneal fluid. With tense ascites, severe clinical signs are possible: respiratory distress due to excessive pressure on the diaphragm and reduced lung capacity; discomfort, restlessness, and anorexia due to bloating and a sense of excessive fullness (as reported by humans with similar disorders); and anorexia, renal insufficiency, and other effects of visceral hypoperfusion due to the abdominal compartment syndrome. In patients with tense ascites, treatment must include both management of the inciting cause and relief of ascites. Acute treatment consists of large-volume abdominal drainage and is described below. Chronic treatment depends on the underlying cause, but with most modified transudates, relies on diuretics and, if palatable, some degree of dietary salt restriction. Diuretics that are typically used include furosemide (0.5 -4 mg/kg PO bid – tid as needed to delay recurrence of ascites without causing dehydration or other adverse effects) or spironolactone 0.5-2 mg/kg PO sid-bid.

Treatment of massive (“tense”) transudative ascites causing clinical signs: large-volume abdominal drainage
The procedure is only performed in patients with serous ascitic fluid causing excessive pressure and convincing, ascites-related clinical signs.

Large-volume abdominal drainage is performed using manual restraint and local anesthesia only. Briefly, a sterile tube is placed transcutaneously into the peritoneal cavity and ascitic fluid is allowed to drain freely and completely over a period of a few hours. Materials necessary to complete the procedure are: hair clippers; surgical scrub solution, rubbing alcohol, and gauze/sponge for prepping skin; 0.5 – 15 ml 2% lidocaine (for local anesthesia), warmed to body temperature (armpit method); sterile alligator forceps (preferable) or mosquito hemostats, 1 pair; a red rubber feeding tube-type (or similar) sterile catheter, size= 5-16 French based on body size; sterile surgical gloves; suture material (e.g. nylon 2-0) and needle (figure 1, D); one #11 sterile scalpel blade; sterile needle holders; sterile suture scissors; an Elizabethan collar; sterile gauze squares (post-procedure); and tissue glue or skin stapler (post-procedure).

In preparing for the procedure, it is important to weigh the animal immediately before procedure (to quantify volume of fluid lost by then weighing the animal post-procedure), to advise owner of possible drawbacks (hair clipping required; procedure
generally is only palliative; underlying problem not corrected; low risk of infection or other complications) and to have an E-collar ready to place on the animal as soon as procedure is complete.

Some points are worth noting prior to the procedure. For example, concerns regarding hypovolemia, hypotension, hypoalbuminemia, and ascending bacterial peritonitis appear unjustified when proper technique is used, given the lack of occurrence in large case series (E. Côté, unpublished data). An Elizabethan collar must be on the patient at all times during drainage, to avoid damage to the tube. Finally, once in place in the standing animal, the tube +/- stopcock may be caught in the grate flooring of the cage when the animal lies down. Covering the grate on the floor of the cage with towels helps prevent this complication.

One performs the procedure as follows. 1) Clip hair widely (ventral abdomen), with umbilicus approximately at center of clipped area. Since the procedure does not require surgical draping, long hair must be trimmed back extensively. 2) Restrain in lateral recumbency. 3) Perform a wide surgical scrub and prep, centered on ventral abdominal midline and just cranial to the umbilicus. 4) Generous lidocaine infusion at planned point of entry. On abdominal midline, generally just cranial to the umbilicus. Use multiple (e.g., 6-8) small subcutaneous boluses. 5) After opening the sterile gloves, the paper wrapper can be kept flat and used as a sterile surface. The sterile gloves are worn from this point onward. 6) The suture scissors are used for making 3-5 additional drainage holes in the red rubber feeding tube, which will reduce the risk of omental plugging during drainage. 7) Using the #11 scalpel blade, a stab incision is made in the skin cranial to the umbilicus on the ventral abdominal midline, at the center of the lidocaine-infiltrated area. NOTE: To avoid an excessively large incision, the #11 scalpel blade should be held between thumb and forefinger. The point at which the blade is held between the thumb and forefinger leaves a maximal width of the exposed blade that is the same as, or just slightly greater than, the diameter of the red rubber feeding tube to be inserted. That is, the fingertips act as a guard to prevent excessive insertion of the blade. 8) After the stab incision has been made, the blade is set aside but kept sterile, in case enlargement of the incision is necessary. 9) The tube is inserted into the abdomen. Tube insertion is facilitated by grasping the tip in the lower jaw of an alligator forceps, closing the forceps, and advancing tube and forceps through the hole. Mosquito forceps are an acceptable alternative. Often, the hole in the skin and the hole in the body wall are not exactly aligned due to imperceptible shifting of the tissue planes. Blunt probing with the tube and forceps may be necessary to find the hole in the abdominal wall. If excessive pressure is required, the incision may need to be enlarged using the #11 scalpel blade. Any sign of discomfort on the patient’s part is an indication for additional lidocaine infiltration at and around the insertion site. 10) Once the tube is inserted appropriately (the clinician may feel a release of pressure as the tube pierces the peritoneum, and then a voluminous flow of ascites immediately emerges through the tube), it is advanced until it protrudes from the abdominal wall by only 5-10 cm. 11) The tube is sutured in place, using 2-0 or 3-0 nylon with both a
circumferential pursestring and a transfixation (suture through the tube) ligatures. 12) If a rapid flow of fluid is occurring, a clamp or partially-closed three-way stopcock (usually requiring a “Christmas-tree”-type adapter to fit most red rubber feeding tubes) can be used for moderating the rate of flow. 13) Complete drainage is possible in 2-6 hours, with the patient placed in a cage with a grated floor to allow drainage and towels on top of the grate to prevent a clamp/stopcock from becoming caught in the grate. In most cases the flow is adjusted a few times so the ascites is fully evacuated after 2-3 hours. CAUTION: An Elizabethan collar is essential to prevent the patient from chewing at, and transecting, the tube. 14) The system may be closed (drainage bag) or open. If open, as is done most commonly, the patient must be monitored for ongoing drainage and the tube needs to be removed immediately when flow ceases, to reduce the risk of ascending infection. 15) When drainage has ended, the patient is again restrained in lateral recumbency and the nylon ligatures are cut. 16) The tube is removed, taking care not to withdraw omentum (a sterile instrument may be needed to push omentum off the tube and back into the abdomen). 17) The skin incision may be dried with a sterile gauze and tissue glue applied to close it. If the incision is > 5 mm, a skin suture or staple may be placed. 18) The patient is weighed and the weight of fluid removed is calculated and recorded.

Large-volume drainage of ascites is not a definitive treatment, and recurrence is inevitable if the underlying process persists. However, in humans with cirrhosis-associated ascites, such drainage procedures are associated with a significantly lower risk of complications than the high doses of diuretics needed to achieve the same goal. Therefore, large-volume abdominal drainage in dogs and cats is reserved for those cases in which serous ascites is present, treatment of the underlying cause has not stopped ascites formation, and ascites has accumulated so substantially that it is clearly responsible for clinical signs.

**Prognosis and outcome**
The results of natural evolution of ascites, and the response to treatment, are highly variable given the wide spectrum of possible causes of ascites. Therefore, few generalities are possible concerning prognosis and outcome. As a rule of thumb, the rate of recurrence of ascites caused by chronic hepatopathies or right-sided congestive heart failure accelerates slowly as the disorder progresses. However, with a dedicated owner who is willing to bring the patient every several weeks for abdominal drainage and who notes a good quality of life for the pet, animals with ascites caused by these disorders have lived comfortably and very satisfactorily in their owners’ opinions for many months to a year or more.

**Pearls**
One of the earliest physical signs of ascites is a slightly more slippery feel of the small intestines than usual, such that the small intestine slides more easily than usual between the examiner’s fingertips.
Urinalysis is required in all cases of ascites (potential abnormalities include those of liver disease, protein-losing nephropathy, and others) but the urine should not be obtained by blind cystocentesis because the “urine” sample may be a sample of ascites. Free-catch, catheter, and ultrasound-guided cystocentesis (only if noninfectious, nonneoplastic cause) are alternatives.

An important -and often overlooked- diagnostic clue regarding the cause of ascites during abdominal ultrasonography is the subjective appearance of the hepatic veins. Ascites caused by right-sided congestive heart failure or caudal vena caval obstruction typically is associated with moderately or markedly enlarged hepatic vessels (on ultrasound), whereas primary intra-abdominal, or systemic, causes of ascites are not.

When the volume of ascites appears to be small, blind abdominocentesis and diagnostic peritoneal lavage generally are low-yield procedures, with multiple possible complications. Diagnostic peritoneal lavage has not lived up to its potential when it was first introduced and is rarely used.
“HOW I TREAT”: POINT-COUNTERPOINT PRESENTATION OF CONTROVERSIES IN SMALL ANIMAL CARDIOLOGY
Etienne Côté  DVM, DACVIM (Cardiology, Small Animal Internal Medicine)
Erin Trageser  VMD
Atlantic Veterinary College, University of PEI
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Evidence-based medicine allows veterinarians1,2 and physicians3 to make decisions based on proof obtained through scientific studies. However, areas of controversy inevitably exist when information is incomplete.

Medical controversy has been used to great, positive effect in human cardiology in the form of point-counterpoint debates that produce informed, constructive results with justifications often supporting both sides of an argument depending on specific clinical applications.4,5 In veterinary cardiology, such debates also have taken place, but much less frequently.6

In these sessions, we propose to offer evidence to support and refute areas of cardiology where controversy continues to exist, in order to clarify the reasons supporting -or failing to support- beliefs that drive diagnostic, treatment, and prognostic decisions in dogs and cats with heart disease.

The following tables present the information that forms the basis for the point-counterpoint presentations.

References
4. Granger CB, Armaganijan LV. Newer anticoagulants should be used as first-line agents to prevent thromboembolism in patients with atrial fibrillation and risk factors for stroke or thromboembolism. Circulation 2012; 125: 159-64.
**Beta-blockade for subclinical hypertrophic cardiomyopathy in cats**

*Reproduced with modifications from Côté E, MacDonald KA, Meurs KM, Sleeper MM. Feline Cardiology (Ames, IA: Wiley-Blackwell, 2011)*

<table>
<thead>
<tr>
<th>Physiologic effects</th>
<th>For beta-blockade</th>
<th>Against beta-blockade</th>
</tr>
</thead>
</table>
| Decrease systolic anterior motion (SAM) of the mitral valve | ● Dramatic reduction in the left ventricular (LV) to aortic pressure gradient is possible in cats with moderate or severe SAM, which may decrease severity of concentric LV hypertrophy  
● By decreasing SAM, mitral regurgitation (MR) is decreased, which may decrease left atrial volume overload and left atrial pressure in cats with moderate MR due to SAM  
● Some cats with moderate to severe SAM have unrecognized symptoms until they are treated, and then the owner observes overt benefits including increased energy and playfulness | ● Indoor cats are sedentary animals that may not have high sympathetic tone until a stressor such as a trip the veterinary office, which magnifies the severity of the SAM far beyond what is seen at home  
● No information is available to confirm whether reduction of SAM leads to improved quality of life, survival, or delays onset of clinical signs |
| Decrease left ventricular (LV) concentric hypertrophy | ● Some cats receiving atenolol may experience reduction in severity of LV concentric hypertrophy  
● Decrease LV hypertrophy may improve diastolic filling properties of the | ● No information regarding effect on morbidity, mortality, or time to onset of clinical signs  
● The reduction in LV hypertrophy appears to be relatively small in a placebo controlled prospective study |
<table>
<thead>
<tr>
<th>myocardium (i.e. decrease LV stiffness), decrease diastolic filling pressures and lessen risk of congestive heart failure</th>
<th>Some cats have improvement in LV hypertrophy without treatment or their hypertrophy never worsens over many years, making the role of medication questionable.</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Suppression of tachycardia</strong></td>
<td>Acute stressful events may cause sinus tachycardia, increasing diastolic filling pressure, and acute development of congestive heart failure in cats, so blunting this acute tachycardic response may be protective</td>
</tr>
<tr>
<td></td>
<td>Indoor cats may not have many acute stressful events that could cause this phenomenon, aside from visits to the veterinary hospital, which would be likely increased during initial monitoring of the cat receiving medications</td>
</tr>
<tr>
<td></td>
<td>Potentially similar effect can be achieved without medication, by instructing owners to avoid situations causing vigorous activity for the cat (e.g., play with laser pointer; minimizing or avoiding outdoor roaming, etc.)</td>
</tr>
<tr>
<td><strong>Effects on diastolic function</strong></td>
<td>Decreased heart rate will increase diastolic filling time, which may be beneficial in cats with stiff hypertrophied ventricles by lessening diastolic filling pressures occurring during tachycardia</td>
</tr>
<tr>
<td></td>
<td>Beta blockers prolong early diastolic relaxation (negative lusitrope), which impairs early diastolic function</td>
</tr>
</tbody>
</table>
more important determinant of increased diastolic filling pressure in cats than early diastolic relaxation since cats have high heart rates compared to other species, which minimizes the overall negative impact of beta blockers effects on slowing diastolic relaxation

<table>
<thead>
<tr>
<th>Medication considerations – owner-related</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Inexpensive</td>
</tr>
<tr>
<td>• Widely available</td>
</tr>
<tr>
<td>• Proactive owners may want to feel that they are giving their cat every opportunity for positive response and/or improvement</td>
</tr>
<tr>
<td>• Lifelong therapy commits owner to q 12 h dosing; cannot abruptly stop the medication after chronic use</td>
</tr>
<tr>
<td>• Lack of perceived benefit (although no visible benefit expected when HCM was discovered incidentally) may cause discouragement &amp; discontinuation of the medication</td>
</tr>
<tr>
<td>• Since cat is receiving medication, owners may be reluctant to continue important follow-up diagnostic testing such as echocardiograms or radiographs if they may perceive there is nothing else to be done</td>
</tr>
</tbody>
</table>
| • Intermittent “on and
“Off” use may be dangerous due to beta receptor up-regulation and possibility for adverse cardiac effects with acute cessation

- Sacrifice of other necessary medications for treatment of non-cardiac diseases due to inability to administer several concurrent medications or prioritization of the beta-blocker over other medications

**Medication considerations – cat-related**

<table>
<thead>
<tr>
<th>Effect</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Well-tolerated; side effects are rare</td>
<td>Most cats will tolerate medication administration if the tablet is concealed in a treat/food or the medication is compounded in a meat-flavored suspension</td>
</tr>
<tr>
<td>May exacerbate bronchoconstriction, especially high doses, in cats with airway disease</td>
<td>Pilling may be difficult/disturbing for cat and owner, and transdermal preparations are ineffective</td>
</tr>
<tr>
<td>Development of medication aversion may make it challenging to medicate if/when life-saving medications are needed later for heart failure or thromboembolism</td>
<td>Difficulty medicating the cat may lead to antagonistic experience with owner</td>
</tr>
</tbody>
</table>

**Morbidity, mortality**
No information available, although no evidence of hastened deterioration in incidentally-found HCM in cats

No information available, although no evidence of greater longevity for incidentally-found HCM in cats

<table>
<thead>
<tr>
<th>ACE inhibition (ACEI) for subclinical myxomatous mitral valve disease</th>
<th>For ACE inhibition</th>
<th>Against ACE inhibition</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Physiologic effects</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Disease substrate</td>
<td>• Importance of tissue ACE now recognized; benefit of ACEI in canine myocardium could be greater than previously thought</td>
<td>• Furosemide treatment strongly activates RAAS, suggesting that the greatest need for ACEI begins with treatment of heart failure, not during pre-heart-failure state</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Does not alter underlying valve disease process; AT II receptors are absent from myxomatous mitral valves in dogs</td>
</tr>
<tr>
<td>“ACE escape”: loss of ACEI efficacy due to activation of parallel pathways for conversion of AT I to AT II</td>
<td>• ACE escape not proven in clinical veterinary medicine</td>
<td>• Cathepsin G, tonin, and chymase account for 70% of AT I → AT II conversion in human heart failure patients taking ACEI; a similar effect can be expected in dogs</td>
</tr>
<tr>
<td></td>
<td>• ACE escape may not be relevant if congestive heart failure is imminent, e.g., with marked left atrial enlargement</td>
<td>• Human ACEI trials show loss of efficacy after 2-5 years, a time period that can be clinically significant (both in relative and absolute terms) in dogs with myxomatous mitral valve disease</td>
</tr>
<tr>
<td>Alternative methods for</td>
<td>• Bradykinin levels</td>
<td>• A synergistic benefit is</td>
</tr>
</tbody>
</table>
| reducing RAAS effects | increase with ACE inhibition, favouring vasodilation (an effect not obtained with AT II receptor blockade)  
- AT II receptor blockers have not been demonstrated to be effective in veterinary cardiology | observed in human heart failure patients treated with both ACEI and AT II receptor blockers |
|-----------------------|--------------------------------------------------------------------------------------------------|----------------------------------------------------------------------------------|
| Initiating treatment only once left atrial enlargement is apparent | - Left atrial enlargement can be considered a surrogate marker of more advanced disease, suggesting greater RAAS activation and therefore increased importance of ACEI treatment  
- Trial of dogs with myxomatous mitral valve disease causing cardiomegaly and left atrial enlargement (VETPROOF) showed interim benefits in some dogs | - Very heterogeneous response: dogs with the same degree of atrial enlargement may vary widely in the duration of their compensated period free of congestive heart failure  
- Left atrial enlargement is easily overdiagnosed on thoracic radiographs of some dogs with normal atrial size, and under-diagnosed on thoracic radiographs of some dogs with mild left atrial enlargement |
| Effects on disease progression | - Early intervention for sustained reduction of multiple adverse effects of AT II (vasoconstriction, cardiac remodeling, aldosterone effects) | - Clinical trials in dogs have produced modest results (VETPROOF) or no evidence of benefit (SVEP)  
- No evidence that adverse effects caused by AT II are reduced in dogs with incidentally-found myxomatous mitral valve disease treated with ACEI |
| Concurrent therapeutic effects | - Benefits for concurrent systemic hypertension and chronic kidney disease, a common | - Antihypertensive effects of ACEI are minimal in dogs |
### Adverse effects

- Few adverse effects, well-tolerated
- Hyperkalemia is possible (but rare) with concurrent spironolactone, and spironolactone is rarely indicated in dogs with incidentally-found myxomatous valve disease
- Cough occurs as a common (15%) adverse effect in humans but virtually never in dogs; ACEI treatment and perceived adverse effect may detract from diagnosis and management of chronic bronchitis, obesity-exacerbated airway disease, or other.

### Medication considerations

- Generic human formulations are inexpensive
- Owner’s perceived benefit of “doing something to help”
- Administering medication may keep owner more closely involved with the dog’s medical care, prompting closer follow-up
- Brand-name veterinary formulations are more costly
- Absence of visible difference during treatment may lead owner to perceive lack of efficacy
- Additional variable to consider (possibility of toxicosis) if any systemic illness occurs

### Positive inotrope treatment for subclinical systolic dysfunction

<table>
<thead>
<tr>
<th>Physiologic effects</th>
<th>For positive inotrope Tx</th>
<th>Against positive inotrope Tx</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increased ventricular systolic function</td>
<td>• Increased systolic function may produce synergistic effects:</td>
<td>• In humans, orally-active positive inotropes have been associated with</td>
</tr>
<tr>
<td><strong>Reduction in ventricular diameter, decreasing wall stress, and improving myocardial perfusion</strong></td>
<td><strong>Increased mortality and are not licensed for use in many countries</strong></td>
<td></td>
</tr>
<tr>
<td>- Calcium-sensitizing agents increase systolic function without increasing myocardial oxygen consumption and therefore without increasing the risk of cardiac arrhythmias</td>
<td>- The positive inotropic effect of the most recently developed and widely used drug of this class, pimobendan, has not been evaluated in graded dosages in dogs with subclinical systolic dysfunction; the optimal dosage for varying degrees of systolic dysfunction in dogs is not known</td>
<td></td>
</tr>
<tr>
<td>- Adverse effects in humans fail to apply to dogs because of a different disease substrate and because a class effect for positive inotropes has been applied to all drugs in the class</td>
<td>- Does not reverse the underlying mechanism</td>
<td></td>
</tr>
<tr>
<td><strong>Loss of efficacy with early initiation of treatment</strong></td>
<td><strong>Undocumented; benefit may be short-lived and/or lost en route to congestive heart failure, reducing the potential for benefit when it is needed most</strong></td>
<td></td>
</tr>
<tr>
<td>- Undocumented; may provide better quality of life immediately, delaying or eliminating decision for euthanasia</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Subgroup variation</strong></td>
<td><strong>No differentiation of immediate positive responders from minimal responders; dogs may continue to be treated unnecessarily (or detrimentally) while dogs with strongly positive initial responses negate adverse responders; subgroups in clinical trials routinely are too</strong></td>
<td></td>
</tr>
<tr>
<td>- Demonstrated benefit of pimobendan treatment in dogs with dilated cardiomyopathy with congestive heart failure; subclinical systolic dysfunction trial results pending</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ventricular volume</td>
<td>• Improved systolic function reduces left ventricular end-diastolic volume</td>
<td>• Positive inotropes do not generally increase lusitropy, and impaired diastolic function is an important feature of dilated cardiomyopathy</td>
</tr>
</tbody>
</table>
| Arrhythmia | • No demonstration of pathologic arrhythmias despite extensive evaluation  
• Anecdotal reports of improvement in arrhythmia (e.g., conversion of atrial fibrillation to normal sinus rhythm) with start of pimobendan treatment | • Unclear arrhythmia effect, since spontaneous arrhythmia is common with systolic dysfunction regardless of treatment, and ultimate effect of malignant arrhythmia – sudden cardiac death – occurs with or without positive inotrope treatment |
| Other adverse effects | • Dependent on specific drug, ranging from well-recognized (digoxin) to more poorly-defined (pimobendan) | • Dependent on specific drug, ranging from well-recognized (digoxin) to more poorly-defined (pimobendan) |
| Medication considerations – owner-related | • Remarkably positive responses in dogs with systolic dysfunction and congestive heart failure may cross over to similar responses in subclinical cases  
• Owners may perceive an absence of clinical signs until effective treatment eliminates clinical signs of heart disease that were | • Not all owners are willing or able to have the diagnosis of systolic dysfunction made by a veterinary cardiologist, and several artifacts (notably cardiomegaly on poor-quality radiographs and low LV fractional shortening on suboptimal echocardiograms) may mimic systolic |
<table>
<thead>
<tr>
<th>attributed to aging or behavior change</th>
<th>dysfunction to an inexperienced clinician</th>
</tr>
</thead>
<tbody>
<tr>
<td>Owner’s perceived benefit of “doing something to help”</td>
<td>Positive inotropes range in price from the inexpensive (digoxin) to the costly (pimobendan)</td>
</tr>
<tr>
<td>Administering medication may keep owner more closely involved with the dog’s medical care, prompting closer follow-up</td>
<td></td>
</tr>
</tbody>
</table>
Aspirin versus clopidogrel for thromboembolism prophylaxis in cats

<table>
<thead>
<tr>
<th>Physiologic effects</th>
<th>For aspirin</th>
<th>For clopidogrel</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mechanism and complementarity</td>
<td>• Permanent inhibition of platelet cyclooxygenase</td>
<td>• Selective inhibition of ADP-induced platelet aggregation without cyclooxygenase effects (=opportunity for synergism with aspirin)</td>
</tr>
<tr>
<td>Duration of effect</td>
<td>• Prolonged (3-day) in vitro effect on feline platelets</td>
<td>• Once daily dosage</td>
</tr>
<tr>
<td>Clinical efficacy</td>
<td>• Clopidogrel efficacy demonstrated in vitro; clinical trial does not show dramatic superiority of clopidogrel over aspirin (blinded); subgroup analysis (some cats may respond, others not) not done</td>
<td>• Aspirin efficacy demonstrated in vitro; clinical trial does not show dramatic superiority of aspirin over clopidogrel (blinded); subgroup analysis (some cats may respond, others not) not done, versus humans where hepatic microenzyme profiling identifies clopidogrel-resistant and –sensitive individuals</td>
</tr>
<tr>
<td>Most effective dosage</td>
<td>• Unknown; wide range of approaches without consensus; lower dosage may produce fewer adverse effects</td>
<td>• Unknown but 18.75 mg dosage investigated consistently</td>
</tr>
</tbody>
</table>

Medication considerations – owner-related

<table>
<thead>
<tr>
<th>Cost</th>
<th>For aspirin</th>
<th>For clopidogrel</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>• Inexpensive</td>
<td>• Relatively more expensive</td>
</tr>
<tr>
<td>Monitoring</td>
<td>• None</td>
<td>• None</td>
</tr>
<tr>
<td>Dosage interval</td>
<td>• Up to 3-day interdose interval may be used for convenience</td>
<td>• Once daily is the recommended dosage interval in cats</td>
</tr>
</tbody>
</table>

Medication considerations
Palatability

- Generally tolerated
- Unpalatable to many cats; requires concealment or encapsulation

Gastrointestinal, hepatic, renal adverse effects

- Possible but uncommon
- Rare

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**Dr. Lisa M. Freeman, DVM, PhD, DACVN**

**ANSWERING OWNERS’ QUESTIONS ABOUT PET FOODS**

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North Grafton, MA

With the hundreds of pet foods available today, an owner's decision about what to feed his or her pet has become a more complicated question than it once was. Nor is there one simple answer since the "best" food for a pet depends on many factors, such as life stage, body condition, exercise (or lack thereof), environment, and health status. Often, owners base their decisions on marketing, rather than on objective nutritional information. Although there are limitations to the information provided on a pet food label, the label can provide important information to help in selecting foods.

The two most useful pieces of information on a pet food label are the nutritional adequacy statement and the manufacturer.

**Nutritional adequacy statement**

A pet food that is intended as a complete and balanced diet must be established as such in one of two ways: 1) By **formulation** to meet the levels established by the Association of American Feed Control Officials (AAFCO) or 2) by **feeding trials**. A diet that is only formulated contains nutrients in amounts that meet AAFCO profiles but the diet has not gone through feeding trials. Therefore, unforeseen problems with bioavailability or nutrient interactions could arise. Feeding trials provide better assurance that the food meets a dog's or a cat's requirements. Although AAFCO feeding trials have their limitations, they should be a minimum level of assurance. When feeding trials have been performed, the label should read, "Animal feeding tests using AAFCO procedures substantiate that Brand X provides completed and balanced nutrition for growth (or maintenance)." If the food is only formulated to meet requirements, the label must read, "Brand Y is formulated to meet the nutritional levels established by the AAFCO Dog (or Cat) Food Nutrient Profiles..." Therefore, the nutritional adequacy statement provides two important pieces of information: 1) Which life stage profile the food meets and 2) how this statement was substantiated. Pet foods are designed to meet the minimums for one of the recognized life stages: growth and reproduction or adult...
maintenance. Foods that are complete and balanced for all life stages meet the requirements for growth, reproduction, and adult maintenance. Be aware that the life stage a food is marketed for may not necessarily be the same stage for which the food is truly meets the minimums. For example, many diets marketed for adult cats actually meet the profiles for all life stages so they have nutrient levels high enough to be fed to kittens or lactating queens. By checking the nutritional adequacy statement, owners can select a food that is most appropriate for their pets' life stage. Also, be aware that over-the-counter foods that state "for intermittent or supplemental use only" are not complete and balanced and should not be fed. Veterinary therapeutic diets may have this "intermittent or supplemental use only" statement because they may be specifically designed not to meet AAFCO minimums (eg, renal diets).

The manufacturer
One of the most important factors to consider is the quality of the pet food manufacturer (not just how persuasive their marketing is). Contact the manufacturer with any questions or concerns. Consider asking the following questions:

- Do you have a board-certified veterinary nutritionist or PhD nutritionist on staff full-time in your company? What is his/her name and are they available for consultation or questions?
- Who formulates your diets and what are their credentials?
- Which of your diet(s) are tested using AAFCO feeding trials, and which by nutrient analysis?
- What specific quality control measures do you use to assure the consistency and quality of your product line?
- Where are your diets produced and manufactured? Can this plant be visited?
- Will you provide a complete product nutrient analysis for any dog or cat food of interest, including digestibility values and caloric density?
- What kinds of research on your products has been conducted, and are the results published in peer-reviewed journals?

In general, the recommendations for selecting a good quality pet food are as follows:

• Feed a pet food manufactured by a well-known, reputable company (based on answers to questions above).
• Feed a complete and balanced food for the appropriate life stage of the pet (growth diet for puppies or kittens, maintenance diet for adults) - this should be substantiated by feeding trials.
• Remember that pets are individuals and may respond better to one diet than to another.

Common Questions:

What is the best food to feed my pet?

There is no best diet, despite all the marketing claims to the contrary. Every pet is unique and the goal is to find the best diet for the individual pet. Expense doesn't necessarily equal quality. There are some inexpensive diets that have years of rigorous scientific testing behind them and some very expensive diets that are lacking in vital nutrients or based on
unsound science. Larger companies generally have more stringent quality control protocols, employ expert nutritionists and food scientists, and strive to increase our collective nutrition knowledge through research. Smaller manufacturers may have less control over ingredient quality, perform less laboratory testing and are less likely to employ full or part time veterinary nutritionists.

A good rule of thumb is that if the marketing of a product sounds too good to be true, the manufacturer cites studies or research that they cannot provide to you or makes claims that cannot be substantiated, then that's a red flag that the diet should be avoided.

How can I pick a good diet to feed to my pet?

Consumers should look for foods made by reputable companies with long histories of producing quality diets. Diets that have an Association of American Feed Control Officials (AAFCO) statement on the label saying that the diets have undergone animal feeding trials for the appropriate life stage are generally preferable to diets that are formulated [by computer] to meet AAFCO nutrient profiles for that life stage. This distinction is particularly important for puppy and kitten diets as well as diets produced by newer and smaller companies with less experience in diet formulation. Ideally, manufacturers should be engaging in both internal and external (such as through a university) research to both improve their products and increase our collective nutrition knowledge. Advertisements and websites should not contain unverifiable claims, perpetuate nutrition myths or promote products solely by bashing other manufacturers' products. In addition, the diet owners select for their pets should be one that the animals do well on.

Is the ingredient list a good way to determine the quality of a pet food?

Although ingredient lists are commonly used by lay people to determine the quality of pet foods, this approach has many pitfalls and is very subjective to intentional manipulation by the food manufacturers. Ingredients are listed on labels in order of weight, including water, so ingredients with high water content (like fresh meats and vegetables) are going to be listed higher than similar amounts of dry ingredients even though they may contribute fewer nutrients to the overall diet. Additionally, ingredients from the same source—chicken meat, chicken fat and chicken by-product meal for example—can be split into component parts, further complicating assessment.

Pets require nutrients, not ingredients; a diet full of great sounding ingredients can be less nutritious than a diet containing less appealing (to people) ingredients. Some manufacturers may add ingredients to diets solely for marketing purposes, to increase the appeal of the diet to consumers. These ingredients may have unproven benefits, be present in miniscule amounts and provide nothing to the diet but added expense. More ingredients also mean more quality control (and more time and expense) is necessary to ensure that the finished product adheres to the desired nutrient formulation.

It is also important to understand that the phrase human grade has no legal meaning in the pet food industry. Once a product is destined for inclusion in pet food, it is no longer fit for human consumption by definition. Moreover, ingredients sourced from the human food chain are not necessarily any more nutritious, wholesome, or safe than ingredients initially destined for pet food. Therefore, manufacturer's claims of human grade ingredients should not be over
I've heard that raw diets prevent and or solve a lot of health problems in pets. Is this true?

Despite anecdotal reports from pet owners and even some veterinarians, there is currently no evidence that raw diets offer any benefits over cooked diets. However, there is substantial evidence that these diets may be associated with dental fractures, bacterial and parasitic infections and other health concerns in pets. There is also potential risk to people, especially those that are immunocompromised such as young children, the elderly and patients with immune mediated diseases or cancer. Pets that eat contaminated raw diets have been demonstrated to shed viable pathologic organisms in their feces and it is likely that areas that they frequent are also contaminated. As numerous recalls and some pathogen surveys in the last few years have proven, all raw meat, regardless of source, should be considered to be contaminated until proven otherwise. For these reasons, the Delta Society has banned raw fed pets from participating in their pet therapy programs.

In addition to food safety concerns, nearly all home-prepared raw diets and most commercially available raw diets are deficient (or excessive) in essential nutrients. It is also common for commercial raw diets to be very high in fat, which may not be tolerated by some animals.

My friend says that grains are bad for dogs, is she correct?

Whole grains, rather than being fillers, contribute valuable nutrients including vitamins, minerals, essential fatty acids and fiber to diets while helping to keep the fat and calories lower than if animal products were used in their place. Even refined grains such as white rice can have beneficial health implications depending on the type of diet and the pet. The vast majority of dogs and cats are very efficient at digesting and utilizing nutrients from grains. While a very small number of dogs are allergic to specific grains, these allergies are no more common than allergies to animal proteins such as chicken, beef and dairy and tend to reflect the prevalence of the ingredient in commercial diets rather than enhanced antigenicity.

It is becoming more common in the saturated pet food market for manufacturers to perpetuate myths to sell diets and increase market share. Grain-free diets are often an example of this strategy. Many of these diets merely substitute highly refined starches such as those from potatoes or tapioca (cassava) in place of grains. These ingredients often provide fewer nutrients and less fiber that whole grains, while costing more.

I read online that by-products can contain hair, hooves and floor sweepings. Is this true?

By-products are commonly vilified, often by diet manufacturers trying to carve out market share for themselves by offering diets that do not contain them. By-products (mainly organ meats and entrails) often provide more nutrients than muscle meats on a per weight basis and are important components and even delicacies of human diets in other countries. The term by-product comes from the fact that they are the leftovers from animal carcasses once the desirable (for Americans) muscle meat has been removed. AAFCO definitions of mammal by-products specifically exclude hair, hooves, horn, hide trimmings, manure and intestinal contents, as well as anything that is not specifically part of the carcass (such as floor sweepings). Like all ingredients, the quality of by-products can vary, so it is important to select
manufacturers who have stringent internal quality control standards.

**Can I use the guaranteed analysis to accurately compare pet foods?**

The guaranteed analysis is required to give only the minimum content of protein and fat in the food and the maximum content of fiber and moisture. Other nutrients may also be listed but are not required. This obviously is only a limited number of nutrients you might be interested in and, in addition, having the minimum or maximum level of a nutrient might not provide all the information you need (for example, in a dog that requires dietary fat restriction, a minimum fat level of 3% listed on the label does not tell you exactly how much fat the diet contains (it might contain 3% but it might contain much more).

Another issue that commonly confuses pet owners is that the nutrient levels in the guaranteed analysis are listed on an "as fed" basis, which includes the water in the food (therefore, the protein level of a dry food will appear to be much higher than the protein level in a canned food, even if the levels on a dry matter basis are exactly the same). A much more accurate way to compare foods is to compare them on a gram or milligram per 100 kcal basis. This information should be available from the manufacturer whose name and address must be listed on the label. The contact information for the manufacturer is one of the most useful pieces of information on the label. A company should be able to provide any other nutritional information that you might require for your patients. If a company cannot or will not provide you with a piece of nutritional information, I would consider that to be a red flag and would not recommend that food!

**Can I feed my pet according to the feeding directions on the label?**

Pet food labels must list feeding directions. Many feeding guidelines overestimate the amount a dog or cat should eat (although some of the pet food companies are beginning to revise their labels to contain more reasonable estimates). Therefore, feeding directions should be used only as a starting point and owners must make adjustments to keep the pet in trim body condition. I typically recommend that owners start at the lower end of the recommended range for most animals (even lower if they have a low activity level) and then carefully monitor body weight for the first several weeks on the new diet so that adjustments can be made as needed.

It seems obvious that the calorie content of the food would be on the label but this is not required information (except for "light" foods). The calorie information is allowed on the label and some pet food companies are starting to include the calorie content. This is very helpful information to be able to compare foods. There are maximum caloric densities for foods that are described as "light," "lite," or "low calorie" (for example, a dry dog food must contain \( \leq 3100 \) kcal/kg). In addition, when a food is described as such, the label must contain a calorie content statement. However, these food still vary tremendously in terms of the calories per cup or can so one must be careful to select one that is appropriate in calorie and nutrient level for the individual pet.

**Should I try to feed a natural or organic pet food?**

The guidelines for the use of "natural" on pet food labels are that the product should not contain any chemically synthesized ingredients. Exceptions can be made
when chemically synthesized vitamins or minerals are used as long as there is a disclaimer on the label (eg, "natural with added vitamins and minerals."). Other descriptors, such as "organic," "holistic," "gourmet," and "human grade" have no legal definition for pet foods and are purely marketing terms.

Nutrition Resources
American Academy of Veterinary Nutrition
www.aavn.org

American Animal Hospital Association Nutritional Assessment Guidelines
http://www.aahanet.org/resources/guidelines.aspx

American College of Veterinary Nutrition
www.acvn.org
(includes a section on Nutrition Resources for nutritional consultations with ACVN Diplomates, including homemade diets)

Association of American Feed Control Officials
www.aafco.org

FDA Animal and Veterinary Site (including pet food)
http://www.fda.gov/AnimalVeterinary/default.htm

National Research Council Pet Nutrition Guides for Owners
http://dels-old.nas.edu/banr/petdoor.html

Pet Food Institute Consumer Guide
www.petfoodreport.org
AGING GRACEFULLY: FEEDING THE SENIOR PET
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General feeding recommendations for pets
With the hundreds of pet foods available today, an owner's decision about what to feed his or her pet has become a more complicated question than it once was. Nor is there one simple answer since the "best" food for a pet depends on many factors, such as life stage, body condition, exercise (or lack thereof), environment, and health status. Often, owners base their decisions on marketing, rather than on objective nutritional information. Although there are limitations to the information provided on a pet food label, the label can provide important information to help in selecting foods. The two most useful pieces of information on the label on which the best decisions can be made are the nutritional adequacy statement and the manufacturer. The usefulness of ingredients, guaranteed analysis, descriptive terms (e.g., holistic, premium, grain free), and nearly all other information on the label ranges from relatively unimportant to useless.

Seniors
It has often been said that aging, in itself, is not a disease. However, aging is often associated with a variety of diseases. Nutrition can be a powerful tool in maintaining health, preventing disease, and in helping to manage disease. However, deciding on the "best" diet for an older dog or cat can be a difficult decision; there is no one best diet for every older animal. Animals are individuals so just because a pet turns 7 or 10 or even 13 years old doesn't necessarily mean it's old. The aging process depends on a variety of factors including breed, genetics, and health problems. Therefore, just because a food is marketed for older animal, doesn't mean it's right for every older dog or cat.

Individual differences aside, there are a number of changes that occur with aging that can affect nutritional requirements. Unfortunately, little research on companion animal aging and nutritional requirements has been done so much of our assumptions are based on research in other species. In other species, digestion and absorption of nutrients can be impaired with aging. Dogs tend to have decreased energy requirements, decreased activity, and to gain fat and lose muscle. Immune function and kidney function also decline with age, although the degree to which this occurs depends upon the individual animal. While these structural or functional changes are thought to occur in older dogs and cats, minimal research has been done on changes in nutritional requirements that can result. In people, there has been a great deal of work on specific requirements for the elderly and these can be very different from younger adults. In the new dietary reference intakes (DRIs) for people, people are now separated into additional age groups: 19-30 years, 31-50 years, 51-70 years, and >70 years for men and women. When enough information is available, recommendations are made for each age group. If there are not enough data to distinguish differences in requirements between these age groups, information is given for a larger, combined age group. Therefore, the direction is to have established requirements for each age group, including people >70 years. Currently, in cats and dogs, adults are considered as a single group, whether the animal is 2
years old, 8 years old, or 15 years old. More specific requirements for elderly animals would be beneficial as the requirements of older dogs and cats are most certainly different from a young adult.

Even if requirements are altered in older dogs and cats, adjustment of the diet may or may not be necessary or even desirable in the average older animal. Many older dogs and cats do very well by continuing to eat a good quality commercial diet designed for adults. Others, however, will benefit from changing to a "senior" diet. It is important to understand that there is no legal definition for "senior" or "geriatric" foods. Although the title generally implies lower protein, lower phosphorus, and a lower caloric content, the levels vary with each company and each company's senior food will have different properties. Therefore, some foods will meet the needs of an individual animal better than others.

"Senior" foods vary depending upon the manufacturer, but there are a number of nutritional adjustments that are common to many:

1. Reduced protein. Although there is a common belief that protein restriction is helpful for older animal, there is little scientific evidence to show that low protein foods are beneficial for the healthy older dog or cat or that moderately high protein foods contribute to the development of kidney disease. Therefore, dogs and cats should not be fed a low protein diet just because they are old (if moderate to severe renal disease is present, then some protein restriction may be beneficial). In fact, foods highly restricted in protein may actually be too low in protein for many older animals and can contribute to muscle loss. The "optimal" protein level for older dogs and cats, however, is still controversial. Some companies manufacture "senior" diets with low protein, some have moderate protein, and some nutritionists actually recommend that older dogs and cats eat a higher protein level than younger animals. The jury is still out but for older animals without significant renal or hepatic disease, it is wise to avoid reduced protein diets.

2. Phosphorus. Phosphorus can contribute to the progression of renal disease, so phosphorus restriction is recommended for animals with significant renal disease. It is not known, however, whether high dietary phosphorus directly contributes to the development of renal disease. Nonetheless, high phosphorus foods may not be ideal for older dogs and cats.

3. Sodium. Sodium levels are often reduced in "senior" foods. Sodium restriction is unnecessary for the general population of older dogs and cats, but may be recommended if cardiac disease is present. Even with early cardiac disease, severe sodium restriction is not necessary and avoiding high sodium intake should be the goal. In more advanced congestive heart failure, more additional restriction of sodium may be beneficial.

4. Calorie adjustment. Many dogs and cats (and people) tend to gain weight as they age. In these obesity-prone animals, decreasing the number of calories eaten (either by feeding less or changing to a food with a lower caloric density) will help to prevent weight gain. Those extra pounds around the middle are not innocuous and can cause or exacerbate other diseases. On the other hand, not all animals gain weight as they age. If a patient is one that is gradually losing weight or muscle with aging and there is no underlying medical
condition, a more calorically dense (and possibly also with some adjustments in other nutrient levels) should be selected to help to prevent weight loss.

5. Fiber. Increased soluble or insoluble fiber intake may be useful for dogs and cats that have decreased intestinal motility and are prone to constipation, but high fiber foods may not appropriate for animals with trouble maintaining weight since high fiber foods are generally low in calories.

6. Supplemental vitamins and minerals. If a good quality commercial food that has undergone AAFCO feeding trials is being fed, supplementation is unnecessary. Some nutritional supplements may be helpful in certain diseases, and future research will help to better define where they can be beneficial and where they can cause problems.

There is an increasing interest in geriatrics among the pet food companies. A new niche has developed for special diets for geriatric companion animals so more and better research into their unique requirements will be done. Hopefully, this will provide the necessary information to develop separate nutritional profiles for elderly dogs and cats. Unfortunately, pet food manufacturers will never be able to design a single food that meets the needs of every older animal. Therefore, it is important to evaluate the individual patient’s condition and health status to determine an appropriate food that will maintain a proper body weight and provide optimal nutrient levels.

In selecting the optimal diet for an older animal, the first thing to consider is overall health. If the animal is healthy, in good body condition, and eating a good quality adult food, there is no reason to change foods. If the patient has one of the diseases often seen with aging such as arthritis, diabetes, cancer, dental problems, cardiac disease, or renal failure, dietary adjustments may help improve clinical signs or even slow progression of the disease. For cats with diabetes mellitus, for example, dietary modifications such as increasing fiber or using a high protein, low carbohydrate food may help to control the disease and reduce insulin requirements. Reduced sodium foods may be useful in dogs with advanced congestive heart failure and can help reduce the diuretic dose required. The best diet (or diets) should be based on the individual animal’s clinical signs, laboratory results, and stage of disease. As the disease progresses and medication adjustments are required, further dietary changes also may be necessary. Dietary modification can help to optimize health in the healthy dog and cat and to modulate disease as animals age. The large variety of commercial diets and their variable nutrient contents provides many choices for optimizing the health of the elderly patient.

**Recommended Reading**


3. WSAVA Nutritional Assessment Guidelines Taskforce: Freeman L, Becvarova I, Cave N,

TALKING TO OWNERS ABOUT UNCONVENTIONAL DIETS
Lisa M. Freeman, DVM, PhD, DACVN
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There is a growing number of people who are using unconventional diets for their pet dogs and cats, whether with the objective of preventing disease or treating an existing condition. Because of this, clinicians are increasingly confronted with clients who have concerns about commercial pet foods and it is not always easy to intelligently answer these questions. There are a number of practices currently being touted that have unproven and even questionable benefits and also may pose some risk to the patient who receives them. This talk will give an overview of unconventional dietary management and will review the rationale and appeal of these diets, the different types of unconventional diets, data supporting the pros and cons of these diets, and, most importantly, how to talk to owners about these diets.

TYPES OF UNCONVENTIONAL DIETS
Vegetarian Diets
Some owners wish to feed vegetarian diets to their dogs and cats. Aside from the issues discussed above, in many cases people choose vegetarian diets due to ethical concerns. There are a number of commercial vegetarian diets available on the market but many people will make a homemade vegetarian diet. There are major concerns over feeding a vegetarian diet to a cat. Cats are obligate carnivores and vegetarian diets cannot meet feline requirements. One commercial vegan cat food states on the label that it is not complete and balanced for cats but this may not be readily apparent to owners. Even commercial vegan cat foods that are intended to be complete and balanced may not be so. For example, one study that analyzed two commercial vegan diets showed that neither met the minimum nutrient amounts in the Association of American Feed Control Officials (AAFCO) Cat Food Nutrient Profiles and thus were not appropriate as a sole source of nutrition for cats. Most feline vegetarian diets are homemade and many recommend the addition of a nutritional supplement intended to provide the nutrients missing in a vegetarian diet. Even with this supplement however, the nutritional adequacy of vegetarian diets has not been proven and is highly questionable.

Some authors will admit that a vegetarian diet is not ideal for cats but claim that dogs are easily able to adapt to a vegetarian diet. There are several commercial vegetarian diets on the market. The long-term nutritional adequacy of these diets for dogs is unclear. Other dog owners who wish to feed a vegetarian diet formulate a homemade diet for their dogs. This introduces the additional problems with homemade diets (see below). One survey study of homemade and commercial vegetarian diets for dogs and cats in Europe showed that nutritional problems were nearly universal with these diets.

Homemade Diets (cooked)
Some owners elect to feed a homemade diet to their pets. Knowing the reasoning behind the desire to feed a homemade diet can be helpful in addressing the positive and negative properties of these diets. There are hundreds of recipes available for homemade diets. These recipes may be empirically based on an owner’s perception of a pet’s nutritional
requirements (i.e., a random combination of meat, grains, and vegetables) or may be obtained from books, magazine articles, or the Internet. In addition, some credentialed veterinary nutritionists will formulate nutritionally balanced homemade diets at the request for owners (see ACVN Nutrition Resources link: www.acvn.org).

While a nutritionally balanced homemade diet can be formulated, most of the recipes used by pet owners are unbalanced - some extremely so. Studies and the clinical experiences of veterinary nutritionists support the fact that most homemade diets, unless very carefully designed and executed, are nutritional unbalanced. Some of these imbalances are severe enough that they could cause serious health problems when used long-term. The most common deficiencies are of calcium, zinc, iron, and other trace minerals but can vary widely between diets. Excesses also can occur but depend upon the type and amount of supplementation used.

**Raw Meat Diets**

A variety of different types of raw meat diets are currently being fed to dogs and cats.

The main three categories of raw food diets are as follows:

1. **Commercially available "complete" raw meat diets:** These diets are intended to be complete and balanced without the need for additional supplements. These diets typically are sold in a frozen form but sometimes are dehydrated.

2. **Homemade complete raw meat diets:** Many recipes for homemade raw meat diets are available in books, articles, and the Internet. The most popular homemade raw food program is the Bones and Raw Food or Biologically Appropriate Raw Food (BARF) diet but there are many others, such as the Ultimate diet and the Volhard diet. The BARF diet advocates a diet “consisting of 60% raw, meaty bones, "with the rest being made up of a "wide variety of foods, based on the type and quantity of foods a wild dog would eat." Those other foods would include "lots of green vegetables (to mimic stomach contents of prey), some offal (liver, kidneys, etc.), meat, eggs, milk, brewer's yeast, yogurt, and small amounts of grains and legumes." The diet is expected to be balanced overall, but each meal is not balanced. For instance, the diet recommends feeding green leafy vegetable meals, starchy meals, grain and legume meals, meat meals, milk meals, offal meals, and food scrap meals during period of 2 to 3 weeks. A typical schedule could include 10 meals of bones combined with 4 meals of green leafy vegetables, 1 meal of starchy food, 1 meal of grains and legumes, 1 meal of meat alone, 2 meals of milk, and 1 or 2 meals of offal during the period of 2 to 3 weeks.

3. **Combination diets:** These consist of commercially available grain-and-supplement mixes. The grain mix is to be fed in combination with raw meat.

Just like standard homemade diets, homemade raw meat diets are likely to have nutritional imbalances. One study showed a variety of nutritional problems, both deficiencies and excesses, in homemade raw diets based on various recipes. Even commercial complete or combination diets had nutritional imbalances that could put the pet at risk for health problems in the animals eating them, especially growing animals. Additional potential problems with raw food diets (both homemade and commercial) relate to safety. The raw bones included in many of these diets carry risks and, while the actual incidence of complications resulting from ingestion...
of raw bones is currently unknown, there are reports of intestinal obstruction, gastrointestinal perforation, gastroenteritis, and fractured teeth that have occurred in animals consuming raw diets. Finally, uncooked meat carries with it the risk of bacterial contamination. Although proponents of the diets argue that dogs are more resistant to bacteria than are people, this has not proven to be true. Raw meat diets can also pose a risk to the pet owners making the diets, especially those that are very young, elderly, or immunosuppressed because of their potential for bacterial contamination (eg, Salmonella, E. coli 0157:H7).

RECOMMENDATIONS

I generally try to find out why owners wish to feed unconventional diets and, if the reasons are based on myths or misperceptions, try to clear these up. Owners may still decide to feed an unconventional diet but my goal is to ensure they make the decision based on facts, rather than inaccurate information. If owners decide to feed an unconventional diet, even after knowing the facts, it’s also important that they understand the potential risks and know what problems to look for. If owners wish to feed a homemade diet, I strongly recommend that it be cooked, that it contain meat, and that is formulated by a credentialed nutritionist. Qualifications for nutritionists are ill-defined so it is important to check credentials. Some board-certified veterinary nutritionists will formulate balanced homemade diets for referring veterinarians or directly to owners – see ACVN Nutrition Resources link: www.acvn.org. Finally, if owners do elect to use a nutritionally balanced, cooked homemade diet, careful monitoring is necessary as subclinical deficiencies still can occur when used long-term.

Additional Reading on Raw Food Diets:


**Top Ten Myths about Raw Meat Diets**

1. **"Their benefits are proven"**
   No scientific studies have shown benefits of raw food diets. Their appeal is based on word of mouth, testimonials, and perceived benefits. For example, raw food diets may result in a shiny coat and small stools because they are generally high in fat and digestibility. However, these same properties can be achieved with commercial cooked diets without risks of raw meat diets.

2. **"This is what animals eat in the wild"**
   Wolves in the wild do eat raw meat (in addition to berries, plants, etc). However, the average lifespan for a wolf in the wild is only a few years. Therefore, what is nutritionally "optimal" for a wolf is not optimal for our pets who we hope will live long and healthy lives.

3. **"Dogs and cats have short gastrointestinal tracts so won't get Salmonella infections"**
   Dogs' and cats' gastrointestinal tracts are not shorter compared to people when viewed in proportion to their smaller body size. Dogs and cats can become infected with Salmonella and other bacteria found in raw meat diets, just as people can (especially young, old, or immunosuppressed individuals).

4. **"Raw food diet ingredients are human grade"**
   Even meats purchased at the best of stores for people can be infected with bacteria so purchasing "human grade" meat does not protect against the health risks of uncooked meats (would you eat raw hamburger?). Also, be aware that the term "human grade" has no legal definition for pet food.

5. **"Freezing raw diets kills bacteria"**
   Most of the bacteria found in raw meat diets can easily survive freezing.

6. **"As long as bones are raw, they're safe"**
   Bones, whether raw or cooked, can fracture dogs' and cats' teeth. Bone also can block or tear the esophagus, stomach, or intestine.

7. **"Cooking destroys enzymes needed for digestion"**
   All the enzymes that dogs and cats (and people) need for digestion are already in the
gastrointestinal tract. Therefore, additional enzymes from food are not required for digestion. In fact, enzymes are proteins so any enzymes that are eaten get broken down by the body and have no benefit in the digestion process.

8. "Grains are added to pet foods as fillers"
Corn, oats, rice, barley, and other grains are healthy ingredients that contain protein, vitamins, and minerals; they are not added as fillers. There is no benefit of potatoes, sweet potatoes, peas, or oatmeal compared to other carbohydrate sources, unless the animal has certain specific health problems.

9. "Most commercial pet foods contain harmful ingredients"
By-products are the animal parts that Americans don't typically eat, such as livers, kidneys, or lungs. By-products have specific legal definitions for what they can and cannot include. For example, by-products must be the clean parts of slaughtered animals and cannot include feathers, hair, horns, teeth, and hooves. Basically, by-products are the organs. Note that some pet foods actually list these ingredients (eg, duck liver, beef lung) but these are really just "by-products."

10. "If bones or chicken necks are added to raw meat diets, they're nutritionally balanced"
Most homemade (and even some commercial) raw meat diets are extremely deficient in calcium and a variety of other nutrients, even if chicken necks, bones, or egg shells are added. This can be disastrous in any animal but especially in young, growing pets.

Also available as a handout on the Massachusetts Veterinary Medical Association's website:
CRITICAL CARE NUTRITION
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The goal of nutritional support is to provide the patient with adequate caloric and nutrient intake in order to prevent the adverse consequences of malnutrition and to optimize patient outcomes. However, in a busy practice it is common to have vaguely written or absent feeding orders and nutritional intake information. The aims of this talk are to review how to assess the nutritional needs of hospitalized patients and how to use that information to develop an individual nutrition plan for each patient. This talk will focus primarily on enteral nutrition, which can be incorporated into every practice.

Why Feed the Hospitalized Patient?
Adequate intake of calories and other nutrients is critical for the optimal care of hospitalized patients. A healthy animal that does not get enough calories will lose primarily fat if not provided with sufficient calories. In contrast, a sick or injured animal will lose lean body mass when it is not given adequate calories because it cannot make the adaptive responses necessary to use fat (instead of protein) as an energy source. Therefore, the sick or injured patient continues to mobilize protein (i.e., stressed starvation):

\[
\begin{align*}
\text{Intake} & < \text{Requirement} \\
\downarrow & \\
\text{Amino acid mobilization} & \\
\downarrow & \\
\text{Decreased protein turnover} & \quad \text{No adaptive response} \\
\downarrow & \\
\text{Preferential fat utilization} & \\
\downarrow & \\
\text{Conservation of lean body mass} & \quad \text{Continued loss of lean body mass} \\
\end{align*}
\]

This loss of lean body mass impairs the animal's strength, immune function, wound healing, and overall survival. When oral intake is not possible, either because of contraindications or because the animal will not eat sufficient amounts voluntarily, nutritional support techniques are then needed to provide some or all of the nutrient requirements. The optimal route for feeding in these patients depends upon a number of patient-dependent issues including the function of the GI tract, the patient's ability to tolerate tube placement, and risk of aspiration, as well as non-patient issues such as cost and technical expertise and support.

Developing the Plan
Proper nutritional assessment will allow the clinician to develop an appropriate nutrition plan which will address who to feed, when to feed, where to feed, and what to feed.

\textit{Who to feed}
Every patient in whom feeding is not contraindicated needs precise written feeding orders and the amount of food consumed must be recorded, which should include route, diet, amount, and frequency (e.g., Feed Diet X orally – ⅓ can TID). There are very few circumstances that require total withholding of food. However, patients who are not hemodynamically stable should first be stabilized before nutritional intervention is initiated. As a general rule, patients who are dehydrated, hypotensive, or hypothermic should not be fed until these gross abnormalities are corrected. It is important to correct major abnormalities in acid-base status, electrolytes, and aim toward normalizing blood glucose but be aware that labwork does not need to be "perfect" before feedings can be instituted. However, recognize the abnormalities, take them into account when developing the nutrition plan and know how to monitor and adjust the plan as parameters change. Patients who are unable to protect their airway or patients otherwise at risk of aspiration should not be fed orally but may still receive nutritional support via an appropriate enteral feeding tube (eg, gastrostomy or jejunostomy tube) or parenteral nutrition. A patient with uncontrolled vomiting may not be able to be fed via the upper GI tract but a distally placed feeding tube such as a jejunostomy tube or parenteral nutrition may meet his nutritional needs. Patients undergoing anesthesia will need to have food withheld. However, patients undergoing frequent anesthesia (e.g. patients undergoing daily wound debridement) need to have a plan that allows adequate time for the patient to eat to meet nutritional needs.

*When to feed*

Given the catabolic stress associated with critical illness, patients anticipated to be anorectic or NPO for longer than 3 days need a nutrition plan. Patients with significant clinical complications in addition to its primary illness such as aspiration pneumonia or sepsis require aggressive nutritional support. These patients are more likely to have a prolonged hospitalization and a greater degree of catabolic stress. In all of these cases, start feeding as soon as the patient is hemodynamically stabilized rather than play a "wait and see game" as to whether they might start eating on their own. Remember that many patients have been partially or fully anorectic at home prior to presentation, so a newly admitted patient may already have been anorectic for several days. Also, if an animal is going to be anesthetized or heavily sedated for diagnostic or therapeutic procedures, be sure to take the opportunity to place a feeding tube if there is any indication that one may be needed.

*Where to feed*

Adequate intake of calories and other nutrients is critical for the optimal care of hospitalized patients. When oral intake is not possible, either because of contraindications or because the animal will not eat sufficient amounts voluntarily, nutritional support techniques are then needed to provide some or all of the nutrient requirements. The optimal route for feeding in these patients depends upon a number of patient-dependent issues including the function of the GI tract, the patient's ability to tolerate tube placement, and risk of aspiration, as well as non-patient issues such as cost and technical expertise and support. Below is a flow chart illustrating one way to help decide which route to use for nutritional support:
Enteral nutrition likely for >5 days
Laparotomy not indicated→PEG or E tube

Patient anorectic→Upper GI→Jejunostomy tube
or unable to eat non-functional

Enteral nutrition→ PPB
Entire GI likely for <5 days non-functional (and normal requirements)

Parenteral nutrition→TPN
likely for >5 days
(or high requirements)

Key: NE tube = nasoesophageal tube, G tube = gastrostomy tube, PEG tube = percutaneous endoscopically-placed gastrostomy tube, E tube = esophagostomy tube, PPN = partial parenteral nutrition, TPN = total parenteral nutrition

Whenever possible, the enteral route should be used because it is the safest, most convenient, most physiologically sound, and least expensive method of nutritional support. In addition, enteral nutrition supports normal GI structure and function. It is critical to think about the route of feeding before doing any procedures on hospitalized animals. Not placing a feeding tube at the time of anesthesia often results in suboptimal feeding - few clinicians want to anesthetize a patient a 2nd time "just to place a tube." If an animal is likely to be anorectic or unable to eat voluntarily and has a functional gastrointestinal tract, consider placing a tube when the animal is sedated or anesthetized for a diagnostic or therapeutic procedure. In cases where the patient is vomiting, has pancreatitis or severe malabsorption, or an inability to guard the airway, parenteral nutrition would be preferred.

Options for Feeding Tubes

There are a number of options for enteral nutrition, which will depend upon the length of time the animal is expected to require nutritional support, ability to tolerate anesthesia, nutritional status, hospital facilities, cost, and the clinician's comfort level with different techniques for tube placement.

- Nasoesophageal tubes. A 3.5-5 Fr feeding tube (silicone, polyurethane, or red rubber) is placed into the distal esophagus through the nares. Placement into the distal esophagus is preferred over placement into the stomach to decrease the risk of gastric reflux. Placement usually does not require sedation (although may require light sedation in very difficult animals). The tube should be secured in place with suture or glue. Nasoesophageal tubes require a liquid diet and are best utilized for short term (ie, <5 days) nutritional support.
• Esophagostomy tubes. These are quick and easy to place with Carmalt forceps or, for large dogs, a Cook placement device or Eld tube (ie, an endoscope is not required). Placement does require general anesthesia or heavy sedation. Post-placement radiographs should be taken to confirm proper placement. This, along with the gastrostomy tube, would be the tube of choice for long-term nutritional support (weeks to months). One advantage of an esophagostomy tube over a gastrostomy tube is that if they are removed earlier than intended, the only risk is cellulitis (vs peritonitis with gastrostomy tubes). Because of the larger tube size (vs nasoesophageal tubes), a larger selection of diets with higher caloric density is possible. A tube size of at least 14 french (and usually at least a 19 french in larger animals) is recommended.
• Gastrostomy tubes. Another excellent choice for long-term enteral nutrition. Gastrostomy tube placement requires surgery or endoscopy (blind placement techniques are possible but are not recommended), but is a quick procedure. Gastrostomy tubes should be left in at least 10-14 days before removal to allow formation of a fistula. Because of the larger tube size (18-24 french), a larger selection of diets with higher caloric density is possible. A gastrostomy tube would be the preferred tube for any animal with esophageal disease. Note: Use the largest tube the animal can comfortably tolerate; this will allow more choice in diet selection, and will decrease the chances of clogging.
• Jejunostomy tubes. These are typically placed surgically, although endoscopic placement techniques are being developed. The use of jejunostomy tubes is primarily restricted to in-hospital use and due to the small luminal diameter, liquid diets are necessary.

Tube Care
The care of enteral feeding tubes is important for their success and carefully securing them in place is the first step. All animals with nasoesophageal tubes should wear an Elizabethan collar at all times. A light bandage should be used for all esophagostomy, gastrostomy, and jejunostomy tubes. The bandage should be changed and the tube site should be checked frequently for redness, swelling, discharge as well as for tube displacement. Some animals with these types of tubes will also need E-collars to prevent them from chewing or dislodging them. Owners should be instructed to bring the patient in for evaluation if they have any concerns about the placement of the tube. NE and E-tubes may dislodge if a patient vomits while displacement of a G-tube or J-tube is a surgical emergency if a fistula has not yet formed. All tubes should be flushed with warm water before and after each feeding to prevent clogging. Keeping the tube capped between feedings also is important. Finally, many medications are incompatible with enteral formulas or may clog the tube.

What to Feed
A number of options are available for diets to use in animals with feeding tubes; the choice depends on patient factors such as concurrent medical conditions which may impact the desired nutrient profile, and non-patient factors such as the type of tube in place, diet availability, and cost. The major classification of commercial enteral diets is either polymeric (containing intact protein, fat, and carbohydrate) or elemental (composed of amino acids,
simple sugars, and triglycerides).

- Veterinary enteral “recovery or critical care” diets - diets that meet canine and feline nutritional requirements and tend to be relatively caloric dense
  - CliniCare/CliniCare RF (Abbott) - liquid diet
  - Iams canine/feline Maximum Calorie
  - Royal Canin Recovery RS
  - Hill's canine/feline a/d

- Blenderized veterinary therapeutic pet foods - blenderized with water and then strained. Generally used when veterinary enteral diets are contraindicated (eg, need a low fat or reduced protein diet)
  - Eg, Royal Canin Low Fat, Hill’s k/d

- Human enteral products: There are a number of human enteral products that could be used in dogs and cats. However, none meet canine or feline requirements as is. This doesn't mean that you can't use them; just that you need to make adjustments [almost all human enteral products require additional protein, B-vitamins, arginine, and taurine for use (for short term use) plus calcium, zinc, iron, and choline (for long term use)].

  Nasoesophageal and jejunostomy tubes require liquid diets (eg, CliniCare). Esophagostomy and gastrostomy tubes, because of their larger size, allow use of a wider variety of diets, including those that are more calorically dense than liquid diets. Iams Maximum Calorie, Hill's a/d, or Royal Canin Recovery are generally the first choices unless a specialized diet is required (eg, a low fat diet or a reduced protein diet). However, the differences in nutrient profile between these diets should be considered.

  Enteral feedings can be given either as a bolus or continuous rate infusion. With either method, be sure to start slowly often feeding to meet 50% of resting energy requirement on the first day then increasing the amount if the patient is tolerating the feedings. Bolus feeding: usually start with 4-6 feedings per day in the hospital administered over at least 10-15 minutes. Food should be fed warmed to between room and body temperature. When sending the animal home, it is best to try to adjust to TID feedings. The maximum bolus size (based on stomach capacity for most animals with tubes) is usually about 20 ml/kg (although some animals will not tolerate this much as a bolus). Continuous feedings are a good choice for patients that don't tolerate bolus feeding (eg, delayed gastric emptying, ileus). This method should be used in nearly all patients with jejunostomy tubes.

Potential Complications and Patient Monitoring

Feeding tubes are generally well tolerated, with few serious complications. However, complications can arise, especially if careful monitoring is not included in the treatment plan. Potential complications include:

- Mechanical
  - Clogged tube
    - When using blenderized diets, be sure to blend completely and put through a sieve twice.
    - Do not administer crushed tablets through NE or J tubes. Check for compatibility when using E or G-tubes (and be sure to crush well).
- Always flush well with warm water after each feeding.
- If a tube does clog, there are 2 main methods for unclogging them:
  - Inject solution of 1/2 tsp Viokase +325 mg sodium bicarbonate in 5 ml warm water. Let sit for 5 minutes, then flush.
  - Inject 5 ml warm soda into tube, let sit for 5 minutes.
- Pulmonary aspiration
  - Always mark the tube where it exits the body with an indelible marker so you can assess whether it has migrated (in or out). Aspiration also can be due to dysphagia, impaired swallow reflex, megaesophagus, or displacement of a more proximally placed tube such as an NE or e-tube should the patient vomit. Aspiration also can occur as the result of misplacement of the tube in respiratory tract-always check the positioning of the tube before feeding and stop if the patient shows any discomfort or coughing while slowly administering the initial water flush.
  - Consider feeding animals at risk for aspiration in an upright position.
- Esophageal erosion (from nasoesophageal or esphagostomy tubes). This is especially when using red rubber tubes.
- Inadvertent removal
  - Always use an E-collar for nasoesophageal tubes and a bandage or stockinette for all other types of tubes.
- Pressure necrosis (with gastrostomy tubes)
  - Be careful not to place too much tension on the tube where it exits the body.

- Metabolic
  - Congestive heart failure (be sure to adjust concurrent IV fluids accordingly when initiating enteral nutrition. Multiple tube flushes can contribute significantly to daily water intake.)
  - Refeeding syndrome (ie, hypophosphatemia/hypokalemia/hypomagnesemia). This is most likely in animals that have not eaten for significant periods of time. This syndrome was more common in the past when very high administration rates were used. Monitor electrolytes during the first 12-72 hours after initiating feedings.

- Gastrointestinal
  - Vomiting
    - Metoclopramide or other anti-emetics are sometimes helpful
    - Inappropriate placement of gastrostomy tube can pull stomach out of normal position and inhibit gastric emptying.
  - Distention/cramps/diarrhea. This can be due to the animal's underlying disease, overly aggressive feeding, or a hyperosmotic diet.
    - Try switching to an isosmotic, reduced fat diet or diluting the current diet with water.
    - Feed smaller, more frequent meals
    - Be sure to warm the diet to room temperature before feeding.
    - Consider continuous rate infusion instead of bolus feeding.
Note that enteral products can become contaminated. Refrigerate all enteral products after opening, and discard opened diets after 48 hours. For gastrostomy tubes, always aspirate the tube before feeding (note that this is not needed for other tubes) and note amount obtained in the record. Residual volumes are not always an indication for discontinuation of feeding, but may justify smaller, more frequent feedings (or even continuous infusion), the addition of medications, or both. Routine monitoring of patient parameters is critical. Monitoring will depend upon the individual case, but at least measure:

- Body weight
- Tube site
- Gastrointestinal signs (vomiting, regurgitation, diarrhea, abdominal discomfort)
- Glucose and electrolytes

**Discontinuing Enteral Feeding**

As soon as the animal is medically able to voluntarily ingest food, it should be offered palatable diet regularly (before each feeding). When the patient is voluntarily consuming at least 60% of its energy requirements, enteral feedings can be gradually decreased. Do not pull the tube until you are sure the animal is eating enough and will continue to do so! Gastrostomy and jejunostomy tubes must remain in place until a fistula forms (typically 10-14 days but may longer in protein depleted, malnourished patients or patients receiving corticosteroids).

**Parenteral Nutrition**

When patients are unable to tolerate enteral feeding, parenteral (intravenous) nutrition should be considered. A veterinary facility with the ability to obtain and maintain aseptic vascular access, to provide attentive 24-hour nursing care, and to perform in-house serum chemistry analysis can provide parenteral nutritional support. Indications for parenteral nutrition include patients with protracted vomiting, severe malabsorption, prolonged ileus, or at high risk of aspiration. Therefore patients with severe pancreatitis, marked protein losing enteropathy, or patients unable to guard their airway exemplify cases in which parenteral nutrition is likely indicated. Parenteral nutrition can be classified as total parenteral nutrition (TPN) or partial parenteral nutrition (PPN). TPN provides 100% of an animal's resting energy requirements and must be administered through a dedicated central catheter, such as a jugular catheter, because of its hypertonicity (generally >1000 mOsm/L). Typical TPN admixtures provide energy, protein, certain water soluble vitamins and trace minerals, and may contain additional electrolytes such as potassium. PPN can provide approximately 70% of energy requirements and 50-100% of protein requirements and can be administered through a dedicated jugular or peripheral vein catheter. Because PPN does not provide all energy requirements, it is only intended for short-term use (e.g., 3-5 days) in a patient that is not already debilitated or in a patient who will concurrently be receiving a portion of his nutritional needs via the enteral route. Animals that are already malnourished, those that have high nutritional requirements, or those with on-going protein loss (e.g., patients with large draining wounds, open abdomen, or severe protein losing enteropathy), or those that have been anorectic for a prolonged period should receive TPN instead.

With either form of parenteral nutrition, it is critical to correct any major fluid, electrolyte, or acid-base abnormalities before initiating parenteral nutrition. Prior to initiating
parenteral nutrition, it should be established that the patient is fluid tolerant for the volume of PN to be administered. Evaluation of the patient for conditions which may cause specific nutrient intolerances (such as the hepatic or renal failure patient who may require decreased protein) should be considered. Similar to enteral nutrition, simple worksheets for calculating TPN and PPN requirements can be made available in the hospital for easy use. TPN should be started gradually (i.e., 50% of total requirements on day 1, 75-100% of total requirements on day 2). Therefore, it will take 2-3 days to reach the animal's total nutritional requirements. Therefore, TPN is typically utilized in patients in which it is anticipated that intravenous nutritional support will be necessary for at least 3 days. Since PPN is only providing a portion of the animal's energy requirements, it can be started at the full rate. All parenteral nutrition should only be administered through an accurate fluid pump.

Catheter care is critical to successfully using parenteral nutrition. Whether using a jugular vein catheter for TPN or a peripheral catheter for PPN, the patient's catheter is a dedicated line. That means that is should not be used for administering medications, collecting blood samples, or measuring central venous pressure. Either a separate dedicated catheter or a clearly labeled dedicated line of a multilumen catheter can be used. Calculations for parenteral nutrition formulas supply 24 hours worth of parenteral nutrition so the bag will need to be changed at the end of every 24-hour period. In between the bag changes, the line from the bag to the animal's catheter should not be broken for any reason. When walking dogs, the line should be removed from the pump, the drip rate should be adjusted (slowed so the dog only receives a small amount during the walk but not clamped off completely which could cause the catheter to clot) and the bag should be carefully carried during the walk. When the bag is changed at the end of every 24-hour period, the lines also should be changed. All handling of the lines and catheters should be done with aseptic technique including sterile placement of catheters and utilizing sterile gloves during bag and line changes to minimize the risk of contamination. The catheter should be carefully rewrapped each day so that the catheter site can be inspected for redness, swelling, or discharge. Finally, catheter type is important to reduce the risk of thrombophlebitis. Long catheters composed of silicone, teflon, or polyurethane should be used for all patients receiving parenteral nutrition.

Routine monitoring is essential to identify potential complications of TPN. The clinical situation should dictate the need as some patients will need more and some less monitoring. Each case is an individual but at least the following should be measured daily in all animals receiving PN:

- Heart/respiratory rate
- Attitude
- Temperature
- Electrolytes (i.e., sodium, chloride, potassium, phosphorus, and magnesium; especially in animals with pre-existing abnormalities, severely ill patients, or in those that have been anorectic >1 week)

Complications of parenteral nutrition are not uncommon but are generally minor. More serious complications can be prevented by recognizing high risk patients and with careful monitoring. Possible mechanical complications include catheter occlusion, line disconnection/breakage, and thrombophlebitis/thromboembolism. The risk for mechanical complications can be reduced by using appropriate catheters, good catheter and line care, and
monitoring the catheter site on a daily basis. Recognize that certain patients such as those with receiving high doses of corticosteroids or those with protein losing conditions are at higher risk of thromboembolism. Metabolic complications are one of the most common occurrences but in most studies, these have been relatively minor and do not require discontinuation of the parenteral nutrition. Hyperglycemia is the most common metabolic complication but can be minimized by using conservative formulations for parenteral nutrition, by starting TPN slowly (PPN can be started at 100%), and by careful monitoring. If glucose concentrations are >200 mg/dL, concurrent insulin administration is recommended. Hypoglycemia can occur with abrupt discontinuation of TPN so patients should be weaned off TPN over 4-6 hours; PPN can be discontinued abruptly. Refeeding syndrome (ie, hypokalemia, hypophosphatemia, and hypomagnesemia) also can occur in animals receiving parenteral nutrition. This occurs most commonly in animals that have not eaten for significant periods of time or in animals with certain predisposing conditions such as diabetic ketoacidosis. This condition usually occurs within 2-3 days of initiating feedings. Therefore, close monitoring and supplementation of additional electrolytes as dictated by the individual patient’s electrolyte profile can prevent serious consequences. Another possible metabolic complication is hypertriglyceridemia. In animals with pre-existing hypertriglyceridemia, the amount of lipid included in the TPN or PPN formulation should be reduced. If hypertriglyceridemia develops, reformulation with a lower lipid content (or without any lipid if triglyceride levels are very high) is necessary.

The complication that is most concerning is sepsis. Fortunately, if careful protocols to compound and administer parenteral nutrition are used, the risk for sepsis is low. Nonetheless, sepsis must be considered in every patient on parenteral nutrition that develops a fever. However, other causes of fever/sepsis should also be considered and ruled out before incriminating parenteral nutrition. If sepsis is suspected, the blood, the parenteral nutrition solution, and the catheter should be cultured.

Once the patient is able to eat, it should be offered food regularly to assess its appetite. The type and amount of food consumed should be carefully recorded. If the gut becomes functional but the patient still will not eat, enteral nutrition techniques should be considered. Oral or enteral nutrition should be used as soon as possible so that gut atrophy can be minimized. When the patient is consuming >60% of its resting energy requirements, parenteral nutrition can be discontinued. For TPN, it should be gradually decreased over a period of 4-6 hours; PPN can be discontinued abruptly.

If a hospital uses parenteral nutrition frequently, it may be most economical to train a technician to compound the parenteral nutrition formulas using sterile technique. If it is not used frequently, it usually is more economical to have parenteral nutrition compounded by a local human hospital or by a home human healthcare company.

Additional Reading

Patient Name ____________________________ Case # ______________________

Date EN Initiated ________________________ Date EN Ended ________________

Current Body Weight _______________________ kg

1. Resting energy requirement (RER)
   70 (weight in kg)\(^{0.75}\) = kcal/day
   or for animals > 3-25 kg, can also use:
   30 (weight in kg) + 70 = kcal/day
   
   RER =

2. Product selected ____________________________

   Contains _______________________ kcal/ml*

   *When diluted appropriately to go through tube

3. Total volume to be administered per day:
   \[
   \text{RER} = _____ \text{ml/day}
   \]
   \[
   \text{kcal/ml in diet}
   \]

4. Administration schedule:
   \[
   1/2 \text{ of total requirement on Day 1} = _____ \text{ml/day}
   \]
   \[
   \text{Total requirement on Day 2} = _____ \text{ml/day}
   \]

5. Feeding schedule: Divide total daily volume into 4-6 feedings
   (depending on duration of anorexia, patient tolerance)
   
   =
   
   ________ feedings/day

6. Calculate volume per feeding:
   \[
   \text{Total volume requirements/day}
   \]
   \[
   \text{Number of feedings} =
   \]
   \[
   \text{ml/feeding(Day 1)}
   \]

   =

   ________ ml/feeding(Day 2)

7. Instructions for bolus feeding:
a. If using a gastrostomy tube, aspirate the tube before feeding (note and record residual volume). This is not necessary for esophagostomy or jejunostomy tubes.
b. Flush tube with _____ml warm water (amount depends on type of tube being used).
c. Administer warmed diet over 3-5 minutes. If any vomiting occurs, stop the feeding and reassess the animal's condition and whether enteral feeding can be continued. Smaller, more frequent feedings or a different diet may be necessary, or it may be necessary to start parenteral nutrition instead.
d. Flush tube with _____ml warm water. Recap the tube and rewrap, if necessary.

Commonly used diets for feeding tubes (dogs or cats):
- Iams Maximum Calorie: 1 can + 25 ml water = 1.8 kcal/ml
- Hill's a/d: 1 can + 25 ml water = 1.0 kcal/ml
- Royal Canin Recovery: 1 can + 25 ml water = 1.0 kcal/ml
- CliniCare Canine/Feline: Use straight from can (1 kcal/ml)
- CliniCare RF: Use straight from the can (1 kcal/ml)
SUCCESSFUL WEIGHT LOSS PROGRAMS
Lisa M. Freeman, DVM, PhD, DACVN
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North Grafton, MA

Obesity is the most common nutritional disorder affecting dogs and cats in the United States (and, now, other countries), and its treatment is extremely challenging. Therefore, it is important to try to prevent obesity – this is much easier than treating obesity once present. Feeding directions are required on pet food labels but the quality of the information varies greatly. Feeding directions should be viewed only as a starting point for an individual animal. The veterinarian and the owner must monitor the individual animal’s response. Owners can be taught to accurately assess body condition at home so that they can adjust the amount of food during growth spurts and plateaus to maintain a trim body condition (between 4 and 5 on a 1-9 scale). Adjusting the amount of food is particularly important after neutering the pet, at which time the energy requirements decrease 10-15% within a very short time of the surgery. Be sure to talk to the owner about treats and table food as these can be a major contributor to obesity. Puppies and kittens should eat a food that has gone through feeding trials for growth until they are 1 year old (18 mos for giant breed dogs). If they are becoming overweight before 1 year of age, they should be switched to a puppy or kitten food that is lower in caloric density but still meets the requirements for growth.

If prevention does not work and the animal becomes overweight, a weight loss program must be designed to achieve the optimum body weight. The key to successful weight reduction is a comprehensive program - this means controlling the calories (from all sources), increasing exercise (if possible), and changing behaviors that contribute to obesity. The bottom line is that to achieve weight loss, a reduction in calories below baseline requirements is necessary. All calories from pet food, treats, and table food must be addressed. To do this, it is critical to get a thorough diet history. Most owners will easily admit to the type of pet food and the amount, but it is often more difficult to get a complete story on treats, table food, and other sources of calories without asking very specific questions about these areas. This history can be obtained by having the owner complete a diet history form while waiting or by having a trained veterinary technician administer it.

Once the diet history is reviewed (additional clarification may then be needed), a plan can be made that will avoid the problems encountered with the individual owner/pet. This plan must control of the quantity and type of pet food, restrict treats and table food, limit access to all other sources of calories (children, other pets, neighbors, grandparents, etc), and provide exercise. First, an initial goal weight should be selected. This should be a reasonable goal for the owner (ie, if a 25 pound cat really should weigh 12 pounds, a reasonable initial goal might be to lose 6 pounds). Owners can easily get discouraged if the initial goal is unreasonable. Also, if they are successful in the initial goal, they are much more willing to continue. Caloric requirements to reach the initial weight goal can be calculated as follows:

\[ \text{RER (in calories/day)} = 70 \times (\text{goal weight in kg})^{0.75} \]

I use the RER for the goal weight as the daily calorie requirement (ie, I do not use an activity factor to calculate a MER). Reducing the number of calories eaten requires controlling both the
pet food and the treats and table food. The total number of calories required per day is then divided by the pet food selected to determine the number of cups or cans required per day:

$$\text{Calories/day} \div \text{calories per can or cup} = \text{cans or cups/day}$$

The total amount of food/day should be divided into at least two meals per day. The new food should be introduced gradually and the owner should monitor for changes in feeding behaviors. If the pet appears hungry in between meals, dividing the food into additional meals (TID or QID) may be helpful. The owner must be instructed to measure the foods exactly at each meal to prevent overfeeding. It is recommended to select a diet that is reduced in calories compared to regular diets. Generally, a diet that is reduced in calories will allow a larger volume of food to be fed which is helpful for most overweight dogs or cats. A reduced calorie food also makes it less likely to have nutritional deficiencies from feeding extremely small amounts of a higher calorie food. Be careful about just making a general recommendation to the owner to switch to a “low calorie” pet food because many of the diets that are marketed as low calorie diets may not be very low in calories. Pet foods marketed as "weight reduction" diets can vary tremendously in terms of calorie density! So, it is important to select a specific diet for overweight cats and to recommend a specific amount to feed (feeding directions often overestimate the amount of food required).

Other properties that may differ between different brands of reduced calorie diets include protein and fiber content. There is speculation that a higher protein content in reduced calorie diets may be helpful in maintaining lean body mass. Certainly, if protein is restricted (this can unintentionally be a problem in cats that require severe calorie restriction in order to lose weight). There also are high protein/low carbohydrate diets marketed for weight loss – these have no magical properties and animals will not lose weight when eating them unless the total calories are sufficiently restricted (which can be difficult with these diets because of their high calorie density). Some reduced calorie diets are low in fiber while others are high in fiber. The effect of fiber on satiety is controversial but high fiber diets may be useful, particularly in animals that appear especially hungry when on a weight reduction diet. Fiber content does alter fecal characteristics and owner preferences may dictate the type of diet selected. Diet changes should be made gradually, particularly when transitioning to a high fiber diet.

Some owners are able to completely discontinue treats and table food but others will need recommendations for acceptable treats. If the owner would like to give treats, work with them to determine a reasonable number of treats/day and the calories provided by the treats should be subtracted from the amount of pet food recommended.

**Monitoring**

Dogs and cats are individuals and can vary tremendously in their calorie requirements. Some may require more calories than you initially estimate in order to have safe, steady weight loss. More commonly, however, they require a reduction in calories from your initial recommendation. Therefore, monitoring is critical to a successful weight reduction program. I recommend that the patient be weighed two weeks after beginning the new diet program. If weight has not changed (or has increased), the owner should first be questioned about possible non-compliance (eg, treats, table food, other pets' food, etc). If compliance is not an issue, then the total amount of pet food should be decreased further. This is often the failure point in a weight loss program. Animals should be weighed every 2 weeks until weight loss (1-2%/week) is achieved. Once the amount of food required for this loss is determined, additional
"weigh-ins" should be done at monthly intervals to ensure slow, steady weight loss. Owners with tractable cats generally bring their cats in for regular weigh-ins but for cats for which the car ride and veterinary office visit is stressful, owners may prefer to purchase a baby scale to weigh the cat at home and then call in with updates.
DIET HISTORY FORM FOR WEIGHT REDUCTION PROGRAMS

Date: _____/_____/

Body weight: _________ lb  _________ lb

Body condition score (1-9):_________________

Current     Usual

The following should be completed by the owner:

1. Is your pet housed: □indoors □outdoors □both □other
2. Please describe your pet’s activity level: □low □moderate □high
   Do you specifically exercise your pet in any way? □yes □no
   If yes, please describe

________________________________________________________

3. Do you have other pets? □yes □no
   If so, how many:   Dogs: ______  Cats: ______  Other: ______

4. How many other people live in your household: ______

5. Who feeds your pet? ____________________________________________

6. How many times per day do you feed your pet?
   □once □twice □three □more than 3 □food is out all the time

7. Do you measure your pet’s food at each meal or estimate the amount? □measure □estimate

8. Do you give your pet any commercial treats or table food? □yes □no
   If yes, are these given: □At regular times each day □In response to begging
   □Other ____________________________________________

9. Do you give any dietary supplements to your pet? □yes □no
   If yes, please list which ones and the doses__________________________________________
   ________________________________________________________________

10. Have you observed any changes in:
    Defecation □yes □no
    Appetite □yes □no
    Activity level □yes □no

11. Have you made any recent changes in diet □yes □no
    If so, please note what the change was and why you made it:
    ________________________________________________________________
    ________________________________________________________________

12. Is your pet receiving any medications? □yes □no
    If yes, please list drugs and doses:
    ________________________________________________________________
    ________________________________________________________________
13. Do you use food (e.g., Pill Pockets, cheese, peanut butter, chicken, etc) to administer pills?
   □ yes □ no If yes, please list what kind(s) and amounts:

14. Please list below the brands and product names (if applicable) and amounts of ALL foods, treats, snacks, and any other foods that your pet eats. This description should provide enough detail that we could go to the store and purchase the exact same food. It should include “people foods” given as treats or at the table.

<table>
<thead>
<tr>
<th>Food</th>
<th>Form</th>
<th>Amount</th>
<th>Number</th>
<th>Fed since</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pro Plan feline adult chicken/rice dry</td>
<td>dry</td>
<td>½ cup</td>
<td>2x/day</td>
<td>Feb, 2004</td>
</tr>
<tr>
<td>Salmon</td>
<td>broiled</td>
<td>2 oz</td>
<td>1x/week</td>
<td>Jan, 2005</td>
</tr>
<tr>
<td>Pounce tarter control chicken treats</td>
<td>---</td>
<td>1</td>
<td>4/day</td>
<td>Nov, 2005</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
TAKE-OUT NUTRITION
Lisa M. Freeman, DVM, PhD, DACVN
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More and more, nutrition is becoming an adjunct to medical therapy. In many diseases, nutrition can not only make the patient feel better but also can help to modulate the disease. Because of the rapidly changing information in this area, it is difficult to keep up to date on the latest information regarding optimal diets for various disease states. One concept that is becoming increasingly important in veterinary nutrition is to view animals as individuals and to feed patients appropriately according to the underlying disease and individual patient characteristics. Not all animals with congestive heart failure (CHF), for example, are the same or require the same diet. Some dogs with CHF will be overweight and some will have lost weight, some will be hypokalemic while others will be hyperkalemic, and a small number will be taurine deficient while most are not. Therefore, the same diet will not meet the requirements of all dogs with CHF. Also, there usually is more than one diet option since several companies now make therapeutic diets so offering the client more than one option is useful so they can find one that the animal likes and eats well. Finally, be careful when giving instructions that the animal eating something less than ideal than to have him not eating the "ideal" diet. Various commercial therapeutic diets will be compared in this talk.

Cats:
Chronic kidney disease

Diet has been shown to be beneficial for slowing progression of CKD in a number of studies. In many cases, nutritional modification is the major treatment for animals with CKD. However, there is no one diet that is the “best” diet for all cats with CKD. Also, there usually is more than one diet option since several companies now make therapeutic diets so offering the client more than one option is useful so they can find one that the animal likes and eats well. If anorexia is an issue, it is preferable to make compromises regarding the "ideal" diet to ensure adequate food intake.

Ideas have changed a great deal in recent years for cats with CKD. It used to be that a highly protein-restricted diet was recommended to "slow progression" as soon as compromised renal function was identified. Now that it has been shown that protein restriction does not slow progression, severe protein restriction is only recommended for advanced renal failure when the animal becomes uremic. In early renal failure, the author typically recommends avoiding high protein diets but does not restrict dietary protein. When disease becomes more severe (the exact cutoffs are not clear but somewhere around BUN>80 mg/dl or creatinine >3.5 mg/dl), then more severe protein restriction can be instituted. When uremia is present, protein restriction can help reduce clinical signs.

Unlike protein, phosphorus restriction does appear to slow progression of renal disease and the level of phosphorus restriction should be related to the degree of renal dysfunction. It is difficult to formulate pet foods that are restricted in phosphorus but not restricted in protein so phosphorus-restricted diets are usually also protein-restricted diets. Newer diets on the
market, however, are now able to provide moderate protein, reduced phosphorus diets. Phosphorus-restricted diets are often not sufficient however, to control hyperphosphatemia. Intestinal phosphate binders are usually required, especially in the more severely affected patients. Because metabolic acidosis is common in animals with CKD, it is important to avoid acidifying diets that will compound this problem. This can be a problem in cats since the majority of commercial feline maintenance diets are designed to produce an acidic urine.

Of greatest importance at any stage of renal failure is to make sure that adequate calories are consumed. This may require a diet change to maintain good food intake. In severe renal failure, it may be impossible to maintain adequate caloric intake with a commercial diet. Some animals will prefer a balanced homemade diet at this stage. Another nutrient that is currently being studied in renal failure is omega-3 fatty acids. Although omega-3 fatty acids may have benefits in renal failure, patient selection, time to institute, and optimal dose are not currently known.

While studies have shown benefits of a diet designed for animals with renal disease for slowing disease progression, it is not clear which components of the diet are most important for these effects so additional research is required. However, the large number of diets that are now commercially available makes nutritional modulation for renal disease much easier. The author generally selects several diets that have the properties needed based on the individual patient’s characteristics and allows the owner and the pet to determine which one they prefer. Treats and table food should also be discussed with the owner so that these do not negate the beneficial properties of the diet.

**Urolithiasis**

Life used to be easy when it could be assumed that all blocked cats had struvite stones so they all could be sent home with an acidifying diet. Now, fewer than 50% of stones are struvite and over 50% are calcium oxalate. Therefore, it becomes more critical to analyze the stones (or at least get a good guess based on urine pH, radiographic density, and urine crystals) to know which diet is most appropriate. In general, the most important goal is to decrease the cat's urine specific gravity. The easiest way of doing this is to use a canned diet. Therefore, the first recommendation for any cat with lower urinary tract disease should be to change to a canned diet. If the cat will not eat canned food, water can be added to the dry food or bottled clam juice can be fed 1-2 times daily to increase water intake.

For struvite stones, a variety of therapeutic diets are specifically designed for cats with struvite stones that produce an acidic urine. One should also note that many other therapeutic diets and even diets from pet stores and grocery stores also produce an acidic urine. However, other factors may also be important in preventing recurrence of struvite urolithiasis. Oxalate stones are more difficult. The mechanism to prevent oxalate stones is not completely clear. Most pet food companies have designed their diets for cats with oxalate stones to produce an alkaline pH. It has not been determined however, whether this alone solves the problem since oxalate stones are not as pH-sensitive. Oxalate stone prevention may require other changes (eg, crystal supersaturation, reducing oxalate precursors such as vitamin C) to alter the solubility of the crystals. In fact, at least two companies now market diets that are designed to prevent both struvite and oxalate stones.

Finally, it also is important to note that the majority of cats with lower urinary tract
disease have idiopathic cystitis. In this case, acidifying or alkalinizing the urine likely has no effect on preventing recurrences. It does appear that, again, changing to a canned food may be the best defense against recurrence in these cats.

**Diabetes mellitus**

In cats, as in dogs, maintenance of ideal body weight should be the primary goal of dietary therapy for diabetes. Although obese cats are predisposed to developing diabetes, diabetic animals often lose weight once they have the disease. If the cat is overweight, a gradual weight loss program can significantly aid in glucose regulation (or, in some cases, correct the problem). In the past, it was recommended that cats with diabetes be fed a reduced calorie, high fiber diet. In some cases, this is still the diet of choice (eg, if the cat is significantly overweight). However, there are now at least two high protein, low carbohydrate commercial therapeutic diets marketed for cats with diabetes. In some cats, use of these diets can be beneficial in reducing insulin requirements or, occasionally, eliminating the need for insulin.

**Dogs:**

**Acute vomiting or diarrhea**

Although the specific treatment of vomiting and diarrhea will depend upon the underlying cause, in general a reduced fat, easily digestible diet is the diet of choice. There are now quite a few commercial diets available for dogs that meet these parameters (Hill's i/d, Purina EN, Eukanuba Low Residue, Royal Canin Lowfat). These diets are preferable to baby food (which is high in protein, fat, and often contains high levels of salt and may contain onion powder) and to homemade diets. Many veterinarians recommend cooked meat and rice for acute vomiting and diarrhea. Although this type of diet is usually not a problem when fed for a few days, it is extremely unbalanced and some owners never change back to a commercial pet food. It also can be difficult to switch a picky pet back to a commercial pet food after eating a homemade diet.

Other factors to consider with acute vomiting and diarrhea are to start with small, frequent meals and to gradually increase the amount fed as tolerated. It is also important to question the owner carefully about the animal's usual diet. If the usual diet was not a good quality, nutritionally balanced diet, this is a good time to encourage the owner to switch, especially if the usual diet played a role in the illness. Also, check on the animal's usual treats and whether the owner is feeding table food as these could also contribute to the current and future problems.

**Chronic diarrhea**

Obviously, a diagnosis is needed to determine the underlying cause for chronic diarrhea (ie, whether it is inflammatory bowel disease, parasites, or other problems). In general, nutrition should be viewed as an adjunct to medical therapy for chronic diarrhea. Occasionally, a diet change will solve the problem (ie, if the owner has been feeding a poor quality or unbalanced diet or if the animal has a food allergy) but this is the exception rather than the rule.

In general, animals with chronic diarrhea fit into three categories in terms of which type of diet provides the best results. Some animals respond best (most often animals with small
bowel diarrhea) to a low fiber, easily digestible diet. Others (often those with large bowel diarrhea) will show some improvement with a high fiber diet. It is not yet clear whether the fiber should be soluble, insoluble, or a mixture however, and much work remains to be done in this area. Finally, a small percentage of animals have food allergy as the root of their diarrhea and these animals may benefit from a novel ingredient diet. Since there is nothing inherently less allergenic about ingredients used in novel ingredient diets (eg, lamb and rice, venison and potato, kangaroo and oats), it is important to find the ingredients to which the animal has not previously been exposed. There also are several therapeutic diets now commercially available that have hydrolyzed protein sources that make the diet (at least in theory) "hypoallergenic."

Urolithiasis

No matter what type of stone is determined to be present in a dog with urolithiasis, the first thing to think about is diluting the urine. This means that a canned food is the first choice for dogs with any form of urolithiasis. It is also important to determine which stone type is present. Struvite stones can be usually be dissolved with treatment of infection, strict use of a dissolution diet, and dilution of the urine (ie, canned diet or dry diet with copious amounts of water added). Dogs with struvite stones may not require long-term use of an acidifying diet; just careful monitoring for recurrent infection and maintenance of a dilute urine. For recurrent struvite crystals/stones, a diet that produces an acidic urine may be beneficial (eg, Hill’s c/d, Royal Canin Urinary S/O).

pH is not the sole factor involved in preventing crystal/stone formation. In fact, while struvite stones are quite sensitive to pH, oxalate stones are relative insensitive to pH. Therefore, the diet of choice for most dogs with calcium oxalate stones is the Royal Canin Urinary S/O (which reduces the risk of recurrence of both struvite and calcium oxalate stones). However, if the dog requires a sodium restricted diet or a low fat diet, this diet would not be indicated as it is high in sodium and in fat.

Another important issue, particularly for dogs with oxalate stones, is to specifically ask the owner if the dog is receiving any dietary supplements. Vitamin C, for example, will acidify the urine and provide oxalate precursors. Other supplements that contain calcium or vitamin D can contribute to hypercalcemia and hypercalciuria.

In a dog with stones, urine should be monitored routinely for specific gravity, presence of crystals, indication of infection, and pH. Note that the Royal Canin Urinary S/O diet typically produces an acidic urine. There is no need to alkalinize the urine in a dog with oxalate stones eating this diet. Clinicians should also discuss treats and table food with owners because these can negate the beneficial effects of the therapeutic diets.

Hepatic failure

Recommendations also have changed for patients with hepatic failure. Not too long ago, a dog with a portosystemic shunt or a dog with chronic hepatitis was immediately put on a very restricted protein diet. Now it is known that this may actually be detrimental to the patient which will break down its own muscle to supply the needed protein. Currently, the recommendation for hepatic disease is to reduce the protein only as much as needed to prevent clinical signs. If clinical signs are not present, there is no need for restriction. If clinical signs are present, medical therapy should be instituted first and then dietary protein should
again be restricted only as much as necessary. There are a number of different diets available that vary in protein content so that a gradual reduction, as needed, can be achieved. Similar to the other diseases discussed, it is of utmost importance to provide adequate calories to maintain optimal body weight.

In animals that do require moderate to severe protein restriction, changing the protein source may also be beneficial. Many animals with hepatic encephalopathy, for example, will tolerate dairy- or egg-based proteins better than meat proteins. In people and experimental models, adjustment of the branched chain amino acids relative to aromatic amino acids ratio may have benefits. If a patient with hepatic disease has ascites, sodium restriction is definitely indicated; otherwise, there is less importance placed on feeding a sodium restricted diet. Commercial diets are available for dogs with moderately reduced protein levels with or without other added nutrients thought to be beneficial in hepatic failure.

**Cardiac disease**

*Body Composition*

Cardiac cachexia, a loss of lean body mass, is common in animals with CHF and has deleterious effects on strength, immune function, and survival. Anorexia, increased energy requirements, and metabolic alterations all contribute to the syndrome of cardiac cachexia. The anorexia may be secondary to fatigue, dyspnea, medication toxicity, or unpalatable diets. However, it is the inflammatory cytokines (eg, tumor necrosis factor) that appear to be the primary mediators of cachexia. These cytokines directly cause anorexia, increase energy requirements, and loss of lean body mass. Therefore, a logical approach to treating patients would be to block these cytokines. One nutritional approach to reducing inflammatory cytokines is supplementation of fish oil, which is high in omega-3 fatty acids. Fish oil decreases cachexia and in some dogs with CHF-induced anorexia, fish oil supplementation improves food intake.

*Potassium*

Angiotensin converting enzyme (ACE) inhibitor therapy has gained widespread use in the management of dogs (and some cats) with CHF. These drugs cause increased serum potassium and some animals develop hyperkalemia. Spironolactone, now used in some dogs and cats with heart disease, is an aldosterone antagonist and a potassium-sparing diuretic. Animals receiving ACE inhibitors or spironolactone can develop hyperkalemia. As some commercial cardiac diets contain increased potassium concentrations to counteract the theoretical potassium loss due to diuretics, these diets can contribute to hyperkalemia.

*Nutritional Deficiencies/Nutritional Pharmacology*
At one time, deficiencies of nutrients such as thiamine and selenium were a common cause of cardiac disease in people. These deficiencies are now uncommon although there may be a number of examples in dogs and cats which still are important. There also are some nutritional deficiencies that can develop secondary to the cardiac disease and its treatment. Finally, supplementing certain nutrients may provide benefits above and beyond their nutritional effects (i.e., nutritional pharmacology). It is not always clear whether the benefits of a nutrient are the result of correcting a deficiency or pharmacologic effects.

Restriction of dietary protein intake used to be made for animals with CHF. However, there is no evidence that protein restriction is necessary for dogs and cats with CHF and, in fact, it probably is deleterious since these patients are predisposed to loss of lean body mass. Unfortunately, many people recommend a diet designed for renal disease for animals with heart disease because many renal diets are restricted in sodium. But protein is restricted even in some diets designed specifically for dogs with cardiac disease. Unless severe renal dysfunction is present, high-quality protein should be fed to meet canine maintenance requirements.

There has been a dramatic reduction in cases of feline dilated cardiomyopathy (DCM) since the late 1980's when increased dietary supplementation of taurine was instituted after the landmark research of Pion, et al. Most of the current cases of feline DCM are not taurine deficient but taurine deficiency should be suspected in all cases of feline DCM. Cats that have been fed a poor quality, homemade, vegetarian, or otherwise unbalanced diets are at risk for taurine deficiency.

Taurine deficiency is now suspected in some cases of canine DCM. Unlike cats, dogs are able to synthesize adequate amounts of taurine and so are not thought to require dietary taurine. Most dogs with DCM do not have taurine deficiency, but low taurine concentrations have been found in some dogs with DCM, most commonly reported for the American Cocker Spaniel, Golden Retrievers, Labrador Retrievers, Newfoundlands, Dalmatians, Portuguese Water Dogs, and English Bulldogs. Taurine deficiency in dogs may be related to dietary factors as it is thought to be more common in dogs eating high fiber or certain lamb meal and rice based diets and has been induced by feeding a low protein, low taurine diet long-term to dogs. Taurine deficiency also may be the result of increased renal or fecal loss of taurine or other metabolic defects present in certain breeds. Taurine supplementation (with or without carnitine supplementation) may be beneficial in some dogs with taurine deficiency but, even in dogs that respond, the response is not as dramatic as in taurine deficient cats with DCM. The exact role of taurine in canine DCM still is unclear and some of the potential benefits may be due to its positive inotropic effects or role in calcium regulation in the myocardium.

The omega-3 polyunsaturated fatty acids, eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), are normally in low concentrations in the cell membrane but levels can be increased by a food or supplement enriched in omega-3 fatty acids. There are a number of potential benefits of omega-3 fatty acids supplementation, including a reduction in inflammation, improvement in appetite, and suppression of arrhythmias. Fish oil supplements (fish oil concentrate capsules with vitamin E but no other ingredients at a concentration of 180 mg EPA + 120 mg DHA per 1 gm capsule) can be prescribed at a dose of 1 capsule/10 pounds body weight. Flax seed oil or cod liver oil should not be used.

Magnesium plays an important role in normal cardiac function. Some cardiac drugs are
associated with magnesium depletion so animals with CHF can be at increased risk for hypomagnesemia. Hypomagnesemia can increase the risk of arrhythmias, decrease cardiac contractility, cause muscle weakness, contribute to renal potassium loss, and can potentiate the adverse effects of certain cardiac medications. Animals with low serum magnesium concentrations should be fed a diet higher in magnesium or may require magnesium supplementation.

**General dietary issues for all diseases**

The process of choosing an appropriate diet for an animal with medical conditions involves examining the patient, the diet, and the owner's feeding practices and considering all the issues at hand. Generally, there is not a single "best" diet for all animals with a particular disease. What might be the best diet for one animal may be contraindicated in another. So, it is important to assess a number of different factors to determine which diet or diets might best suit a particular patient.

**The Patient**

In general, the nutrients of concern in cardiac patients are calories, sodium and chloride, protein, potassium, and magnesium. However, patients with cardiac disease vary tremendously in terms of their clinical signs, laboratory parameters, and food preferences and these will all affect diet selection. For example, dogs with asymptomatic heart disease require less severe sodium restriction than those with CHF. Overweight cats require a less calorically dense diet than would a thin cat. Laboratory results (eg, hypo- vs hyperkalemia) and concurrent diseases also influence diet choice.

**The Diet**

Based on these and other patient parameters, a diet or diets can be matched to the individual patient. For example, in an animal with cardiac disease without CHF (ie, a dog with chronic valvular disease with no clinical signs or a asymptomatic cat with hypertrophic cardiomyopathy), the author recommends only mild sodium restriction and counsels the owner to avoid diets high in sodium and to avoid treats or table food that are high in sodium. Most owners are unaware of the sodium content of pet foods and human foods and need very specific instructions regarding which foods are appropriate. When CHF first arises, additional sodium restriction is recommended. Diets designed for dogs with renal disease are not recommended for cardiac patients (unless there is concurrent severe renal failure) because of the protein restriction inherent in these diets. Above all, the diet must be palatable enough for the animal to eat it willingly.

**Feeding Practices**

Although the pet may have dietary preferences and dictate the types of food eaten to a certain degree, the owner is the major determinant of the pet's diet. Therefore, meeting the owner's expectations in terms of diet is important for animals with medical conditions. Their pet's quality of life is of tremendous importance to owners so providing diets that are palatable and readily eaten is critical. Most dogs (and many cats) receive treats and the majority of dog owners give the medications with "people food." Including this information in the overall diet plan is important to achieve success with nutritional modification. Also, cost preferences should be considered as veterinary therapeutic diets may be out of the price range for long term use by some owners; in this case, lower priced alternatives should be offered.
**Important Points in Diet Selection**

1. As mentioned previously, there is usually not a single "best" diet for any patient. The author typically determines several diets that would be appropriate for an individual patient based on the patient, diet, and feeding practices. These diets are offered as choices for the owner and for the pet. Once they determine which diet the pet prefers, this diet can be used. Having specific dietary choices is particularly beneficial for more animals with severe disease (advanced CHF or renal failure), in which a cyclical or selective loss of appetite is common.

2. Don't change the diet while the patient is hospitalized. Once the animal is home, feeling better, and stabilized on medications, a gradual change to a new diet can be made. Forced dietary changes when the animal is sick can induce food aversions.

3. Since most owners give treats (either pet treats or "people food"), be sure to specifically discuss treats with the owner. Most owners are unaware of treats that would be contraindicated (eg, high salt or high protein treats or table food). The author typically provides a list of foods that are appropriate and foods to avoid as treats to assist the owner in wise selection.

For more information on nutrition and cardiac disease, please see the HeartSmart website

[www.tufts.edu/vet/heartsmart](http://www.tufts.edu/vet/heartsmart)
DIFFICULT WOUND CLOSURE
Once in a while, the veterinary surgeon is confronted with a difficult wound to treat or with a challenging skin closure after extensive cutaneous resection. This presentation is an overview of different means of addressing wound closure under tension.

SKIN STRUCTURE
- **EPIDERMIS**
  - Stratum basale, stratum spinosum and stratum corneum
- **DERMIS**
  - Collagen and elastic fibres
  - Matrix of mucopolysaccharides
  - Cells (fibroblasts, macrophages, mast cells, plasma cells)
  - Hair follicles
- **HYPODERMIS**
  - Hair follicles
  - Fat
  - Collagen
  - Elastic fibres
  - PANNICULUS MUSCLE
    - *Panniculus carnosus m.*
    - Head and neck
      - Platysma, sphincter colli (superficialis et profundus)
    - Trunk
      - *Cutaneous trunci, supramammarius, preputialis*

VASCULAR SUPPLY TO THE SKIN
- **VERY IMPORTANT**
- Originates from the deep layers of the skin and branches out superficially
- Capillary network is divided in plexuses
  - Superficial
  - Middle
  - Deep or sub-dermal
    - THE MAJOR VASCULAR NETWORK TO THE SKIN
    - WITHIN THE PANNICULUS CARNOSUS MUSCLE
    - IMPERATIVE TO PRESERVE ITS INTERRELATION WITH THE SKIN
    - IF NOT
      - NECROSIS WILL OCCUR
• Arterial supply originates from the aorta
• Perforating arteries
  o Direct cutaneous arteries
  o Determine angelsomes
  o Arborisation within the *paniculus carnosus* muscle
    ▪ Sub-dermal plexus

**WOUND CLOSURE PLANIFICATION**
• ALWAYS PLAN SURGERY, DON’T IMPROVISE
  o Plan at least 2 or 3 ways of closing the proposed wound
    ▪ Am I capable of performing wound closure?
    ▪ Position the patient properly
    ▪ Clipped enough?
    ▪ Use skin tension lines

**WOUND RELIEVING TECHNIQUES**
The presentation will illustrate different means of achieving wound closure under tension with the following techniques:

• **SKIN MOBILISATION**

• **WALKING SUTURES**

• **TENSION SUTURES**

• **SKIN STRETCHING/EXPANSION**

• **TENSION RELIEVING INCISIONS**
  o SIMPLE
    ▪ LINEAR
    ▪ V-Y
    ▪ Z
  o MULTIPLE

• **SKIN FLAPS**
  o INDIRECT
  o DIRECT
    ▪ ADVANCEMENT
    ▪ ROTATION
    ▪ TRANSPOSITION
    ▪ SKIN FOLD
- AXIAL PATTERN FLAP
  - OMOCERVICAL
  - CAUDAL AURICULAR
  - THORACODORSAL
  - SUPERFICIAL BRACHIAL
  - CRANIAL SUPERFICIAL EPIGASTRIC
  - CAUDAL SUPERFICIAL EPIGASTRIC
  - LATERAL THORACIC
  - DEEP CIRCUMFLEX ILIAC
  - GENICULAR
  - REVERSE SAPHENOUS
  - LATERAL CAUDAL

- SKIN GRAFTS
  - FREE
    - Classification
    - Survival
      - Adherence, plasmatic imbibition, inoscutation and ingrowth of vessels
    - Types
      - Pinch, punch, strip, stamp, mesh
  - VASCULARIZED

REFERENCES

Laser Surgery: How Does it Work? Indications?
Bertrand Lussier DMV MSc Dipl. ACVS

LASER USE
Laser is used in many fields
- Medical
  o Surgery, ophthalmology, plastic surgery, lithotripsy, wound healing, dentistry
- Industrial
  o Cut, weld, treatment of materials, fabrication
- Defence
  o Target marking, missile guidance, countermeasures, radars, weapons
- Research
  o Spectroscopy, laser ablation, laser annealing, interferometry, LIDAR, laser capture, micro dissection
- Commercial products
  o Laser printers, CD and DVD players, barcode readers, thermometers, pointers, wireless computer mouse
- Shows
  o lighting
- Aesthetics
  o Hair removal, acne...

HISTORY
Albert Einstein was the first to describe the concept of laser energy in 1917. The ancestor of the laser was developed by CH Townes in 1957 (MASER). Theodore Maiman invented the first functional laser in 1960.

LASERS IN VETERINARY MEDICINE
As reported by Eeg in 2008, 3000 clinics use CO$_2$ lasers to some level. There are about 58 000 clinics in the USA: 5% of clinics can market lasers, 14% are AAHA certified

ADVANTAGES?
It has been reported to seal nerve endings, capillaries and lymphatics. The use of laser facilitates certain procedures and decreases inflammation and pain.

UNDERSTANDING LASERS
- “An understanding of laser light properties and interaction with tissue will result in optimal patient outcomes without increased risk to the surgeon, staff, or pet. For veterinarians to fully appreciate the advantages of laser energy, they must first understand how laser energy interacts with and affects living tissues before recommending or attempting to apply this technology to companion” - Barb Gores DVM DACVS

WHAT IS LASER?
Acronym for **Light Amplification by the Stimulated Emission of Radiation.**

Laser light is produced by stimulating a medium and concentrating the resulting energy called the beam. Laser beam has 3 properties: monochromatic, collimated and coherent.

Once produced, the beam has to be transmitted: this can be done via a hollow reflective guide, fibre optics or with mirrors.

**TISSUE INTERACTION**
When released, the laser beam can be reflected, scattered, transmitted or absorbed by tissues. Absorption results in energy transfer to tissues. The energy transferred is dependant on the absorption coefficient of the tissues irradiated. Tissue composition varies in water, haemoglobin and melanin content. The most commonly used laser in small animal practices is the CO₂ laser. The wavelength emitted by the CO₂ laser is 10 600nm. This wavelength is absorbed selectively by water (90%) and less than 10% by haemoglobin and melanin.

Tissue damage varies upon transmitted energy which is dependant on Power and Time

- ENERGY (joules) = POWER (watts) x TIME (secs)

**BIOLOGICAL EFFECTS**
Absorbed energy can induce several biological effects; they are called photopyrolysis, photovaporolysis and carbonisation.

- Photopyrolysis
  - Tissues between 60-100°C
  - Coagulation of proteins
  - Tissue contraction

- Photovaporolysis
  - Tissues > 100°C
  - section

- Carbonisation
  - Tissues > 150°C
  - Blackened tissues
  - More energy absorption
  - Detrimental

**HOW CAN WE MAXIMIZE THE EFFECTS OF LASER WHILE MINIMISING COLLATERAL DAMAGE TO SURROUNDING TISSUES?**
The first way is to optimise the power density

- A common problem in laser surgery is using low power densities. This creates the retention of heat, does not provide vaporolysis, does not permit tissues to cool down which results in heat accumulation, tissue dehydration, desiccation and carbonisation.

The second way is by using settings that will permit tissues to cool down
• The use of super pulse mode permits thermal relaxation time which is the time necessary for target tissues to cool down by 50% through transfer of heat to surrounding tissue via thermal diffusion.

TYPES OF LASER SCALPELS
The perfect, all-around surgical laser does not exist. The CO₂ laser is used in small animal practices because it is affordable, has adjustable settings, is reliable and mobile. The laser is named on the irradiated milieu: CO₂, diode, Nd:YAG, Ho:YAG, HeNe, Argon, XeF.

LASER SAFETY
Laser safety is of prime importance. When using laser scalpel, there are risks to the patient, to the user and the personnel.
• Risks to the patient
  o Laser burns
    ▪ Local
    ▪ Reflection
    ▪ Erratic manipulation
    ▪ Endotracheal tube
• Risks to the user and personnel
  o Laser burns
    ▪ Hands, eyes
  o Plume
    ▪ Irritating
    ▪ Cancer?
    ▪ Viral DNA
• PRECAUTIONS
  o Never look at the beam
  o Never interpose objects between the beam and target tissues
  o Avoid uncontrolled reflections
  o Always wear wavelength specific protective eyewear
  o Work in a closed environment
  o Avoid contact of beam with flammable material and/or wet surrounding tissues of susceptible zones
  o Aspirate vapours and plume

INDICATIONS OF LASER SURGERY IN SMALL ANIMAL PRACTICE

Use laser surgery on tissues that have high water content: soft tissues. The laser scalpel can be used for:
• Incision
• excision,
• cytoreduction,
• wound sterilisation,
• wound irradiation after excision of neoplasm
• decrease wound contamination

The following is a partial list of the procedures that can be performed using a CO₂ laser:

OVH
Castration
Onychectomy
Nail shortening
Cutaneous mass excision
Pinna or ear canal mass excision
Entropion, ectropion, palpebral mass excision
Oral, labial, gingival mass excision
Correction of stenotic nares, excision of nasal masses, laryngeal saccules excision,
ventriculocordectomy, soft palate resection, laryngeal mass excision
Excision of perianal masses, circumanal adenomas, anal sacculectomy
Vaginal hyperplasia correction, excision of vestibular/vaginal masses
Cystotomy
**APPROACHES TO THE THORAX: WHICH ONE SHOULD I USE?**

**DIAPHRAGMATIC HERNIAS: TYPES AND SURGICAL CORRECTION/MANAGEMENT**

Bertrand Lussier DMV MSc Dipl. ACVS

**APPROACHES TO THE THORAX: WHICH ONE SHOULD I USE?**

The answer is quite easy: what am I doing in the thorax?? Dependant on what procedure needs to be performed, the approach can vary. It is recommended to use the least invasive approach with the lowest morbidity. As an example, for persistant ductus arteriosus (PDA) ligation, a single intercostal thoracotomy is indicated because it permits adequate exposure with the lowest morbidity.

There are mostly 5 approaches to the thorax:
- Transdiaphragmatic
- Intercostal
- Rib pivot/resection
- Median sternotomy
- Thoracoscopy

**TRANS DIAPHRAGMATIC**

This is an approach only seldom used. It has been anecdotally reported for transdiaphragmatic epicardial lead implantation for a pacemaker. It has been abandoned because of the use of transcutaneous endocardial leads with SQ pacemaker (cervical). Exposure to the cavity is poor. Mostly used for the surgical correction of diaphragmatic hernias.

**INDICATIONS**
- transdiaphragmatic epicardial lead implantation for a pacemaker
- diaphragmatic hernias for thoracic drain placement
- omentalisation of thorax

**INTERCOSTAL**

This is the most frequently used approach to the thorax. It provides adequate exposure and has a low morbidity rate. The procedure is simple, quick, not too painful, post-operative pain management is simple and efficient.

Intercostal thoracotomy provides good exposure to the structures in the vicinity of the thoracotomy, however visualization of structures farther from the approach are limited and the exposure to the contralateral side is null.

This approach is used when exposure of a thoracic region has been previously determined by imaging techniques (radiographs, ultrasound CT MRI) or via thoracoscopy.

The site of intercostal thoracotomy can be between the 3rd and the 10th intercostal spaces.

**INDICATIONS**
The indications are numerous. The following table adapted from Fossum summarises these indications.

<table>
<thead>
<tr>
<th>STRUCTURE</th>
<th>DISEASE</th>
<th>LEFT (space)</th>
<th>RIGHT (space)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Heart</td>
<td>Right atrium haemangiosarcoma</td>
<td></td>
<td>4 (5)</td>
</tr>
<tr>
<td></td>
<td>Cor triatriatum dexter</td>
<td></td>
<td>4 (5)</td>
</tr>
<tr>
<td></td>
<td>Persiant ductus arteriosus</td>
<td>4 (5)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Persiant right aortic arch</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pulmonic valve</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Pericardium</td>
<td></td>
<td>4,5</td>
</tr>
<tr>
<td>Lungs</td>
<td>Cranial lobe</td>
<td>4,5</td>
<td>4,5</td>
</tr>
<tr>
<td></td>
<td>Middle lobe</td>
<td></td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Accessory lobe</td>
<td></td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Caudal lobe</td>
<td>5 (6)</td>
<td>5 (6)</td>
</tr>
<tr>
<td>Esophagus</td>
<td>Foreign body</td>
<td>3,4 cranial</td>
<td>7,9 caudal</td>
</tr>
<tr>
<td></td>
<td>tumour</td>
<td>3,4 cranial</td>
<td>7,9 caudal</td>
</tr>
<tr>
<td>Cranial thorax</td>
<td>thymoma</td>
<td>3,4</td>
<td></td>
</tr>
<tr>
<td>Caudal thorax</td>
<td>Chylothorax with thoracic duct ligation</td>
<td>7,9 (cat)</td>
<td>7-9 (dog)</td>
</tr>
<tr>
<td>---------------</td>
<td>-----------------------------------------</td>
<td>----------</td>
<td>----------</td>
</tr>
<tr>
<td>Cranial vena cava</td>
<td>(4)</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Caudal vena cava</td>
<td>(6-7)</td>
<td>6-7</td>
<td></td>
</tr>
</tbody>
</table>

**SURGICAL TECHNIQUE**
- Curvilinear skin incision (parallel to the ribs) from the costovertebral junction to the sternum
- Incision of muscles *latissimus dorsi, scalenus* or external abdominal oblique, *serratus ventralis*, external and internal intercostals (at the center of intercostal space to avoid the nerves and vessels that are located at the caudal aspect of each rib)
- Incision of parietal pleura – mention it to the anaesthetist (thorax is opened) and make sure the patient is well ventilated
- «perform thoracic surgery»
- Closure:
- thoracic drain placement
- local intercostal blocks (bupivacain) including the space of the thoracotomy and 2 spaces cranial and caudal (5 spaces total)
- prepass a series of simple interrupted suture (resorbable suture of your choice) including the rib on each side of the thoracotomy (beware of vessels and nerves!!)
- The ribs are approximated by the assistant and the sutures are tied
- The muscles, SQ tissues and skin are sutured accordingly
- Reestablishment of thoracic negative pressure via the preplaced drain
- A pleural block can also be accomplished at this point (the patient it turned on the side of the thoracotomy and local block (bupivacain) is injected through the thoracic drain)

**RIB PIVOT/RESECTION**

This is an extended intercostal thoracotomy. It provides a larger exposure to the thoracic cavity:
- By disarticulating a rib at the costochondral junction and replacing it with a cerclage wire
- By resecting a rib

**INDICATIONS**
Same as intercostal thoracotomy
SURGICAL TECHNIQUE
Same as intercostal thoracotomy except that we incise and elevate the periosteum of the implicated rib and proceed to either the disarticulation/pivot of this rib or resection of it.
Closure:
  for disarticulation/pivot: drill hole on each side of the costochondral junction replace rib and twist cerclage appropriately, then suture soft tissues
  for rib resection: we do not need to replace the rib by a graft or a prosthetic/biologic mesh, suture soft tissue

I have used this approach only twice:
  in a dog to remove a large tumour
  in a horse for exploration of a mass

MEDIAN STERNOTOMY
It is the only approach that provides thorough exploration of the thoracic cavity. It can also be combined to a cervical approach (cranial partial sternotomy) or an exploratory laparotomy (caudal partial sternotomy).
In contrast to intercostal thoracotomy, this approach is more complicated, needs more specialized instrumentation (power saw), takes longer to perform, is painful, post-operative pain management is more complicated and not as efficient.
Median sternotomy provides excellent exposure to all structures of both hemi thorax but has a higher morbidity rate (pain, wound drainage/infection/dehiscence, not airtight closure).
We use this approach when exposure of a thoracic region has not been previously determined, that we need a large exposure or that multiple procedures must be performed.

INDICATIONS
Exploration of the thorax
Bilateral partial pulmonary lobectomies
Thoracic duct ligation
Thymoma resection
Intrahepatic portosystemic shunt ligation (sometime used with exploratory laparotomy)
Cardiac surgery
Pyothorax
Tracheal resection

SURGICAL TECHNIQUE
- Ventral midline skin incision from the manubrium to xyphoid process
- Sharp and blunt dissection (use cautery if possible) to expose the sternum. Elevate musculature on each side to expose the sternebrae.
- Use a power saw (bone saw or special sternal saw) staying midline. This ensures 1) an easier closure providing solid bone-bone contact and 2) avoid internal thoracic arteries which are
located abaxially on the thoracic side of the sternebrae.
- Try to keep 2 sternebrae intact (2 cranial for caudal approach/2 caudal for cranial approach).
This will provide easier more solid closure avoiding cranial-caudal slippage/shifting of the sternum resulting in pain and delayed healing
- «Perform thoracic surgery»
- Closure:
  - Thoracic drain placement
  - Prepax a series of cerclage (20ga) wires (dog > 15kg) or heavy (#2) non resorbable
    (polypropylene, polybutester) suture (cats and dogs< 15 kg) in an alternating cruciate pattern
    around each sternebra
  - The sternebrae are approximated and the sternotomy closed.
  - The muscles, SQ tissues and skin are sutured accordingly
  - Reestablishment of thoracic negative pressure via the preplaced drain
  - A pleural block can also be accomplished at this point (the patient it turned in sternal
    recumbancy and local block (bupivacain) is injected through the thoracic drain)

THORACOSCOPY/ VIDEO-ASSISTED THORACIC SURGERY (VATS)
The use of VATS has been more reported in the last few years.
INDICATIONS
It is mostly used for exploration, helping us to
1) plan surgery: which side/space to perform an intercostal thoracotomy or
   should we do a median sternotomy
   i. pyothorax
   ii. pulmonary disease (bullae, blebs)
   iii. fine needle aspiration of tumours
2) perform simple thoracic procedures
   i. foreign body retrieval (porcupine quills, vegetal material)
   ii. thoracic exploration and lavage with thoracic drain placement in cases of
      early pyothorax
   iii. pericardiectomy
   iv. lung biopsies via small thoracotomy
3) perform more advanced procedures
   i. thoracic duct ligation
   ii. PDA ligation
   iii. Endoscopic lung lobectomy

SURGICAL TECHNIQUE
• With the dog positioned in dorsal recumbancy a small skin incision is performed at the
  tip of the xyphoid
• A 5mm diameter 10cm long rigid sheath (screw type) with a sharp trocar is inserted
• A 5mm laparoscope/thoracoscope is inserted in the thoracic cavity
• 2 intercostal ports(one left/one right) are established with 10mm pliable sheaths
• The caudal mediastinum is sharply incised with endoscopic Metzenbaum scissors (cautery assisted)
• Thoracic exploration is performed; a 25°oblique scope can be used to explore had to get places
• “Perform VATS “or plan appropriate thoracotomy
• Closure :
• Thoracic drain placement under direct visualisation
• Remove all sheaths
• For each port use one or two cruciate sutures with 2-0 polydioxanone (PDS), then close SQ and skin accordingly
• Reestablishment of thoracic negative pressure via the preplaced drain
• A pleural block can also be accomplished at this point through the thoracic drain

DIAPHRAGMATIC HERNIAS: TYPES AND SURGICAL CORRECTION/MANAGEMENT

It is a structural defect of the diaphragm permitting communication between the abdominal cavity and the thoracic cavity. There are 2 types : congenital and acquired

CONGENITAL HERNIAS
The incidence of congenital diaphragmatic hernias (DH) represents only 10-15% of all hernias. The most commonly described type is the peritoneopericardial hernia which are rather easy to correct. Also reported are pleuroperitoneal hernias which can be very tedious to repair.

Peritoneopericardial hernia is present at birth and animals are frequently asymptomatic. There is continuity between the peritoneum and a defect in the fusion of the caudal mediastinum, permitting direct communication of abdominal content into the pericardial sac.

Pleuroperitoneal hernia is also present at birth, but patient are more symptomatic. There are multiple anomalous findings:

• Sternal malformation
• Cranial abdominal hernia
• Umbilical hernia
• Hair “whirls” at the xyphoid appendix
• Cardiac malformations
• Pulmonary malformations

TRAUMATIC HERNIAS
This is the most frequent type of diaphragmatic hernias. They are caused by:
• Being hit by car, falls, kicked by horse, by moose, hit by snowmobile, fights, big dog/little dog encounters

There are multiple injuries associated with traumatic hernias:
  Haemothorax
  Pneumothorax
Pulmonary contusions
Pleural Effusion
Rib fractures
Long bone fractures (approximately 2% of animals presented for long bone fractures also have a traumatic diaphragmatic hernia)

PREOPERATIVE EVALUATION
This evaluation is imperative. It must include:
- Thorough physical examination
- Orthopaedic/neurologic examination
- CBC/Chemistry panel UA
- Thoracic and abdominal radiographs
- Abdominal ultrasound (if possible)
- ECG (arrhythmias reported in 12% patients)
- Aggressive treatment for shock and monitoring of recovery
- Analgesia

RECOMMENDATION
It is imperative to stabilize these patients before anaesthesia and surgery. The majority of these trauma patients need to be stabilized and observed. Diaphragmatic hernia is not a surgical emergency unless:
- There is a hollow dilated organ in the thorax impeding on respiratory function
- There is internal bleeding

PROGNOSIS
Boudrieau et al Comp contin educ pract vet 1987
- If surgery if performed less than 24 hours from trauma, the mortality rate is high *(33%-50%)
- If surgery if performed more than 36 hours from trauma, the mortality rate is low *(10-15%)

- Cats- 82,3% survival
- Mortality was not associated with duration of hernia, hernia content, CBC chemistry, panel abnormalities, hypotension, use of positive inotropes or glucocorticoids, surgeon's level of training.
- Mortality was associated with age, respiratory rate at the time of hospital admission, and multiple concurrent injuries.

- Evaluation of chronic DH in 36 dogs and 16 cats (> 2weeks)
- Diagnosis on radiographs in only 66%
  - 33% required other diagnostic imaging
- Adhesions requiring either lung, liver or intestinal resection in 14 patients
- Median sternotomy required in 14/50 patients (28%)
- 86% survival

- **Objective**—To determine the survival rates of dogs and cats that underwent surgical treatment for traumatic diaphragmatic hernia within 24 hours of admission and determine whether timing of surgery affected perioperative survival rate
  - 89.1% of patients (dogs and cats) with acute or chronic DH were discharged from the hospital
  - 89.7% of acute DH operated on within 24 hours of presentation were discharged from the hospital


- Retrospective study of peritoneopericardial DH in dogs (8) and cats (31)
- The overall mortality was 5.1% (2/39),
  - involving one (3.2%) of the 31 cats
    - congestive heart failure secondary to chronic, hypertrophic, obstructive cardiomyopathy diagnosed on echocardiography 9 days postoperatively;
  - one (12.5%) of the eight dogs.
    - cardiopulmonary arrest the morning following surgical correction

**SURGICAL CORRECTION OF DIAPHRAGMATIC HERNIAS**

**CONGENITAL**

*Peritoneopericardial*

See Acquired

*Pleuroperitoneal*

Difficult to address for the unexperienced surgeon. Must be prepared to repair a large defect in the diaphragm

- Partial suture of the diaphragm dorsoventrally
- Suspension of the diaphragm around the costal arch
- If needed, free diaphragm from costal arch and advance axially or use bilateral transversus abdominis flap
  - If defect too large
    - Prosthetic mesh (Prolene™ or Marlex™)
    - Biological mesh (Vetbiosis™)
    - Omental reinforcement

**ACQUIRED**

Perform ventral midline coeliotomy

- Simple technique with excellent visualisation of surgical field
- Low morbidity
- Can be combined with caudal median sternotomy

Perform visual examination of the entire abdominal cavity (note missing viscera!!)

Examination of diaphragm
Localisation of rents/tears
Visualisation of heart and pericardial sac
Reduction of ectopic structures
  Beware of adhesions!!!
  If needed, the hernia can be enlarged to
    further permit inspection of the herniated structures
    break down adhesions
    help in reduction of structures
Reestablish pulmonary expansion of possible atelectatic lungs (in traumatic hernias, not necessary in peritoneopericardial hernias if sac is not perforated)
INSERT THORACIC DRAIN (and suture it) WHILE THORAX IS OPENED, IT’S EASIER!!!
Perform herniorrhaphy
  Grasp the border of the hernia with retention sutures or Babcock/Allis tissue forceps
    Try to close the defect
    If necessary perform diaphragmatic reconstruction
Suture the diaphragm
  -multiple horizontal mattress sutures with non-absorbable (silk, nylon, polypropylene), can be followed by a simple continuous over sew in large dogs
    OR
  -vest over pants
    If the diaphragm was directly torn from the costal arch, pass suture around ribs to anchor the diaphragm to the chest wall.
Evacuate air from the thorax
  Different methods
  Maintain pulmonary expansion while tightening last stitch
  Via transdiaphragmatic thoracocentesis
  Through the thoracic drain previously placed
Close abdominal cavity

POSTOPERATIVE RECOVERY
  • Intensive care!!!!
  • Radiographs (effusion, pneumothorax, atelectasis, edema, thoracic drain position)
  • Oxygen therapy
  • Analgesia
  • Care to thoracic drain
**Dr. Jacqui Neilson, DVM, DACVB**

**ACUTE MANAGEMENT OF BEHAVIOURAL PROBLEMS**

Jacqueline C. Neilson, DVM, DACVB

**Introduction:**

When dealing with undesirable behaviours the overall objective is to implement therapy to modify the pet so that it no longer offers/exhibits the behaviour. Behavioural modification processes often involve techniques such as desensitization and counterconditioning; these are not “quick fixes”. In fact, protocols often involve weeks or months of behavioural modification. It may be beyond the capacity of the primary veterinary clinic to provide this type of guidance. Even if provided, immediate management is still critical for overall success.

This time delay to treatment completion/success necessitates a more immediate method of controlling the problem. For example, the behaviour in question may be dangerous for the family or pet; the behaviour may be damaging to the environment or expression of the behaviour may be affecting undesirable learning in the pet. All of these problems can fracture the family/pet bond and result in relinquishment or euthanasia. So while implementing a therapeutic treatment program is important for ultimate treatment success, it is just as important, if not more so, to offer techniques for immediate control of the problem behaviours. In some cases, these immediate interventions will control the problem sufficiently for the pet and the owner so that no additional treatment is necessary.

**Avoidance:**

If a discrete trigger(s) for the undesirable behaviour can be identified, efforts should be made to avoid the trigger(s). Later in the treatment process those triggers are often reintroduced to the pet, usually in a gradient fashion. One of the reasons that avoidance is such a critical step is because animals continue to perform behaviour that are rewarding to them; this occurs through the process of operant conditioning. A reward may be in the form of positive reinforcement (application of something positive) or negative reinforcement (removal of something aversive). Avoidance serves the important function of stopping the animal of practicing and being rewarded for performing the undesirable behaviour. This “time-off” from practicing the undesirable behaviour also gives the owner an opportunity to work on foundation exercises — these are the building blocks of establishing a new behavioural response in the pet.

Some owners may be frustrated with this advice saying that avoidance is too passive an approach to the problem or perhaps is simply catering to the problem pet. It is critical to explain to these owners that this is a temporary proposition that will prevent further deterioration. In some cases the owners may discover that the avoidance management technique is so successful that they will choose to continue to utilize it permanently.

**Avoid punishment:**

Punishment is often the owner’s first response to problematic behaviours. Unfortunately punishment is often applied inappropriately therefore ultimately making the problematic
behaviour worse instead of better. If punishment is to be considered some general principles of punishment must be met: it needs to be applied immediately (within 1-2 seconds of initiation of the behaviour), it needs to be consistently applied (every time the undesirable behaviour occurs) and it needs to be effective (strong enough to inhibit the behaviour but not so severe that it causes intense fear/anxiety/aggression). These are difficult criteria for most owners to meet, therefore the punishment is ineffective at the very least and, in many cases, punishment escalates the problem behaviour. This escalation may be due to the fact that many problematic behaviours actually stem from anxiety. If the pet starts to anticipate a punishment in addition to the inherent anxiety about the trigger stimulus, the anxiety is amplified in the future. Even apparent success (suppression of the problematic behaviour) is not necessarily true success if the pets overall anxiety level is elevated. So in most cases punishment is not a good option for management of problematic behaviours.

Redirect the pet:
This is actually the initial portion of many behavioural modification plans and, in some cases, it can also be a wonderful acute management tool. For many animals, no one has ever given the animal direction about what TO DO. The owners have instead focused their efforts on telling the pet what NOT TO DO, “no, bad dog, no”. This provides the animal with little direction. By teaching/practicing some very simple but practical commands, the owner may be able to successfully redirect the pet to offer an acceptable behaviour. I advocate using lure training to expedite the process. Lure training involves finding something that the pet loves (often delectable food treats but for some dogs it will be a special toy) and using that reward to lure the pet into the desired behaviour. If the lure is small enough (as would be true with tiny, delectable food treats) it should be contained in an enclosed fist- this creates a “target hand”- dogs will quickly learn to follow that target hand (closed fist) wherever it takes them.

The three most important commands that the problem dog should master are: sit, watch me and u-turn. These should all be mastered in non-distracting circumstances (inside house, in yard, on walks with no distractions) before they are attempted when the dog is distracted/aroused.

Redirection when owner is absent during expression of the behavioural problem:
When the problematic behaviour occurs when the owner is absent (e.g. separation anxiety) redirecting the pet with the above commands is not an option. In these cases, if avoidance is not possible (e.g. dog can’t go to work or doggie daycare) then a simple counterconditioning technique may work – redirecting the dog onto an activity that is incompatible with anxiety during the owners’ absence. This is essentially redirection without anyone present. The simplest way to accomplish this is by giving a food motivated dog a long-lasting food treat at departure. If a dog is profoundly anxious, this may not be successful unless combined with some other strategies (e.g. pheromone/drug therapy).

What to do when the pet exhibits undesirable behaviour:
Despite the best efforts at avoidance or redirection, there will be situations where the pet exhibits the undesirable behaviour. The best response from the owner in most of these
situations is to have the owner remain calm and simply remove the pet from the situation or clean up the mess. Animals don’t learn well when highly aroused...imagine a child in the midst of a temper tantrum. This is an opportunity for owners to consider how they could improve their management strategies to avoid future bad experiences.

**Tools:**
There are certain products that can help quickly, either to modify the pet’s behaviour or to provide control.
Head collars- Calms some pets; provides enhanced control; may help handler effectively redirect pet
Leash – pets with problematic behaviours that occur when they are on leashes should not wear extension leashes; instead they should be on 4-6 foot leashes so that the pet is always in close proximity to the handler for greater control.
Basket Muzzles- provide a barrier between teeth/skin in case there is an inadvertent lapse in management/avoidance; these are essentially portable baby gates
Calming Cap: reduces intensity of visual stimuli by
Baby gates/Crates: For segregation
Food puzzle toys: Busy Buddy Line ([www.premier.com](http://www.premier.com)) Twist n’ Treat; Squirrel Dude

**Pheromones:**
The product, Dog Appeasing Pheromone (DAP or Comfort Zone), uses a calming pheromone to treat anxiety. The DAP is a synthetic analogue of the pheromone released from the mammary area of the lactating bitch. The heated plug in diffuser releases calming pheromones into the dog’s environment. The DAP collar has a body heat activated release of pheromones.

**Drugs:**
Drugs may provide acute management. In most countries there are very limited options for approved drugs to treat behavioural conditions. When there is an indicated use for a given condition, these medications should be tried first. If a drug is used off-label, full disclosure should be made to the owner and risks explained.

Adjunct drug therapy should be considered if:
- the drug therapy is appropriate for the diagnosis
- there are no identified contraindications identified for drug usage
- behavioural modification alone cannot control the problem
- the animal’s welfare is compromised
- the drug can facilitate the behavioural modification process (risk/benefit analysis)

There are two categories of drugs to consider: event drugs and daily drugs. As the name implies, event drugs are given for specific predictable events, ideally administered about 60 minutes prior to the event. They tend to be short acting. In contrast, daily medications are given on a daily basis and tend to have longer half lives/duration of action.

**Event Drugs:**
Event drugs for home use for anxiety related conditions such as noise phobias or separation anxiety. These fast acting anxiolytics (e.g. benzodiazepines) may be helpful for anxious animals that do not exhibit aggression (treatment of fear-aggression with benzodiazepines may result in disinhibition of aggression).

### Key Drug-General

<table>
<thead>
<tr>
<th>Drug</th>
<th>Drug Class</th>
<th>Canine Dose (mg/kg)</th>
<th>Route</th>
<th>Frequency</th>
</tr>
</thead>
<tbody>
<tr>
<td>alprazolam</td>
<td>Benzo</td>
<td>0.05-0.1</td>
<td>Oral</td>
<td>PRN</td>
</tr>
<tr>
<td>diazepam</td>
<td>Benzo</td>
<td>0.5-2.0</td>
<td>Oral</td>
<td>PRN</td>
</tr>
<tr>
<td>lorazepam</td>
<td>Benzo</td>
<td>0.1-0.2</td>
<td>Oral</td>
<td>PRN</td>
</tr>
<tr>
<td>clonazepam</td>
<td>Benzo</td>
<td>0.1-1.0</td>
<td>Oral</td>
<td>PRN</td>
</tr>
<tr>
<td>trazodone</td>
<td>SARI</td>
<td>2-5</td>
<td>Oral</td>
<td>PRN or BID</td>
</tr>
</tbody>
</table>

**Benzo=** benzodiazepine; **SARI =** serotoninantagonist/reuptake inhibitor

### Daily medications

The group of drugs that currently receive the most attention and application by veterinary behaviourists are drugs that influence serotonin activity. This includes drugs such as amitriptyline, clomipramine, fluoxetine, paroxetine and sertraline. Serotonin (5-HT or 5-hydroxytryptophan) is a neurotransmitter in the brain. The role of serotonin in the brain is not completely understood. Among other functions, it plays a role in endocrine regulation, temperature control, feeding, sexual behaviour, and sensorimotor arousal and anxiety. Many canine behavioural problems have anxiety as an underlying problem including cases of aggression, separation anxiety and obsessive-compulsive disorders. Therefore, serotonin-enhancing medications may have beneficial effects when used to treat these conditions. While it can take up to several weeks to appreciate the behavioural benefits of these medications often one can appreciate effects very early in the course of treatment, within the first week. If serotonergic drug therapy is used as part of the treatment plan, the goal is to use the drug for a sustained time period (2-6 months) during which time the behavioural modification is also employed. The animal should learn appropriate behaviour in previously problematic situations. When the dog has demonstrated improvement for at least 1-2 months, drug therapy can be stopped.

### Serotonin Enhancing Drugs

<table>
<thead>
<tr>
<th>Generic Name</th>
<th>Drug Class</th>
<th>Canine Dose (mg/kg)</th>
<th>Common Side Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>amitriptyline</td>
<td>TCA</td>
<td>2.2-4.4 SID</td>
<td>Lethargy, Inappetance, Dilated pupils</td>
</tr>
<tr>
<td>buspirone</td>
<td>Serotonin Agonist</td>
<td>0.5-1 BID</td>
<td>Intercat irritability, Owner affection</td>
</tr>
<tr>
<td>clomipramine</td>
<td>TCA</td>
<td>1-2 BID</td>
<td>Sedation, GI upset</td>
</tr>
<tr>
<td>Drug</td>
<td>Class</td>
<td>SID</td>
<td>Side Effects</td>
</tr>
<tr>
<td>------------</td>
<td>-------</td>
<td>-----</td>
<td>--------------------</td>
</tr>
<tr>
<td>Fluoxetine</td>
<td>SSRI</td>
<td>1-2 SID</td>
<td>Dry mouth, Lethargy, GI upset</td>
</tr>
<tr>
<td>Paroxetine</td>
<td>SSRI</td>
<td>1-2 SID</td>
<td>Dry mouth, Lethargy, GI upset</td>
</tr>
<tr>
<td>Selegiline</td>
<td>MAOI</td>
<td>0.5-1 SID</td>
<td>GI upset, Agitation</td>
</tr>
<tr>
<td>Sertraline</td>
<td>SSRI</td>
<td>1-3 SID</td>
<td>GI upset, Lethargy</td>
</tr>
</tbody>
</table>

**Summary:**
Providing owners with techniques to quickly and safely control their pet may create a window of opportunity for owners to implement behavioural modification programs that provide long term changes in their pet’s behaviour.

**References/Suggested Reading**


CANINE AGGRESSION TO OWNERS: ALPHA OR ANXIOUS?
Jacqueline C. Neilson, DVM, DACVB

Alpha vs. Anxious
Dominance is a term that almost every dog owner has encountered. Another term for a dominant dog is an “alpha” dog – the alpha implying that they are in the lead position in the pack. Many behavioural problems ranging from aggression to coprophagia are blamed on “dominant” or “alpha” behaviours. Behaviours such as rushing out the open door that occur due to pure positive reinforcement from the dog’s perspective have been labelled incorrectly as “dominant behaviour.” In fact, appeasement behaviours such as jumping up to lick the face have been misconstrued as dominant behaviours. Perhaps one of the greatest misconceptions is that aggression equates with social dominance. In fact, the opposite is true – dominance rank is not positively correlated with aggressive events, in fact higher-ranking wolves have few aggressive encounters. Dogs with aggression are often labelled as dominant or alpha despite obvious fearful postures during or after an event. Unfortunately, overuse and misuse of the idea of dominance has resulted in needless canine suffering and death.

Perhaps the most frightening aspect of labelling this wide range of behaviours as “alpha” behaviours is the subsequent actions and reactions that humans feel compelled to employ to correct the “dominance”. It is a relative license to be pushy/forceful/abusive to the dog. Forceful actions are taken to correct the dog – at best the dog may become conditioned to avoid further aversive actions from the owner; at worst the dog will become more fearful, more aggressive, dangerous and perhaps even euthanized. This may be due to a misunderstanding about the true motivation behind the behaviour.

One question that may arise with the human-dog relationship is if there really is a pack dominance hierarchy ever formed between these different species. Considering humans and dogs “speak” different languages, there may be a fundamental barrier to establishment of a true hierarchy. When was the last time a person put their ears back in communication to a dog? And recent research supports the idea that canine hierarchies are more flexible than previously considered and that they are based primarily on appeasement behaviours – essentially active displays of submission and deference keep the peace. When a dog trembling/growling/snarling over a Kleenex tissue is extrapolated to a “dominant” dog the jump is big and probably wrong. Firstly, the dog didn’t steal the Kleenex as an act of defiance/leadership; it stole the Kleenex for the immediate benefit of tasty human nasal secretions or perhaps attention. And yes, that dog wants to retain access to that Kleenex. But it is in a highly aroused/emotional and perhaps even fearful state – yelling/physically punishing the dog may make it more nervous/aggressive next time it finds an off-limits valuable treat. The dog is getting conditioned to respond aggressively due to the human action/reaction and its basic desires – this does not make the dog a dominant dog, it makes it a trained dog (trained to do the opposite action of what most owners’ desire).

Despite this, dominance aggression is frequently diagnosed in the canine population, ranging between 20-59% of behavioural caseloads. In cases of dominance aggression, family members or very familiar people are usually the targets of the aggression. The dog is often described as
having a superior position in the social hierarchy and the dog is using aggression to manage situations where his/her status is threatened. However when cases of “dominance” aggression in dogs are examined, these dogs are often fearful or submissive. Owners report signs that are ambivalent or submissive surrounding attacks. These behaviours are in conflict with a truly dominant/confident personality. Also they often first see the signs at a fairly young age, six months of age or less. These puppies are often the easily trained in certain settings: often labelled the “smartest” dog in their puppy class. If they are astute students at learning “good” behaviours, they will also be astute students at learning what we may consider “bad” behaviours – it is all operant conditioning.

If the dog is not dominant, then what is occurring? Pending rule outs of underlying medical disease such as hypothyroidism, consider that the dog may actually be anxious. Dogs that bite owners may do so in contexts related to social dominance but their motivation may be based almost entirely in anxiety. Two broad groups emerge: dogs that reach anxiety threshold and resort to default of aggression to control situation; and dogs that are unsure of their social role and use aggressive behaviours to deform the social system to get badly needed information about expectations. Since aggression is often very successful at terminating the uncomfortable situation, the dog learns that aggression is a good way to manage situations of conflict.

If the motivation behind the aggression is anxiety and not an overly confident/dominant dog, then the treatment plan must reflect this. This may explain why neutering these dogs often has little effect on aggression – reducing testosterone is unlikely to alter fear/anxiety. Domination techniques (e.g. alpha roll over) in response to conflict aggression is contraindicated, as they would only serve to increase the anxiety of the dog. Many owners report an escalation in the aggression when they attempt these domination techniques and this is understandable if the dog is truly in a state of anxiety/fear. Employing these techniques will only serve to escalate the fear/anxiety and subsequently escalate the aggression.

**Interventions**

Important treatment principles for the dog with conflict aggression include: avoiding confrontation, having a safe way to handle the dog and establishing consistent dog-owner interactions. If there is a specific trigger situation, desensitization and counterconditioning to that trigger can be implemented.

Many owners are concerned that if they avoid confrontations, they are letting the pet “win.” However, this is not the case. Any animal in a highly aroused emotional state is not a good candidate for learning. The dog will be taught acceptable behaviour when he is calm and relaxed. The owners also want to avoid being placed in situations where the dog’s aggression is successful, thereby reinforcing the unwanted aggressive behaviour. By avoiding triggers for aggression, this unwanted learning will not occur. To avoid aggressive situations, sometimes the owners will have to modify their behaviour (e.g. don’t get near the dog when he is eating) or modify the environment (e.g. if the dog has been aggressive with toys, remove them from
To establish consistent dog-owner interactions, it is often necessary to terminate all casual interactions between the dog and the owner. Predictable, structured interactions should become the mainstay of owner-dog interactions. Generally, owners are instructed to give the dog a command prior to all interactions. If the dog responds appropriately to the command, the interaction can proceed. If the dog does not respond, the owner should ignore the dog. In addition to these lifestyle interaction changes, the dog and owner should practice obedience training that rewards obedient, relaxed behaviour in the dog. A benefit of a well-run obedience program is that it provides another source of structured, predictable interactions where the dog is reinforced for relaxed, obedient behaviour. But caution is necessary, not all obedience classes are created equal – poor timing, aversive techniques etc. may actually sabotage improvement.

If a specific trigger for the aggression is identified, gradual desensitization and counterconditioning to that trigger can be implemented. The dog is being conditioned to respond a different way to the trigger stimulus. It is not learning to be submissive or less dominant – it is being taught that the “threat” is no longer a threat.

In anxious dogs, drug therapy may be helpful in reducing anxiety and associated aggression. There is no medication licensed to treat canine aggression, so all medication use is considered off-label and carries the risks inherent with off-label use. Low serotonin has been associated with aggression. In one study, aggressive dogs had lower metabolites of serotonin in the CSF, suggesting lower levels in the brain. A study of fluoxetine administered to dogs exhibiting aggression to owners showed a reduction in the aggression with drug therapy. A study on the effects of clomipramine in cases of owner directed aggression did not show benefits over that of a placebo. Serotonin enhancing drugs such as fluoxetine 1-2 mg/kg once daily; paroxetine 1-2 mg/kg once daily; amitriptyline 2.2-4.4 mg/kg once daily or sertraline 1-3 mg/kg once daily can be considered as an adjunct to behavioural modification in these cases.

Pheromone therapy may also be helpful. Dog Appeasing Pheromone (DAP) is a synthetic analogue of a pheromone produced by the lactating bitch and it is supposed to calm the nursing puppies. The DAP (Adaptil™) diffuser can be plugged into an electrical outlet for a constant diffusion in the environment or a DAP ((Adaptil™) collar can be worn by the pet.

Alternative therapies such as homeopathic remedies, special diets, pressure body wraps, touch therapies, etc. have been advocated by some but lack strong research to support the claims. That said, consideration of these techniques may be valuable in the right client/pet.

**Conclusion**

It is important for clinicians to consider the fact that most dogs presenting with aggression are not confident/dominant dogs since it has a huge impact upon the treatment plan. Kind, gentle and consistent handling will reap more rewards than harsh, challenging and threatening behaviours in these dogs in conflict.
References


Reisner, IR; Mann, JJ. Comparison of cerebrospinal fluid monoamine metabolite levels in dominant aggressive and non-aggressive dogs. Brain Research 1996; 7(14): 57-64.
SEE SPOT RUN, SEE SPOT BITE
Jacqueline C. Neilson, DVM, DACVB

Introduction
The incidence of dog aggression directed towards people is hard to quantify since the many incidents go unreported. Aggression is the most frequent reason owners seek the advice of board certified veterinary behaviourists, constituting about 70% of the caseload. Millions of dogs are relinquished to shelters each year and behavioural reasons for relinquishment include aggression. This aggression epidemic is most likely due to a constellation of factors including, but not limited to, living environments, exercise regimes, socialization, genetics, miscommunication and mismatched expectations. Owners often are distraught that their pet has shown aggressive behaviour. Issues of public safety and liability are a concern when canine aggression is involved.
It is important to remember that aggression is often a normal behaviour. Aggression is a form of communication. In order to try to minimize and control aggression, it is important to understand the underlying motivation for the aggression. As with most problematic behaviours, the treatment success depends upon many social and environmental factors. Considerations such as owner expectations, breed, accuracy of diagnosis and appropriate treatment all play a role in the successful control of the problem. Sometimes, despite our efforts, the risks associated with the aggression are significant enough to warrant euthanasia of the animal.

Pathogenesis & Diagnosis:
The veterinarian’s role in canine aggression should involve prevention and intervention advice. When addressing aggression in a patient, the clinician should establish a diagnosis and determine if the aggression is abnormal behaviour (excessive fear/poor impulse control) or normal behaviour that is just unacceptable. Then a treatment plan that includes management, behavioural modification, pheromones and/or drug therapy can be implemented. If there is persistent significant risk to the safety of others, euthanasia or re-homing should be considered.
A motivational diagnosis (why the dog is exhibiting aggression) is important to establish as it allows for a targeted treatment plan. Focus on these four areas to arrive at a diagnosis
- Dog’s health status – underlying illness may cause or contribute to aggression
- Dog’s general temperament – confident, shy, fearful
- Dog’s aggressive events – triggers/targets, behaviour, recovery
- Evolution of the aggressive behaviour over time

In the popular media, aggressive dogs have often been characterized as dominant. While aggression can be a component of dominant behaviour, the circumstances where it is utilized are often during ritualized events with conspecifics where the dominant dog is very confident and controlled. Most dogs that present in as patients for aggression do not fit this description. In fact it is now believed that the majority of dogs that exhibit aggression do so secondary to fear. The reason that it is so important to dispel the dominance myth is that people are likely to
use force or domination to “treat” a dominant dog. These techniques are contraindicated in a fearful dog.

While many dogs may appear very offensive (lunging, growling, snapping, biting) in their actions during an aggressive event, the underlying motivation for that aggressive event is often a defensive strategy, essentially “fight or flight.” If the dog feels that it can’t flee or that it by fleeing certain resources would be jeopardized, the fight (aggressive) option may become the chosen response. When utilized as a defensive manoeuvre to a perceived threat, the fight response is often successful, thus establishing it as the default response in similar future situations.

Some common categories of aggression directed toward unfamiliar people are described below:

- **Fear related aggression:**
  - Often targeted at a group or class of individuals such as men, unfamiliar people, unfamiliar dogs, large dogs, veterinarians or children. It may also be associated with certain events such as grooming, car rides, etc.
  - The dog often exhibits some postures either before, during or after the aggressive event that are consistent with fear such as a tucked tail; active retreat; ears pulled back; bimodal piloerection.
  - Historical accounts of early aggressive events may include more fearful postures/retreat behaviour.

- **Territorial aggression**
  - The dog responds aggressively to unfamiliar people coming onto its territory. Usually the home/yard and the car tend to be the areas where territorial aggression is most pronounced.
  - The aggressive behaviour is often rewarded because the target of the aggression is successfully “driven away” (this often is not a direct result of the aggression- it just so happens that the individual is passing by or delivering mail).
  - Some cases may truly be fear related, not just territorial protection.

- **Predatory aggression**
  - This type of aggression lacks the conflict present in other types of aggression and is associated with the act of chasing/capturing/killing/consuming.
  - Often targeted at cats, squirrels, livestock but the chasing portion of the predatory sequence can be triggered by fast moving objects like bicycles, cars, joggers in some dogs.
  - Infants may trigger a predatory response in some dogs

In many cases the aggression may not be abnormal, just unacceptable to the owners. Normal aggression may respond well to management and behavioural modification. Abnormal aggression (rooted in excessive fear/anxiety or with poor impulse control) will also require management and behavioural modification but may also benefit from additional therapy such as pheromones or drugs. Since aggression can be heritable, dogs with aggressive
tendencies and/or unstable temperaments should not be bred. Since many aggressive responses are based in fear, appropriate and continuous socialization may be protective against the development of aggressive behaviour. In addition, consistent and calm leadership within a household may help to provide a predictable environment that protects against the development of aggression.

Any medical problem can contribute to irritability and aggressive responses. Chronic diseases may be controlled but aggressive responses may still remain due to learning or residual fear or anxiety. Although hypothyroidism is often linked with behavioural conditions, including aggression, robust data to support the causative link is lacking. In fact a controlled study at University of Pennsylvania did not find an association between hypothyroidism and aggression.

Aggression based in fear is associated with activation of the sympathetic nervous system and the consequential cascade of hormones and neurochemicals that elicit the fight/flight response. Studies have linked low serotonin levels with increased aggressive behaviour. Expression of the aggression and the subsequent consequences is almost always likely to promote additional aggression through operant conditioning. Therefore management to prevent aggressive displays is paramount in the treatment protocol.

**Treatment**

- **Management** is an important first step to prevent further aggressive episodes
  - The first step is to provide safety from the aggressive pet. After separating the pet from individuals, use confinement, muzzles, leashes, and head collars, as needed, to create a safer environment.
  - Keep the pet away from either the triggers and/or the victims and block visual access
  - Confinement must be secure: a crate, a room with a lock, a locked yard with a fence the dog cannot jump or climb. An adult should put the pet into confinement.
  - Many dogs are unaccustomed to confinement; training to be confined must be part of the plan for it to be a realistic and workable option.
  - Confinement must occur every time the trigger for the aggression might be encountered; many families are unable to ensure this happens.
  - The pet does not have to go to all places; if problems occur on walks, in parks, in the car etc. these areas must be avoided until new tasks are learned. Walks can occur in industrial complexes instead of neighbourhoods and parks
- **Behavioural modification**
  - The first step in trying to change an unwanted behaviour is to teach the pet to settle and relax on a verbal command.
  - This is not a simple obedience task. The goal is for the dog to be calm and quiet as evidenced by physiologic changes, such as slower respiration and relaxed body postures and facial expressions.
• Change the pet-owner relationship to include a command-response relationship. The pet is to earn all things by performing a command including access to food, outdoors, attention, play etc.
• Physical and verbal reprimands must be avoided. They often increase rather than decrease emotional arousal and can cause fear and anxiety as well as an escalation of aggression.
• A reward gradient should be established for the patient. What are the favoured rewards that the pet will work for? These can be play, food, petting etc.
• A stimulus gradient must be established for the behaviour. How does the behaviour vary with various characteristics of the stimulus such as distance, speed of approach, size of stimulus, sound, location, etc.
  • Learning will not occur when the stimulus is strong enough to elicit underlying fear/anxiety or aggression.
  • Low level stimuli that the animal can learn to perceive as non-threatening are essential for learning to occur
• Three tasks are most useful when trying to change behaviours: “sit”, “watch me” and an escape command such as “back up” or “let’s go” or “go to your crate”.
  • These must be learned prior to any encounters with the stimulus. Once they are well learned both the pet and the owner will have more confidence and know what to do
• Once relaxation and control techniques have been learned, they can be used to begin desensitization exercises to the problematic target stimuli. To properly desensitize the dog, the target stimuli must be kept at a low enough intensity so that the dog can respond in this relaxed manner. This is accomplished by developing a stimulus gradient, including all the relevant details of the stimulus, such as size, location, speed of approach, and noise level. With success, the stimulus is gradually intensified until the dog can respond to the target stimuli in a relaxed manner.
• Other techniques include classical conditioning-associating a pleasant stimulus with one that provokes an unwanted response, i.e. associating food with the approach of a stranger. Hopefully over time strangers will predict food and the underlying emotional state will change.
• Owners must be very aware of the reactions of the dog and stop training and leave the situation before the pet becomes emotionally aroused, anxious or aggressive.
• Pheromone and drug therapy
  • Synthetic analogues of pheromones may be helpful in fear related aggression cases.
  • There are no drugs licensed to treat interdog aggression. Serotonin enhancing drug therapy may be indicated in dogs that have an underlying anxiety/fear component or poor impulse control. Examples include: fluoxetine 1-2 mg/kg/day; clomipramine 2-4 mg/kg/day; sertraline 1-3 mg/kg/day.
• Alternative therapies
  • Nutracueticals, homeopathy, touch therapy, body wraps and other interventions may provide some benefit to individual cases but they lack rigorous scientific data on efficacy.
Conclusion:
Most aggression towards unfamiliar people is fear based aggression. When a dog chooses to bite, it becomes evident that biting is a behavioural strategy the dog is willing to use at least in that circumstance. This may mean that dog presents a higher risk for future biting than one that never has bitten. Biting behaviour is rarely cured, rather it is controlled, and with safety precautions may become less of a risk but still may occur.

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**Feline House-soiling**
Jacqueline C Neilson, DVM, DACVB

**Introduction:** While cats are the most popular pet in the United States, their most common behavioral problem, house-soiling with urine (periura) and/or feces (perichezia) is very unpopular with their owners. In fact, house-soiling may lead to outdoor banishment, relinquishment or euthanasia. Studies support this regrettable outcome, with behavioral problems, primarily house-soiling, being a leading cause of relinquishment of cats to shelters.\(^1\) In one study, over 23% of the cats relinquished to a shelter had daily or weekly incidents of house-soiling.\(^2\) A recent study by Marder found that the most common problem identified post adoption of a cat from a shelter was house-soiling with 9% of the cats in her study exhibiting this behavior within three months post adoption.\(^3\)

A common misconception of owners is that the house-soiling cat is spiteful or vindictive. In fact, marking behavior is considered to be a normal feline communication tool. Inappropriate toileting occurs due to preferences or aversions and is not motivated by personal grievances. That said, owner actions/behavior can influence marking and inappropriate toileting. Marking behavior has been hypothesized to be associated with stress/anxiety. An owner that creates a hectic environment for her cat by having lots of house-guests, a new baby, fostering/adopting multiple animals, etc. may trigger the marking behavior in her cat. But the cat’s motivation to mark is not vengeance but a reaction to the household circumstances. And in those circumstances, the marking behavior may be very adaptive for the cat, as marking may be important in spatial organization. When considering inappropriate toileting, an owner that purchases/provides an inferior litter product may find that the cat rejects that litter (litter aversion) and finds an alternative inappropriate toileting spot. But the cat isn’t trying to “get even” with the owner for buying the inferior litter, the cat is simply coping with this unfortunate situation. An owner that fails to scoop the litterbox on a regular basis may discover periuria/perichezia secondary to lack of cleanliness (litterbox aversion). Once again the cat isn’t getting back at the owner for poor box hygiene, the cat is creatively coping with a untenable situation, a filthy litterbox. So while the house-soiling cat may be engaging in the perichezia/periuria as a consequence of an owner’s actions or lack of actions, it is not a vengeful act but a way to cope with the circumstances.

Understanding the true underlying motivations for house-soiling and making a definitive diagnosis is critical since it allows us to design a targeted treatment plan. Targeted treatment plans are likely to be more successful than shotgun therapy.

**Terminology:** Unfortunately, universally accepted diagnostic terminology does not exist to describe feline house-soiling. For the purposes of this paper and presentation, the inappropriate deposition of urine (periuria) or feces (perichezia) with the intent of evacuation of the bladder/bowels will be called inappropriate *toileting* and periuria/perichezia deposited with the intent of communication will be called *marking*. The term house-soiling will encompass both inappropriate toileting and marking behaviors.
**Diagnosis:**
There are three main diagnostic categories for periuria:

![Diagram](image)

Toileting and Marking are both behavioral diagnosis and are considered as diagnosis of exclusion...primary medical causes must be ruled out before arriving at a behavioral diagnosis. Cats with primary medical problems that result in dysuria, pollakiuria, stranguria, polyuria, constipation, diarrhea, or difficulty accessing the litterbox (e.g. arthritis) may present with house-soiling. A thorough history, physical examination and appropriate diagnostic testing based upon the signalment and presenting complaint (periuria vs. perichezia) is important to rule out/address any underlying medical causes.

The most common medical diagnosis for cats between the ages of 1-7 years that are demonstrating periuria are feline idiopathic cystitis and uroliths. Diagnostic testing should be aimed at the most likely conditions, thus a physical examination, urinalysis and imaging studies are recommended. Less than two percent of these adult cats with periuria suffer from bacterial bladder infections, however it is very common for veterinarians to dispense antibiotic therapy. Once medical problems have either been ruled out or are being treated, the behavioral aspects of the house-soiling should be addressed. These generally fall into one of two categories: inappropriate toileting due to aversions or preferences or marking as a communication tool.

**Behavioral Diagnosis:** Urine marking is a form of communication. Some cats may feel more compelled to communicate if their territory is threatened or there is some other stressor in their life. Urine marking is a normal behavior that is considered unacceptable in our homes. About 10% of prepubertally castrated male cats and 5% of prepubertally spayed female cats show problem urine marking. Territorial marking behavior may be stimulated by multiple cats sharing a common living area, breeding season or the arrival of new cats into a territory. Situations that evoke anxiety or stress in a cat such as the addition of a new family member or a dramatic change in work schedules, may also lead to urine marking.

Toileting problems are often triggered by medical causes, aversions, preferences or anxiety. Litterbox aversion is a common cause of inappropriate toileting. Litterbox cleanliness, location, style and litter type/brand can all impact acceptance/rejection of the litterbox. A negative experience accessing or in the litterbox (e.g. ambushed by a person/another animal when in the box) can create a litterbox aversion. Preferences may involve substrate preferences and location preferences. When a cat develops a substrate preference it is selecting a substrate (e.g. carpet) that is more pleasing to the cat than the substrate that the owner is providing in the litterbox. If the historical information suggests that the cat is always choosing a certain substrate for elimination then this possible cause should be explored more carefully.
The author has found that the following four questions are helpful for discerning toileting from marking:

**LOCATION OF PERIURIA?**

<table>
<thead>
<tr>
<th>VERTICAL MARKING</th>
<th>HORIZONTAL MARKING OR TOILETING</th>
</tr>
</thead>
</table>

**CONCURRENT PERICHEZIA?**

<table>
<thead>
<tr>
<th>YES TOILETING</th>
<th>NO MARKING OR TOILETING</th>
</tr>
</thead>
</table>

**SUBSTRATE PATTERN TO PERIURIA?**

<table>
<thead>
<tr>
<th>YES TOILETING</th>
<th>NO MARKING</th>
</tr>
</thead>
</table>

**LITTERBOX SET-UP?**

<table>
<thead>
<tr>
<th>GOOD MARKING</th>
<th>POOR TOILETING</th>
</tr>
</thead>
</table>

Marking involves urine sprayed on vertical surfaces or puddles of urine deposited on horizontal surfaces with special social significance. One tends *not* to see a particular pattern of substrate use, in fact the periuria is often found in areas with different substrates underfoot. Inappropriate defecation is rarely involved. The cat continues to use the litterbox for both urination and defecation and there is no evidence of litterbox avoidance.

In contrast, the cat with inappropriate toileting usually deposits significant quantities of urine and/or feces on horizontal surfaces. A substrate-use pattern is often identified: for example, the cat always targets a certain type of carpet. The cat shows decreased or absent usage of the litterbox. Historical collection may reveal a pattern of inappropriate litterbox cleaning, box type, litter type or box placement.

The estimated volume of the urine deposit(s) is not particularly helpful at discerning marking from toileting. And while intercat tension or aggression may certainly be a causative factor of periuria and will need to be addressed in the treatment if it exists, it is not particularly helpful in the diagnostic portion as intercat aggression can result in marking or toileting problems.

**Treatment of Inappropriate Toileting:** The treatment for inappropriate toileting should focus on providing a very attractive litterbox while reducing the attractiveness or accessibility of inappropriate target spots. The most attractive litterbox set up for the majority of cats includes the **ABC’s** of the best box:

- **A= Accessible**
• **B** = Big

• **C** = Clumping Clay, Carbon, Clean

To find the ultimate litterbox for an individual cat, a cafeteria of different options is the best strategy; the cat is given a variety of options and then preference is determined by usage. Most cats like boxes that follow the ABC’s listed above which are discussed in greater detail below.

**Accessible:** Placement of the litterbox (location) has been hypothesized to contribute to litterbox rejection; the box is located in an area that is too noisy, too far away, too trapped, etc. and the cat seeks an alternative toileting site. It is generally recommended to avoid dead ends in litterbox placement where there is only one exit point for the cat. This may be especially critical in multiple cat households with intercat tension/aggression. Since covered litterboxes only provide the research on box location is often defined by the presence of the box in the core location vs. non-core location. Different researchers use different definitions for “core” but a reasonable definition is that the core area is the zone where the cat spends at least 75% of its time. In free-ranging cats one study showed that they were more likely to eliminate in a non-core area than in their core area. This poses the question that if the litterbox is placed in a core area, does this predispose cats to litterbox rejection. In a study comparing 20 cats with litterbox rejection issues and 20 cats without litterbox rejection issues the litterbox location (core vs. non-core location) was analyzed to see if it was a factor in box rejection. Box location (core vs. non-core placement) was not found to be a factor. But it should be noted that 37/40 boxes in the study were located in a non-core area.

**Big:** It has been hypothesized that one cause of feline toileting problems is a litterbox aversion secondary to small box size. Some experts recommend that litterboxes should be 1.5 times the body length of the cat. However, to the author’s knowledge, there is a lack of evidence to support this claim. A study was conducted to identify if cats had a preference for a certain box size when all other variables were equal. In order to determine litterbox size preference, 32 cats housed in four colony rooms in a shelter were given three litterboxes equal in every parameter except box size for a 21-hour test period. Excrement deposited in the boxes was collected, counted, weighed and recorded. Both parametric (ANOVA) and non-parametric (Friedman) tests were run on the data blocking by room. Although statistical significance was not achieved, the trend was that the large boxes were preferred over the medium and small boxes for the number of deposits and total weight of excrement.

Box style (covered vs. uncovered) has also been hypothesized as a risk factor for litterbox rejection however data has not supported this finding. That said, most covered boxes are relatively small in size so this may be a negative factor inherent to the majority of covered boxes.

**Clay Clumping:** Studies have established that, when given a variety of litter options, cats preferentially use finely particulate sand-like ("clumping" or "scoopable") litter for elimination.
Clean: Although regular box scooping is regularly advocated by behaviorists a study by Sung that examined 20 cats with inappropriate toileting vs. 20 cats without inappropriate toileting did not find box cleaning frequency to be a risk factor for inappropriate toileting.21 Eleven cats without house-soiling had their boxes only scooped 1-3 times per week while 10 cats with inappropriate toileting had the same cleaning schedule. This data could reflect individual differences between cats with the more fastidious cats developing the inappropriate toileting. Another possible explanation is that the critical level of soiling that makes a cat reject its litterbox is scooping less than once weekly; this particular study had no households with that infrequent a scooping schedule.

Carbon: Cats may consider a heavily soiled box aversive, perhaps due to the strong malodor that is associated with the fecal excrement. While regular scooping and discarding of any solid waste is advised, additional techniques to control fecal odor are desirable. A patented process of adding activated carbon to litter has been incorporated into some litters, in an attempt to reduce fecal malodor. While humans can readily appreciate the effectiveness of activated carbon in odor reduction, a study was conducted with cats to see if they appreciated/preferred a litter with activated carbon over a litter without the activated carbon. The results showed a trend for cats to use the litter with the activated carbon, suggesting that it provides superior odor control and may help in prevention and treatment of litterbox problems.19 Another study was performed comparing two different litter odor control additives, activated carbon and bicarbonate of soda, in a population of cats.24 These particular odor control additives are used in two different top selling national litter brands. Clorox company, the makers of Fresh Step litter products hold a patent on the carbon additive. Cats housed in four colony rooms at a shelter were given access to two identical litterboxes with the exception of the litter odor control additive. Excrement was collected from the boxes over four consecutive 12-hour overnight test periods. The amount of excrement was analyzed via both parametric (ANOVA) and non-parametric (Friedman Test) tests using a randomized block design. Cats showed a significant (p<0.05) preference for the litterbox with the activated carbon additive, suggesting that felines prefer activated carbon over bicarbonate of soda as an odor control additive. A follow up study using the actual commercial brands of litter (Fresh Step Scoop® vs. Arm and Hammer Super Scoop®) showed a statistically significant preference for the litter containing activated carbon.25 A study comparing Fresh Step Scoop® to Tidy Cats Instant Action® also showed a statistically significant preferential use of the Fresh Step Scoop litter. The impact of fragrance in litters is not clear. In one study, scented litter was found to be a risk factor for elimination problems but in another study, scented litter was not associated with elimination problems.21, 22 A prospective study by the author (in publication) offering scented and unscented litter to a group of cats showed no preference in their usage of one litter over the other. Both the aroma and the intensity of the fragrance may be a factor in a cat’s response to a fragrance. There is little published information on scent preferences in cats but a 2007 pilot study showed that cats preferred cedar and fish odors and showed avoidance behavior to citrus and floral scents. A follow up study with a larger population of enrolled cats (18 cats) and a modified scent palate that included bleach, cedar, citrus, fish and floral scents showed that cats preferred bleach and fish scents to the other offered scents.23 These fragrances were not presented in the context of litter/elimination and in that context, the
results may be different. In cats with elimination problems that are currently offered a scented litter material, it is advised to offer a non-scented litter. Future research will hopefully elucidate a litter fragrance that is both pleasing to the owner and the cat.

The soiled areas should be cleansed with an enzymatic cleanser. Sometimes the cat will have to be confined away from areas in the house where s/he has chosen to eliminate. Alternatively, those soiled areas can be made aversive with plastic, upside down contact paper, aluminum foil, food, etc. If the cat has chosen one or two areas in the house to eliminate, the new attractive litterbox should be placed at those locations. If the cat uses the box, it can gradually (1 inch per day) be moved to a more appropriate location, if necessary. If anxiety is associated with the inappropriate toileting, anxiolytic drug therapy may be instituted. However, in most cases of inappropriate toileting, drugs are not necessary or indicated for treatment success.

**Treatment for Urine Marking:** Marking animals should be neutered. Ninety percent of intact males show a significant decrease in marking behavior after castration.5

Once neutered, marking treatment should follow the D,E,F,Gs:

- **D = Drugs**
- **E = Environmental Enrichment**
- **F = Feliway**
- **G = Get Rid of Triggers**

Drug therapy has been long used to help control urine marking, however, to date, no drugs have been licensed by the FDA or other regulatory bodies to treat urine marking in cats. The two drugs that have the most information published regarding their efficacy in the treatment of urine marking are clomipramine and fluoxetine.11,12,13 The relative treatment success for both of these drugs is comparable and it is high, with approximately 80% of treated cats having a significant (>75%) reduction in urine marking when receiving the medication. Response to treatment can be rapid, within days of initiating treatment, but continued treatment shows a steady incremental increase in efficacy. Studies have evaluated cases on drug therapy up to 32 weeks and found that urine marking is controlled over these prolonged treatment durations.14 Side effects are usually mild and self limiting but may include lethargy, gastrointestinal upset and paradoxical anxiety. Severe or persistent side effects may warrant dose reduction or termination of treatment. See table 1 for doses and other drug options.

**TABLE 1: Drug options for treating feline urine marking**

<table>
<thead>
<tr>
<th>Drug Name</th>
<th>Dose</th>
<th>Dosing frequency</th>
<th>Dosing route</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fluoxetine</td>
<td>0.5-1.0 mg/kg</td>
<td>Once daily</td>
<td>Oral</td>
</tr>
<tr>
<td>Clomipramine</td>
<td>0.25-0.5 mg/kg</td>
<td>Once daily</td>
<td>Oral</td>
</tr>
<tr>
<td>Drug</td>
<td>Dosage</td>
<td>Administration</td>
<td>Route</td>
</tr>
<tr>
<td>----------------</td>
<td>--------------</td>
<td>----------------</td>
<td>---------</td>
</tr>
<tr>
<td>Paroxetine</td>
<td>0.25-0.5 mg/kg</td>
<td>Once daily</td>
<td>Oral</td>
</tr>
<tr>
<td>Amitriptyline</td>
<td>0.5-1.0 mg/kg</td>
<td>Once to twice daily</td>
<td>Oral</td>
</tr>
<tr>
<td>Buspirone</td>
<td>0.5-1.0 mg/kg</td>
<td>Twice daily</td>
<td>Oral</td>
</tr>
<tr>
<td>Cyproheptadine</td>
<td>0.5 mg/kg</td>
<td>Once daily</td>
<td>Oral</td>
</tr>
<tr>
<td>Diazepam</td>
<td>0.2-0.5 mg/kg</td>
<td>Twice daily</td>
<td>Oral</td>
</tr>
</tbody>
</table>

**Environmental Enrichment:** An “environment of plenty” should be created in multiple cat households. This involves creating multiple feeding areas, multiple elimination areas and multiple single cat sleeping perches at different vertical heights throughout the home. Positive interaction time (e.g. playing with a feather, grooming) should be spent with each cat on a daily basis.

Adequate environmental management of soiled areas and litterboxes may help to reduce marking. A study examined the effects of environmental management on the frequency of vertical urine marking. The owner cleaned urine marked spots with an enzymatic cleanser, provided one litterbox per cat plus one additional, scooped the box daily and changed the box weekly. These four steps significantly reduced the number of urine marks.

**Feliway:** Other forms of marking such as bunting (facial marking) and scratch marking should be encouraged. To encourage scratch marking, scratching posts and/or pads should be placed around the home, with the highest concentration in areas where the marking is occurring. To encourage facial marking, there is a product available called Feliway™. Feliway is a synthetic analog of the feline facial pheromone. Three proposed principal functions of the facial pheromone are: 1) spatial organization 2) relationships with other cats and 3) emotional stabilization. It is proposed that by increasing emotional stabilization Feliway results in the resolution or decrease of urine marking. Several studies support Feliway’s efficacy in the treatment of urine marking. 7,8,9,10

**Get Rid of Triggers:** To treat urine marking the clinician should be trying to reduce or remove conflict and stress in the environment. Stray cats and neighborhood cats should be discouraged from entering the territory of the resident cat. The owner may need to block the view from windows if their cat is aroused by the presence of other cats outside the home. If there is tension between cats in a household, the cats may need to be separated for time periods during the day or one cat may need to wear a bell so that the other cat can avoid interactions. If there are other social (e.g. visitors/children) or environmental triggers (e.g. noises) either avoidance or a program of desensitization and counterconditioning should be implemented.

**Conclusion**
In conclusion it is critical that the veterinarian establish a definitive diagnosis for cats with periuria and consider that it will either be medical, toileting or marking. By establishing a definitive diagnosis they can provide a targeted treatment plan which will increase treatment success and ultimately preserve the human-animal bond.

**References:**
12. Landsberg GM, Wilson AL Effects of Clomipramine on Cats presented for Urine Marking *JAAHA* 41: 3-11 2005
THE LINK BETWEEN BEHAVIOR AND MEDICINE: FELINE IDIOPATHIC CYSTITIS AND FELINE OBESITY
Jacqueline C. Neilson, DVM, DACVB

Introduction
For comprehensive care, behavioural interventions, not simply nutritional interventions, need to be considered in the management and treatment of two common feline conditions: feline idiopathic cystitis (FIC) and obesity. By providing comprehensive, multi-modal therapy, treatment success is enhanced.

Stress Reduction
Stress has been implicated in the pathogenesis of FIC. Compared with controls, cats affected with FIC show several aberrant stress-related factors including high concentrations of circulating catecholamines, malfunctioning alpha-2 adrenoreceptors, smaller adrenal glands, and suboptimal responses to a challenge with synthetic ACTH (Reche 1998, Westropp 2007). These findings have prompted interest in stress reduction as a part of the treatment plan for cats suffering from FIC. One study measured a variety of parameters in FIC-affected cats when exposed to stressful versus enriched environments. Catecholamine concentrations and urinary bladder permeability decreased during the enrichment phase, suggesting environmental enrichment may have a beneficial effect for cats with FIC (Westropp 2006).

Three areas of stress reduction should be considered for cats with FIC:
- Creating a cat considerate environment
- Resolving rotten relationships
- Administering anxiolytic agents

Creating a Cat Considerate Environment
A cat considerate environment provides an abundance of feline resources spread throughout the home that offer outlets for species typical behaviours. A standard rule is to provide as many resources as cats plus an additional one. For example, if there are four cats, there should be five litterboxes in different locations. Cat resources include: scratching surfaces, sleeping/resting perches, food and water stations, litterboxes and opportunities for play and exploration.

Resolving Rotten Relationships
Periuria can be very frustrating to owners and can break the pet-family bond. Punishment delivered to the cat upon discovery of a soiled location is not only ineffective, but it may worsen the problem. Owners should be advised to avoid punishing their cat if periuria is observed or discovered. In general, predictable routines and interactions may help reduce stress in the cat.
Intercat aggression or tension can be stressful. Some simple interventions that can improve feline relations include: creating a cat considerate environment with an abundance of resources spread throughout the environment (see previous section); placing a cat-safe belled collar on aggressor cat(s) and partial or full segregation of the cats. For severe cases, a
comprehensive behavioural modification program that includes desensitization and counter-conditioning should be considered.

**Administering Anxiolytic Agents**

Synthetic feline facial pheromone therapy (Feliway®) in the environment has been shown to reduce stress in cats and may be helpful for general stress reduction. In a randomized controlled study of 12 cats with FIC, there was a trend for cats exposed to Feliway® to have less severe episodes and fewer recurrences of cystitis (Gunn-Moore 2004). These differences were not statistically significant; however, these preliminary findings indicate the need for additional study.

Nutritional supplements such as L-theanine, a structural analogue of glutamate, are marketed as anxiolytic agents for cats. These therapies lack rigorous scientific evidence of efficacy and have not been specifically evaluated for the treatment of FIC. No drug therapy has been identified to successfully treat or control clinical signs of FIC.

**Best Box**

FIC or other causes of lower urinary tract disease (e.g., urolithiasis) may initiate periuria but behavioural issues may maintain periuria despite resolution of the underlying cause. There are two main causes at the root of persistent periuria secondary to FIC: litter box rejection and inappropriate preferences.

If a cat develops a litter box aversion secondary to FIC or experiences an urgency that causes elimination elsewhere, the possibility exists for development of a secondary toileting location or substrate preference. To resolve this problem, the preferred inappropriate site should be made less attractive (for example, by placing double-sided tape on the surface) or unavailable while the litter box is improved to meet the preferences of the cat. Offering a litterbox cafeteria can help to identify individual preferences. In general it can be helping to remember the ABC’s of the Best Box: Accessible, Big, Clean and Clumping Clay (litter).

**Obesity**

Most people grasp the protocol to achieve weight loss: adjusting caloric output to exceed caloric intake. One may think that achieving weight loss in a cat would be fairly simple since most owners can control calories provided. But while the concept of weight loss may be simple, the application is very challenging for several reasons. First and foremost, there is the human factor: owners may not even recognize that their cat is overweight, or, if they do, part of their emotional bond with their cat may be based upon food/feeding making it difficult to alter their behaviour. Secondly, even the most compliant owners who recognize the need for dietary management may abandon a dietary treatment plan when the cat on a calorically restricted diet awakens them at 3 am for a snack. Finally trying to increase caloric output from a cat can be a challenge. Exercising a cat is fundamentally different than human or canine exercise programs which tend to focus on endurance, making it difficult for owners to implement.
Feeding Enrichment
At first glance this may seem contradictory – why would we want to enrich feeding in overweight cats? The answer is that by creating a feeding program that recognizes and embraces typical feline behavioural ingestion patterns, we will maximize cat welfare, create opportunities to expend energy and minimize excessive consumption.

Feral cats eat 10-20 small meals throughout the day and night. These meals consist of small prey (rodents/birds/reptiles and insects) that require active hunting to secure the prey. Cats tend to avoid hunting and eating near other cats, it is a solitary endeavour. Not all hunting excursions are successful, and while gender, prey size, prey density and weather can all play a role in hunting “success” it is estimated that less than 50% of hunting expeditions are successful.

In contrast to the wild cat, our household cats tend to be fed a commercial dry pet food either as ad libitum (free choice) or in 2 to 3 servings (meals) daily. About two-thirds of owners supplement the dry food diet with some moist (canned) food; however very few owners feed moist (canned) food exclusively. In one study of 550 cat owners (owning 1177 cats) it was found that feeding most often occurred in the kitchen (79%) and usually all the cats were fed in the same room together (Heidenberger 1997). Twenty-four percent (24%) of the cats had communal food bowls.

Creating feeding enrichment that is attractive to the owner and the pet is critical. There is no single solution for every cat or household, feeding enrichment needs to be custom tailored to each situation. We do know that many people and cats don’t fare well in the “all you can eat” buffet line. Case in point: the average weight gain for passengers on a weeklong cruise with seemingly endless buffet options is 8 pounds. In the face of a highly palatable food, cats are likely to indulge in more calories than are necessary, setting them up for weight gain. So the options include portion control or making the energy expenditure to get the food counter the calories consumed. The following suggestions for feeding enrichment should be considered:

“Will Work for Food”
It has been established in cats that prey capture/killing is independent of prey consumption. The first two activities (prey capture and killing) are not necessarily related to hunger (Laundre’ 1977). Cats are compelled to hunt, even when they aren’t hungry. We can capitalize on this natural tendency to hunt in our feeding regimes both to expend calories and mitigate rapid food consumption. There are several food puzzle toys on the market designed for delivery of dry food through toy manipulation. The varying styles may appeal to different cat personalities and hunting styles. For owners that do not want to use puzzle toys there are other options. A food treasure hunt (hide little allocations of food throughout the home, including on elevated perches), a food toss (toss kibble across the room) or use of a timed automatic feeder may help to mediate the intake of food by the cat.

“Multiple Small Meals”
Mimicking the more natural feeding patterns of cats (smaller, more frequent meals) was
found in one owner survey to be a successful intervention for feeding related problems (Heidenberger, 1997). Another study found that limiting quantity and increasing frequency of feeding positively impacted weight loss success (Russell, 2000).

“A Table For One, Please”
The impact of social interactions between household cats on food intake has not been rigorously studied. We know that cats do not appear to experience the same social facilitation that occurs with dogs or other group hunters/eaters. For cats, the stress of having cats within sight when they are eating may be an issue. Keep in mind that a lack of overt aggression is not equal to comfort/happiness. While exceptions primarily with related cats have been noted, most cats hunt and eat alone. Therefore, setting up a food delivery scenario that allows for privacy when eating may be beneficial for house-cats.

Activity Enrichment
Play time should be scheduled daily when the cat is active and alert. Since cats tend to be most active at dawn and dusk and since most owners are home at those hours, those may make good play periods for the majority of cats. Cats tend to expend energy in short bursts of high levels of activity. So when engaging with a cat, think sprinter, not marathoner. The timing of play and the rotation of toys may impact interest in the games. One study found that rotating toys during a play session may spark a renewed interest in play (Hall 2002). Short breaks (5 minutes) between toys seemed to enhance play with second item while prolonged intervals (25-45 minutes) resulted in a decreased interest in the second toy. A good model for a cat play period may be commercial breaks during an evening TV show. Every 5-7 minutes there is a 2-3 minute commercial break – toss a new toy every commercial break!

Three Dimensional Space
It has been documented in several species, that increasing housing space alone did not change levels of activity. Making the space variable, entertaining and multi-dimensional increases the quality of the space and activity levels. Cats in laboratory settings (kennels) tend to select elevated perches over the bottom of the kennel and cats prefer upholstered perches over slick surfaced perches, regardless of their height. Other studies show that they prefer resting places that are warm, dry, and protected on two sides and situated in the corners or edges of an enclosure where they can watch without the possibility of being approached from behind. Every household should provide plenty of cat friendly perches. Single cat sized perches may be particularly important in multi-cat households as they may provide a way to gain fairly secure physical separation from other cats.

Outdoor Enclosures/Leashed Walks
There are ways to provide the outdoor world to an indoor cat - namely secure enclosures or leashed walks. There are specialty products to cat proof yards (www.catfencein.com, www.purrfectfence.com) or some owners build their own enclosures using chicken wire or other fencing materials. If leash walking a cat, the cat should first be acclimated to a well fitting harness (such as the Come with Me Kitty® harness by Premier) and leash indoors before
venturing outside.

**Conclusion**
FIC and Obesity are multi-factorial problems that necessitate multi-modal approaches to achieve treatment success. While nutritional intervention may be important in both of these conditions, behavioural interventions should also be part of the treatment plan.

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Dr. Joane Parent, BSc, DVM, MVetSc, ACVIM Neurology
Seizing again! How to evaluate the dog with seizure activity!

The seizing cat! Recognizing, evaluating and treating appropriately!

Dizzy dogs and cats! The vestibular system revisited!

On the ‘catwalk’! Localizing the ataxia!

The dog trot! Expanding the perspective of lesion localization!

Evolution of the treatment of intervertebral disc disease.
Dr. Anthony Pease, DVM, MS, DACVR
Imaging Overview: Choices for the Veterinarian in the Digital Age
Anthony Pease, DVM, MS, DACVR

Diagnostic imaging has seen a huge technology shift in the last 10 years. Modalities that were not accessible to the small animal patient, such as magnetic resonance imaging, are now considered the modality of choice for neurologic examinations. This technology shift has caused a lot of confusion as well as questions about what modalities are used for which diseases and why. The purpose of this article is to explain the different modalities including conventional radiography, ultrasound, nuclear medicine, computed tomography and magnetic resonance imaging, their uses and the pros and cons of each.

Radiography is the oldest and widest used diagnostic imaging modality available. Since its discovery by Wilhelm Conrad Roentgen on November 8, 1895, several changes have been made. These changes include the use of screens to minimize patient dose while increasing the efficiency of information transfer from the x-rays to film. In addition, automatic processors were invented to speed the development of the film to generate an image. Computed radiography (CR) and digital radiography (DX) have been created to optimize contrast resolution and create a virtual image that can be stored in a computer, rather than on a shelf by creating a digital image. These modalities can be further divided into direct and indirect imaging. Direct imaging occurs when the x-ray photon directly strikes a detector to create an image. This will provide the greatest spatial resolution for digital images, but it is still less than screen-film combinations. Indirect imaging is when the x-ray photon interacts with a phosphor in the screen to transform the x-ray photon into light. The light can then expose the imaging plate with greater efficiency and minimal loss of resolution.

The choice of which system to buy will be guided by your needs as a practitioner. Digital, indirect radiography such as a charged coupling device, is inexpensive but provides a rapid digital image. This system generally comes with an x-ray table and a large device that works similar to a digital camera. Other forms of indirect and direct digital radiographic systems may have an imaging plate but are considerably more expensive. In exchange, for the added expense more detail and better imaging quality is obtained. Computed radiography is an indirect cassette based system much like conventional radiographs. When the cassette is exposed it is placed in a reader to generate the image. This can take around 45-60 seconds, but is mildly less expensive (depending on the number of cassettes required) and more versatile than most DX systems.

The main thing to avoid is the high pressure salesperson talking of resolution. People will use the terms megapixels, pixel depth, and even line pairs per millimeter. The thing to remember is that all digital systems (with the exception of digital mammography) will have less spatial resolution than most film screen combinations. That said, it is not the spatial resolution we care about. Spatial resolution, the ability to see to objects of similar opacity next to each other, is not as importance as contrast resolution. Contrast resolution is the ability to see two structures of slightly different opacities next to each other. This is where digital imaging (direct and indirect) is superior. Because it is possible to adjust the grey scale on the images after exposure, the ability to identify small fragments, areas of mineralization or nodules within the lungs, is far greater with digital imaging modalities compared to conventional film. The choice
of which vendor and technology is right for your clinic is difficult and it is recommended that you seek help from a board certified radiologist or advice from colleagues who have the system you are interested in, to guide your purchase choice.

Radiography is the method of choice for rapid evaluation of the skeletal system and the thorax. Pulmonary edema can only be evaluated with radiography (be it computed tomography, digital radiography or conventional) and fractures, though seen with ultrasound and nuclear medicine, can best be evaluated with some form of radiographic technique. In addition, radiography can be used to give an overview of the abdomen. Unlike ultrasound, which will be discussed next, radiographs can help look at large gas filled structures that are not easily evaluated with ultrasound. Examples include gastric dilation with volvulus and mechanical obstructions. It is possible to identify these with ultrasound as well, but radiography remains faster and easier to make the diagnosis.

Ultrasound is a rapidly growing, non-invasive method to evaluate any portion of the body. Ultrasound involves sound waves that enter the body and are reflected in various degrees that allow the generation of an image. The main strength of ultrasound is to be able to differentiate soft tissue compared to fluid. With radiography, soft tissue and fluid have the same opacity. With ultrasound, it is possible to see the cortex compared to the medulla of the kidney as well as tell the difference between the portal vein and the hepatic veins of the liver. The main drawback to ultrasound is that it is a technical skill that requires practice and patience as well as guidance to perform a good quality examination.

Use for ultrasound centers around any structure that the probe can be placed on. From eyes, to the heart, to lungs, abdomen and musculoskeletal system, ultrasound can provide diagnostic information. The musculoskeletal ultrasound, though not used in veterinary medicine to the degree as in human medicine, is a useful tool for joint swelling, aggressive bone lesions, and muscle damage, especially when guiding a needle or biopsy to get a sample of the lesion. Ultrasound has virtually eliminated the need for all contrast medium procedures as well. At Michigan State University, we rarely perform barium procedures such as upper gastrointestinal tract examinations, excretory urography and even cystography. The use of ultrasound has replaced these more time consuming and invasive procedures with a rapid, non-invasive modality that provides a large amount of detail centering on soft tissue evaluation and differentiation. By far, the most common use of ultrasound is for evaluation of the abdomen. Being able to examine the internal architecture of organs and imaging through a peritoneal cavity full of fluid to identify a mass has allowed abdominal ultrasound to replace radiography as the routine screening procedure for abdominal pain. One other use of ultrasound worth mentioning is the evaluation of the brain. If MRI and CT are not available to you, imaging through an open fontanel or through the foramen magnum can aid in identification of hydrocephalus as well as increased intracranial pressure with the use of pulse wave Doppler. The applications for ultrasound are quite vast, but it is a rapidly growing, inexpensive and exciting field of diagnostic imaging that can provide a large amount of information regarding a patient.

Nuclear medicine has some uses in small animal medicine mainly centered on portosystemic shunt evaluation. Nuclear medicine works similar to radiography in that radiation is used; however, instead of beaming radiation through a patient, we are administering a radioactive material and observing where it goes. This is done by binding
radioactive material to another substance that will allow us to observe bone turn-over and remodeling or how well the lung is being perfused. For portosystemic shunt evaluations, 99m Technetium pertechnetate is administered without binding it to another substance. This is injected either into the rectum or into the spleen and the blood flow to the liver and heart is evaluated. This method provides a qualitative analysis will provide the idea if a macroscopic portosystemic shunt is present, but not the location. Generally ultrasound or more recently CT and MRI have been used to give the surgeons an idea of location. Binding the 99m Technetium pertechnetate to methylene diphosphonate (MDP) allows for evaluation of bone remodeling seen with metastatic neoplasia (like osteosarcoma) or discospondylitis. Though this is very sensitive to detect changes, bone generally remodels for up to 3 years after an injury, so the clinical importance is difficult to assess. 99m Technetium pertechnetate can also be bound to macro-aggregated albumin (MAA) to allow for evaluation of right to left shunts and for pulmonary thromboembolism. By attaching the radioactive Technetium to a large molecule like MAA, the Technetium will stop at the first capillary bed. If that happens to be the left kidney, then there is a right to left shunt (assuming you injected the radiopharmaceutical in a vein). If there is a focal area where no activity is seen in the lung it is considered secondary to an infarcted area likely due to pulmonary thromboemboli.

Computed tomography is an advanced form of radiography. An x-ray tube and detector is used, but the difference is the tube spins around the patient to allow a cross-sectional image of the patient to be generated. This eliminates the superimposition of structures seen with conventional radiography. This method of imaging is fast, easy to perform and provides a large amount of information rapidly. With the newer CT scanners, an entire dog and be imaged in less than 2 minutes allowing for sedated CT examinations rather than placing the patient under general anesthesia. Computed tomography requires specialized equipment and training usually only found at referral hospitals and institutions, but mobile CT facilities and outpatient centers are starting to become more prominent. The use of CT in the veterinary patient centers on musculoskeletal abnormalities, and is considered the gold standard to look for pulmonary metastatic disease due to the lack of superimposition and superior contrast resolution. Other examinations including the brain and soft tissue masses can be performed with administration of intravenous contrast medium. The spinal cord can also be evaluated, but the evaluation is limited to evaluating mineralized disc material and neoplastic masses.

Magnetic resonance imaging has slowly gained acceptance as the modality of choice for neurologic disease. Magnetic resonance imaging works by aligning all the atoms in the patient into one direction, then, using a radio frequency pulse “tuned” into the hydrogen atom, we are able to image various tissues based on their water composition. We call these sequences T1 and T2 weighted based on the time it takes for the atom to return to the resting state. This means that MRI can selectively look at free fluid and alternatively see edema and other soft tissues in an anatomic as well as a physiologic state. Since bone has little water present, it also has little signal on MRI and is difficult to evaluate.

Many options are open to the practitioner involving imaging that can be done to help provide a diagnosis. The goal of this lecture was to familiarize you with the different modalities available to you as a clinician to help guide you with management and diagnostic decisions that you make every day. Not every modality is the best choice in every case, nor is it feasible based on cost of the procedure. Therefore, careful consideration of all the technology at your
disposal is necessary in the ever changing world of medicine.
Thoracic Radiography: How to See the Pattern Through the Trees
Anthony Pease, DVM, MS, DACVR

Thoracic radiographs are the mainstay of diagnostic imaging. The debate between two view and three view thoracic radiographs may continue, but no one argues that imaging the thoracic is the most complicated and more informative radiographic procedure available. With the contrast provided by the lungs, soft tissue opacities and radiographic changes within the lungs are easy to see, but hard to interpret. By far, a normal thoracic radiograph is still the most difficult to interpret.

Radiographic technique and positioning is the most important thing to thoracic radiographic interpretation. The first priority is the proper radiographic technique. Due to respiratory motion, the kVp setting is set high (generally 100 or 120) and the mA is also maximized to keep a small exposure time to minimize motion artifacts. Recumbency is also a major factor in radiographic interpretation. The lung needs to be aerated in order to see radiographic changes since the soft tissue opacity of the lesion needs to contrast with the aerated lung. Therefore, if a lesion is in the right cranial lung lobe, then a left lateral radiograph is needed. Alternatively, if the lesion is in the left caudal lung lobe, especially in the dorsal aspect, a dorsoventral projection should be performed.

Once the radiograph is obtained, the next step is to determine if the lungs are too white, too dark or normal. The second question is if this change is secondary to technique or pathology. To determine if the increased opacity is secondary to technique, one should evaluate the degree that the first and second thoracic spinous process can be seen, also the degree of contact between the diaphragm and the heart as well as the ability to see the pulmonary vasculature and the superimposed triceps musculature on the thoracic inlet.

Once you decide a lesion is present, the next debate that is currently going on is the importance of pulmonary patterns versus location. Pulmonary patterns are divided into alveolar, interstitial and bronchial lung patterns. These patterns were based on air bronchograms, increased opacity to the lung fields or increased thickness of the bronchial wall creating increased lines and rings, respectively. That said, generally, it is easier to consider the radiographic pattern as a degree of severity with alveolar being the most severe, interstitial is moderate and bronchial being mild pulmonary disease. The alternative way to evaluate the lungs is to decide on the location and the distribution of the pathology identified.

For location, you can divide pulmonary disease into cranioventral, caudodorsal or diffuse disease. Cranioventral disease has 3 differential diagnoses: bronchopneumonia, hemorrhage or neoplasia. If it is caudodorsal there are 2 differential diagnoses: cardiogenic and non-cardiogenic pulmonary edema. Diffuse can be any of the five diagnoses. If the lesion is not occupying a lung lobe and is more structured, it can have a focal or multifocal distribution. A focal pulmonary lesion can be a tumor, granuloma, abscess or bulla (if radiolucent), whereas multifocal lesions tend to be neoplasia, fungal granulomas or pulmonary osteomas (which are < 5 mm soft tissue to mineral opacities throughout the lungs, generally seen in Collies).

If pleural fluid is present, retraction of the lung lobes away from the body wall can be seen. In cats, if this retraction remains after the fluid is removed, restrictive cardiomyopathy is considered most likely. If a cranial mediastinal mass is suspected, a standing horizontal beam radiograph can be obtained with the dog or cat standing on their hindlimbs and a ventrodorsal...
projection obtained to cause the fluid to be caudal to the heart. If pleural fluid is seen, the first thing to evaluate is the ribs, as rib tumors are a frequent, overlooked cause for pleural fluid. Also, radiographs can help identify a site to obtain a sample of the fluid, which can provide insight to the cause.

The cardiovascular structures of the lungs can also be evaluated to provide further information if a cardiogenic pulmonary edema is suspected. The cardiac silhouette is comprised of the heart and the blood within the heart as well as the surrounding pericardium. Since fluid and soft tissue have the same opacity, a difference between these structures cannot be identified. If the heart is enlarged, generally chamber enlargement is seen such as the left atrium or right atrium. Cardiac changes are generally vague and only occur when the changes are severe. When the heart hypertrophies, it undergoes concentric or eccentric hypertrophy. Concentric hypertrophy is secondary to a pressure overload. If the heart can compress hard enough, it can push the blood out of the chamber. The heart then hypertrophies the muscle to create a smaller lumen. The heart shape remains the same and therefore cats with hypertrophic cardiomyopathy and dogs with pulmonic or subaortic stenosis will not have radiographic signs of cardiomegaly until the disease is very advanced. Alternatively, eccentric hypertrophy is secondary to a volume overload. No matter how strong the contraction, the fluid cannot clear the chamber so the hypertrophic muscle is formed on the outside of the lumen. This change can be seen radiographically, but is a rare condition, mainly occurring with dilated cardiomyopathy.

Pulmonary vasculature can also be evaluated to help to determine the cause for a caudodorsal lung pattern. If the pulmonary artery is dilated, the primary cause is pulmonary hypertension from any cause. In adult dogs, the main cause is secondary to heartworm disease or pulmonary thromboembolic disease. If the pulmonary vein is enlarged, then generally it is a sign of left-sided heart failure. This vein enlargement is first seen in the right caudal lung lobe and then progresses to the remaining lung lobes with time. If both the arteries and veins are enlarged, then that is caused by over circulation, such as a patent ductus arteriosus or ventricular septal defect. Small vasculature is a rare finding, but may be secondary to hypovolemia, hypoadrenocorticism or severe pulmonic stenosis.

Radiographic interpretation of cardiac disease is considered difficult and numerous studies have tried to identify the easiest methods to simplify the interpretation. Using vertebral heart score, inverting the image so that black is white and white is black, even rotating the image to look for rib lesions. All these methods have found that nothing is better than experience at image interpretation and practice. In addition, with the rapid expansion of digital imaging, bronchial lung patterns are being over diagnosed due to the increased image resolution. Having normal radiographs and evaluating the entire image, included the surrounding musculature and skeletal structures is essential to make accurate diagnoses.

Thoracic radiography is considered a challenging region to interpret not because the lesions are difficult to see, but rather because the lesions identified are generally non-specific and are difficult to interpret. Generally, most practitioners see a cranioventral alveolar lung pattern and diagnose aspiration pneumonia and a caudodorsal lung pattern as pulmonary edema. In truth, the thoracic radiographs should be evaluated as a whole, is there a megaesophagus or history of vomiting? Is there heart murmur, enlarged pulmonary veins or enlargement of the left atrium of the heart? These questions should be asked prior to
starting therapy with the hope the diagnosis is correct. Thoracic radiographs are not obtained to determine if a disease is present, but rather to identify the extent of disease and determine the progression or regression. Bearing in mind that radiographic improvement may lag behind clinical improvement by several days.

Although thoracic radiography is challenging, this lecture will provide an overview of normal anatomy as well as case examples of common disease processes to help provide the participant with an increase in knowledge and level of comfort interpreting pulmonary and cardiac changes.
Abdominal Radiography in the Acute Abdomen
Anthony Pease, DVM, MS, DACVR

Abdominal radiographs are a rapid, readily available method to give an overview of the abdomen. Though most people believe ultrasound is the new modality of choice for abdominal evaluation, the limitations of ultrasound not being able to penetrate gas as well as the technical ability and time to acquire images still make abdominal radiographs a great first modality in the patient with acute abdominal pain.

Ultimately, the question for the clinician with an abdominal patient is whether surgery is indicated or if medical management is the best course of action. With radiographs providing an overview of the entire abdomen, and the use of the gas within the bowel to provide contrast, abdominal radiographs can be useful as a triage tool that can be augmented and finding further characterized using abdominal ultrasound.

When evaluating the stomach, generally most abdominal radiographs include a right lateral and ventrodorsal projection. The question always arises on why this is performed. These two views have become the standard since a right lateral projection places gas in the fundus of the stomach and fluid in the pyloric antrum. To evaluate the pylorus, a ventrodorsal projection is used to put fluid in the fundus and gas in the pyloric antrum. At Michigan State University, we take 3 view radiographs of all abdomens to include a right lateral to seen the fundus, a left lateral to evaluate the pylorus and look for pyloric outflow obstructions and a ventrodorsal to provide more information about the pylorus and to better evaluate the colon.

With the availability of ultrasound, the use of contrast medium for upper gastrointestinal contrast medium procedures is not routinely performed. However, in clinics without the benefits of ultrasound, barium or iodinated contrast medium procedures still provide some use to evaluate if a luminal obstruction exists, if the bowel wall is think or infiltrated, look at overall motility or assess for a rupture. The main drawback to this procedure is that if any of those differential diagnoses are suspected, an exploratory laparotomy is indicated rather than a contrast procedure that could delay surgery by 3-6 hours.

Barium contrast medium is the most universally used for gastrointestinal imaging. It is safe, the dose is 6-10 milliliters per pound and generally is administered through a gastric tube. If aspirated, barium causes physical obstruction of the airways with no inflammatory component, but may cause granulomas if it leaks into the peritoneal or pleural cavity. For this reason, barium is contra-indicated if a ruptured bowel or ruptured esophagus is suspected. Iodinated contrast medium is generally used intravenously but can be administered orally. The main limitation is that it has a bad taste, is hypertonic so it will draw fluid into the bowel and since it is hypertonic, will cause an inflammatory reaction if aspirated into the lungs.

Positional radiography can also be used to evaluate for free gas in the abdomen. Since an air/liquid interface is needed to help to see gas within the peritoneal space, a horizontal beam projection with the dog on its left side and obtaining a ventrodorsal projection will put the gas in the right lateral abdomen near the pyloric antrum. Since the pylorus is small, the gas accumulation will be identified caudal to the diaphragm.

For gastric dilation with volvulus, the main feature is to obtain a right lateral radiograph. No other projection is needed. If the pylorus is seen in the craniodorsal abdomen, a GDV is
confirmed. Numerous times people have been fooled by the normal appearance of the ventrodorsal projection and decided the case was just gastric dilation. Nothing else can put the pylorus in the craniodorsal abdomen except for a GDV.

Small intestinal wall thickness is also something frequently evaluated on survey radiographs. This cannot be done. Since soft tissue and fluid are the same opacity, it is impossible to know if the structure observed is a thick wall or just a combination of fluid summating with the small intestinal wall.

The abdomen is divided into two spaces, peritoneal and retroperitoneal. The retroperitoneal space contains the adrenal glands, kidneys and sublumbar lymph nodes and the peritoneal space contains the remaining organs. This determination is important since it will aid in the differential diagnoses of a mass that is present or the cause for gas within the abdomen. The retroperitoneal space is dorsal to the colon. Therefore if a soft tissue mass displaces the colon ventrally, then the mass is likely retroperitoneal indicating it is either arising from the kidney or adrenal glands. If gas is present in the retroperitoneum, this is likely secondary to a pneumomediastinum rather than a rupture of the gastrointestinal tract.

Radiographs are useful to determine if a surgical obstruction or mass is present or at least provides a general overview of the abdomen. Though barium contrast medium can be used, this has largely been replaced with ultrasound or exploratory surgery. By the end of this lecture, the audience will seen numerous examples of radiographs for surgical and non-surgical lesions and how a better understanding of the limitations and benefits of abdominal radiography.
Radiography of Bones: Is it more than a Fracture?
Anthony Pease, DVM, MS, DACVR

The evaluation of the musculoskeletal system is difficult due to the numerous soft tissues as well as the bone structures involved. Rapid assessment of the bone structure is routinely performed using radiographs; however, the subtlety of disease and joint compared to bone pathology can be confusing. The purpose of this lecture is to cover the identification of aggressive compared to non-aggressive bone lesions as well as erosive compared to non-erosive joint pathology.

When evaluating the skeletal system, the first thing to determine is if the lesion is aggressive or non-aggressive. A non-aggressive lesion diagnoses include callous, malunion fractures, bone cysts, osteomas, osteochondritis dessicans, panosteitis, fragmented medial coronoid process, osteoarthritis or metabolic disorders. Aggressive lesions are due to neoplasia or osteomyelitis.

When deciding about aggressive lesions, there are 6 radiographic signs that are used: bone lysis, periosteal reaction, rate of progression, zone of transition, cortical lysis. Bone lysis has three different appearances, geographic (focal) moth-eaten and permeative. The difference between the degree of lysis is mainly on the rate of progression. It requires approximately 50% of the bone per unit area to be destroyed before it is visible on radiographs. This is because the bone is a three dimensional object viewed from two dimensions. Because of this, bone is superimposed on itself, making subtle lesions hard to detect. The more lysis that is present, the easier it is to see on radiographs. Also, by the time lysis is seen on a radiograph, the lesion is quite severe.

Periosteal reaction can either be smooth (continuous) or interrupted. The easiest way to determine this is if you could trace the outline of the periosteal reaction with a pencil and never have to lift the pencil from the radiograph. Smooth periosteal reactions are generally associated with trauma whereas interrupted periosteal reactions are due to an aggressive process.

Rate of progression is probably the most overlooked method to assess an aggressive lesion. By the time a questionable aggressive lesion is seen on a radiograph, the lysis is quite substantial. Therefore, the rate of progression in 2-4 weeks will also be dramatic. If a question exists between an aggressive and non-aggressive lesions, supportive medical management for 2-4 weeks then repeat radiographs to look for progression can aid in determining if the lesion is aggressive.

Zone of transition is a more nebulous sign, but the idea is that if a clear-cut demarcation between normal and abnormal bone is seen, then the lesion is more likely non-aggressive. If there is a long zone of transition, the difference between normal and abnormal bone is blurred and the lesion is more likely to be aggressive. In addition, cortical lysis as opposed to overall bone lysis can be used to determine aggressive bone lesions. If the cortex is thin, but no lysis is present, then it is more likely that the lesion is non-aggressive.

After determining these radiographic signs, the next clue is based on the location of the lesion. If the lesion is generalize in that it effects all bones equally, then the primary differential diagnosis is a metabolic or nutritional abnormality. If only one bone is involved, this is a focal or monostotic lesion and a primary bone tumor or soft tissue tumor with secondary bone
involvement is considered most likely. If multiple bones in the same region (locally extensive),
different bones that are not in close proximity or multiple areas in the same bone are involved,
this generally indicates a hematogenous spread disease as bacterial osteomyelitis or metastatic
neoplasia. A soft tissue tumor with secondary bone involvement is possible with locally
extensive lesions, such as aggressive lesions that cross a joint.

Anatomic location is also a key into the differential diagnoses. If the lesion is epiphyseal
or physeal in origin, then it is likely secondary to infection, trauma or potentially a nutritional
abnormality. These lesions are generally in juvenile dogs and cats. If the lesion is in the
metaphyseal region, then a primary bone tumor or hematogenous infection is most likely due
to the proximity of the nutrient foramen. If the lesion is diaphyseal, then the lesion is likely
metastatic neoplasia, a soft tissue mass with secondary bone involvement or a focal infection
related to a penetrating trauma.

After all these signs and locations are taken into account, then the differential diagnoses
are prioritized based on the signalment and history of the patient. A 2 year old hunting dog
with an aggressive bone lesion in the proximal metaphysis of the humerus is more likely to have
a fungal infection; however an 8 year old Rottweiler with the exact same radiographic findings
is more likely to have osteosarcoma. These considerations should be made when assessing
aggressive lesions. Since osteosarcoma is a common tumor type, it is not uncommon for
clinicians to see an aggressive lesion, even if it is locally extensive and crosses a joint, and
consider a primary bone tumor like osteosarcoma. However, other tumors such as malignant
histiocytosis, synovial cell (histocytic) sarcoma, or even fibrosarcoma, chondrosarcoma and
metastatic neoplasia can all be considered possible. Biopsy (excisional or incisional), thoracic
radiographs and history may aid in further prioritizing the lesion.

Lesions centered on joints are similar to those in bone. These lesions are centered on
the epiphysis of both sides of the joint. Just as aggressive and non-aggressive lesions exist in
bone, erosive and non-erosive lesions are in joints. A non-erosive lesion is osteoarthritis. Everything else is considered erosive. Osteoarthritis is a degenerative condition due to joint
instability or trauma. It is characterized by the presence of osteophytes and enthesiophytes.
An osteophyte is smooth bone production within the joint capsule that serves as a buttress to
tighten ligaments and stabilize the joint. Enthesiophytes are bone production at the
attachment of the joint capsule and ligaments due to abnormal tension that is present on the
soft tissues from joint instability.

Erosive lesions in small animals are usually infectious and mostly autoimmune in origin.
Causes of erosive arthropathy also include chronic hemarthrosis or neoplasia, but these are less
likely in small animals. Just as with bones, joint lesions are characterized by the number
involved. A monoarthrosis (one joint) is usually osteoarthritis or a traumatic infection, such as a
puncture wound. A polyarthropathy (multiple joints involved) usually indicates a
hematogenous infection or immune mediated disease.

The radiographic signs for an erosive arthropathy include subchondral bone lysis,
presence of osteoarthritis, decreased joint space (especially when weightbearing), luxation or
subluxation of the joint and fragmentation of adjacent bone. Based on these signs, and the
presence of one or multiple joints involved, a arthrocentesis can be performed to determine
the cause for the erosive arthropathy.

Radiographic findings of joint an bone lesions can be confusing if one does not consider
the vast number of differential diagnoses possible and then makes an educated decision to prioritize the lesion. This is generally done by the clinician automatically due to the geographic location and the likelihood of disease in a given area. If a dog in southern Michigan presents to Michigan State University for an aggressive bone lesion, neoplasia is more likely. However, if the patient is from northern Michigan, then fungal osteomyelitis should be considered possible. At the end of this lecture the hope is that the veterinarian will have numerous examples and a better overall appreciation of how to evaluate a radiograph for aggressive and erosive lesions.
Abdominal Ultrasound: The Most Useful, Under Used Tool in Your Practice
Anthony Pease, DVM, MS, DACVR

The use of radiography to examine the abdomen is full of complications. Radiographs are very good at determining the difference between bone and gas, but soft tissue and fluid are the same opacity. When dealing with intra-abdominal lesions, the main goal is to differentiate one soft tissue mass from a normal soft tissue structure from abdominal fluid. Ultrasound uses high frequency sound waves to accomplish what radiographs cannot. With ultrasound, fluid and soft tissue can be clearly distinguished from one another, where bone and gas cannot. The purpose of this proceeding is to describe the benefits and uses of abdominal ultrasound to the general practitioner.

Abdominal ultrasound is a unique diagnostic test in veterinary imaging. Unlike blood work, radiographs, computed tomography or magnetic resonance imaging, ultrasound requires the sonographer to both acquire images as well as interpret them. This unique combination is why physicians have allowed technicians to acquire the ultrasound images and that leaves radiologists free to perform other studies and interpret the images acquired. This model has not been accepted in veterinary medicine as yet.

So the first stage to abdominal ultrasound is gaining the technical skill to acquire images. This requires patience and time, but is relatively easy with practice. Where ultrasound skill comes into play is with adaptation for disease processes. It is necessary to know that if you suspect portal hypertension, you need to look behind the left kidney for acquired portosystemic shunts. If you see a thrombus in the splenic vein, you need to evaluate the portal vein for thrombosis as well. This is the degree of medicine that keeps the ultrasound probe in the hands of the veterinarian.

Ultrasound examinations have nearly replaced abdominal radiographs at Michigan State University. As an example, on a given day, we performed 14 ultrasound examinations and 3 abdominal radiographic series, generally performed during emergency hours. This replacement has occurred because ultrasound provides better detail and more information about the abdomen compared to plain radiographs. Though we have virtually replaced radiography, radiography is more rapid and gives a better overview of the abdomen compared to ultrasound. For example, a gastric dilation with volvulus can be diagnosed with ultrasound, but it would be easier and more accurate to use radiography to identify the gas filled pylorus displaced dorsally and to the right.

Once the images have been acquired, the next step is interpretation. When ultrasound was first used, it was the first non-invasive, cross-sectional imaging modality. This means that rather than just seeing the outline of an organ, you can now see the portal vein within the liver and the medulla within the kidney. Ultrasound images were compared to gross necropsy examination, but done in a much less invasive manner. Since the image generated can see into the organ, it is very sensitive to find morphologic changes such as masses, cysts, abscesses and tumors. However, unlike gross pathology, you no longer have color and smell to aid in your diagnosis. For this reason, an abscess can look just like a tumor which can look just like a blood clot. This is why we considered ultrasound very sensitive for disease, but not very specific. The benefit of ultrasound is the ability to identify a lesion in an organ of interest as well as aid in obtaining a sample, either with fine needle aspiration or with a biopsy to help determine the
true nature of the lesion.

Common lesions identified using ultrasound include: foreign body obstruction, mucocele formation, splenic hemangiosarcoma and urinary tract disease will be shown for the purpose of illustration. Previously, a foreign body obstruction could only be identified if it was completely obstructive, was radiopaque or radiolucent and if there was marked dilation orad from the lesion. With the superimposition of other organ structures, sometimes barium was used to evaluate wall thickness and motility. Ultrasound has virtually eliminated the need for barium studies and allows the evaluation and identification of foreign material, whether completely or partially obstructed, within the gastrointestinal tract. This is because that any foreign material, whether it is made from wood, cloth or metal, will absorb sound and cast a dark shadow deep to the lesion. That coupled with the increased ability to identify small intestinal distension and wall layering, makes the determination between a foreign body obstruction and a neoplastic mass easily distinguished.

A mucocele is a chronic form of cholecystitis. Generally, a patient presents with a chronic history of intermittent vomiting followed by an acute onset of collapse or severe, unrelenting vomiting. Ultrasound is the only method available to non-invasively examine the gallbladder and bile duct for evidence of obstruction or mucocele formation. A mucocele has the unique appearance of linear striations that radiate from the center of the lumen. These radiations are thought to be bile salts trapped within a thick, hypoechoic (dark) mucosal wall. At this stage, the gallbladder is considered a nidus for infection and a surgical emergency since it has a high risk of rupture if left in place.

Large splenic masses are generally easily identified on radiographs or ultrasound (as well as physical examination). The difference is in the dog that presents with acute collapse and a hemoabdomen. It is true that with a hemoabdomen and no history of trauma, you can perform an exploratory surgery to find the source of the bleeding, but this is usually difficult to do. Instead, ultrasound evaluation of the abdomen to look for a mass as well as metastatic disease is considered the non-invasive method of choice to help with the surgical planning.

Lastly, urinary tract abnormalities such as hydronephrosis, perinephric pseudocysts, transitional cell carcinoma and cystitis can all be evaluated without the use for contrast medium or general anesthesia in a rapid non-invasive way using ultrasound. Examination of the kidneys will show if the renal pelvis is dilated or the kidney is surrounded by fluid. With radiographs, since fluid and soft tissue are the same opacity, it is not possible to make this determination without contrast medium, which is considered nephrotoxic. Instead, ultrasound can show the architecture of the kidney as well as help find a distended ureter if one is present. The wall of the urinary bladder is also a dilemma with ultrasound since the urine will obscure the luminal margin. With ultrasound, small areas of mineralization within the mass as well as proliferation of the wall seen with cystitis can be quickly and accurately identified, though differentiating tumor versus inflammation is difficult without obtaining a sample with traumatic catheterization.

Abdominal ultrasound in the general practice has the potential to provide a practitioner with rapid information to help facilitate referral or further diagnostic tests especially in the vague, chronically ill patient. With practice, guidance and perseverance, it is possible to use this modality as a triage tool as well as method to determine the progression and regression of disease.
Computed Tomography: How can it Help the General Practitioner?
Anthony Pease, DVM, MS, DACVR

The use of computed tomography (CT), though not readily available at every institution, is becoming a more widespread modality for use in the small animal patient. Computed tomography affords a rapid evaluation of skeletal images with a small slice thickness that can be as small as 0.625. Computed tomography in chondrodystrophic breeds of dogs is being performed instead of myelography due to the rapid acquisition and ability to see mineralized disc material. Due to the increased availability of multislice CT scanners, sedated CT scans are becoming possible due to the fast acquisition time. The purpose of this proceeding is to describe the physics and benefits of CT imaging in the small patient and the new uses that this modality provides.

Computed tomography is a method of image acquisition similar to radiography. An x-ray tube is used to generate photons that pass through a patient and is read by a detector (similar to digital radiography). Where CT differs is the fact that the x-ray tube spins around the object being imaged to provide a cross-section of each section of the object without superimposition of structures as occurs with conventional radiographs. This elimination of superimposition allows for enhanced ability to detect fracture fragments, improves the ability to localize lesions (such as dental disease and sinus lesions) and increases the ability to see smaller lesions, when compared to conventional radiography.

Computed tomographic technology is generally talked about in terms of what “generation” a scanner is considered. For example, a third generation scanner (also called a rotate-rotate scanner) has a series of detectors that spins with the x-ray tube. A fourth generation scanner (also called a rotate-stationary scanner) has a spinning x-ray tube and a complete ring of transducers that do not move with the x-ray tube. A sixth generation scanner is the most widely available today and this is also called a helical scanner. The difference is that a fourth generation scanner generally has a large number of detectors, which means better resolution, when compared to the third generation scanner. A helical scanner allows for continuous acquisition of an image while the table moves to decrease the scan time required.

The way an image is generated in a normal acquisition is that the x-ray tube completely circles the desired area and an image is generated. This provides the best possible resolution, but when three-dimensional reconstruction is performed, the image can look very irregular. This is because, like slices of bread, each slice will not perfectly align with the image cranial and caudal making a stepped appearance. During a helical acquisition, the table continues to move through the gantry allowing only 66% of a complete revolution around the object. The remaining 34% of the image is then extrapolated from the slice in front of the image and behind the image. Though this means that the image is not completely accurate, but it provides for a smoother transition between slices, smaller slice thickness and a more rapid acquisition.

Due to the use of x-ray technology, the use and interpretation of CT is more intuitive then other modalities like ultrasound or magnetic resonance imaging. Computed tomography works on the basis of attenuation of the x-ray beam. Due to this difference in attenuation, it is possible to see acute hemorrhage, mineralization, small fragments of bone secondary to a fragmented medial coronoid process, as well as the difference between fluid and soft tissue when contrast medium is given.
Computed tomography’s main benefits are rapid acquisition and lack of superimposition. The average CT study can take approximately 2-3 minutes per acquisition. Multislice CT scanners are similar to single slice, except that the multislice scanners have multiple rows of detectors allowing more than one slice to be acquired at a time. Multislice scanners range from 2 slice to 256, or infinite, slice. The more detectors that are present, the faster the acquisition and the smaller the possible slice thickness. However, just like with radiographs, a small slice thickness means more energy is required, which would adversely increase the patient’s radiation dose. A complete head CT of a dog using a multislice CT scanner generally will take 20 seconds.

Due to the rapid acquisition and the amount of image detail, CT is considered the first imaging modality to evaluate the nasal cavity, the elbows and the thorax for pulmonary metastatic disease. Computed tomography is also ideal for trauma patients or patients that are paralyzed and are not stable enough for longer procedures like MRI. With the ability to reconstruct images in multiple planes as well as generating three-dimensional images, CT can provide rapid guidance for surgeons as well as being used by oncology for radiation therapy planning.

The most common use for CT is evaluation of the head and cranial vault. Though CT does not have the soft tissue detail as seen with MRI, CT with administration of contrast medium, has the ability to see morphologic lesions of the brain that would require radiation therapy or surgical removal. Computed tomography is also superior to evaluate the bone structures of the head including the tympanic bulla and temporomandibular joints. When precise evaluation of fractures is needed, CT can provide a large amount of detail into the amount of fragmentation as well as fracture lines that are present in the bone, which may not be visible using conventional radiography.

Computed tomography is also useful in evaluating the vertebral canal for lumbosacral disease, intervertebral disc herniation and spinal tumors. If a myelogram has been performed, CT is able to provide more information about the spinal cord even hours after the contrast medium has been administered. Since fluid and soft tissue have the same attenuation, the spinal cord silhouette is seen comprising the spinal cord and subarachnoid space. However, when contrast medium is given intrathecally, then compressive lesions of the spinal cord can be properly assessed. Subtle lytic lesions seen with discospondylitis can also be seen on CT before they are visible with radiography.

In the thorax, CT has become the gold standard for pulmonary metastatic disease. This is because CT provides higher contrast resolution, which is the ability to see two objects of different opacities as different, compared to radiographs. Therefore, small soft tissue nodules are easily seen in the lungs due to the lack of superimposition. In addition, mediastinal masses and rib tumors can be easily identified and assessed using CT due to the lack of superimposition seen with radiographs.

The newest technique that is gaining more favor is CT angiography. Using timed boluses of contrast medium allows for the vasculature to be critically evaluated. Combine this with the rapid acquisition and CT is becoming the modality of choice for portosystemic shunt evaluations. Also, arterial versus venous phases can be evaluated for masses in the liver and pancreas and show promise in helping to determine neoplastic masses from hypertrophy. However, more research is needed before definitive assessments can be made. Computed
tomographic angiography is also helpful for adrenal gland masses to assess for involvement of the surrounding vasculature. Due to the large size of the masses and the central location of the caudal vena cava, accurate evaluation of vessel involvement is sometimes not possible. Using CT and contrast medium, filling defects can be seen in the vasculature to indicate a thrombus or extension of the mass into the vascular structure or even provide information about how vascular a mass is prior to surgical removal.

Computed tomography is faster and provides better bone detail than any other modality currently in use. The ability to produce sub-millimeter slice thickness that can be rapidly reconstructed into any image plane prior to performing surgery makes this the modality of choice when time is of the essence. Patients with metal implants can be imaged using CT and since ultrasound cannot penetrate gas, abdominal CT is becoming recommended instead of ultrasound especially to evaluate the abdomen in large breed dogs.

Computed tomography is more readily available due to the decreased cost of purchasing and maintaining the machine compared to magnetic resonance imaging. With newer CTs being made, multislice CT scanners (4-16 slice) are being used in veterinary medicine on a routine basis. The speed of acquisition, the possibility of using sedation rather than general anesthesia and the lack of superimposition makes CT the modality of choice for nasal imaging or lesions that are suspected to have bone involvement. Though MRI is still superior to look at soft tissue lesions or for neurologic localization, CT remains the first modality to provide rapid assessment and determination of acute surgical lesions compared to other modalities.
Dr. Erin Trageser, VMD, MSc Candidate

Feline hypertrophic cardiomyopathy: update 2012
Erin Trageser, VMD; Etienne Côté, DVM, DACVIM (internal medicine, cardiology)

Hypertrophic cardiomyopathy (HCM) is the most common acquired heart disease in cats and an important cause of morbidity and mortality. Manifestations of HCM vary from subclinical disease that may remain compensated for years to clinical signs of congestive heart failure (CHF), aortic thromboembolism, and cardiac arrhythmias.

Prevalence In subclinical disease, an auscultable heart murmur may be the first or only sign of cardiac disease. Recent studies have reported a prevalence of heart murmurs in apparently healthy cat populations between 15 and 34%.\textsuperscript{1-3} In one study in which cats with no clinical signs of heart disease underwent cardiac auscultation and echocardiogram, the prevalence of HCM was 15.5%, and the positive predictive value of a murmur leading to a diagnosis of HCM was only 31%.\textsuperscript{4} This, coupled with recent similar evidence in a UK-based population of cats, suggests that although highly prevalent, HCM may not account for up to two thirds of auscultable murmurs in cats.\textsuperscript{4,5}

Consider these two important differentials for an incidentally identified heart murmur in an apparently healthy cat:

- Hypertrophic cardiomyopathy (mild, moderate, or severe)
- Physiologic murmur, particularly \textit{dynamic right ventricular outflow tract obstruction} (DRVOTO), a benign but well recognized physiologic phenomenon

Definitive identification of the source of a murmur requires echocardiography, which likely entails referral to a veterinary cardiologist. A conversation between the referring veterinarian and owner of a cat with an incidentally identified murmur should include the option of referral for and possible outcomes of diagnostic evaluation, the high prevalence of occult heart disease in otherwise healthy cats, and the role of early identification of HCM for proper management and prognosis. The optimal time for referral, (or at least, discussing the option of referral with the owner) is upon first identification of a murmur since HCM, like most forms of acquired heart disease in cats, is irreversible and often progressive.

Biomarkers Given the time and expense often incurred by owners pursuing specialty care, recent research interest has focused on the role of biomarkers in diagnosis and prognosis of HCM. These blood-based tests potentially provide a widely available, cost efficient, minimally invasive screening tool for HCM. The most heavily researched biomarkers include cardiac troponins and the natriuretic peptides.

\textit{Cardiac troponins} The clinical utility of serum concentrations of cardiac troponins as a screening tool for HCM has not been reliably established. Cardiac troponins are sarcomeric proteins released from myocytes in the presence of myocardial damage such as hypoxia, ischemia, and pressure and volume overload. Serum concentrations of cardiac troponin I (cTnI) provide a sensitive and clinically useful measure of myocardial damage in various human and canine
cardiac conditions. There is evidence that cats with moderate to severe HCM have higher serum [cTnI] than normal controls, but the difference between normal and mildly affected cats remains ambiguous.6,7 Likewise, there is conflicting evidence regarding serum [cTnI] in cats with and without congestive heart failure. In a study investigating the role of [cTnI] measurements in dyspneic cats, plasma concentrations ≥ 0.2 ng/mL demonstrated 100% sensitivity but only 58% specificity for identifying a cardiogenic source of dyspnea, thus necessitating additional discriminatory diagnostic tests (e.g. radiograph +/- echocardiogram).7 These results suggest an overlap of dyspneic cats with and without cardiac disease exhibiting a [cTnI] between these cutoff values, thus limiting the clinical utility of the test.

*The natriuretic peptides* Atrial natriuretic peptide (ANP) is synthesized in and released from atrial myocytes in response to mechanical stretch secondary to volume load. It functions to induce natriuresis, diuresis, and mild vasodilation. Fragments of ANP’s precursor molecules (pre-proANP and proANP) have been measured in cat serum using multiple commercially available human ELISA or RIA kits.8-11 Elevated atrial pressures, such as in CHF, increase the amount of ANP and its precursor molecules in the circulation of humans with congestive heart failure. There is evidence that serum concentrations of N-terminal pro-ANP differ between healthy controls, cats with subclinical cardiomyopathy, and cats with cardiomyopathy and congestive heart failure.8,10 However, other studies have found no difference between NT-proANP levels between healthy controls and cats with cardiomyopathy.11

| At this time, conflicting evidence and the lack of a clinically available feline assay preclude the use of NT-proANP as a screening tool for HCM in cats. |

A commercially available clinical assay detecting the amino-terminal of the prohormone form of B-type natriuretic peptide (NT-proBNP) in feline plasma has recently generated a great deal of interest in the role of this biomarker in identifying cats with occult cardiomyopathy. B-type natriuretic peptide is expressed constitutively in myocytes and released preferentially from ventricular myocytes under conditions of stress (stretch, hypoxia, or ischemia). Studies have demonstrated significant differences in plasma [NT-proBNP] between cats with dyspnea secondary to respiratory disease and cats with cardiogenic dyspnea.12,13 Two studies out of the University of California-Davis suggest that plasma [NT-proBNP] is not a useful screening test for HCM because although plasma [NT-proBNP] was significantly higher in severely affected cats versus normal controls, it failed to distinguish those with mild disease.14,15 Results of a recent study out of the University of Munich differ greatly, reporting that a plasma concentration of 100 pmol/L had a 92.4% sensitivity and a 93.9% specificity for detecting even mild HCM as compared to normal controls.16 Most recently, a multi-center study compared the [NT-proBNP] in 114 normal, healthy cats with the [NT-proBNP] in 113 cats with occult cardiomyopathy (e.g. no clinical signs of echocardiographically confirmed cardiomyopathy.)17 In this population of
cats, a plasma [NT-proBNP] >46 pmol/L distinguished normal cats from those with occult heart disease with a specificity of 91.2% and a sensitivity of 85.8%. Furthermore, [NT-proBNP] >99 pmol/L was 100% specific and 70.8% sensitive for identifying affected cats. The investigators concluded that this biomarker reliably discriminates normal cats from those affected with occult cardiomyopathy and that the degree of increase is associated with particular echocardiographic parameters of disease severity.\textsuperscript{17}

It is worth noting the contrast in results between these studies, which may be due to variable population characteristics, different criteria for the echocardiographic diagnosis of cardiomyopathy, or differences in sample handling and evaluation. Until the current clinical sampling protocol is identical to that in the most recent studies, and until these results are duplicated in the general cat population, extrapolation of these results to clinical practice should be done cautiously.

\begin{quote}
Recent evidence suggests that plasma [NT-proBNP] may differentiate normal cats from those with occult cardiomyopathy and may be associated with the severity of disease. Though more clinical studies are necessary, [NT-proBNP] shows promise as a screening tool for HCM.
\end{quote}

On the heels of a study documenting an early and sensitive increase in biomarkers of collagen metabolism in humans with HCM, we are currently conducting research at the Atlantic Veterinary College to investigate whether the same biomarkers of collagen metabolism can be measured in HCM affected cats.

**Genetic testing** Over 600 mutations have been identified in humans with HCM, though a limited number (fewer than a dozen) accounts for more than half of HCM cases. A similar situation may exist in cats, making the potential value of genetic testing attractive, particularly for breeding purposes. In 2005, Meurs et al. described a single point mutation in the gene encoding myosin binding protein C3 (MYBPC3) in a colony of Maine Coon cats.\textsuperscript{18} Subsequent work identified a similar mutation in the Ragdoll breed\textsuperscript{19} and, currently, both mutations can be identified on commercially available genetic tests (Veterinary Genetics Laboratory Veterinary in Raleigh, NC: www.cvm.ncsu.edu/vhc/csd/vcgl/index.html). Prevalence of mutation carries varies between 22–41.5% of Maine Coon cats depending on geographic distribution of the population studied.\textsuperscript{20-22} Not all carriers demonstrate echocardiographic changes consistent with HCM at the time of genetic testing, likely due to age-related penetrance and/or variable criteria for echocardiographic diagnosis.

Currently:

- many cardiologists recommend that mutation carriers be removed from the breeding pool to mitigate the risk of passing the mutation onto some (heterozygotes) or all (homozygotes) future progeny.
- Some cardiologists propose that in the absence of sufficient long term evidence of phenotypic expression among heterozygotes, a genetic test result identifying a cat as a heterozygote is open to discussion between veterinarian and breeder about the cat’s potential desirable breeding qualities vs. the risk of propagating a known genetic
mutation.

- Mutation carriers should always be evaluated by echocardiography to identify the presence and severity of disease and to decide whether intervention is warranted.

Alternatively, a result indicating that the cat is not a carrier should be interpreted carefully. Specifically, a “negative” result on one of these two tests identifies the cat as a “non-carrier” of one single identified mutation on one gene. There are undoubtedly other yet-unidentified genetic mutations associated with HCM, and breeders and owners alike should be educated that a negative test does not mean the cat does not have or will never develop HCM. If a cat is showing clinical signs of heart disease, further discriminatory diagnostic tests (such as radiographs and echocardiography) is recommended regardless of genetic testing status.

**Treatment**  Currently, treatment of HCM is considered in both subclinical (asymptomatic) and decompensated (“symptomatic”) disease.

**Heart rate control**  In subclinical disease, there is a theoretical benefit to avoiding bursts of tachycardia since tachycardia compromises diastolic filling time. This is accomplished clinically by modulating heart rate with either beta-blockers (such as atenolol) or calcium-channel blockers (such as diltiazem). Earlier evidence demonstrated a clinical benefit in cats treated with diltiazem, though the logistics of treatment (i.e. thrice daily dosing) can decrease owner compliance and minimize the benefit.22 Extended release diltiazem is clinically available but has been shown to produce unreliable serum concentrations.23 Despite a paucity of evidence, the use of beta-blockers for the same purpose of preventing tachycardia has gained traction among practicing cardiologists. The authors’ preference is atenolol at a dose of 6.25mg PO q12-24 hours as determined by an exam room heart rate obtained 12 hours after administration. If the exam room HR exceeds 170, then the dosage frequency is increased to q12 hours.

The role of owner compliance and cat tolerance as determinants of treatment in the subclinical HCM cannot be overestimated, particularly in light of the lack of evidence documenting a clear clinical benefit. If the cat experiences undue stress (and, thus, tachycardia) in the process of receiving treatment, the purpose of treatment has been negated and should be abandoned and reserved for after the development of clinical signs.

Owners should be educated that once initiated, a dedicated schedule of treatment delivery is essential and abrupt discontinuation is potentially harmful to the cat. If the owner’s lifestyle cannot accommodate timely and consistent treatment, beta-blockade should not be pursued and treatment should be reserved for after the development of clinical signs. It is prudent to remember that beta-blockade should never be initiated in acute congestive heart failure. Beginning a beta-blocker (because, for example, the heart rate is very rapid) when a cat presents for acute CHF is likely to acutely reduce cardiac output and can readily be fatal. Beta-blockade initiation is always reserved for clinically stable patients. If a cat is already receiving a beta-blocker at the onset of CHF, the dose of the beta-blocker can either be maintained or cut in half while treatment is initiated to resolve critical clinical signs (ie. furosemide for pulmonary edema or thoracocentesis for pleural effusion, etc). The decision to maintain or reduce the dose should be made in light of the cat’s clinical stability.

Treatment with diuretics and ACE inhibitors are reserved for after the onset of congestive heart
failure and are almost invariably life-long. Anticoagulation is warranted in cats with evidence of thromboembolic disease or those with left atrial enlargement, the latter representing the key predisposing factor in the formation of intra-cardiac thrombi.

This can be accomplished with generic, uncoated baby aspirin at a dose of 81mg PO q72 hours OR with clopidogrel at a dose of 18.75mg PO q24 hours. Clopidogrel (Plavix®) irreversibly binds to ADP receptors on the platelet membrane and functions to inhibit platelet aggregation and cross-linking by fibrin. Although anecdotally endorsed by many practitioners, evidence of a clinical benefit of clopidogrel over aspirin is still lacking. A large prospective, blinded study is currently underway to compare the outcomes of cats treated prophylactically with either aspirin or clopidogrel. One major side effect of clopidogrel is poor palatability and inappetence in cats receiving it. These side effects may be minimized by having the owner coat each tablet fragment with a thin film of butter, petroleum jelly, or wax prior to administration, or placing the tablet in a treat or tasteless gel capsule.

Anticoagulation for the purpose of mitigating the risk of aortic thromboembolism is only indicated once a cat has experienced thromboembolism, radiographic or echocardiographic evidence of left atrial enlargement is identified, or both.

Aggressive anticoagulation is recommended in cats with clinical evidence of thromboembolic disease or those with echocardiographically demonstrable left atrial spontaneous contrast or thrombus. Warfarin, unfractionated heparin, and low molecular weight heparin have all been investigated in cats with aortic thromboembolism and all are plausible treatment strategies given that the practitioner is familiar with the indications, doses, and complications of each. Active thrombolytic therapy with recombinant tissue plasminogen activator (rTPA) has been shown to resolve emboli faster than the endogenous thrombolytic system but confers no demonstrable clinical benefit, presumably because of the high risk of reperfusion injury. In addition, rTPA is often cost prohibitive.

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Overview of blood types and crossmatch testing in dogs and cats
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Overview of the Issue
- Pretransfusion crossmatch testing minimizes the risk of acute hemolytic transfusion reaction.
- However, a negative crossmatch test reaction does not exclude the possibility of delayed-onset hemolytic reactions, nor does it predict whether or not non-hemolytic transfusion reactions will occur.
- There are currently over 12 recognized canine blood groups (and several possible individual canine blood types), but only one blood group (dog erythrocyte antigen (DEA) 1) is of major clinical significance in canine transfusion.
- There is currently 1 feline blood group with 3 possible blood types, which have clinical significance.

Key Clinical Diagnostic Points
- A crossmatch is ideally done before all transfusions
- A crossmatch is not necessary before a patient’s first transfusion or those within 2 days of the first transfusion, though, blood type-matched RBCs must absolutely be used in cats
- A crossmatch should definitely be done before all subsequent transfusions

Key Therapeutic Points
- Transfuse to relieve clinical signs of anemia, not to attain a specific packed cell volume (PCV).
- Use blood component therapy (e.g., packed-RBCs) when indicated and available.
- PCV measurement alone is an unreliable indicator of when a transfusion is necessary.
- In addition to the patient’s PCV, other important parameters (e.g., clinical signs of anemia, duration of the anemia) should be considered in order to determine if the use of a potentially harmful treatment, i.e., a transfusion, is truly warranted.
- Two general rules of thumb regarding PCV can be applied when trying to decide if a transfusion may be indicated: (1) transfuse when the PCV is <15% or (2) when there has been acute RBC loss resulting in a 50% or greater reduction from the original (or previous) PCV.

Species-Specific Key Therapeutic Points
Dogs
- If the transfusion recipient’s blood type is unknown, at least use DEA 1 negative RBCs or blood, though, it is preferable to use a universal donor (DEA 4 positive and neg. for other DEAs).
- If recipient’s DEA 1 blood type IS known (DEA 1-positive Vs. DEA 1-negative), then use DEA 1 type-specific blood that is major crossmatch-compatible.
- Use type-specific blood for multiply transfused IMHA patients.
**Cats**

- Use type-specific blood (give Type A to Type A or Type B to Type B).
- If type-specific blood is used a crossmatch is not necessary for a first transfusion. But, if it is not possible to blood type the patient and the donor, a crossmatch is absolutely necessary even before the first transfusion.

**Additional Detail**

**Feline:** Within the single feline blood group, there are three blood types, A, B, and AB. **Type A** blood (75-95% of cats) is much more common than **Type B** (2-25% of cats). The incidence of each blood type varies significantly with breed and geographic region, e.g., type B incidence ranges from 0-60% in some breeds (see table below). **Type AB** blood is very rare, seen in <1% of cats. Type AB blood occurs only in breeds in which type B has been detected. Reported incidence rates are as follows: US & Canada: 13/9239 cats (.14%), Australia: 7/1895 cats (.4%).

The nature transfusion reactions is dictated by the anti-RBC antibodies present, which, in cats, are naturally occurring, preformed antibodies. The reactivity of the antibodies depends on the cat’s blood type. The half-life of completely matched RBCs, that incite no transfusion reaction, is 33 days.

**Severe transfusion reaction and death are possible when as little as 1 mL of type A blood is given to a type B cat.** The reaction causes rapid intravascular hemolysis of the donor RBCs within minutes to hours. This very rapid RBC destruction can lead to severe clinical signs of anaphylactic shock associated with hypotension, apnea, AV block, and death. **When type A cats receive type B blood, minor transfusion reactions can occur that result in primarily extravascular removal of the donor RBCs within 2 to 4 days.**

To maximize the safety of transfusion, type-specific, crossmatched blood should be used. However, in an emergency situation, card blood typing alone - without a crossmatch, is adequate before a first transfusion. A crossmatch should be performed before ALL second transfusions, as these individuals may have been sensitized to foreign RBC antigens during their first transfusion.

**Feline neonatal isoerythrolysis (NI)** is very rare. It has been reported in Persians and Himalayans. It occurs when a type B queen has type A or AB offspring due to colostral transfer of naturally occurring, maternal anti-A antibodies. NI is believed to be a major cause of fading kitten syndrome. The signs are variable and range from unapparent hemolysis to severe hemolytic anemia with hemoglobinuria. If there is concern that NI may result from a particular mating, the breeding pair may be blood typed prior to mating to avoid this potential.

**Feline blood type incidence by United States geographic region**

<table>
<thead>
<tr>
<th>Region</th>
<th>n</th>
<th>A (%)</th>
<th>B (%)</th>
<th>AB (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Northeast</td>
<td>145</td>
<td>99.7</td>
<td>.3</td>
<td>0</td>
</tr>
<tr>
<td>North central</td>
<td>506</td>
<td>99.4</td>
<td>.4</td>
<td>.2</td>
</tr>
<tr>
<td>Southeas t</td>
<td>534</td>
<td>98.5</td>
<td>1.5</td>
<td>0</td>
</tr>
<tr>
<td>-----------</td>
<td>-----</td>
<td>------</td>
<td>-----</td>
<td>---</td>
</tr>
<tr>
<td>Southwes t</td>
<td>483</td>
<td>97.5</td>
<td>2.5</td>
<td>0</td>
</tr>
<tr>
<td>West Coast</td>
<td>812</td>
<td>94.8</td>
<td>4.7</td>
<td>.5</td>
</tr>
</tbody>
</table>

**Blood type B frequency in United States purebred cats**

<table>
<thead>
<tr>
<th>Frequency (%)</th>
<th>Breeds</th>
</tr>
</thead>
<tbody>
<tr>
<td>25-50</td>
<td>Exotic shorthair, British shorthair, Cornish Rex, Devon Rex</td>
</tr>
<tr>
<td>5-25</td>
<td>Abyssinian, Himalayan, Birman, Persian, Somali, Sphinx, Scottish fold, Japanese bobtail</td>
</tr>
<tr>
<td>&lt;5</td>
<td>Maine coon, Norwegian forest, DSH, DLH</td>
</tr>
<tr>
<td>0</td>
<td>Siamese, Burmese, Tonkinese, Russian blue, Oicat, Oriental shorthair</td>
</tr>
</tbody>
</table>

**Characteristics of feline anti-erythrocyte antibodies**

<table>
<thead>
<tr>
<th>Type A cats</th>
<th>Type B cats</th>
<th>Type AB cats</th>
</tr>
</thead>
<tbody>
<tr>
<td>Preformed antibody present in 35%</td>
<td>Preformed antibody present in 95%</td>
<td>Usually NO preformed antibodies</td>
</tr>
<tr>
<td>Low titer</td>
<td>High titer</td>
<td></td>
</tr>
<tr>
<td>Directed against RBC B antigen</td>
<td>Directed against RBC A antigen</td>
<td></td>
</tr>
<tr>
<td>Hemolysins</td>
<td>Hemolysins</td>
<td></td>
</tr>
<tr>
<td>IgM = IgG</td>
<td>IgM &gt; IgG</td>
<td></td>
</tr>
<tr>
<td>Responsible for minor transfusion reactions</td>
<td>Responsible for severe acute transfusion reactions and neonatal isoerythrolysis</td>
<td></td>
</tr>
<tr>
<td>RBC removal: ~2 days</td>
<td>RBC removal: ~1 hour</td>
<td></td>
</tr>
</tbody>
</table>

**Canine:** At least 12 canine blood groups have been identified, though not all are internationally recognized. In the United States the blood groups are currently referred to using the dog erythrocyte antigen (DEA) system. Each group is designated by ‘DEA’ followed by a number for the locus.allele, e.g., DEA 1.1, DEA 1.2. (A change to this nomenclature has been proposed, that is not yet universally accepted, though it is used by some. It is detailed in the table below). This is an active area of research. Various investigators describe other blood groups, which may not be included in the current system and/or for which commercial antisera may not be available for typing. Unlike in cats which have a single recognized blood group, a given dog’s overall blood type is dependent upon the combination of blood group antigens that are expressed on its RBC surfaces. There may be antigens of one or more groups on the surface of a single individual’s RBCs, except for DEA 1.1 and DEA 1.2, which are mutually exclusive, (because they are of the same group; i.e., 1.1 and 1.2 are different alleles at the same locus).
Among the canine blood groups, only DEA 1 is of major clinical significance. Approximately 60% of U.S. dogs are DEA 1-positive (DEA 1.1: 45%, DEA 1.2: 20%). It is in the 40% of dogs that are DEA 1-negative that a severe hemolytic RBC transfusion reaction is possible. The nature of transfusion reactions is dictated by the anti-RBC antibodies present. In dogs, anti-RBC antibodies are not preformed and occur only after the immune stimulation caused by exposure to another’s blood. Severe transfusion reactions are possible only in sensitized individuals that have been transfused previously with DEA 1-positive blood or in those that have been pregnant previously with DEA 1-positive offspring. If sensitized by exposure to DEA 1-positive blood, a DEA 1-negative individual will likely produce high titer anti-DEA 1 antibodies. (Antibodies directed against one of the DEA 1 group antigens can cross react with other antigens in the group). Anti-DEA 1.1 antibody is a strong agglutinin and hemolysin of DEA 1.1 positive RBCs. (It is also a variable agglutinin of DEA 1.2 and 1.3 positive RBCs). Transfusion reactions caused by this antibody result in severe acute hemolysis of donor RBCs with hemoglobinuria, bilirubinemia, and removal of transfused RBCs within 12 hours. (Transfusion of a DEA 1.1 positive recipient with plasma containing this antibody can cause hemolysis of the recipient’s RBCs due to the donor’s plasma). Anti-DEA 1.2 antibody can be formed by a sensitized DEA 1.2 negative individual. Transfusion reactions caused by this antibody result in severe acute hemolytic transfusion reaction with hemoglobinuria, bilirubinemia and removal of transfused RBCs within 12-24 hours.

To maximize the safety of transfusion, crossmatched blood should be used. However, since clinically significant preformed anti-RBC antibodies have not been demonstrated, unmatched first transfusions of the dog are usually safe. A crossmatch should be performed before ALL second transfusions, as these individuals may have been sensitized to foreign RBC antigens during their first transfusion. If the transfusion-recipient’s DEA 1 blood type is known (DEA 1-positive Vs. DEA 1-negative), then DEA 1 type-specific blood that is major crossmatch-compatible can be used.

Naturally occurring canine neonatal isoerythrolysis is EXTREMELY rare. Sensitization of a DEA 1.1 negative bitch by previous transfusion or pregnancy can result in NI in DEA 1.1 positive offspring of subsequent pregnancies.

### Current internationally recognized canine erythrocyte antigens

<table>
<thead>
<tr>
<th>Current designation</th>
<th>Proposed change</th>
<th>Typing sera available</th>
<th>Clinically significant</th>
<th>United States incidence (%)</th>
<th>In dogs that don’t have the antigen</th>
</tr>
</thead>
<tbody>
<tr>
<td>DEA 1.1</td>
<td>Aa₁</td>
<td>Yes</td>
<td>Yes</td>
<td>45</td>
<td>0 Acute hemolytic</td>
</tr>
<tr>
<td>DEA 1.2</td>
<td>Aa₂</td>
<td>Yes</td>
<td>Yes</td>
<td>20</td>
<td>0 Acute hemolytic</td>
</tr>
<tr>
<td>DEA 1.3</td>
<td>Aa₃</td>
<td>No</td>
<td>No</td>
<td>Australia</td>
<td>0 Unknown</td>
</tr>
<tr>
<td>DEA 3</td>
<td>Ba</td>
<td>Yes</td>
<td>Somewhat</td>
<td>6&lt;sup&gt;a&lt;/sup&gt;</td>
<td>≤20 Decreased RBC $t_1/2$, potentially severe reaction</td>
</tr>
</tbody>
</table>
Transfusion reactions may be classified as immunologic or non-immunologic; and as acute or delayed. **Immunologic reactions** may be hemolytic or non-hemolytic. **Acute hemolytic** reactions occur due to preformed anti-RBC antibodies. In cats, they occur at a rate of <1 reaction per 100 transfusions, (reactions are especially common in type B cats). In dogs, they occur at a rate of 1 reaction per 2000 transfusions. **Delayed hemolytic** reactions occur 2-21 days post-transfusion, due to de novo synthesis of new anti-RBC antibodies. **Non-hemolytic** reactions are due to antibodies directed against WBCs, platelets, plasma proteins.

There are several possible **non-immunologic transfusion reactions**. Since blood products are colloids, it is possible to cause iatrogenic intravascular **volume overload** and congestive heart failure with a transfusion. **Vomition** may be observed during a transfusion. **Septicemia** can be caused by bacterial contamination of the blood product. **Hemoglobinemia/uria** can occur due to a transfusion for many reasons other than immune-mediated destruction of RBCs. These reasons include bacterial contamination of the blood product which causes RBC destruction and transfusion of physically damaged RBCs due to improper collection, storage, or administration, (such as freezing/overheating of the blood product, excessive infusion pump pressure during administration, or mixing the RBCs with hyper- or hypo- tonic solution for administration). **Air embolism** associated with blood storage in glass bottles, (which are no longer routinely used), has been observed. **Transfusion of a very large volume of blood**, i.e., more than one blood volume (e.g., dogs: >8% BW<sub>kg</sub> = >80 mL/kg) can cause **dilutional coagulopathy** or **electrolyte abnormalities**. **Hypocalcemia** can occur due to citrate anticoagulant toxicity and result in tremors or arrhythmias. A pre-existing hyperkalemia may be exacerbated by hemolysis and the release of intracellular potassium. **Transmission of blood borne diseases** is also possible.

The risk of transfusion reactions can be minimized by using compatible, crossmatched blood, an appropriate donor (discussed later), component therapy when possible (e.g., pRBC, FFP), and proper techniques for collection, storage, and administration. **Blood crossmatching** (procedure at end of follow) is used to detect the presence of hemolyzing or hemagglutinating antibodies in the serum of donor and recipient animals. It should be performed ideally before all transfusions of any animal, **definitely** before a second transfusion of any animal, and when it is unknown whether or not an animal has received a prior transfusion. The test uses saline washed RBCs that are incubated with serum and then examined for hemolysis and/or agglutination (gross or microscopic). (Agglutination is the aggregation of RBCs which persists after a saline wash. It occurs due to the presence of surface bound immunoglobulin. Whereas, rouleaux dissipates with saline washing). A test reaction which results in hemolysis and/or

<table>
<thead>
<tr>
<th>DEA 4</th>
<th>Ca</th>
<th>Yes</th>
<th>No</th>
<th>≤98</th>
<th>0</th>
<th>None&lt;sup&gt;b&lt;/sup&gt;</th>
</tr>
</thead>
<tbody>
<tr>
<td>DEA 5</td>
<td>Da</td>
<td>Yes</td>
<td>Somewhat</td>
<td>Low&lt;sup&gt;a&lt;/sup&gt;</td>
<td>10</td>
<td>Decreased RBC t&lt;sub&gt;1/2&lt;/sub&gt;</td>
</tr>
<tr>
<td>DEA 6</td>
<td>Fa</td>
<td>No</td>
<td>No</td>
<td>≤100&lt;sup&gt;a&lt;/sup&gt;</td>
<td>0</td>
<td>Unknown</td>
</tr>
<tr>
<td>DEA 7</td>
<td>Tr&lt;sup&gt;f&lt;/sup&gt;</td>
<td>Yes</td>
<td>Somewhat</td>
<td>40-54</td>
<td>20-50</td>
<td>Decreased RBC t&lt;sub&gt;1/2&lt;/sub&gt;</td>
</tr>
<tr>
<td>DEA 8</td>
<td>No</td>
<td>No</td>
<td>No</td>
<td>40-45</td>
<td>Unknown</td>
<td></td>
</tr>
</tbody>
</table>

<sup>a</sup>DEA 5 and 6 vary with breed and geographic region, (20-30% of Greyhounds DEA 3 &/or 5 positive)

<sup>b</sup>Case report questions this belief about DEA 4 (JVIM 2003 Nov/Dec; 17:931-3)
agglutination is interpreted as a **positive result**. A positive result indicates that preformed anti-RBC antibodies were detected and that an acute hemolytic transfusion reaction is likely. A test reaction which causes neither hemolysis nor agglutination is interpreted as a **negative result**. A negative result indicates ONLY that preformed anti-RBC antibodies were not detected and that an acute hemolytic transfusion reaction is unlikely. (Though, the crossmatch test may lack sufficient sensitivity to detect some anti-RBC antibodies). Additionally, a negative crossmatch result DOES NOT exclude the potential of a delayed hemolytic transfusion reaction or of other immunologic and non-immunologic transfusion reactions. Crossmatching does not detect antibodies directed against other blood components, e.g., WBCs, platelets, and plasma proteins.

The minor crossmatch reaction tests for the reaction that the donor serum may cause against the recipient’s RBCs. This reaction is referred to as ‘minor’ because, during a transfusion, the volume of transfused donor serum is relatively small once diluted within the recipient patient’s total serum volume. A positive minor crossmatch reaction indicates that a small amount of hemolysis of recipient RBCs may occur due to the presence of the donor’s serum in the transfusion. When absolutely necessary, a minor reacting donor can be used for a transfusion.

### Crossmatch reactions

<table>
<thead>
<tr>
<th></th>
<th>Major</th>
<th>Minor</th>
</tr>
</thead>
<tbody>
<tr>
<td>donor RBCs</td>
<td>donor serum</td>
<td>donor serum</td>
</tr>
<tr>
<td>recipient serum</td>
<td>recipient RBCs</td>
<td></td>
</tr>
</tbody>
</table>

| **Reaction detects** | Preformed recipient Abs directed against donor RBCs | Preformed donor Abs directed against recipient RBCs |

### Crossmatching procedure

1. Obtain an EDTA anticoagulated (purple top) and a non-anticoagulated (red top) blood sample from both patient and donor. The EDTA will serve as the source of RBC (antigen). The non-anticoagulated sample will serve as the source of serum (antibody).
2. Centrifuge and separate the plasma or serum from the RBCs.
3. Wash the RBCs by adding .9% saline or phosphate buffered saline (PBS) to a small amount of pRBCs, mixing, and centrifuging. Decant the saline and repeat 3 times, filling the tubes with saline, mixing, centrifuging, and decanting.
4. After the last wash, decant the supernatant and resuspend the cells with .9% saline to result in a 2-4% suspension of RBCs (dilute tomato juice color). The suspension may also be calculated, for example, .1 mL of RBCs in 2.4 mL of saline yields a 4% suspension.
5. Make the following mixtures by adding the indicated amount of well-mixed RBC suspension and serum into 12 x 75 mm tubes:
   a. **Major crossmatch**
      i. 1 drop donor RBC suspension
      ii. 2 drops patient serum
   b. **Minor crossmatch**
      i. 1 drop patient RBC suspension
4. 2 drops donor serum
   c. Patient autocontrol
      i. 1 drop patient RBC suspension
      ii. 2 drops patient serum
   d. Donor autocontrol
      i. 1 drop donor RBC suspension
      ii. 2 drops donor serum
6. Incubate tubes 15 - 30 minutes at 37 deg C
7. Centrifuge for 15 seconds, 3400 rpm/1000 x g
8. Read tubes:
   a. Macroscopic: Examine the tubes for hemolysis. Then rotate the tubes gently
      and observe them for cells coming off of the red cell “button” at the bottom of
      the tube. In a compatible reaction, i.e., where there is no antigen-antibody
      reaction, the cells should float off freely, with no clumping/hemagglutination
      (compare to control tubes). Rouleaux formation can be falsely interpreted as a
      reaction. If rouleaux is suspected, a saline replacement technique (below) can
      be used. Proceed to the microscopic examination in those tubes with weak or
      no obvious macroscopic reactions.
   b. Microscopic: After the tubes have been inspected macroscopically, place a drop
      of the cell and serum mixture on a slide, coverslip, and examine microscopically.
      The RBCs should normally appear as individual cells, with no agglutination or
      rouleaux formation.

Saline replacement procedure (use as needed when reading the crossmatch reaction (step 8
above)
   1. Re-centrifuge the RBC and serum mixture when rouleaux is suspected.
   2. Remove the serum.
   3. Replace the serum with an equal volume of .9% saline (i.e., 2 drops) and gently mix.
   4. Centrifuge the mixture for 15 seconds at 1000 x g.
   5. Resuspend the RBCs as for macroscopic inspection and observe for agglutination.
      Rouleaux will disperse; agglutination will persist.

References and Additional Reading
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4. Feldman BF, Zinkl JG, Jain, NC, eds. Schalm’s Veterinary Hematology. 5th ed.
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   and 'Candidatus Mycoplasma haemominutum' in blood of cats used for transfusions. J Feline


Overview of coagulation testing
Heather L. Wamsley, BS, DVM, PhD, DACVP (Clinical)
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University of Florida, Gainesville, Florida, USA

Objectives of presentation
- The clotting cascade will be reviewed from two perspectives: the classic cascade model and the cell-based model.
- Common hemostatic tests will be reviewed, including PT, PTT, ACT, FDP, D-dimer, and thromboelastography.

Details
There are two models of coagulation, the class coagulation cascade and the cell-based model. The classic cascade is helpful when considering traditional in vitro laboratory tests of hemostasis, e.g., PT, PTT, ACT. The cell-based model is a newer model that is thought to be more representative of events in vivo when the hemostatic plug is generated; it is useful to consider this model when discussing thromboelastography, a test of hemostatic test in which there is renewed interest. With the classic cascade, coagulation is divided into the intrinsic the extrinsic arms, which meet at the common portion of the pathway. The cell-based model describes coagulation in three major steps: initiation, amplification, and propagation (“thrombin burst”). In this process numerous reactions occur simultaneously on the surface of damaged endothelium and on the surfaces of platelets. This model highlights features of the coagulation cascade that, in concert, amplify the clotting reaction and highlights the critical role that thrombin has, not only in generating the final product of coagulation – a stable, cross-linked fibrin plug, but also in promoting continued activation of the clotting cascade. Thrombin is also an important activator of fibrinolytic reactions that help keep coagulation localized to the site of injury.

The PT (prothrombin time) is a test of the extrinsic and common pathways. A 70% clotting factor deficiency is required before a prolonged PT can be detected. Prolongation indicates inhibition of deficiency of clotting factor 7 or common pathway factor(s). The PT is a good screening test for Vitamin K antagonism or deficiency because, of the vitamin K-dependent clotting factors, factor 7 has the shortest half-life. The PTT (partial thromboplastin time) is a test of the intrinsic and common pathways. A 70% clotting factor deficiency is required before a prolonged PTT can be detected. Prolongation indicates inhibition or deficiency of any intrinsic or common pathway factor(s). Decreased clotting factor 12 will prolong the in vitro test, but is not associated with in vivo bleeding – some animals do not even have factor 12. It is common to see a mildly prolonged PTT test result due to problems with the sample, e.g., under filled citrate tube – excess anticoagulant, erythrocytosis. The ACT (activated clotting time) is a test of the intrinsic and common pathways. This test is easy to do in-house and is used to determine the time it takes for the blood to clot once it has been collected into a specialized tube. This test uses the patient’s own clotting factors, calcium, and platelets. This test is less sensitive than the PTT; a 95% clotting factor deficiency is required before a prolonged ACT can be detected. Prolongation indicates inhibition or deficiency of any intrinsic or common pathway factor(s). Since this test relies on the patient’s own platelets for
the test reaction, it is important to keep in mind that severe thrombocytopenia (<10,000/µL) can slightly prolong the ACT. Therefore, prior to doing an ACT, it may be useful to determine the patient’s platelet count. The ACT is inexpensive and simple and can be performed anywhere using the specialized blood collection tube. After the blood is collected and mixed, it is incubated at 37°C. A heatblock is used in the lab, but a human axilla can be used instead.

There are two fibrinolysis products that can be commonly measured to help determine if there is inappropriate activation of the clotting cascade, e.g., thromboembolic disease, and to help determine if there are conditions associated with increased fibrinolysis, e.g., disseminated intravascular coagulation (DIC). Fibrin(ogen) degradation products (FDP) and D-dimers are commonly available tests. FDPs are formed by plasmin degradation of fibrinogen, fibrin monomers, crosslinked fibrin from stable clot. Since FDP can form from breakdown of fibrinogen, elevated FDP does not necessarily indicate that the clotting cascade has been activated. D-dimers are only formed by plasmin degradation of crosslinked fibrin from a stable clot, so this test is considered more specific for activation of the clotting cascade. FDP elevations can occur due to breakdown of fibrinogen (fibrinogenolysis) and/or due to breakdown of fibrin (fibrinolysis). Fibrinogenolysis causes increased FDP, but should not increase D-dimers. Conditions associated with fibrinogenolysis include certain snake envenomations, heatstroke, hypotensive shock, and surgical trauma. Fibrin is formed after activation of the clotting cascade. Initially, unstable, fibrin monomers are formed. Once the clotting cascade has progressed fully to completion, stable, cross-linked fibrin aggregates are formed. Proteolysis of stable, cross-linked fibrin aggregates causes increases in FDPs and D-dimers. Conditions associated with increased D-dimers include disseminated intravascular coagulation, sepsis and probably other inflammatory conditions, internal hemorrhage, and thromboembolic disease. Higher D-dimer values (>500 ng/mL) are suggestive of thromboembolic disease; however, animals with hemorrhagic effusion may also have very high D-dimer concentrations (Nelson and Andreasen, 2003).

<table>
<thead>
<tr>
<th>D-dimer (ng/mL)</th>
<th>&lt;250</th>
<th>250-500</th>
<th>500-1000</th>
<th>1000-2000</th>
<th>&gt;2000</th>
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</thead>
<tbody>
<tr>
<td>Control</td>
<td>30</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neoplasia</td>
<td>9</td>
<td>1</td>
<td>4</td>
<td>1*</td>
<td>1*</td>
</tr>
<tr>
<td>Heart failure</td>
<td>8</td>
<td>1</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Liver disease</td>
<td>4</td>
<td>4</td>
<td>4</td>
<td>1*</td>
<td></td>
</tr>
<tr>
<td>Renal failure</td>
<td>5</td>
<td>2</td>
<td>1</td>
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</tr>
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<td>Postsurgery</td>
<td>11</td>
<td>3</td>
<td>6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thromboembolic dz</td>
<td></td>
<td>4</td>
<td>9</td>
<td>7</td>
<td></td>
</tr>
</tbody>
</table>

From Nelson and Andreasen, JVIM 2003

If available in your area, thromboelastography (TEG) may be another useful test to evaluate a patient’s potential for inappropriate clot formation (i.e., hypercoagulability) or for bleeding (i.e. hypocoagulability). There has been renewed interest in TEG, which is a whole blood-based test of hemostasis and has been thoroughly reviewed elsewhere (Kol and Borjesson, 2010; McMichael and Smith, 2011). TEG has been validated in dogs, cats, and horses. It is a promising companion to other traditional hemostatic tests and may have a particular benefit in predicting hypercoagulability and for critical care patients with conditions that predispose to hypercoagulability (e.g., malignant neoplasia, immune-mediated disease, canine parvovirus infection). Though, results of prospective research and
publications that characterize the test’s sensitivity and specificity are currently limited. Numeric and graphic data, i.e., time to form the clot and strength of the clot, respectively, are derived from this test, which involves recording information obtained from a torsion wire that oscillates within the blood sample as it clots after being mixed with reagents in a cup. When considering TEG, care should be taken to employ a lab with its own established reference ranges and with personal experienced in running and interpreting this test, especially the factors that influence the assay (e.g., time between collection and analysis and the patient’s fibrinogen, platelet, and erythrocyte concentrations) (Kol and Borjesson, 2010).

References
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Tests of proteinuria in dogs and cats
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University of Florida, College of Veterinary Medicine
Gainesville, Florida, USA

OBJECTIVES OF THE PRESENTATION
• Discuss diagnostic tests used to diagnose proteinuria
• Provide information about revised guidelines for interpretation of urine protein to creatinine ratios and microalbuminuria

KEY CLINICAL DIAGNOSTIC POINTS
• The duration and magnitude of proteinuria and whether or not there is concurrent azotemia should guide the level of response to the proteinuria.
• Revised action values for UPC in azotemic animals are <0.5 for azotemic dogs and <0.4 for azotemic cats.
• Normotensive, non-hypercortisolemic, nonazotemic animals with persistent renal microalbuminuria, UPC <1 should be monitored, particularly if they are breed-predisposed for renal disease, have pre-existing renal disease, or have received treatments that may cause renal damage.

ADDITIONAL DETAIL
Urine dipstick protein testing is performed to screen for diseases that cause excess protein loss by the urinary tract (e.g. glomerular diseases) or that cause excess systemic production of protein with overflow into urine (e.g. multiple myeloma). However, since other nonpathologic or pathologic causes of proteinuria occur more commonly, a positive protein reaction should be interpreted judiciously.

The dipstick protein reaction is most sensitive to albumin. Other proteins (e.g. hemoglobin, immunoglobulin light chains [Bence Jones proteins]) must be present at very high concentrations to react with dipstick reagents and cause a positive reaction. The sulphosalicylic acid protein precipitation test is a facile method that detects all types of protein and permits of verification dipstick results. The test is very easy to perform and the single reagent used in the test has a long shelf-life. All types of protein are detected by this method, therefore Bence Jones proteinuria, which would most likely go undiagnosed by urine dipstick, can be identified.

Additional data from the urinalysis (i.e. specific gravity, pH, sediment examination) are needed to determine the significance of proteinuria detected by the dipstick reaction. A positive protein reaction should be interpreted in the context of the urine specific gravity, since a small amount of protein (trace to 1+, <0.30 g/L) can be a normal finding in a single well concentrated urine (specific gravity >1.030) sample; however, persistent trace or 1+ proteinuria should prompt further investigation (Lees et al., 2005). A similar protein concentration in dilute urine or in an animal receiving potentially nephrotoxic drugs regardless of urine concentration would be abnormal. Consideration of the urine pH is necessary, since urine that is either markedly alkaline (pH >9) or moderately alkaline (pH 7.5) and well concentrated (specific gravity >1.035) will likely cause a false positive dipstick protein reaction. In one study, false positive dipstick protein reactions were more common in cats than in dogs (Grauer et al., 2004).
The specificity of the dipstick protein reaction was 31% in cats and 69% in dogs when compared to a species-specific albumin ELISA. False positive reactions may have occurred due to the presence of non-albumin proteins or other interfering substances. Contamination of the urine sample with cleanser residues or improper dipstick usage or storage can also cause false positive reactions.

Knowledge of the urine sediment is requisite for accurate interpretation of urine dipstick protein. The most common pathologic causes of increased urine protein concentration are urinary tract inflammation, infection, hemorrhage, or some combination of the three that most often arises from the lower urinary or genital tracts. In these instances, microscopic examination of the urine sediment will likely disclose pyuria, bacteriuria, or hematuria. In order to attribute dipstick proteinuria entirely to hemorrhage, the heme reaction must be at least 3+ (large) and macroscopic hematuria should be present. If the heme reaction is less than 3+ and no other causes of a persistently positive protein reaction (e.g. pyuria, bacteriuria, spurious) are detected, then underlying disease (e.g. nephrotic syndrome, multiple myeloma, proximal renal tubular defect) may be present.

When significant dipstick proteinuria has been verified as persistent based upon a second urinalysis and when other sources of a positive protein reaction (e.g. pyuria with or without bacteriuria) have been excluded, measurement of the urine protein-to-creatinine ratio is necessary to more precisely determine the severity of proteinuria. The urine protein-to-creatinine ratio should be <1.0; though recent advances in canine and feline urine albumin testing have prompted re-evaluation of this reference value. Some investigators now favor use of <0.5 as the urine protein-to-creatinine ratio reference value for nonazotemic animals, <0.5 for azotemic dogs, and <0.4 for azotemic cats (Lees et al., 2005). A urine protein-to-creatinine ratio that is >1.0 in a sample obtained by cystocentesis should rouse concern for glomerular disease (e.g. glomerulonephritis, glomerulosclerosis, or canine amyloidosis), Bence Jones proteinuria, or much less commonly tubular proteinuria. Serum biochemistry and serum or urine protein electrophoresis are useful initial tests that will help localize the cause of proteinuria to preglomerular versus renal causes, and these results may direct the course of future diagnostic testing (e.g. bone marrow biopsy versus renal biopsy).

Serial measurement of the urine protein-to-creatinine ratio can be used to stage the progression of renal disease and to evaluate the response to therapy. Determination of the urine protein-to-creatinine ratio may also be performed to help establish the prognosis for newly diagnosed cases of canine chronic renal failure (Jacob et al., 2005). A urine protein-to-creatinine ratio that is >1.0 at the time of initial chronic renal failure diagnosis is a negative prognostic indicator. Compared to patients with a urine protein-to-creatinine ratio that is <1.0, canine chronic renal failure patients with a ratio that is >1.0 experience more rapid progression of renal disease, greater likelihood of uremic crisis, and greater risk of death due to either renal or nonrenal causes. The rate of disease progression and risk of complications were directly proportional to the magnitude of urine protein-to-creatinine ratio elevation. Similarly, proteinuria predicts reduced survival times in healthy, nonazotemic cats (Walker et al., 2004) and in cats with chronic renal failure (Syme and Elliot, 2003). Urine protein-to-creatinine ratios >0.3 in healthy nonazotemic cats or >0.4 in cats with chronic renal failure were significant predictors of reduced survival times in these two studies.

An assay to detect microalbuminuria (E.R.D.-Screen™ Urine Test) is available.
Microalbuminuria refers to increased urine albumin that remains beneath the detection limit of the urine dipstick protein reaction (i.e. urine albumin >0.01 g/L but <0.30 g/L). The prevalence of microalbuminuria has been reported as 15 to 19% in clinically normal dogs (Gary et al., 2004; Jensen et al., 2001) and 14% in clinically normal cats. Increased prevalence has been reported with older age or the presence of nonrenal disease (Heska Corporation unpublished data available at http://www.heska.com/erd). In canine experimental models of progressive glomerular disease, the prevalence of microalbuminuria was 75-100% and persistent microalbuminuria preceded the development of overt proteinuria (Grauer et al., 2002; Lees et al., 2002). Controlled studies to establish reference ranges for clinically normal animals, animals with nonrenal disease, and animals with early glomerular disease or controlled clinical studies to correlate identification of microalbuminuria in apparently healthy animals with the subsequent development of renal disease have not yet been published. However, these preliminary investigations suggest that, similar to humans, detection of persistent microalbuminuria in dogs may aid in early diagnosis of occult glomerular disease prior to the development of a positive dipstick protein reaction or increased urine protein-to-creatinine ratio.

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Cytologic evaluation of urine sediment
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OVERVIEW
Urine samples are labile; components of the urine sample may change or may be lost during the delay between sample collection and sample analysis. Ideally, urine samples should be examined soon after collection by a trained individual. Routine urinalysis is a test that may improved by performing it in-house.

INTRODUCTION
Complete urinalysis includes assessment of several physical and chemical characteristics of urine. It is a simple, economical test that requires minimal specialized equipment and can be easily performed by trained staff in general veterinary practice. With proper sample handling and testing, data generated by urinalysis rapidly disclose vital information about the urinary tract and also provide a general screen of other body systems (e.g. endocrine, hepatic). Taking advantage of the full potential of in-house urinalysis can rapidly provide you with information that will benefit you and your patients.

DETAILS
Urine Sample Collection Method, Timing, and Handling Prior to Analysis
In addition to biologic variability of the patient, urinalysis results are influenced by the urine collection method, the timing of urine collection, administration of therapeutic or diagnostic agents prior to collection, and how the sample is handled prior to analysis. There are several methods of urine collection each with their own advantages and disadvantages. Ideally at least 6 mL of urine should be collected prior to the administration of therapeutic or diagnostic agents so that baseline information can be established. In most situations, either naturally voided urine, collected midstream into a sterile container or urine obtained by cystocentesis is preferred. Since urine collection method can have a significant effect upon urinalysis results, it is important to record the collection method in medical records. It should also be indicated whether or not therapeutic or diagnostic agents (e.g. parenteral fluids, antimicrobials, glucocorticoids, diuretics, antihypertensives, radiographic contrast) have been administered prior to urine collection. If so, the type of agent and timing and duration of administration relative to urine collection should be recorded.

Ideally, urine should be collected into a sterile, opaque, sealable, labeled container and analyzed within 60 minutes of collection. If it is not possible to analyze the sample within 60 minutes, the sample should be preserved by refrigeration soon after collection for up to approximately 12 hours (i.e. overnight). Samples intended for routine urinalysis should not be frozen. Also, though numerous chemical preservatives of urine exist (e.g. boric acid, formalin, Mucolexx™), routine use of these preservatives is not recommended, since each may affect different components of the urinalysis. It is important that sealed containers are used for urine sample storage since certain volatile compounds may evaporate (e.g. ketones), and samples should be protected from light since certain compounds may degrade due to light-exposure.
Urine Sediment Wet-Mount Findings

**Crystalluria** occurs when urine is saturated with dissolved minerals or other crystallogeenic substances that precipitate out of solution to form crystals. Crystals may form *in vivo* for either pathologic or nonpathologic reasons, or crystals may precipitate in urine *ex vivo* due to cold temperature or prolonged storage, postcollection alterations of urine pH, or evaporation of water from the sample. To increase the likelihood that crystals present in the urine sample actually represent those that may be present in the patient, fresh, nonrefrigerated urine samples should be analyzed within approximately one hour of collection.

In most instances, crystalluria does not necessarily indicate the presence of uroliths or even a predisposition to form uroliths. For example, a small number of magnesium ammonium phosphate or amorphous phosphate crystals are frequently observed in clinically normal dogs and cats. Detection of crystalluria may be diagnostically useful when abnormal crystal types are identified (e.g. ammonium biurate, calcium oxalate monohydrate, cystine); when large aggregates of magnesium ammonium phosphate or calcium oxalate dihydrate crystals are found; or when crystalluria is observed in a patient that has confirmed urolithiasis. Evaluation of the type of crystals present may be useful to estimate the mineral component of the urolith(s), while awaiting results of complete urolith analysis. Uroliths are often heterogenous; therefore crystalluria is not a definitive indicator of urolith mineral content. Sequential evaluation of crystalluria may aid in monitoring a patient’s response to therapy for urolith dissolution. Specific types of common urine crystals are discussed here. For a complete discussion including uncommon types of crystalluria, consult a text devoted to urinalysis such as, *Urinalysis: A Clinical Guide to Compassionate Patient Care* (Osborne and Stevens, 1999).

**Magnesium ammonium phosphate crystals** are referred to as struvite crystals, triple phosphate crystals (a misnomer), or infection crystals (an older term). They are colorless and frequently form variably sized casket cover-shaped crystals. They also form three to eight sided prisms, needles, or flat crystals with oblique ends. Magnesium ammonium phosphate crystals most commonly form in alkaline
urine, which often occurs in association with bacterial infection. They may develop after collection in refrigerated, stored urine samples (Albasan et al., 2003), or in those that become alkaline during storage due to bacterial overgrowth or contamination of the sample with cleanser residues, for example. When magnesium ammonium phosphate crystals are detected in a stored urine sample, the finding should be verified by prompt examination of a freshly obtained urine sample that has not been refrigerated. Magnesium ammonium phosphate crystals are very commonly seen in dogs and occasionally in cats. When found in significant number, they are most frequently associated with bacterial infection by urease-producing bacteria, such as Staphylococcus or Proteus. However, in cats they can occur in the absence of infection, likely due to ammonia excretion by the renal tubules. Magnesium ammonium phosphate crystals may also be seen in clinically normal animals that have alkaline urine for reasons other than infection (e.g. diet, recent meal), animals that have sterile or infection-associated uroliths of potentially mixed mineral composition, or with urinary tract disease in the absence of urolithiasis.

**Calcium oxalate crystals** occur in two forms, dihydrate and monohydrate. Calcium oxalate dihydrate crystals occur much more commonly. They are colorless, variably sized, octahedrons that resemble a small, gift card type envelope or a Maltese cross and most commonly form in acidic urine. They may develop after collection in stored urine samples with or without refrigeration (Albasan et al., 2003) or in those that become acidic during storage due to bacterial overgrowth, for example. When calcium oxalate dihydrate crystals are detected in a stored urine sample, the finding should be verified by prompt examination of freshly obtained urine that has not been refrigerated. Calcium oxalate dihydrate crystals may be seen in clinically normal animals. They also occur with calcium oxalate urolithiasis, hypercalciuria (e.g. due to hypercalcemia or hypercortisolemia), or hyperoxaluria (e.g. ingestion of vegetation high in oxalates [e.g. Brassica family], ethylene glycol, or chocolate). They have been reported with increased frequency in cats as a complication of urine acidification to manage magnesium ammonium phosphate formation.

Calcium oxalate monohydrate crystals are colorless and variably sized. They may be flat with pointed ends and resemble picket fence boards. They may also form spindle or dumbbell-shaped crystals. Although either calcium oxalate monohydrate or dihydrate crystals can be seen with acute ethylene glycol intoxication, the monohydrate form with picket fence board morphology is more diagnostic of intoxication, since this form is usually only seen during acute ethylene glycol toxicity. Formation of these crystals is time dependent and occurs only during the early phase of intoxication. Crystalluria may be observed within 3 hours of ingestion in cats and within 6 hours in dogs and may last up to 18 hours post-ingestion. Calcium oxalate monohydrate crystals with spindle or dumbbell morphology are uncommonly observed with other causes of hyperoxaluria (e.g. chocolate ingestion).

**Calcium carbonate crystals** are variably sized, yellow-brown or colorless, variably shaped crystals (tic-tac-shaped, dumbbell-shaped, or spheres with radiant striations) that are found individually or in clusters usually within alkaline urine. They are seen in clinically normal horses, elephants, goats, rabbits, and guinea pigs. Anecdotally, they may very rarely be seen in dogs. Sulfonamide crystals, which can be seen in dogs and cats after sulfa-containing antibiotic administration, may form globules with radiant striations and could be mistaken for calcium carbonate crystals. Calcium carbonate crystals look similar to the crystals observed in 2007
secondary to melamine-contaminated pet foods.

**Bilirubin crystals** may precipitate as orange to reddish-brown granules or needle-like crystals. A low number of crystals are routinely observed in canine urine, especially in highly concentrated samples from male dogs. When bilirubin crystals are found in other species or in persistently large quantity in a canine patient, a disease associated with icterus (i.e. hemolytic or hepatobiliary disease) may be present.

**Amorphous phosphate and amorphous urate crystals** are similar in shape and may form amorphous debris or small spheroids. Amorphous phosphates are distinguished from amorphous urates in two ways: phosphates are colorless or light yellow and form in alkaline urine, while urates are yellow-brown to black and form in acidic urine. Amorphous phosphates are commonly observed in alkaline urine of clinically normal animals, and they are not clinically significant. Conversely, amorphous urates are an uncommon abnormal finding in most breeds. They may be seen in animals with portovascular malformation, severe hepatic disease, or ammonium biurate urolithiasis. Amorphous urates are routinely found in Dalmatians and English Bulldogs and may represent a predisposition for urate urolithiasis in these breeds.

Compared to other breeds, Dalmatians excrete a larger amount of uric acid in their urine and are therefore prone to form **uric acid crystals**. Uric acid crystals are colorless; flat; variably, but often diamond-shaped; six sided crystals. Most other breeds convert uric acid to a water soluble compound (i.e. allantoin) for excretion. Dalmatians have defective purine metabolism, preventing this conversion, so that uric acid is excreted in its native form into the urine. Also, Dalmatians have decreased tubular resorption of uric acid compared to other breeds. Uric acid crystals can also occasionally be seen in English Bulldogs. They are rarely seen in other dog breeds or cats and, when observed, have the same significance as amorphous urate or ammonium biurate crystals.

**Ammonium biurate crystals** are golden-brown and spherical with irregular protrusions, which engender a thorn-apple or sarcoptic mange-like appearance. In cats, they may form smooth aggregates of spheroids. Ammonium biurate crystals are seen in animals with portovascular malformation, severe hepatic disease, ammonium biurate urolithiasis, and uncommonly in clinically normal Dalmatians and English Bulldogs.

**Cystine crystals** are colorless, flat hexagons that may have unequal sides. Cystine crystalluria is an abnormal normal finding seen in animals that are cystinuric due to an inherited defect in proximal renal tubular transport of several amino acids (i.e. arginine, cystine, lysine, ornithine). Crystals are prone to develop in cystinuric patients that have concentrated, acidic urine. Cystinuria is a predisposition for the development of cystine urolithiasis. Among dogs, male Dachshunds, Basset Hounds, English Bulldogs, Yorkshire Terriers, Irish Terriers, Chihuahuas, Mastiffs, Rottweilers, and Newfoundlands are affected with increased frequency. Uroliths often lodge at the base of the os penis and may be missed on survey radiographs since they are relatively radiolucent. Female dogs and other breeds also may be affected. In cats, this disease has been recognized in male and female Siamese and American Domestic Shorthairs.

**Iatrogenic crystalluria** can be seen with administration of some antibiotics,
allopurinol, and radiocontrast medium. Sulfonamide crystals are pale yellow crystals and may form haystack-like bundles or globules with radiant striations. The latter morphology may be mistaken for calcium carbonate crystals.

**Renal tubular casts and pseudocasts**

Renal tubular casts are formed by proteinaceous plugs of dense, mesh-like mucoprotein (Tamm-Horsfall mucoprotein) that accumulate within the distal portion of the nephron. A low number (<2 per low power field) of these proteinaceous hyaline casts can occasionally be observed in urine of normal animals. Diuresis of dehydrated animals or proteinuria of pregglomerular or renal etiology can cause an increased number of hyaline casts to be present in urine. Renal tubular epithelial cells that die and slough into the tubular lumen can be entrapped within this dense mucoprotein matrix. If present, inflammatory cells associated with renal tubulointerstitial inflammation may also be entrapped. During microscopic sediment evaluation, cellular casts are further classified as either epithelial, leukocyte, or erythrocyte casts, if the constituent cells can be discerned. Once locked within the proteinaceous matrix, cells continue to degenerate, progressing from intact cells, to granular cellular remnants, and finally to a waxy cholesterol-rich end product. A cast may dislodge from a given renal tubular lumen at any time during this degenerative process and may be observed in the urine sediment. However, in clinically normal animals only granular casts are rarely found (<2 per low power field). Other material can lodge within the proteinaceous matrix, such as lipid from degenerated renal tubular epithelial cells, hemoglobin during hemolytic disease, and bilirubin.

**The number of casts observed in the sediment** does not correlate with the severity of renal disease or its reversibility; and the absence of casts from urine sediment cannot be used to exclude the possibility of renal disease, especially since casts are fragile and prone to degeneration, particularly in alkaline urine. When hyaline or granular casts are present in increased numbers or when other cast types are observed, one can only conclude that the renal tubules are involved in an active disease process of unknown severity or reversibility. When present, the type of cast observed may provide additional information. Leukocyte casts indicate active renal tubulointerstitial inflammation. Waxy casts reflect a chronic tubular lesion. To recognize the onset of nephrotoxicity in patients receiving aminoglycoside antibiotic therapy, it is useful to monitor urine sediment for the appearance of tubular casts, which should prompt withdrawal of the antibiotic. Other abnormalities seen with aminoglycoside-induced nephrotoxicity include isosthenuria, proteinuria, glucosuria, aminoaciduria, all of which may precede the onset of azotemia.

Structures such as mucus threads or fibers may resemble casts and should not be mistaken for them during microscopic examination. Mucus threads are distinguished by their variable width and tapered ends. Fibers are typically much larger than the surrounding cells and may contain a repetitive internal structure, suggesting a synthetic origin.

**Epithelial cells**

Epithelial surfaces along the length of the genitourinary tract undergo constant turnover, therefore it is routine to see a low number of epithelial cells (<5
per low power field) in normal urine samples. A greater number of epithelial cells are seen in urine samples collected by catheterization or in patients with inflamed, hyperplastic, or neoplastic mucosa. Using wet mount preparations, it can be challenging to distinguish the different types of epithelial cells, since transitional cells are highly pleomorphic and many types of epithelial cells will become rounded once sloughed into fluid and degenerate when exposed to urine. Cell morphology is best appreciated in freshly formed and collected urine that is promptly analyzed. When evaluation of cell morphology is critical, the sediment pellet can be evaluated by Diff Quik-stained, dry-mount cytology of the urine sediment pellet. Other methods to diagnose structural lesions within the urinary tract (e.g. ultrasonography, catheter biopsy) are often more reliable and conclusive than urinalysis.

**Squamous epithelial cells** line the distal third of the urethra, the vagina, and the prepuce. They are large, flat, or rolled cells that have angular sides and usually a single small, condensed nucleus or they may be anucleate. A variable number of squamous epithelial cells are most commonly observed with lower urinary tract contamination of voided or catheterized samples. Squamous epithelial cells should not be present in samples collected by cystocentesis. A significant number of squamous epithelial cells are very rarely seen in cystocentesis samples due to squamous cell carcinoma of the bladder or due to squamous metaplasia of the bladder, which can occur with transitional cell carcinoma or chronic bladder irritation. Squamous epithelial cells may also be found if the uterine body of an intact female was unintentionally penetrated during urine sample collection.

**Transitional epithelial cells** line the renal pelves, ureters, bladder, and proximal two-thirds of the urethra. They are highly pleomorphic, variably sized cells that are smaller than squamous epithelial cells and two to four times larger than leukocytes. They may be round, oval, pear-shaped, polygonal, or caudate and often have granular cytoplasm with a single nucleus that is larger than that of squamous epithelial cells. There should be <5 transitional epithelial cells per low power field in normal urine sediments. A greater number of transitional epithelial cells are seen in urine samples collected by catheterization or in patients with inflamed, hyperplastic, or neoplastic mucosa. Transitional epithelial cells with caudate morphology specifically line the renal pelves. Caudate transitional epithelial cells are rarely observed in urine sediments and are an abnormal finding that can sometimes be seen in patients with pyelonephritis, renal pelvic calculi, or other pathology involving the renal pelves.

Cuboidal-to-low columnar **renal tubular epithelial cells** often become small round cells once they have exfoliated into urine and are not always easily distinguished from leukocytes or small transitional epithelial cells. Unless these cells are found within a tubular cast, observation of renal tubular epithelial cells is not considered a dependable indicator of renal disease, since a low number of tubular cells are sloughed normally and since other similarly sized cells (e.g. small transitional epithelial cells, leukocytes) may be mistakenly identified as renal tubular epithelial cells in wet mount preparations. Though, observation of a very large number of these cells with their cuboidal-to-low columnar morphology intact (not rounded) is a rare abnormal finding that would indicate active renal tubular disease.

**Neoplastic epithelial cells** are occasionally identified in urine sediment. In a patient that has a bladder or urethral mass, the urine sediment finding of atypical transitional epithelial cells in the absence of inflammation is suggestive of transitional cell carcinoma. Neoplastic transitional epithelial cells may exfoliate in cohesive sheets or individually. They are identified
by their malignant features, such as high nuclear-to-cytoplasmic ratio, variable cell and nuclear size, clumped chromatin with prominent nucleoli, and mitotic activity. However, when inflammation is present, it is not possible to distinguish hyperplastic epithelial cells, which develop similar cytologic features, from neoplastic epithelial cells. Since it is quite common for bladder tumors to become secondarily inflamed, definitive diagnosis using urine cytology alone is often not possible. In these instances, additional diagnostic information (e.g., imprint cytology or histology of catheter biopsy material) may be helpful in making a definitive diagnosis. Other less commonly observed tumors include rhabdomyosarcoma, urothelial papilloma, and squamous cell carcinoma.

**Blood cells, infectious agents, and other sediment findings**

Highly alkaline or dilute urine or improper sample storage may significantly reduce the number of cells in the urine sediment. During microscopic examination care should be taken to distinguish erythrocytes from lipid droplets. **Lipid droplets** are quite variably sized, refractile, greenish discs that are usually smaller than erythrocytes, often float above the plane of focus, and never exhibit the biconcave appearance of erythrocytes. They are frequently observed in feline urine. Beyond their potential to be misidentified as erythrocytes, they are of little significance.

**Erythrocytes** are quite translucent and may be pale orange due to their hemoglobin content. Erythrocyte shape varies with the toxicity of the urine. They may maintain their biconcave disc morphology; shrivel, becoming crenated in concentrated urine; or swell, becoming rounded in dilute urine. There should be <5 erythrocytes per high power field. However, the number observed can be influenced by collection method. Hematuria can be a component of pathology seen with hemorrhagic diathesis (e.g., thrombocytopenia), infection, inflammation, necrosis, neoplasia, toxicity (e.g., cyclophosphamide), or trauma.

In a sample collected by cystocentesis, there should be <3 leukocytes per high power field. In a sample collected by catheterization or midstream voiding, there should be <8 leukocytes per high power field. Being larger than erythrocytes and smaller than epithelial cells, leukocytes are intermediate in size compared to other cells that may be present in the sediment. They are usually round with a stippled appearance and greyish internal structure that transmits less light than erythrocytes; segmented nuclei are frequently visualized. Some leukocytes contain granules that are occasionally visible as refractile structures within the leukocyte. These cells may be referred to as **glitter cells**.

Observation of pyuria with concurrent **bacteriuria** indicates active urinary tract inflammation with either primary or secondary bacterial infection. Urine culture is useful to definitively identify microorganisms and to determine their antimicrobial sensitivities. Pyuria is also seen with other causes of genitourinary tract inflammation, such as urolithiasis, neoplasms, prostatitis, pyometra, and less common infections by viruses, mycoplasmas, or ureaplasmas. Cystocentesis may avoid contamination of the urine sample by leukocytes from the genital tract and aid in localizing the source of pyuria.

The absence of pyuria does not exclude the possibility of a urinary tract infection; therefore **urine sediment evaluation alone cannot be used to definitively exclude the possibility of infectious urinary tract disease**. Silent urinary tract infections (i.e., those lacking a detectable
inflammatory response) can be seen with hyperadrenocorticism/hypercortisolemia, diabetes mellitus, and other immunosuppressed states. Also, leukocytes and bacteria may be diluted below the detection limit of light microscopy in polyuric conditions when large volumes of dilute urine are produced (e.g. pylonephritis). At least 10,000 bacilli/mL or 100,000 cocci/mL are required for detection by light microscopy.

Bacteria may be observed in urine sediment for reasons other than urinary tract infection (e.g. overgrowth after collection). When microorganisms are observed in stained wet mounts (e.g. sedi-stain, new methylene blue), it is necessary to distinguish them from contaminants by confirming their presence in unstained wet mounts or by cytologic examination of the dry-mount sediment pellet. The latter is a more sensitive and specific method to detect bacteria than wet mounting and permits more accurate identification of bacterial morphology (Swenson et al., 2004). Urine cultures can occasionally be negative, even though bacteria were presumptively detected during urinalysis. Reasons for this disparity include antimicrobial administration prior to sample collection, prolonged urine storage prior to culture, improper culture technique, bacterial contamination of the sample during urinalysis, misidentification of nonbacterial structures. Also, uncommon infections by viruses or highly fastidious microorganisms (e.g. Mycoplasma, Ureaplasma) may result in negative urine culture though a urinary tract infection is present.

Cystocentesis is the ideal collection method for urine culture. The likelihood of representative culture results may be enhanced by collection of a randomly-timed urine sample (which will likely consist of freshly formed urine that has not stagnated within the bladder) along with inoculation of a Culturette™ tube immediately after collection. Afterward, the Culturette™ should be refrigerated to prevent overgrowth of robust bacteria.

With routine urine culture, results are reported that simply identify the bacteria present and their respective antimicrobial sensitivities. Quantitative urine culture is an alternative that may be helpful to determine if the bacteria cultured from a urine sample likely represent a true infection or if they are likely contaminants of the sample. In addition to bacterial identification and elucidation of their antimicrobial sensitivities, quantitative culture enumerates the bacteria as colony forming units (CFU)/mL. Quantitative urine culture can be used even when the urine sample has been collected by transurethral catheterization or by collection of midstream voided urine, instead of cystocentesis. Quantitative urine culture results must be interpreted using published guidelines that are based upon the urine collection method.

**Other sediment findings**

Dependent on geographic distribution, other infectious agents are occasionally identified in urine sediment, such as fungi (e.g. Candida, Aspergillus, Blastomyces dermatitidis, Cryptococcus); algae (e.g. Prototheca); and nematode ova, larvae, or adults (e.g. Capillaria, Dioctophyma immitis, Dioctophyma renale). Trichuris (whipworm) parasite eggs appear very similar to Capillaria parasite eggs. These two eggs can be distinguished by the positioning of their bipolar caps and the texture of their outer shells. The bipolar caps of Capillaria ova are slightly askew, rather than being perfectly bipolar as they are in Trichuris ova. And, the shells of Capillaria ova have a granular appearance, rather than perfectly smooth as they are in Trichuris ova. Capillaria are usually an incidental finding in the urine of asymptomatic cats. However, Capillaria eggs are rarely identified in cats that present with hematuria, which resolves after fenbendazole administration.
Common contaminants of urine samples include sperm, talc, glass chips, plant pollen, hair, and fibers. Aside from sperm, these contaminants can be mistaken for urine crystals (i.e. talc, glass chips), transitional epithelial cells (i.e. plant pollen), or casts (i.e. hair, fibers).

Urinalysis: wet-mount versus dry-mount

Urinalysis performed in-house by a trained staff member, soon after urine sample collection is a great way to reduce or avoid artifacts that can arise during the delay between sample collection and sample analysis. Keeping such analysis in-house may increase productivity and potentially improve your urinalysis laboratory results. In addition to traditional wet-mount urinalysis, dry-mount cytology of urine samples can be very useful in suspected cases of urinary tract infection (Swenson et al., 2004) or urinary tract neoplasia. The method is described below here. Slides prepared using this method can be evaluated in-house, or they can be readily sent to an outside laboratory for review by a pathologist. The benefit is that diagnostic material on slides prepared this way will not degrade the same way that it would in a liquid urine sample during transport to an out-of-house, reference laboratory. Often when fluid urine samples are sent to a reference laboratory for pathologist review to diagnose neoplasia, cells deteriorate to the point where cytologic diagnosis no longer possible.

Method to prepare urine sediment for dry-mounting and routine cytologic examination

1) Centrifuge the urine as is done for wet-mounting.
2) Use a transfer pipette to aspirate the pellet from the bottom of the conical centrifuge tube.
3) Place a small drop of the aspirated material onto a clean, glass microscope slide.
4) Use a second clean, glass microscope slide to spread the material in a monolayer.
5) Allow the slide to air-dry. Heat fixation is not necessary and would alter cell morphology.
6) Stain as a routine cytology using Diff Quik® or other similar stain. Alternatively, the slide can be stored in a covered container at room temperature and sent to a outside diagnostic laboratory for evaluation by a pathologist.

REFERENCES

Dry-Mount Fecal Cytology
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INTRODUCTION
There are several diagnostic tests of feces that may be necessary during complete evaluation of patients with clinical signs referable to the gastrointestinal tract. Optimal fecal assessment, including potential tests (e.g. wet-mount fecal cytology, dry-mount fecal cytology, bacterial culture, fecal antigen detection methods, fecal flotation, fecal sedimentation, Baermann technique, etc.) and their required sample handling, diagnostic indications, and interpretations, have been compiled in a great review article that is available online (Broussard, 2003). Dry-mount fecal cytology is one component of a thorough diagnostic evaluation of patients with gastrointestinal signs. Since some fecal pathogens can be morphologically indistinguishable from incidental nonpathogens, results of dry-mount fecal cytology should be interpreted in the context of the patient’s clinical presentation and results of other diagnostic tests, such as wet-mount fecal cytology, bacterial culture, and fecal antigen detection methods.

DETAILS
Sample collection, processing, examination, and other considerations
For fecal cytology, there are a few possible sampling methods, including collection of unadulterated fecal material, rectal saline lavage, and rectal scraping. A small amount of fresh fecal material may be obtained during digital rectal examination or using a moistened cotton-tipped applicator or fecal loop if digital rectal exam is not possible. Immediately collected, voided feces may also be used; however, voided samples may be more representative of the luminal portion of the rectum, which is not ideal for fecal cytology. Usually, if a small amount of fecal material is obtained using a moistened cotton-tipped applicator, the cotton tip of the applicator can be gently rolled on the slide to prepare a direct thin film preparation that is not excessively dense. Otherwise, if unadulterated fecal material has been obtained, slight dilution of the feces may be used to prepare a thin film preparation by placing a drop of sterile, normal saline on a clean, glass microscope slide, adding a very small amount of fecal material (no larger than a match-head), mixing with a sterile, wooden applicator, and spreading as for other fluid thin film preparations. Prior to spreading, newsprint should be legible though the saline-diluted drop of fecal material. Rectal scraping is a somewhat more invasive method of sampling the rectal mucosa by direct scraping of its surface with a swab or blunt spatula. Rectal scraping is typically required to identify some infections that localize to the deeper portion of the mucosa (e.g., histoplasmosis, protothecosis) or to characterize deep mucosal cellular infiltrates.
Dry-mount fecal cytology can be useful to examine the microorganism flora and any host cells that may be present (e.g., epithelial, inflammatory) and to detect other pathogens that may be present (e.g., bacterial, fungal, algal, oomycetal, or protozoal). In some cases, evaluation of dry-mount fecal cytology may permit one to determine the primary cause of a patient’s gastrointestinal signs. However, more often, abnormalities detected using dry-mount fecal cytology may or may not be contributing to or exacerbating the ongoing gastrointestinal signs. Many abnormalities, particularly those involving the background flora, are nonspecific, representing incidental findings associated with other underlying diseases, physiologic processes, or previous antimicrobial treatments. When evaluating dry-mount fecal cytology, it may be useful to apply a systematic approached aimed at evaluation of attendant background cells and detection of abnormal eucaryotic cells and pathogenic microorganisms.

REFERENCES

Key points to assess during systematic evaluation of dry-mount fecal cytology
1. Evaluate the background bacterial and yeast flora using 100x objective
2. Determine the number of spore-forming bacteria per 100x objective
3. Examine the smear for other potential pathogens using 50x or 100x objectives
   a. Algal (e.g., Prototheca)
   b. Bacterial (e.g., gull-wing and spiral shaped bacteria)
   c. Fungal (e.g., Histoplasma, Aspergillus, Blastomyces, Candida, Cryptococcus)
   d. Oomycetal (e.g., Pythium)
   e. Protozoal (e.g., Cryptosporidium, Giardia, Entamoeba, Trichomonas, Ballintidium)
   f. Rare findings (e.g., nematode and trematode eggs and larvae are rarely diagnosed by this method)
4. Scan the smear for the presence of host cells
   a. Inflammatory cells – observe types and relative quantities
   b. Epithelial cells – assess number and morphology (i.e., normal, hyperplastic, or neoplastic)
   c. Other atypical or neoplastic host cells
Normal flora, pleomorphic bacilli

Low number of incidental yeast

Closer mag. Incidental yeast

Fragments of plant

Large sheet of epithelial cells and individualized squamous cells

Abnormal flora: numerous cocci, several budding *C. guttulatus*

Budding *Cyniclomyces guttulatus*

Abnormal flora: numerous cocci with a degenerate neutrophil

Large aggregate of several Neutrophils

A single small lymphocyte (left) along with three well-differentiated low columnar epithelial cells

Very large, tightly cohesive, multicellular sheet of low columnar epithelial cells

Abnormal flora: absent background bacilli; presence of *Candida* pseudohyphae and blastospore
An increased number of spore-forming bacilli (>5 per 100X objective field). These bacilli may either be *Bacillus* sp. or *Clostridium* sp.

Malachite green is a stain available in microbiology laboratories that specifically stains bacterial spores.

Neutrophil with two miniscule, pleomorphic, gull wing-shaped bacteria (arrowheads). Miniscule, pleomorphic, gull wing-shaped bacteria (arrowheads) near a pigmented, squamous cell.

*Campylobacter* bacteria (arrowheads). Miniscule, pleomorphic, gull wing-shaped bacteria (arrowheads) near a pigmented, squamous cell.

*Giardia* trophozoites: It is uncommon to diagnose *Giardia* using dry-mount fecal cytology.

*Two Entamoeba histolytica* with a single neutrophil

*Three budding* (top) and one non-budding (lower right) *Cryptococcus*

*Several non-endosporulated Prototheca* are seen intracellularly in a macrophage (rectal scrape).
Cytologic evaluation of synovial fluid
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College of Veterinary Medicine
University of Florida, Gainesville, Florida, USA

Summary
Synovial fluid analysis can be an important diagnostic test in cases of suspected polyarthritis. The primary goal of synovial fluid analysis is to classify the fluid one of three ways: normal, noninflammatory (mononuclear inflammation), or inflammatory (neutrophilic inflammation) joint fluid. If necessary, the critical parts of synovial fluid analysis can be performed on as little as one drop of fluid. Results of synovial fluid analysis seldom yield a specific, definitive clinical diagnosis; results should be interpreted in light of other clinical and diagnostic findings (e.g., history, radiographs, culture, and serology). Complete synovial fluid analysis includes the following steps: Examination of the fluid’s physical features (Volume, Color, Transparency, Viscosity), Protein concentration measurement, Total nucleated cell count per microliter, Differential leukocyte count and microscopic examination.

Arthrocentesis
Those joints that are obviously effusive should be sampled. However, in dogs and cats, even if only one joint appears clinically affected, fluid should be collected from multiple joints in most cases. This is because polyarthropathies are more common than monoarthropathies in small animals; and in order to diagnose a polyarthropathy, one must document inflammation in more than one joint. Some suggest that a suspected polyarthritis warrants collection from at least 6 joints. The carpi and stifles are often sampled, because doing so is technically easy. Most immunologic arthropathies are more prominent in distal joints (i.e., carpal and tarsal). An exception to this generalization is lymphoplasmacytic synovitis, which usually affects the stifle joints. Small animal septic (i.e., bacterial) joint disease is more often found in the larger, proximal joints, such as the hip.

Slides should be made soon after collection. Apply one drop of fluid per clean microscope slide, spread as for other cytologic samples, air dry (do NOT heat fix), and stain with Romanowsky-type stain (e.g., Diff Quik®) or save the unstained slides for shipment to a referral laboratory. Observe the color and turbidity of the fluid and estimate its viscosity as the fluid drops from the syringe to the slide. Normal joint fluid should be highly viscous and form a string that is at least one inch long before breaking as the syringe is pulled away from the slide (string test). If only a few drops of fluid are obtained, microscopic examination is sufficient to complete the critical components of analysis (i.e., estimation of viscosity, nucleated cell count, and differential cell count). One drop should be saved for culture. It can be washed from the syringe by aspirating sterile saline or enrichment broth (e.g., thioglycolate) into the syringe and then emptying the fluid it into a sterile tube or culture medium (i.e., aerobic ± anaerobic ± mycoplasma). If an additional volume of fluid is obtained, the fluid should be separated into 2 aliquots. One aliquot should be placed in a purple top, EDTA tube, because inflamed or blood contaminated fluid will likely form a fibrin clot. The other aliquot should be placed in a red top, plain tube, because EDTA degrades mucin and interferes with microbial culture and pH measurement.
**Routine synovial fluid analysis** includes assessment of the 1) physical features of the fluid (i.e., volume, color, transparency, viscosity), 2) refractometer protein concentration, 3) total nucleated cell count, and 4) differential cell count with microscopic exam.

**Viscosity**

1. Gross observation (the string test): One can visually assess the viscosity during collection and preparation of the microscope slides. As the fluid drop is applied to the microscope slide, pull the tip of the syringe away from the slide. Normal synovial fluid will form at least a 1-inch long string before breaking as the syringe and slide are pulled apart.

2. Microscopic observation: Microscopically, normal synovial fluid has a very dense granular eosinophilic background, which is due to mucin in the sample. Decreased density of the granular eosinophilic background suggests decreased joint fluid mucin content.

**NOTE:** EDTA degrades mucin and therefore spuriously decreases the viscosity of joint fluid and interferes with the mucin clot test. Viscosity should be assessed when the microscopic slides are made or using the sample aliquot that is in the plain, red top tube.

**Total nucleated cell count**

**Slide estimation:** This method employs the same formula that is used to estimate the leukocyte count from a peripheral blood film:

- Nucleated cells/µL = (average # of nucleated cells per field) × (microscope objective power)²
- The average nucleated cell count per field should be determined using at least 10 **REPRESENTATIVE fields.** (A representative field is considered an area of the cytologic preparation where the nucleated cells are not clumped or trapped within a clot and where the sample is neither too thinly nor too thickly applied to the slide. Areas that are too thick will be extremely eosinophilic and the nucleated cells will be compressed by the surrounding fluid. This makes it impossible to identify the individual types of leukocytes).
- When sufficient sample volume is not available for the actual cell count per microliter, slide estimation of the nucleated cell count can be used as a substitute. **If properly done, the slide estimate of the nucleated cell count can provide a reliable indication of whether the nucleated cell count is normal or increased.**

**Manual and automated counts:** The cells can be directly counted using either a manual hemacytometer or an automated CBC machine. However, counts obtained by automated cell counting machines are often inaccurately high, since electronic cell counts are significantly higher than counts obtained manually from the same sample. The marked viscosity of synovial fluid can cause error with either method due to imprecise pipetting and cell clumping. To decrease the fluid’s viscosity, hyaluronidase can be added to an aliquot of the fluid sample prior to performing the cell count by hemacytometer or CBC machine. **(Joint fluid should absolutely NOT be put through a CBC machine without the addition of hyaluronidase).** Alternatively, if
performing a manual count using a hemacytometer, a Unipette® can be used to lyse the erythrocytes and decreased the fluid’s viscosity by dilution. Though, this method may be less accurate than the hyaluronidase procedure.

**Differential cell count and microscopic exam**

Normal synovial fluid contains ≥90% mononuclear cells and <10% mature, nondegenerate neutrophils that may be windrowing (arranged linearly) on a dense granular eosinophilic background, which may contain erythrocytes and/or platelets, depending upon the degree of blood contamination. The mononuclear cells should consist predominantly of macrophages that have pale basophilic, minimally vacuolated cytoplasm. A lesser number of small, well-differentiated lymphocytes and a low number of synoviocytes (a.k.a. clasmatocytes) may also be present. Synoviocytes are round to oval with abundant basophilic cytoplasm and an eccentrically placed oval nucleus that has condensed chromatin. Microscopic examination is the most important component of synovial fluid analysis. If necessary, the mucin content (viscosity) and the nucleated cell count can be estimated from the single drop of fluid used to prepare the slide. Microscopic examination is the sole method used to classify an arthropathy as noninflammatory or inflammatory.

**Normal synovial fluid in dogs and cats**

<table>
<thead>
<tr>
<th></th>
<th>WBC/µL</th>
<th>Mono %</th>
<th>Neut %</th>
<th>Volume (mL)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>Range</td>
<td>Mean</td>
<td>Range</td>
</tr>
<tr>
<td>Dogs</td>
<td>430</td>
<td>0-2900</td>
<td>98.6</td>
<td>88-100</td>
</tr>
<tr>
<td>Cats</td>
<td>161</td>
<td>2-1134</td>
<td>96.4</td>
<td>61-100</td>
</tr>
</tbody>
</table>
## Synovial fluid response in canine and feline diseases

<table>
<thead>
<tr>
<th>Condition</th>
<th>WBC/µL</th>
<th>Mono%</th>
<th>Neut%</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>DJD</td>
<td>&lt;5000</td>
<td>&gt;90</td>
<td>&lt;10</td>
<td>Reactive monocytes</td>
</tr>
<tr>
<td>Trauma</td>
<td>2500-3000</td>
<td>&gt;90</td>
<td>&lt;10</td>
<td>Neut% depends on peripheral blood</td>
</tr>
<tr>
<td>Hemarthrosis</td>
<td>2500-3000</td>
<td>&gt;90</td>
<td>&lt;10</td>
<td>Neut% dep. on peripheral blood; erythrophagia/hemosiderin</td>
</tr>
<tr>
<td>Bacterial (septic) arthritis</td>
<td>15,000-267,000</td>
<td>5-23</td>
<td>77-95</td>
<td>~25% have intracellular bacteria; +/- degenerative neutrophils</td>
</tr>
<tr>
<td>Rickettsial polyarthritis</td>
<td>38,800-50,000</td>
<td>20-40</td>
<td>60-80</td>
<td>≤1% neutrophils contain morulae (E. ewingii, A. phagocytophilum)</td>
</tr>
<tr>
<td>Lyme disease</td>
<td>average 46,300</td>
<td>15</td>
<td>85</td>
<td>Rarely see spirochetes</td>
</tr>
<tr>
<td><em>Mycoplasma</em> polyarthritis</td>
<td>&gt;1000</td>
<td>Predom.</td>
<td></td>
<td>Occasionally, lymphs predominate</td>
</tr>
<tr>
<td>Bacterial L-form associated</td>
<td>&gt;1000</td>
<td>Predom.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calicivirus arthritis</td>
<td>&gt;1000</td>
<td>monos + lymphs</td>
<td></td>
<td>Rarely, neutrophils predominate</td>
</tr>
<tr>
<td>Idiopathic, nonerosive immune-mediated polyarthritis</td>
<td>4000-370,000</td>
<td>&lt;10</td>
<td>&gt;90</td>
<td></td>
</tr>
<tr>
<td>Reactive polyarthritis</td>
<td>&gt;3000</td>
<td>&lt;10</td>
<td>&gt;90</td>
<td></td>
</tr>
<tr>
<td>SLE-associated polyarthritis</td>
<td>5000-371,000</td>
<td>7-85</td>
<td>15-93</td>
<td>Neuts usually predom.; occ’ly, monocytes predom; ANA+</td>
</tr>
<tr>
<td>Lymphoplasmacytic synovitis</td>
<td>5000-200,000 (rarely, &gt;200,000)</td>
<td>Lymphs predom.</td>
<td></td>
<td>Lymphs &amp; plasma cells predom.; CCL rupture associated</td>
</tr>
<tr>
<td>Eosinophilic synovitis</td>
<td>Increased</td>
<td></td>
<td></td>
<td>Eosinophils predom. (rare condition)</td>
</tr>
<tr>
<td>Idiopathic, erosive polyarthritis (rheumatoid)</td>
<td>2900-80,000 (avg. 30,000)</td>
<td>5-80</td>
<td>20-95 (avg. 74)</td>
<td>Monocytes occ’ly predominate</td>
</tr>
<tr>
<td>Feline chronic progressive polyarthritis</td>
<td>4000-70,000</td>
<td>1-75</td>
<td>25-99</td>
<td>When chronic, lymphs and plasma cells predom.; FeSFV+, +/- FeLV+</td>
</tr>
</tbody>
</table>
Heart diseases are frustrating diseases for the bovine practitioner. They are often presented in the terminal stage of the disease with clinical signs of heart failure. An early diagnosis may be limited in the field due to the lack of ancillary tests that can be done directly in a farm context. However, it is important to remember that ancillary tests are not reserved to veterinary teaching hospital and can be used to reach a diagnosis and therefore may help in a better management of the cases.

The four major heart diseases of cattle consist in pericarditis, bacterial endocarditis (BE) and cardiac form of enzootic lymphoma. In younger animals, congenital heart defect (CHD) is a frequent cause of cardiac disease differentiating these clinical entities is important in order to reach an early diagnosis avoiding therapeutic attempts in cases with a poor prognosis (e.g.: lymphoma) or to treat animals with a better prognosis (e.g.: idiopathic hemorrhagic pericarditis).

Moving from this introduction we can now discuss more in detail of the four main cardiac heart diseases as well as their clinical management in the field and the specific ancillary tests that can be used to reach a diagnosis. The complete cell blood count and chemistry panel can be mainly used as a first step to distinguish inflammatory (pericarditis and endocarditis) vs. non inflammatory cardiac diseases. Chronic inflammatory changes (hyperglobulinemia, non-regenerative anemia and hyperfibrinogenemia) are commonly observed in cases of bacterial endocarditis.

**Pericardial effusion : Septic vs. Neoplastic vs. Idiopathic.**

The main clinical finding associated with pericardial effusion is muffled heart sounds. The splashing sounds can be found in cases of septic pericarditis when a gas-liquid interface is observed due to bacterial gas production. Due to the increased pericardial pressure and
abnormal diastolic function of the heart, clinical signs of heart failure are due to increased cardiac preload and decreased cardiac output.

The assessment of pericardial fluid is a critical step of the workup diagnosis in these cases. Transthoracic echocardiography (TTE) can be useful to determine the quantity of pericardial effusion. The echogenic appearance of the pericardial effusion can also be useful for the clinician. Anechoic pericardial effusion is classically associated with pericardial fluid with a low proteic and cellular content (e.g. : hydropericardium, neoplastic pericardial effusion and idiopathic hemorrhagic pericarditis). By contrast highly heterogenic content with hypoechoic to echoic pericardial fluid is more commonly observed with septic pericarditis secondary to hardware disease. To the author’s knowledge the sensitivity and specificity of these findings still need to be determined. For these reasons pericardiocentesis is the next step to reach a diagnosis.

The pericardiocentesis is an easy procedure that is performed using an 18G 4’ needle on the 5th left intercostal space. When TTE has been performed, it may help to choose the best spot that will yield a positive result. A macroscopic examination of the sample will be enough in cases of septic pericarditis (cloudy liquid with foul odour). In cases of obtention of hemorrhagic sample, the sample should be submitted for a cytological analysis to differentiate neoplastic effusion (due to cardiac lymphoma) from idiopathic hemorrhagic pericarditis. This is of critical importance since the prognosis of IHP is good by contrast to other types of pericardial effusion. The exact etiology of this disease remains unknown and it is not known if IHP could represent an early stage of bovine leukosis virus (BLV) infection.

Although pericardial drainage with a chest trocard is a simple procedure that will cure IHP cases, it is usually not successful to manage traumatic pericarditis. The TTE may also be useful to monitor the therapeutic efficiency of pericardial drainage. In cases with confirmed traumatic pericarditis, the chest trocard drainage does not allow an effective pericardial drainage due to multiple fibrin clots contents. In these cases, the 5th rib resection allows a more complete pericardial drainage. This surgery can be performed in the farm under with anesthesia. It should be remembered that the prognosis still remains reserved despite this intervention especially due to the constrictive pericarditis complication.
**Cardiac Lymphoma**: 
Bovine enzootic lymphoma is the most common neoplasm secondary to BLV infection in North America. Cardiac form of lymphoma is commonly observed in the right atrium of the heart for unknown reason.\(^5\) Two different types of cardiac lymphoma presentation have been reported.\(^1\) The first one is characterized by massive amount of neoplastic effusion that will lead to clinical signs of tamponade and congestive heart failure. For these reasons, it may be difficult to distinguish it from other types of pericarditis. The second form characterized by the absence of significant pericardial effusion but by a mass effect in the right atrium. The diagnosis of cardiac lymphoma may be easy when the animal presents other typical signs of lymphoma (e.g. polyadenomegaly). However, in the absence of such typical signs the diagnosis of lymphoma must be reached by the visualization of neoplastic lymphocytes (pericardiocentesis, abdominoacentesis...). The prognosis is poor when the diagnosis is made. It is not recommended to send the cow to slaughter due to that generalized condition, high risk of condemnation and for consumer safety.

**Bacterial Endocarditis**: 
This cardiac disease is maybe the most insidious heart disease since clinical signs of heart failure are not common in these cases. Pooled sensitivity of clinical signs of heart failure was 37.7\% (95\% confidence interval 21.6-57.0\%) in a recent meta-analysis using a random-effect model on published studies reporting necropsy confirmed bovine BE cases.\(^2\) The classical presentation of BE cases is a cow with a history of chronic inflammatory process with undulating fever. One of the big problems is that there are a lot of cow developing this problem without having BE. For these reasons it is important for the clinician to diagnose BE. The prognosis of BE is low to poor especially in non high-genetic merit cows due to the extended required antimicrobial treatment and extended withdrawal time. A thorough cardiac auscultation is required since heart murmur sensitivity in cows with a definitive BE diagnosis is 60.3\% (51.8-68.3\%). The sensitivity in early stage of the disease may be lower so the absence of a murmur does not rule out a BE. Again TTE may be useful ancillary tests for diagnosing BE. Its pooled sensitivity was 84.3\% (60.4-95.0\%) in confirmed cases of BE.\(^2\) The TTE needs to focus on each valve especially the tricuspid valve since it is the most common site of valvular BE in cattle. The tricuspid valve is the easiest
valve to observe even for an operator non used in TTE, it can be seen from the right side of the thorax in the 4th intercostal space as well as the mitral valve (four heart cavities views). The observation of pulmonary and aortic valves may require a smaller probe to be able to manipulate it in the narrow intercostal space. Since BE is frequently accompanied with periodic blood emboli from the infected valve, blood culture has been used for diagnosing BE. This ancillary test may be of interest to determine the etiology of the endocardial infection and may help in the therapeutic decision for high valuable cows. Its pooled sensitivity was good at 86.9% (39.1-98.6%). However, the data were limited to only two studies reporting 58 and 28 cases of BE. Blood culture may be perceived as non-practical test in the field since the result is delayed until bacterial growth and identification is obtained. However, it is a simple ancillary test to perform in the field. Special care is required to aseptically prepare the vessel that will be punctured (traditionally the jugular vein) to avoid contamination from skin bacteria. This test still has its place as an interesting test for BE diagnosis and may be helpful when other tests are doubtful. It still remains unknown what is the best moment to perform this test in regard with the presence or absence of fever. Such information is unfortunately not available in large animals.

**Congenital Heart Diseases (CHD)**

The most common CHD in cattle is ventricular septal defect (VSD).³ The typical presentation of this disease is a young calf presented for chronic respiratory problem. The auscultation is quite typical with a holosystolic cardiac murmur with a point of maximal intensity on the right side of the thorax and palpation of a precordial fremitus. In a previous retrospective study, pneumonia was a common necropsy finding which may explain why these animals frequently have a history of respiratory problems.³

Again TTE is the more interesting ancillary test to diagnose VSD. The VSD are typically observed in the membranous part of the interventricular septum, just below the aortic root. The best view to observe VSD is the Left Ventricular Outflow Tract view in which both ventricles, atria and the aortic root are observed.

Other congenital heart diseases included the atrial septal defect and tetralogy of Fallot (which include the four different anomalies: VSD, right ventricular hypertrophy, overriding aorta, and
pulmonary stenosis). Other types of anomalies can also be found but are uncommon. Their diagnosis using TTE requires a used operator as well as a cardiac probe to obtain specific cardiac views. A simple test that can be performed when suspecting right-to-left shunting anomalies is the bubble test. A small volume sterile saline solution is injected after being agitated through jugular vein. The operator simultaneously observes the right and left heart. In cases of right to left shunt the echoic saline solution will be observed in the left heart (left ventricle and/or atrium).

The prognosis of CHD depends on the type of the defect as well as its repercussion on cardiac hemodynamic. Due to its uncertainty and sometimes suspected (although not proved) hereditary component, culling is a common suggestion. However, if the CHD is an incidental finding, for example a small VSD unrelated to the reason of presentation, it still remain unknown if suggestion of culling is a good suggestion. Adult productive cows have been mentioned having a VSD as well as horses with good athletic performance. One of the big challenge in calves diagnosed with CHD is that as soon as a heart disease is diagnosed the risk of being culled is so high that even calves with a benign CHD are culled due to the lack of objective predictive landmarks.

In conclusion, the field approach of heart diseases is mainly based on the diagnostic tests to reach a definitive diagnosis. An early diagnosis allows to spare time and money in low value cows. On the other side it allows to propose rapidly a specific treatment in high value cows. The TTE is the more interesting ancillary tool to monitor the repercussions of the disease on the heart as well as on cardiac function. With a good practice and knowledge of the cardiac anatomy, TTE is a very interesting ancillary tool that is not reserved to internists practicing in hospital settings.

References


Dr. Elizabeth Doré, DVM, DACVIM
Bovine Paratuberculosis.
Environmental sampling and culture: a simple and efficient diagnostic tool.

A general review on paratuberculosis concerning the agent, the mode of transmission, the importance of the disease and diagnostic tests used especially at the herd level will be done. The emphasis will be made on environmental sampling for detection of Mycobacterium avium subsp. paratuberculosis. Results of research done in Québec dairy herds will be presented.

Paratuberculosis is a chronic intestinal disease affecting ruminant species worldwide and it is caused by the bacteria Mycobacterium avium subsp. paratuberculosis (MAP). Cattle get mostly infected at a young age when they are more susceptible to the infection. A meta-analysis revealed that 73.7% of calves exposed to MAP before the age of 6 months developed lesions of Johne’s disease. The same study also concluded that lesions of Johne’s disease developed in 19.3% of cattle exposed after 12 months of age. MAP is mostly transmitted by ingestion of contaminated feces. Contaminated colostrum or milk by the MAP can also be involved in the infection of young calves. MAP can be excreted in milk of symptomatic and asymptomatic cows. The infection can also be transmitted in utero. A meta-analysis concluded that 9% of fetuses from subclinical cows were infected with MAP while 39% of fetuses from clinical cows were infected. Shedding of MAP in the feces occurs a long time after infection because of the long incubation period of this disease that is reported to range between 2 and 10 years. Only a few infected cattle will exhibit clinical signs which consisted of chronic weight lost with normal appetite, chronic diarrhea without a fever and unresponsive to treatment and submandibular edema consecutive to severe hypoproteinemia.

Since there is not treatment for this disease, animals showing clinical signs will have to be culled or euthanized. Economic losses with paratuberculosis are mostly attributable to animals infected subclinically. Productivity of these animals is affected at different levels: their can have less milk production than expected, they can have a significant increase in open days and they are more at risk of being culled from the herd. A Canadian study including 8 out of the 10 provinces showed that for a herd with 61 dairy cows with a seroprevalence of 12.7%, average economic losses were of 2992$ annually or 49$ per cow and 409$ per seropositive cow. This disease is also a concern because of the zoonotic potential of MAP. A systematic review conducted by a team of Canadian researchers found that the zoonotic potential of MAP could not be ignored despite the fact that there is little solid evidence to prove it.

Screening for MAP in a herd can be done using serological tests (ELISA) on serum or milk or using bacteriological culture on individual or pooled feces or on environmental samples. In recent years, PCR have also been validated for detection on MAP directly in fecal samples. The sampling of each individual can be costly and requires a lot of manipulations. A simulation model for the herds in the U.S. Midwest has shown that the culture of the environment is the most profitable in terms of cost benefit compared to ELISA and culture of individual or pooled feces. The sensitivity of this method in the model was ≥ 99% for herds with a prevalence of ≥
16%. The sensitivity was below 95% in herds with a prevalence of 5%, which is comparable to the use of ELISA test with confirmation by fecal culture\textsuperscript{13}. Environmental cultures were assessed as a diagnosis tool to detect MAP within the herd. Raizman and his colleagues took environmental samples on 62 free-stall farms and 46 tie-stall farms, in Minnesota, from May to September 2002. They isolated the MAP in the aisles of cows (77% of herds), manure storage (68%), calving area (21%), sick cow pen (18%), water runoff (6%) and weaned calves areas (3%)\textsuperscript{14}. The detection of MAP in 23 Californian herds by Berghaus and colleagues was similar for the three diagnostic methods used: environmental sample cultures (74%), individual fecal culture of 60 cows (70%) and ELISA on more than 60 serums (65%). Environmental cultures were more often positive in the "lagoon" where wastewater and manure are stored (15/23) compared to sick or fresh cow pen (8 / 22) and the alleyway where cows exited from the milking parlor (9 / 23)\textsuperscript{15}. A study conducted in 21 US states by Lombard and colleagues found at least one positive environmental sample by culture on 70.4% of herds infected with MAP\textsuperscript{16}.

Culture of environmental samples could also be a qualitative way to estimate the prevalence within herds. In a study done on Minnesota dairy farms, the fecal pool prevalence was estimated to be between 0.3 and 4% when cow alleyways and manure storage both cultured negative. Inversely, in herds where both areas cultured a heavy load of bacteria, the fecal pool prevalence was estimated to be between 53 and 73%\textsuperscript{14}. On large Californian dairy herds, the proportion of ELISA positive cows in each herd was significantly correlated with the proportion of positive environmental cultures. In this study the environmental samples were collected form 3 different locations: the return alleyway where cows exit from the milking parlor, the alleyways of the sick and fresh cow pens and the lagoon which is the wastewater storage\textsuperscript{15}. In seven Michigan’s herds, where environmental cultures were done every 6 months for 4 years, the primary manure storage area or the lactating cow floor cultured positive for MAP 75% of the time when the herd prevalence was above 2%. When the herd prevalence, determined annually by fecal culture of all adult cows, was equal or below 2% the 2 sites never cultured positive for MAP\textsuperscript{17}.

There is briefly how to perform environmental sampling on a dairy herd based on the USDA Johne’s Control Program\textsuperscript{18}. Six composite samples from 3 different sites are required and each sample should consist of 4 subsamples. The first site corresponds to an area of manure concentration (e.g. gutters). The second site is a manure storage area (e.g. manure pit). Finally, the third site is another manure concentration area but different from the first site (e.g. sick cow pen).

Optimal storage conditions of manure samples are essential to ensure the detection of MAP. Waiting for the culture, manure can be stored several days at 4 °C or -70 °C for long-term preservation. Storage at -20 °C significantly alters the viability of MAP and reduces chances to isolate it by bacteriologic culture\textsuperscript{19}.

A program for prevention and control of paratuberculosis was launched in November 2007 in the province of Québec. The first step of the program consisted of a visit at the farm by the veterinarian to assess the management practices by a questionnaire. The questionnaire was composed of 42 questions in 5 different sections: 1) Calving area and newborn calf care, 2)
Calves before weaning, 3) Post-weaned calves, 4) Heifers, 5) Adult cows. After completing the questionnaire and identifying risk factors that can increase the risk of MAP transmission, the veterinarian gave recommendations to the producer. After one year in the program, environmental cultures were done on participating farms and are repeated yearly in herds that still adhered to the program.

RÉFÉRENCES


Uniform Program Standards for the Voluntary Bovine Johne’s Disease Control Program, September 2010.


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The PK/PD Approach to Antimicrobial Therapy in Cattle
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Learning Objectives
- Understand how pharmacokinetics (PK) describes drug absorption, distribution, metabolism and elimination.
- Understand how pharmacodynamics (PD) describes drug action.
- Gain an appreciation of how PK and PD changes in disease, so that your drug therapy can change appropriately.
- Understand how we are intersecting PK/PD to develop the most effective antimicrobial dosage regimens.

Overview of Pharmacokinetics
Drug Distribution: volume of distribution (Vd): (L/kg) is a mathematical term used to describe the apparent volume of the body in which a drug is dissolved. The numerical value of Vd gives a rough indication of the distribution of the drug in the body. For most drugs we use, drug distribution is determined by the drug's ability to cross biological membranes and reach tissues outside the vasculature system. The physical characteristics of the drug molecule (ionization, lipid solubility, molecular size, etc) determine its ability to cross membranes.
Drug elimination refers to the irreversible removal of drug from the body by all routes of elimination. Elimination may be divided into two major components: excretion and biotransformation. Drug excretion is the removal of the intact drug. Most drugs are excreted by the kidney into the urine. Other pathways include the excretion of drug into bile, sweat, saliva, or milk. Biotransformation or drug metabolism converts the drug in the body to a metabolite that is more readily excreted. Enzymes involved in biotransformation are mainly located in the liver.

Clearance (Cl): measures drug elimination from the body without reference to the mechanism of elimination. It is the most important PK parameter, it is always reported in pharmacokinetic papers, but it is rarely interpreted for clinical use of a drug. It is the most important PK parameter because it is the only parameter that controls the overall drug exposure. But the units are cumbersome and its value has to be related to the species.

Elimination Half Life (t½): is the time required for drug concentration to decrease by one half. T½ determines the drug dosage interval; how long a toxic or pharmacologic effect will persist; drug withdrawal times for food animals or performance animals. It is the PK value most prone to error, since it's calculated from data. But it is more easily understood than clearance.

Drug Accumulation: Drugs are often given in multiple-dosage regimens. To predict plasma drug concentrations, it is necessary to decide whether or not successive doses of a drug will have any effect on the previous dose. The principle of superposition assumes that early doses of drug do not affect the pharmacokinetics of subsequent doses. For most drugs, as equal doses are given at a constant dosage interval, the plasma concentration-time curve plateaus and a steady-state is reached. At steady state, the plasma drug concentrations fluctuate between a Cmax and
Clinical antimicrobial Pharmacodynamics

Overview

Clinical consequences of dosage interval < t1/2:
1. Cmax at steady-state is greater than the peak concentration after a single dose.
2. There is minimal fluctuation between Cmax and Cmin.
3. Missing a dose will not affect plasma concentrations greatly.
4. There is a lag time to reach the desired plasma concentration, and there will be a lag time for plasma concentration to change in response to a dose change.

Clinical consequences of dosage interval > t1/2:
1. Cmax is less than when the dosage interval was < t1/2. As the dosage interval increases, Cmax becomes closer in value to the peak concentration of a single dose. If the dosage interval is >10 t1/2's, the time required to eliminate 99.9% of the previous dose, drug accumulation does not occur.
2. There is marked fluctuation between Cmax and Cmin (peak and trough).
3. Missing a dose will affect plasma concentrations greatly.
4. There is minimal lag time to achieve the desired plasma concentration.

Overview of Pharmacodynamics

Pharmacodynamics is what a drug does to the body (or in the case of anti-infectives, what the drug does to the pathogen), whereas pharmacokinetics is what the body does to a drug. The majority of drugs either mimic or inhibit normal physiological/biochemical processes or inhibit pathological processes in animals or inhibit vital processes of parasites and microbial organisms. Pharmacodynamics involves receptor binding (including receptor sensitivity), postreceptor effects, and chemical interactions. Integration of PK and PD helps explain the relationship between the drug dose and clinical response.

PK/PD Integration and Antimicrobial Efficacy

A successful antimicrobial dosage regimen depends on both a measure of drug exposure (PK) and a measure of the potency of the drug against the infecting organism (PD). New information is rapidly emerging regarding the PK/PD relationships that determine antimicrobial efficacy in both human and veterinary patients. The PK parameters used in drug dosage design are the area under the plasma concentration versus time curve (AUC) from 0 to 24 hours, the maximum plasma concentration (Cmax), and the time the antimicrobial concentration exceeds a defined PD threshold (T). The most commonly used PD parameter is the bacterial minimum inhibitory concentration (MIC). In relating the PK and PD parameters to clinical efficacy, antimicrobial drug action is classified as either concentration-dependent or time-dependent.
**Concentration Dependent Antimicrobials**

For antimicrobials whose efficacy is concentration-dependent, high plasma concentrations relative to the MIC of the pathogen (Cmax:MIC) and the area under the plasma concentration-time curve that is above the bacterial MIC during the dosage interval (area under the inhibitory curve, AUC0-24 hr:MIC) are the major determinants of clinical efficacy (Figure 1). These drugs also have prolonged PAEs, thereby allowing once a day dosing while maintaining maximum clinical efficacy. For fluoroquinolones (enrofloxacin, orbifloxacin, difloxacin, marbofloxacin), clinical efficacy is associated with achieving either a AUC0-24 hr:MIC >125 or a Cmax:MIC>10. For aminoglycosides (gentamicin, amikacin), achieving a Cmax:MIC>10 is considered optimal for efficacy. Other antimicrobials that appear to have concentration-dependent activity include metronidazole (Cmax:MIC>10-25) and azithromycin (AUC0-24 hr:MIC>25). For some pathogens with very high MIC values, such as *Pseudomonas aeruginosa*, achieving the optimum PK/PD ratios may be impossible with label or even higher than label dosages. In such cases, under dosing is ineffective and merely contributes to antimicrobial resistance.

![Dose-Dependent Antimicrobial Activity](image-url)

which the antimicrobial activity (T>MIC) (Figure 2). All concentrations should be above the MIC is still being debated and is likely specific for individual bacteria-drug combinations. Typically, exceeding the MIC by 1-5 multiples for between 40-100% of the dosage interval is appropriate for time-dependent killers. The T>MIC should be closer to 100% for bacteriostatic antimicrobials and for patients that are immunosuppressed. So these drugs typically require frequent dosing or constant rate infusions for appropriate therapy. In sequestered infections, penetration of the antimicrobial to the site of infection may require high plasma concentrations in order to achieve a sufficient concentration gradient. In such cases, the AUC0-24 hr:MIC and/or Cmax:MIC may also be important in determining efficacy of otherwise time-dependent antimicrobials. The penicillins, cephalosporins, most macrolides and lincosamides, tetracyclines, chloramphenicol and the potentiated sulfonamides are considered time-dependent antimicrobials.

![Time Dependent Antimicrobial Activity](image-url)
Summary
The integration of PK/PD data for dosage design is now the standard in human medicine and is rapidly developing in veterinary medicine. The benefits of the PK/PD approach include increased efficacy of our drug therapy while minimizing adverse effects and for antimicrobials, minimizing antimicrobial resistance.
Inflammation and pain are very common clinical problems in veterinary medicine. It is a tremendously expanding area in human medicine (all the baby boomers are getting older and suffering from their physical activities like bungee jumping and mountain biking!). We have a much better understanding of the mechanisms of pain and inflammation and many of the human drugs are being explored for use in animals. Practitioners need a basic understanding of the action of these drugs in order to appreciate clinical differences between them.

**Classification of Pain**
- Anatomic: somatic or visceral
- Temporal: acute vs chronic
- Mechanistically:
  - Inflammatory: tissue trauma and inflammation
  - Neuropathic: nerve injury
- Physiological: noxious input (high-threshold), discrete (localized), transient, protective
- Pathological: follows severe trauma, non-noxious input (low threshold), diffuse, prolonged, no protective function

**Pharmacological Considerations**
Species differences in pharmacokinetics and pharmacodynamics are extremely important. Drug actions may be highly selective, but rarely specific. Many of the transmitter and receptor systems in the central nervous system undergo developmental changes.

**Neural Plasticity**: ability of the nervous system to modify function in response to different environmental stimuli. “Sensitization” can be peripheral or central.

**Limited Tissue Trauma and Inflammation**
- Pain is:
  1. discrete
  2. proportionate and protective
  3. resolves once inflammation resolves

**Extensive Tissue Trauma and Inflammation**
- Peripheral and central sensitization occurs. Pain is:
  1. diffuse
  2. disproportionate
  3. debilitative
  4. continues beyond the resolution of inflammation

**Peripheral Sensitization**
1. With trauma, inflammatory mediators are released from damaged cells (H⁺, K⁺), plasma (bradykinin), platelets (serotonin), mast cells (histamine), and macrophages (cytokines).
2. Damaged cell membranes start the arachidonic acid cascade with the resulting production of prostaglandins and leukotrienes.

3. Nociceptor nerve endings release substance P and calcitonin-generated peptide, which cause mast cell degranulation, vasodilation, edema and further sensitization and activation of nociceptors.


5. Increased excitability in injured peripheral nerves causes a death of GABAergic inhibitory interneurons over weeks, further removing tonic pain inhibition.

So you get a “sensitizing soup” of chemical mediators that convert high-threshold nociceptors into low-threshold pain receptors.

Central Sensitization

1. Repetitive stimulation of peripheral nociceptors leads to sustained release of glutamate and neuropeptides from afferent neurons.

2. Sustained activation of dorsal horn projection neurons leads to progressive cellular depolarization and activation of additional types of glutamate receptors.
   a. These receptors activate 2nd messenger systems, increasing intracellular calcium, and phosphorylating ion channels.
   b. These changes cause an activity-dependent increase in the excitability of dorsal horn projection neurons to subsequent nociceptive input (called “windup”).
   c. Windup is the physiological trigger for central sensitization.

3. Further stimulation of peripheral nociceptors causes additional long-term cellular changes in projection neurons that lead to an expansion of receptive fields and reduction in activation thresholds for nonnoxious stimuli (hyperalgesia, allodynia).

Principles of Effective Pain Therapy

1. Patients presented for elective surgery (dehorning, castration) will have a normal physiological response to the surgical pain and can be routinely treated for pain.

2. Patients presented for acute nonelective surgery (fracture repair) that involves substantial tissue trauma and inflammation will have a pathological response to pain that will require more aggressive pain management.

3. Patients presented for chronically painful conditions (laminitis) that have extensive tissue trauma and inflammation will have an extremely pathological response to pain that may be refractory to “standard” pain treatments.

4. “Pre-emptive” analgesia: analgesic therapy given before causing pain and inflammation is more effective than giving the same drug after causing pain and inflammation.

5. “Multimodal” analgesia: simultaneous administration of 2 or more classes of analgesics is more effective and safer than monotherapy. Select drugs that sequentially block
descending nociceptor pathways (NSAID, local anesthetic, opioid) and activate
descending antinociceptor pathways (alpha-2 agonists).
6. “Mechanism based” analgesia: choose drugs that target specific molecular mechanisms
involved in nociception and antinociception.
7. Effective nonsteroidal anti-inflammatory drug therapy
   a. Cyclooxygenase (COX) selectivity has not been proven clinically relevant for
efficacy or toxicity in veterinary species.
   b. Analgesic effects are from central effects as well as anti-prostaglandin effects.
   c. NSAIDs are more effective as analgesics when given prior to the onset of the
inflammatory processes or insult.
   d. The time to onset and duration of analgesic properties of NSAIDs does not
   correlate well with their anti-inflammatory properties. The analgesic effect is
   likely to have a more rapid onset and shorter duration of action than the anti-
inflammatory action.
   e. Dosage regimens for effective analgesia may need to be different than for anti-
inflammatory action.
8. Combination drug therapy:
   a. Synergism vs Addition
   b. Combining drugs from the same drug class that differ in their onset and duration
of action.
   c. Combining two or more drugs from different classes, such as an opioid with an
NSAID.
   d. Combining drugs delivered through different routes, such as a topical agent
(lidocaine) with an oral agent (gabapentin).

Caution: “…adding more medications into a treatment regimen does not always lead to
better pain relief and/or functional recovery, but it surely increases the cost and possibly
side effects of drug therapy.”

**Chirality:** Chiral compounds are nonsuperimposable mirror images. An important concept in
understanding the pharmacokinetics and pharmacodynamics of drugs (especially the alpha-2
adrenergic receptor agonists and new nonsteroidal anti-inflammatory drugs) is that some drugs
exist as stereoisomers (enantiomers). Stereoisomers are compounds with the same molecular
formula, but because of asymmetrically oriented chemical groups in space, they produce
nonsuperimposable mirror images and are known as “chiral” compounds. This means that they
are like your hands - superimposable palm to palm, but not palm to back. There are several
(and sometimes confusing) ways of referring to the configuration of asymmetric molecules. For
the nonsteroidal anti-inflammatory drugs it is common to use the “S” (sinister) and “R” (rectus)
designation for each of a pair of enantiomers. Sometimes “+” and “-” are used and sometimes
“levo-” (eg, levofloxacin) and “dex-” (eg, dexmedetomidine) are used.
Although each member of a pair of enantiomers differs in three dimensional orientation, their
physical properties (melting and boiling points, refractive index, solubility, etc) are identical. But
it is very important to realize that biological systems are highly chiral environments. The pharmacokinetics and pharmacodynamic effects of each of a pair of enantiomers may be very different. Therapeutic efficacy and/or toxic effects may be related specifically to one enantiomer. However, many drugs are formulated as racemic mixtures, containing equal (50:50) amounts of each enantiomer, because chemical synthesis of pure enantiomers is very expensive. It is estimated that 25% of the drugs used clinically are chiral compounds. This includes many of the plant alkaloids and glycosides (morphine, digoxin) and the nonsteroidal anti-inflammatory drugs (NSAIDs). Stereospecificity may occur in the pharmacokinetic processes of absorption, distribution, metabolism and excretion, especially if the process involves a carrier protein. If the fit of a drug molecule into the binding site on a protein, enzyme or receptor involves the chiral centre, then the affinity for attachment will be different for each of a pair of enantiomers. To further confuse the issue of sorting out the different pharmacokinetics for each enantiomer, some enantiomers can undergo “chiral inversion”, as hepatic enzymes convert one form of the enantiomer to the other form. The degree of chiral inversion for any drug varies between species, and cannot be predicted from one species to another. Therefore, it is very hazardous to extrapolate dosages for chiral compounds from one species to another.

**Therapy of Pain and Inflammation in Cattle**

Unfortunately, treatment options for pain and inflammation in food-producing animals is more limited than in other species mostly due to the limited number of approved products for food animals. Some quality assurance programs (eg. swine) require that drugs must be licensed for use in a food animal species before they can be used. In food animal production, cost is always a large concern. Although glucocorticoids and local anesthetics are cheap, some NSAIDs are expensive, especially if large volumes are required or if they are used for long-term therapy. Frequency of administration is more of a concern in food animals than companion animals. Animals on pasture or in a large herd may be difficult to catch. Drugs with short half-lives (such as aspirin) aren’t very practical in these situations. Withdrawal times in meat and milk can limit the utility of pain medications. These can vary from very short (0 milk withdrawal, 24 hr meat for ketoprofen in cattle) to very long (CgFARAD recommends a meat withdrawal of 60+ days for phenylbutazone in cattle and the use of phenylbutazone in dairy cows 20+ months of age is illegal in the US). These limitations should not stop you from using pain medication in these animals! Anti-inflammatory drugs often make good economic sense, as inflammation and pain will reduce food intake and production. As well, cost of medication should not be used as an excuse when animal welfare is involved.

**Lameness:** While foot rot is a leading cause of lameness in cattle, it is not the only cause of lameness. A careful diagnosis should be made before administering drugs that will prevent salvage for slaughter. Local anesthetics are injected or applied to a specific area to block neuronal conduction of pain in order to localize lameness or perform minor surgical procedures. Lidocaine is the most common local anaesthetic used in veterinary patients. Mepivacaine (Carbocaine®) is similar to lidocaine, but has a slower onset of action but a longer duration of action. The efficacy of a local anesthetic is highly dependent on the local tissue pH. Infected tissues are extremely difficult to successfully “block”. Anti-inflammatory drugs can be
useful for supportive therapy for lame cattle. They do not solve the problem and the underlying cause should be determined and treated appropriately (eg. antimicrobials for bacterial footrot, proper hoof-trimming for sole abscesses, etc.) For some valuable animals (like breeding bulls), NSAIDs like ketoprofen (Anafen®), meloxicam (Metacam®) or flunixin (Banamine®) may help decrease the interdigital swelling, thus relieving pain and helping the animals use the affected limb. While phenylbutazone is inexpensive and effective, it is not recommended due to human health concerns and extremely long withdrawal recommendations from the CGFARAD. Although cheaper and longer-acting, glucocorticoids are not the best choice for supportive care of footrot because of immunosuppressive effects. They may be useful for intra-articular injection in cases with DJD.

**Downer cows/musculoskeletal injury**: Although many downer cows are likely to have a metabolic predisposing cause (such as hypocalcemia, hypomagnesemia, hypokalemia, etc.), traumatic injuries are also common. These cows may be bright and alert, and may try to stand or “creep” along the ground with their front legs. Traumatic causes include nerve (sciatic/obturator) damage after calving, breeding injuries (both cow and bull), slipping on concrete, or entrapment in a stall. Downer cows may also have more exotic causes (like BLV tumours impinging on nerves) or may be idiopathic. Regardless of the cause, all downer cows require intensive care, with lots of bedding and placement in sternal recumbency. Pharmacology is of secondary importance, but anti-inflammatories (NSAIDs or glucocorticoids) are helpful, especially if given within a short time of the injury. Don’t forget to look for and treat any underlying cause like milk fever or toxic metritis/mastitis!

**Bovine respiratory disease/”shipping fever”**: Most anti-inflammatories have a label claim for the supporting care of bovine respiratory disease. The NSAIDs are useful for treating pyrexia, but again are just supportive therapy. Due to their immunosuppressive effects, the use of glucocorticoids for BRD is controversial. Other anti-inflammatory “supportive care” uses include metritis and pinkeye.

**Endotoxemia/toxic shock**: Severe conditions like septic mastitis or metritis can result in endotoxemia or toxic shock. Ketoprofen (Anafen®) and flunixin (Banamine®) are specifically labeled for endotoxemia in cattle, but meloxicam (Metacam®) is also useful. Isoflupredone (Predef® 2X) also has a “shock” claim, but it is an intramuscular acetate formulation, not an immediate-acting IV formulation (such as methylprednisolone sodium succinate) so it is unlikely to be effective without primary therapy such as intravenous fluids.
Livestock production in Canada depends on drugs and other chemicals to protect the health of animals. Additionally, food animals may on occasion be exposed to environmental contaminants or become the object of bioterrorism. To protect the Canadian public against adverse health effects, there are federal programs charged with the detection of chemical and drug residues in foods of animal origin. To proactively reduce residues risks, veterinarians need to be able to provide producers with accurate information withdrawal times for drugs or chemicals in animals prior to slaughter or shipment of eggs or milk. With initial financial support from the Agriculture and Agri-Food Canada’s Canadian Adaptation and Rural Development Fund (CARD Fund) and continuing funding from veterinary medical and livestock producer groups, the Canadian global food animal residue avoidance databank (GgFARAD) was established at the Western College of Veterinary Medicine in Saskatoon, SK in 2002 to provide information on residue avoidance to veterinarians.

Historical Background

The FARAD concept was established in 1982 as a cooperative project between four US veterinary colleges and the US Department of Agriculture’s Food Safety and Inspection Service as a way to reduce the rate of residue violations in animal products through education and information. The founding philosophy of FARAD was that information about residue avoidance from all sources should be immediately available from a scientific source. The FARAD was developed to not only contain information about approved animal drugs but to also include information on extralabel drug use and environmental toxins. For this “one-stop shopping” information service to work, the FARAD information was collated into a searchable computer database, with residue and pharmacokinetic data analyzed and interpreted by veterinary pharmacologists and toxicologists. For many years, the US FARAD centers provided limited consultation on residue avoidance to Canadian veterinarians but encouraged development of a Canadian databank to suit our specific needs.

Canadian gFARAD

Initially, the Canadian gFARAD was composed of two regional centers: the Western College of Veterinary Medicine in the west and the Faculté de médecine vétérinaire in the east. Unfortunately, in 2005 the eastern centre became unsustainable and since then, all services were handled by the WCVM centre’s clinical pharmacology residents under the supervision of Patricia Dowling, DVM, MSc, Diplomate ACVIM and ACVCP and Sarah Parker, DVM, Mvetsci. In 2011, a new eastern centre at the Ontario Veterinary College opened, supervised by Ron Johnson, DVM, PhD, Diplomate ACVCP. This expansion increases our resources and allows us increased educational and research efforts. The Canadian gFARAD provides expert-mediated decision support for any inquiry related to drug or chemical residues in food animals. Canadian gFARAD personnel assist veterinarians or government agencies with inquiries related to environmental contamination or bioterrorism. Extralabel drug withdrawal information is only
provided to licensed veterinarians because of their privilege and responsibility in using or prescribing drugs in an extralabel manner. The purpose of the CgFARAD is not to promote extralabel drug use, but to protect the public safety when it is necessary for veterinarians to use drugs in such a manner. Accurate information regarding the condition being treated is also critical for CgFARAD personnel to determine the impact of disease on residue depletion. Because the CgFARAD withdrawal recommendation is not an official withdrawal time and is based on data that has not been reviewed nor approved by the Veterinary Drugs Directorate, responsibility for residue violations rests with the prescribing veterinarian. In the unfortunate event where a residue results from extralabel drug use, it is still advantageous for a veterinarian to have recommended a withdrawal interval that is based on the best scientific evidence available. Since starting operations in October of 2002, the CgFARAD has answered 14,700 requests for withdrawal recommendations in food animals. The bulk of the requests concern the extralabel use of drugs in poultry and swine feeds. This practice is not allowed by law in the United States and in-feed antimicrobials have been banned by the European Union. This presents a unique and sometimes problematic case load for CgFARAD personnel as we attempt to provide adequate withdrawal recommendations with often limited data. We are greatly assisted in our efforts by colleagues at the Canadian Food Inspection Agency’s Centre for Veterinary Drug Residues, who provide information on analytical methods and limits of detection for many veterinary drugs.

How to Contact the CgFARAD

When a veterinarian specifically uses a drug in an extralabel manner, the best way to obtain a withdrawal recommendation is to submit a request through our website at www.cgfarad.usask.ca. Drug depletion data is constantly emerging, so CgFARAD recommendations may change rapidly. Therefore we recommend that a withdrawal recommendation be requested for each specific case of extralabel drug use. North American Compendiums generously supplies the CgFARAD with the generic and trade names of all the drugs and chemicals in the Canadian Compendium of Veterinary Drug Products (CVP), so veterinarians can pick the specific drug product from drop down menus. The web format provides additional necessary information such as the disease being treated, as pathophysiology may affect the final withdrawal recommendations. Routine inquiries are typically answered within a few working days, while complex residue problems may require a longer period of time as personnel search for information upon which to make a recommendation. If the request involves chemicals or products not in the CVP (eg, toxins, human drug products) or general information is being sought, the website provides an email message service and a phone number where practitioners can leave a voice message.
Dr. Gilles Fecteau, DVM, Diplomate ACVIM  
Farm animal service in Saint-Hyacinthe  
10 rules we feel help us over the years  
Gilles Fecteau DMV, DACVIM and André Desrochers DMV, MS, DACVS, DECBHM  
St-Hyacinthe, Québec

HISTORICAL BACKGROUND INFORMATION:
The farm animal service at St-Hyacinthe Veterinary School is a referring center for Eastern Canada bovine practitioners. Approximately 1000 cases are referred each year to the hospital. Amongst the clinician team, there are 3 Diplomates of the ACVIM, 2 Diplomates of the ACVS and 2 Diplomates of the ECBHM. The classical patient is an Holstein dairy cow from one of the 2 following population: a commercial cow from a client near the Veterinary school or a purebred cow from a anywhere in the province of Québec, Ontario or the Maritimes. The small ruminant case load is proportional to its economical importance in Québec.

(1) **Complementary expertise and team effort:** One important aspect is that the different clinicians should have complementary expertise. All can perform most tasks and correctly manage any type of cases, however we feel that natural interest for particular subject and type of cases is useful to broad the service offer to the clientele. By diversifying the interests, the group ensures that the literature search and body of knowledge remains current. In fact, not only the level of expertise is current but the development of new technique or new approach will emerge in some specific fields of interest. The achievement of the common goal has to be a team effort. This means that the individual goals may be a bit behind the actual collective objective. Always remember the reason we are there; teach veterinary medicine while treating true patients...

(2) **Define clearly who your clients are:** As the team develops and evolves (new hiring and retirement), it is extremely important to remember our mission. The primary goal of our work and as a consequence, the primary client is the *veterinary student*. The society needs you to graduate enough veterinarians each year to maintain the steady state between retirement and new graduates in the profession. Therefore, our primary and most important client is the veterinary student. A secondary and also extremely important client is the referral veterinarian. Since our policy is to accept only referrals, we would loose our clientele if the contact with the referral was inadequate. Of course, another important client is the producer, owner of our patient. Remember that they play an important role in supporting the veterinary school through funding that becomes available. Of course, the patient is also your client and patient care has to be amongst the most important criteria that directs the decision of your services. The interns and residents also clients and future colleagues, deserve time and attention. They are close from your staff and students and often offer valuable suggestions to improve the delivery of services.

**Refuse case direct from the client:** It can be very tempting to have client directly calling you instead of having the referral veterinarians involved. We systematically refuse that approach and found that in the long run, the service is winning. If a client calls us directly, we remain
polite and ask him to confirm with his primary veterinarian that the patient be referred to us. In other words, we explain that we work in team with his veterinarian and that we need to talk and discuss the case prior to the hospitalisation. The type of case evolved over the years and very few cases are now considered routine case.

(3) Today’s farm animal students become tomorrow's referral: It is important that students interested by farm animal practice realise and understand how a referred case is managed and what services can be offered to bovine practitioners. They often think naively that referral veterinarians are practitioners with fewer competencies and in fact they will do better once they graduate. It is our role to demonstrate that it is in fact a proof of competency to refer a case for care that cannot be provided at the farm. Teach them their limits and what we can do for them as a referral veterinarian. We also explain in rounds that it is always easier to criticize a case while you come later. The reality of the situation in which the primary veterinarians was working cannot be described and understood easily after the case is referred to us. The condition may also evolve after the first examination was performed. Treat them professionally because they will be your referral for the next 20 years.

(4) Availability: phone (pager): Referral veterinarians need you at any time of the day or night. Most phone calls will NOT result in a case coming through your clinics. However, if you are not answering all phone calls, you will miss the phone call related to a referral case. We must accept that most phone calls are in fact free consult or free continuing education. It is the price to pay to see interesting cases and maintain clinical activities. We are extremely easy to reach during the day and multiple team members are in fact available to answer the phone. There is a price to pay and how many publications or grant are lost while you are on the phone over the years is difficult to estimate?

(5) Referral letters and phone calls: One extremely important factor for referral veterinarians is that they need to be kept informed. This is actually even more important if the primary diagnosis changes after your assessment of the case. This could be misinterpreted by the owner as a mistake by the referral veterinarians. For each referral, a summary (very brief) is sent by fax or e-mail the day of discharge. Our hospital software system does not allow any possibility to easily share information with veterinarians. The summary must be sent quickly even if it means that it is not as complete or perfect or informative, as you would like it to be. Many producers will call the referring veterinarians as soon as the cow returns home. It is very uncomfortable for a veterinarian to answer questions from his clients if he does not know the details of the case. Cases where the producer appears unhappy with the care provided by his referring veterinarians will be approached with caution. Phone contact will be made with the referring veterinarian to explain the situation, what was said and not said. We do not believe that long and fastidious referral letters are appreciated by our referral veterinarians if they arrive late.

(6) Be on site for emergency: Emergency situations are always problematic situations. There is a lot of tension in the air. Moreover, your personal is reduced (AHT, barn crew, students, colleagues). It is probably the worst time for a junior Faculty or residents to be initiated to
manage a case by himself. “Be on site” to take the pressure off the shoulder of the residents; we can share responsibilities and alternate roles during the next emergency. The senior staff takes care of client communication (often a bit more tricky) or technical aspects of surgery. We tend not to let the residents manage emergencies by themselves until the final year. We feel that the client often makes their idea of our quality of service based on the level of care they received after hours. It is difficult for us to let an inexperienced veterinarian in charge of emergencies because of they are all referral and complicated cases.

(7) Monitor the billing policy and ensure possibilities of academic reductions of fees: I know this is easier said than done but in order to maintain some cases you need some flexibility with billing. All cases cannot blindly bill in the same manner. In some situations extraordinary case needs extraordinary cares and extraordinary bills as well. The subject of billing is always prone to conflict with administration and this should be addressed with tenacity. It is the yin and yan theory. They want us to charge more; we want them to charge less... It is a constant battle. The only thing we can say is if you loose this battle, you will be left without a caseload and it will likely cost more to the veterinary school to achieve its primary goal: train veterinary students how to practice! However, the billing reductions need to be monitored and rules needs to be established. We don’t want a clinician to have a reputation to charge less than others. We also feel that some opportunities are good to increase the income.

Seek opportunities and accept the challenges: Often, our service is challenge by unusual type of cases. This forces us out of our comfort zone and at first the temptation can be to say No! Think twice since some of those challenges can be tomorrow’s case load. As examples, the down cows and tank did great for us; the cloned calves also. The secret is to do it in a way that creates a win-win situation. We charge for what it is worth and adapt our clinics and time to feel good about the services provided.

(8) Support for government agencies (epidemio-surveillance): We feel that support from government agencies is very important. As a referral center, we could provide sentinel surveillance to detect strange cases or exotic diseases. Referral veterinarians tend to call us if they see cases with unusual presentation. This is in fact a service that some government agencies should be offering? Collaboration with government agencies must be encouraged. Sometimes funding becomes available to establish surveillance and monitoring of specific diseases.

(9) Contact with referral veterinarians through CE and publications: The proximity with referral veterinarians can be maintained by CE and workshops. In our experience, in the week following a conference or a workshop, some of the veterinarians who attended the session refer cases. Almost as if they were reminded that that possibility exists. It is important to present challenging cases but not to become too esoteric. Cases series or retrospectives studies with a good clinical description and some ideas in percentage of the prognosis are excellent. Practitioners appreciate presentation of all the mistakes that you did yourself before diagnosing the problem and realising that you were facing a new disease or clinical entity.
(10) **Match clinical needs and academic objectives:** As Faculty member, it is important that you prepare your summary for promotion as well as making sure the clinics work. It is an equilibrium and what we found works best is to try to match those 2 objectives instead of making them in opposition. That would mean clinical cases become teaching material, CE material and your research material. We try to stay away from research grants that keep you away from the clinics unless it is your professional objective. Make it a mission or a section statement so the administration will acknowledge it and most likely consider it in the process of promotions. At the same time it will be more rewarding and encourage others to do the same.

**Conclusion:** There is no secret recipe to maintain a case load. It needs time and commitment. The time in clinics should be a priority. Recruiting a good team with a common goal, means you’re on the road for success. If you are looking for big grant and zillions of publications in prestigious journal, you may be disappointed with a heavy commitment in clinics. However, do not think that your life will be boring...
In cattle, more than thirty species of mycoplasmas have been identified. They are the cause of many diseases including respiratory, articular, mammary, genital, ocular and auricular infections [1]. Among them, two species are particularly pathogenic: *Mycoplasma mycoides* subsp. *mycoides*, which is the cause of the contagious bovine pleuropneumonia (North America is free of CBBP); and *Mycoplasma bovis*. The importance of *M. bovis* in dairy cattle no longer needs to be proven and diseases linked to *M. bovis* are the source of significant economic losses. Major outbreaks of *M. bovis* associated diseases have been reported in different parts of the world secondary to its introduction into naive herds. Thus, biosecurity recommendations for *M. bovis* infection have become necessary. Since *M. bovis* is, by far, the most pathogenic and frequent mycoplasma isolated in Canada, this presentation will mainly focus on it. Additionally, little to no information on the epidemiology of other mycoplasma infections is available.

Most of the information presented herein has been recently published in a complete review of literature performed by Maunsell et al. [2]. Readers may refer to this article for further information.

1. Source and transmission of mycoplasma

1.1 Source of infection

**Infected animals and shedding.** The introduction of an asymptomatically infected animal is reported to be the main source of infection in a herd. However, since the transmission and development of clinical signs may be delayed, identification of the primary source of infection may be difficult. Outbreaks of *M. bovis* associated diseases have also been reported in apparently closed herd, making other means of infection possible. *M. bovis* can be isolated from the upper respiratory tract (URT), the mammary gland, the conjunctiva and the urogenital tract of healthy and sick cattle [2]. The URT mucosa and the mammary gland are certainly the most important sites of persistence and shedding of *M. bovis* [2]. Persistence and duration of shedding is not known, but some cattle have been reported to shed intermittently *M. bovis* in mammary or nasal secretions for many months [2]. Factors associated with increase shedding are not known. However, clinical diseases and stressful events (such as transportation, co-mingling, entry into a feedlot, or thermic stress) have been associated with increase shedding [2].

**Environment and fomites.** *M. bovis* is well known to persist for a long period in the environment, particularly in cool and humid conditions. *M. bovis* has also been isolated from air and flies in barns containing sick calves [3, 4]. However, the exact role of these reservoirs in *M. bovis* epidemiology is not known. As an example, *M. bovis* has been reported to survive for a
long period in recycled sand bedding [5], but in an experimental study, transmission from contaminated sand bedding to naive calves did not occur [6].

1.2 Transmission

**Mastitis.** Mycoplasma mastitis are considered as a contagious mastitis, with udder-to-udder spread being the major means of transmission [7]. Milking equipment, milker’s hands or contaminated intramammary treatments have all been implicated in the spread of mycoplasma mastitis [7]. It is not known if internal dissemination to the mammary gland from other body sites plays an important role in the epidemiology of mycoplasma mastitis. Mechanical transfer from contaminated nasal or vaginal secretions and/or fomites to the mammary gland is also highly suspected [7, 8].

**Pneumonia.** Feeding calves with whole milk of infected cows in their mammary gland (with or without clinical signs of mastitis) is reported to be an important source of infection in young calves [9]. Direct transmission of *M. bovis* in nasal secretions could occur by aerosols and nose-to-nose contacts. Indirect, mechanical transmission could also be possible from contaminated feed, water, housing or other fomites such as nipples, buckets or tube feeders [2]. Co-infections with other pathogens (virus or bacteria) are frequently reported in cases of *M. bovis* pneumonia or otitis media [2]. However, the exact role of these co-infections in the development of clinical signs, severity of disease, or shedding is not known.

Key points:
- Introduction of asymptotically animal is an important risk factor for the development of mycoplasma associated diseases outbreak.
- Mycoplasma may be transmitted from one cow to another during the milking period.
- Feeding calves with contaminated milk is an important source of infection for calves.
- Direct and indirect contaminations from infected nasal secretions are likely to occur.

2. Identification of clinical cases and asymptotically infected animals

2.1 Clinical signs associated with mycoplasma diseases

As mentioned earlier, mycoplasmas are mainly the cause of respiratory, articular, mammary, genital, ocular and auricular infections. *Mycoplasma bovis* is a cause of mastitis, pneumonia, arthritis and otitis media and, in a less extent, of keratoconjunctivitis, meningitis, decubital abscesses, endocarditis and genital diseases.

**Mastitis.** Cows of all ages and at any stages of lactation may be affected, but cows in early lactation have been reported to be more severely affected [7]. Clinical cases of *M. bovis* and *M. bovigenitalium* mastitis were also reported in prepubertal heifers [10, 11]. Many intramammary mycoplasma infections are subclinical [7]. Clinical mycoplasmal mastitis can vary from mild to severe clinical mastitis. Clinical signs are inconstant and quite similar to mastitis caused by
other organisms. However, some aspects of the clinical presentation may help to differentiate
them: (1) Swollen and hard udder, but without heat or pain; (2) Poor response to treatment; (3)
More than one quarter affected; (4) Drastic decrease in milk production; (4) Signs of systemic
illness usually relatively mild; (5) Concomitant arthritis, synovitis and/or joint effusion within a
herd or within the same animal.

**Pneumonia.** Pneumonia due to *M. bovis* may occur in cattle of all ages. Clinical signs of
mycoplasmal respiratory affections are indistinguishable from other cause of pneumonia in
cattle. Chronicity, poor response to treatment, poor weight gain or weight loss are frequent [2].
Otitis media and/or arthritis may be observed within the affected animals or other animals
within the herd [2]. Even not specific, these features are highly suggestive of *M. bovis*
pneumonia.
A specific syndrome, chronic pneumonia and polyarthritis syndrome, is described in weaned
beef calves and in beef cattle [2].

**Otitis media/interna.** Clinical signs associated with *M. bovis* otitis media/interna are due to ear
pain (head shaking and scratching, or rubbing ears) and cranial nerves VII (ear droop, ptosis,
eyelid paresis with secondary epiphora and exposure keratitis) and VIII (head tilt) deficits. Purulent
aural discharge may be present if the tympanic membrane is ruptured. Clinical signs of
pneumonia are frequently observed in calves with otitis media. In advanced cases of otitis
interna, clinical signs of meningitis (depression, convulsion) may develop. Spontaneous
regurgitation, loss of pharyngeal tone and dysphagia have also been reported in calves with *M.
Bovis* associated otitis media/interna. Calves with chronic otitis media have poor weight gain
and become emaciated.

**Septic arthritis.** All age categories may be affected by *M. bovis* arthritis. Clinical signs are
typically those of septic arthritis in cattle. Distension of the tendon sheath and periarticular soft
tissue swelling is frequently observed in cases of *M. bovis* arthritis. More than one joint may be
affected. No or poor response to treatment is frequent.

2.2 What diagnostic tests are available for the detection infected animals?

**Culture.** *Mycoplasma* culture is fastidious and requires specific culture media and technical
skills. Some precautions should be taken when sending samples and the laboratory must be
specifically advised to perform culture for mycoplasmas. Growth may be usually detected after
48h but an incubation period of at least 7 to 10 days is required to consider a culture to be
negative [2]. The identification of the species is essential and used to be made by a serological
method or by growth inhibition, but these techniques have been replaced by PCR.

**PCR.** Polymerase Chain Reaction (PCR) techniques are more and more used for the diagnosis of
*M. bovis* infections. They have the advantages of being rapid, specific and now less expensive
than culture. PCR is also less or not affected by storage contrary to culture. PCR is expected to
be more sensitive than culture (ie to be able to detect mycoplasma at lower bacterial
concentration or CFU/ml). One inconvenient of PCR techniques is that they are usually specific
to *M. bovis*. However, non specie-specific mycoplasma PCR (ie PCR that detect all mycoplasma species without identification) are available. Since some cattle may be healthy carriers of mycoplasma, the interpretation of the detection of mycoplasma genes must be made with caution and always correlated with the clinical signs.

**Detection of antigens.** The diagnosis of mycoplasmosis may also be made by direct detection of *M. bovis* antigens from clinical materials using direct immunofluorescence, immunoelectrophoresis, immunoblotting or enzymatic capture (Waites, 1999). At necropsy, immunohistochemistry may be useful allowing detection of *M. bovis* antigens along with histological lesions.

**Serology.** Antibodies are usually detected by 6-10 days following experimental infection [12]. Antibodies titers can remain elevated for months and maternal antibodies results in high titers in calves. More, a poor correlation between antibody titers and infection or disease is reported [2]. Consequently, interpretation of antibody titers on an individual basis is difficult or even impossible. However, serology may be useful for the detection (or absence) of infection in a group of animals, before the introduction of animals in a herd, or for epidemiological survey [2].

2.3 *How can these tests be used to confirm a clinical case?*

**Mastitis.** Since mycoplasmas are shed intermittently in milk of infected mammary gland, confirmation of infection may be difficult. Sensitivity of a single milk culture for the detection of mycoplasma subclinical infection is reported to be low (<30%) [7, 13]. However, in clinical cases the number of bacteria shed is usually higher increasing the sensitivity of culture. It is not clear if individual or composite milk samples may increase or decrease the yield of positive culture [13]. On one hand, in most cases, multiple quarters are affected making composite milk sample potentially more “effective” samples. On the other hand, if only one quarter is affected, the infected milk will be diluted by the milk from the 3 other non infected quarters. Based on this low sensitivity and inconsistent shedding pattern, multiple milk samples should be taken before considering a cow not infected.

**Pneumonia.** Confirmation of *M. bovis* associated pneumonia on a live animal is also challenging. A poor correlation has been reported between the isolation of *M. bovis* either from the URT (i.e. nasal, nasopharyngeal or tonsil swabs) or the lower respiratory tract (i.e. transtracheal wash or BAL samples) and clinical disease [14-17]. However, transtracheal wash or BAL samples are probably the most appropriate samples for confirmation of *M. bovis* associated pneumonia [2].

**Septic arthritis/tenosynovitis.** Diagnosis of mycoplasma septic arthritis is based on culture or detection by PCR of mycoplasma in synovial fluid or membrane samples.

**Otitis media.** Since collection of fluid from the middle ear is not easy, confirmation of mycoplasma otitis media is difficult on live animals. Mycoplasmas can be culture or detected from purulent secretion present in the external ear secondary to tympanic membrane rupture.
2.4 What diagnostic tests can be recommended before the introduction of an animal and how to detect subclinical or healthy carriers?

Direct detection of mycoplasma. As mentioned earlier, sensitivity of a single milk culture for the detection of subclinical infected cow is low. It is not known how PCR could be more sensitive in the detection of such animals. Additionally, the absence of a low SCC could not be used as an indicator of absence of mycoplasma infection since infected cows have been reported to have normal SCC. However, keeping in mind this low sensitivity, when purchasing lactating cows or introducing heifers that have been raised off sites, milk samples should be submitted for mycoplasma culture or PCR. Animals should be isolated until results are obtained [18, 19]. Nasal swabs are commonly used in epidemiological studies and had been recommended to determine the M. bovis status of a group of calves [20]. However, the sensitivity of this test is not known. On an individual basis, the sensitivity of a URT swab for the detection of healthy carrier is not known. Based on these considerations, it is difficult to recommend any specific tests or procedures for the detection of healthy carriers. On the other hand, culture and PCR are 100% specific and indicate presence of the bacteria.

Serology. As mentioned earlier, serology may be used to select M. bovis-free groups of cattle. In Ireland, serology was utilized successfully to reintroduce free cattle following BSE depopulation. Nicholas et al. reported that “its use in maintaining M. Bovis-free herds by testing newly bought-in animals before introduction to the herd should also be seriously considered”[18]. Nevertheless, in some area (as in Québec) it could be difficult to find seronegative individual animal or group of animals. More, an animal could be seronegative secondary to the delay between infection and the increase in antibody titers. Finally, since there is a poor correlation between antibody titers and infection or disease, the infected status of an animal cannot be based on a single serology result.

Data from the herd of origin. Since no diagnostic test can determine with a high confident level the M. bovis status of an individual or a group of animals, it is important, when possible, to obtain some information on the herd of origin. The objective of these data (bulk tank milk culture or PCR history, mastitis clinical case history, calf health records) is to determine if M. bovis infections are reported in the herd of origin in order to evaluate the potential risk of introducing M. bovis infected cattle.
Key points:

- *M. Bovis* associated diseases can be suspected in cases of: (1) chronicity; (2) failure or poor response to the antibiotic treatment commonly used in first intent; (3) combination of clinical signs of pneumonia-arthritis, pneumonia-mastitis and mastitis-arthritis within a herd or within the same animal.
- Identification of *M. bovis* infected cattle is difficult because of the low sensitivity of the available tests and the intermittent shedding.
- Milk of newly introduced cows or heifers should be submitted for mycoplasma culture or PCR.

3. Biosecurity rules

3.1 *Isolation/segregation of infected animals*

Maintaining a closed herd is probably the best way to prevent *M. bovis* infection. When it is not possible, purchased animals should be tested (with the limitations mentioned earlier) and isolated (quarantine) [2]. Since transmission could occur via aerosols or fomites, strict isolation of sick animals is recommended, with no or at least limited contact with healthy animals. Therefore, once identified, sick animals should be moved to a separate hospital area. In our hospital, cattle with suspected or confirmed *M. bovis* associated diseases are isolated in individual boxes. The ventilation of these boxes is separated from the rest of the hospital. In herds with mycopasmal mastitis, strict milking parlor hygiene is essential to reduce udder-to-udder transmission of *M. bovis*. Culling all infected cows is still the strongly recommended course of action [2]. When not feasible immediately, strict segregation of infected and clean cows may be effectively employed to limit new infections [7]. Maunsell et al. recommended that cows were segregated at all times not just in the milking parlor [2].

3.2 *Proper management practices*

Proper sanitization of buckets, housing and other equipment between uses, wearing gloves, handling sick cattle last and strict milking parlor hygiene are recommended. As an example, in our hospital, treatments of infected cattle are all performed last; students or technicians wear gloves and clothes only dedicated to the infected animal. Dairy cows are milked last and with designated milking unit.

3.3 *Disinfection of materials and environment.*

Sanitized pen, hutches buckets and other equipments between uses is necessary. *M. bovis* is highly susceptible to heat and to most commonly used disinfectants. Peracetic acid or iodophor based disinfectants are reported to be highly effective [21]. Chlorine-, chlorhexidine-, acid- or iodine-based teat dips have all been reported to be effective [22].

4. Control and prevention [23, 24]
4.1 Reduction of the level of exposure to M. bovis

Control milk and colostrums quality by using milk replacer or pasteurized whole milk. M. bovis are inactivated by batch pasteurization of milk at 65.5°C for 30 min or 70°C for 3 min [25] or high-temperature (74-76°C) short-time (flash) pasteurization [26].

Reduce exposition to infected animals by identification and culling of infected animals, segregation of infected animals, separated hospital area, all-in-all-out practices, prompt treatment of infected animals.

Reduce exposition to fomites and aerosol by proper sanitize practices, wear gloves, look after youngest calves and clean animals first, all-in-all-out practices, adequate ventilation system.

Prevent introduction of M. bovis in the herd by screening and putting in quarantine purchased animals.

4.2 Reduction of potential risks factors

Make sure that management practices are adequate, including milking practices, alimentation (quality and quantity), ventilation, as well as lavage and disinfection procedures.

Make sure that protection against other major pathogens is adequate by adequate colostrums management, adequate vaccination protocols, as well as control and eradication of BVD infection.

4.3 Institution of specific protection against M. bovis

Use specific vaccines. Different commercial vaccines are available in USA, but not in Canada. Several companies also produce autogenous M. bovis vaccines. To date data are lacking on the efficacy of these vaccines and recent studies have reported limited to no efficacy of some of them [27].

Use methaphylaxis. Maunsell et al. stated that “metaphylactic use of antibiotics is probably justified when high levels of morbidity and mortality due to M. Bovis associated diseases [bovine respiratory disease] are being sustained or can be expected in high risk cattle, although M. Bovis-specific efficacy data and economic analyses are needed” [2].

References


22. Boddie, R.L., et al., *Germicidal activities of representatives of five different teat dip classes*


INTRODUCTION
Animal welfare is the state of an animal as it attempts to cope with its environment. Humans interact or affect the animals through their activities and husbandry practices. Veterinarians have been advocates for animal welfare for years, long before it was ever defined as “animal welfare.” Just a few years ago, many of us confused the term with being in line with radical animal rights groups. Today, one of the biggest areas of research interest in universities is related to animal welfare.

Over the last several years, there have been many video segments aired on the media outlets that have presented deficiencies in animal welfare on production animal facilities. The animal production industry has many animal production methods that are not particularly pleasing to the everyday consumer, i.e. gestation stalls for sows and veal crates for young calves to name a couple. Whenever one of these videos surface and consumers see how animals “are raised in the real world” (according to the anti-agriculture activists), there is a renewed effort calling for improved animal welfare on animal production farms. The dairy industry is not immune to these challenges and we need to work together and tell our story about how well dairy animals are raised. This has resulted in the creation of programs such as the National Dairy FARM Program: Farmers Assuring Responsible Management Animal Care Guidelines being adopted by many dairy processors. This has been done to assure consumers that their dairy producers use best management practices in the production of dairy products.

Despite the many advances in research affecting dairy calf management, calf rearing continues to be an area that challenges management. According to the last NAHMS Dairy report, death loss in pre-weaned and weaned calves has changed very little over the last twenty years. These surveys have shown that pre-weaned mortality has ranged from a low of 7.8% in the 2007 survey to a high of 10.8 in the 1996 survey. Weaned calf mortality has ranged from 1.8 – 2.8% in the last four NAHMS Dairy surveys (USDA, 2009). These numbers represent a huge loss to the dairy industry and challenges producers and their consultants to create strategies to improve the health of these animals. As a result, these changes should provide dairy farmers with more replacements allowing for genetics progress and potentially eliminating the need to purchase outside replacements.

FACILITIES
Facilities used to raise calves have made an about-face over the last twenty years. Individual hutches or pens for calves that are placed outdoors, providing increased bio-security for the individual animal, have become common place. These often have replaced poorly ventilated barns where calves were often group raised. Now, many dairy farms are again raising calves in buildings to improve the working conditions for the people that manage the calves. The advent
of computer feeders is also renewing interest in raising calves in groups. Group feeding strategies appear to stress calves less through the weaning period, however, they do increase the risk for increased morbidity and mortality over calves raised in individual hutches. No matter whether producers use hutches or group housing, raise the calves indoors or outdoors, there are a few basic tenants that must be in place in order for these operations to be successful.

Calves should be removed from their dams immediately following birth to reduce the potential for contracting an infectious agent in the calving pen. Calves should be transported in clean carts to a receiving area to allow the calf time to dry and acclimate to its new environment. The use of towels to help dry the calf and/or the use of a heat box assures that the calf does not become hypothermic. Drying of the head, ears, nose and mouth stimulates the calf to breath. As soon as a suckle reflex is present, the calf can be fed colostrum.

The calf should be placed in a hutch or pen that provides a minimum of 28-32 ft² of space per animal. If calves are group housed, they still appear to get a better start if they are housed individually for a few days. Solid plastic partitions between adjacent calves provide for the most protection against calf to calf spread of disease. However, they also require special considerations to properly ventilate. When ambient temperatures are below 58 °F, sufficient bedding should be provided to allow the calf to nest into the bedding and conserve heat. Additionally, consideration must be given to minimize heat stress during the warmer months of the year. Calves spend more than 75% of their time lying down between birth and weaning, so there is tremendous opportunity to expose calves to infectious agents via the bedding material. Deep bedded straw provides the best ability to nest and should be sufficiently deep to allow the calves legs to be hidden while lying down. Other bedding materials such as corn stalks, bean stubble, and wood shavings can be suitable bedding materials during warmer weather. Materials with smaller surface areas are more prone to retaining moisture and manure and, therefore, can give rise to higher bedding bacteria counts.

In both individual and group housed barns, positive pressure ventilation systems have been shown to be effective at reducing airborne bacteria levels, thus reducing the risk for respiratory disease. Building and pen design play a large role in determining the ventilation system requirements and design. Buildings with individual pens that have solid partitions between calves produce individual pen microenvironments that need to be individually ventilated. In group housed barns, natural or negative ventilation systems do not provide sufficient air movement at the level where the calf is housed to properly replenish fresh air. Positive pressure ventilation systems that provide at least 15 cubic feet per minute of additional air flow improve air quality at the calf level and reduce respiratory disease morbidity and mortality (Nordlund, 2007).

When calves are housed in group pens, limiting group size to 7-10 animals per group has a positive effect on reducing calf morbidity and mortality from respiratory disease. It is unclear whether this effect is due to improved social welfare of the calves or the effect of stocking density per unit of area of the barn (Gorden, 2010). During design and construction of calf
barns, sufficient space must be included to allow for small pens even during the periods with the highest calving rates. In order to achieve these recommendations, space should be sufficient to accommodate up to 140% of the weekly calving rate. Additionally, accommodations should be made to allow for a calf pen to be managed in an “all in-all out” format with sufficient time between groups to allow for proper cleaning and sanitation.

NUTRITION
Once the calf has developed a suckle reflex after birth, three to four quarts (10-12% of the calf’s body mass) of clean, good quality colostrum should be delivered to the calf within 4-6 hours. Jersey calves should be limited to three quarts of colostrum. While this recommendation has been common place for years in the literature, the evidence of successful implementation of this practice is lacking. According to the NAHMS Dairy 2007 survey which reported 19.2% of dairy calves experience failure of passive transfer (USDA, 2010). The desired goal of colostrum administration is to deliver enough colostrum to achieve a serum immunoglobulin 1 (IgG1) concentration of at least 1000 mg/dL. In order to achieve this goal, the calf must ingest 150-200 g of immunoglobulin through the delivery of good quality colostrum or a colostrum replacement product. Good quality colostrum has a minimum concentration of 50 g/L, thus the recommendation of 3-4 quarts. Colostrum quality is extremely variable and can be measured in the field but, this procedure has limited use. At a minimum, colostrum should be collected within 4-6 hours of birth as lactogenesis will dilute the colostrum and reduce the quality.

Bacterial contamination of colostrum interferes with intestinal absorption, so it is essential that excellent udder preparation practices are in place prior to collection of colostrum. In addition, the equipment used to collect, feed, and store the colostrum should be maintained in a sanitary manner in order to reduce bacterial contamination. Unused colostrum can be stored in a good quality refrigerator for a period of 3 days and can be frozen for a period of 1-2 years without sufficient loss of quality. Storage vessels should be thin walled and small in order to minimize insulation effects and allow for rapid cooling. Slow cooling allows for bacteria in the colostrum to incubate during storage. In addition, these types of storage vessels will make re-heating the colostrum more rapid.

Reduction of bacterial numbers can be achieved through pasteurization or acidification of colostrum with the use of products like formic acid. Colostrum should be pasteurized at 140 F for one hour. Bacterial contamination can be monitored by completing total bacteria counts (TBC) and coliform counts. As a goal, TBC of colostrum should be less than 1 million colony forming units (cfu)/mL and a coliform count of less than 10,000 cfu/mL (McGuirk, 2004).

Calves will typically not ingest 3-4 quarts of colostrum in one feeding on their own, so the use of an esophageal feeder is essential to assure adequate colostrum delivery. The use of a four quart esophageal feed will assure employee compliance of this recommendation. It should be noted that calves will typically not be very hungery at the next feeding but this should not deter farms from utilizing the recommendation of feeding 3-4 quarts of colostrum.

Over the past 10-15 years, we have gained tremendous intellect in the area of calf nutrition.
Traditional feeding programs limit fed calves in an attempt to get the calves eating starter early in their life and lead to early weaning. These feeding programs have not been overly successful in promoting good calf health. Experience has repeatedly shown that increasing the plane of nutrition of calves during the milk feeding period has reduced morbidity and mortality rates. When looking at nutritional needs of calves, it is important to understand the nutritional needs of the immune system. Studies have shown that nutrient consumption increases dramatically during bacterial challenge. Moderate infections have been shown to increase gluconeogenesis rate 150-200%. Sepsis in humans has been demonstrated to increase basal metabolic rate 25-55% while sepsis in laboratory rodents has resulted in loss of 40% of total body protein and a reduction in the rate of protein synthesis (Lochmiller, 2000). If calves do not have sufficient body reserves when challenged, they are at an increased risk to show clinical disease.

According to the Dairy Calf and Heifer Association’s Gold Standards, calves should double their birth weight through the milk feeding period. Therefore, a calf with an 85 lb. birth weight will need to gain 1.30 lbs./day assuming a 60 day milk feeding period. Utilizing the NRC software program, expected growth rates can be predicted for different feeding programs. In addition, estimates of the impact of cold stress can be demonstrated. The program does not do a good job of predicting growth during periods of heat stress. As an example, if a producer feeds one pound of a 20% protein:20% fat milk replacer to an 85 lb. calf during thermoneutral periods along with 0.25 lbs./day of starter, calves will only gain 0.67 lbs./day with protein being the growth limiting nutrient. If the ambient temperature drops to 32 F, the expected growth rate drops to 0.12 lbs./day with energy now becoming the growth limiting nutrient. By comparison, feeding 4 quarts of whole milk per day would only result in a gain of 0.57 lbs./day at 32 F and just over one pound of gain per day during thermoneutral periods.

Increasing caloric intakes in calves during the milk feeding period is best accomplished by adding an additional feeding per day. In many operations, the extra labor requirement to provide an extra feeding is not available so extra calories are delivered by increasing the volume of milk per feeding and/or increasing the concentration of milk replacer powder per volume of water, up to a maximum of 18% solids. Feeding consistency is important for maintenance of calf health. In order to minimize digestive disorders, changes in feeding should be made slowly with a minimum of 3-5 days between changes.

Assuring that plenty of fresh water is always available is essential for minimizing digestive upsets and for the consumption of calf starter. Calves will not eat starter if they do not have water available. Starter consumption, not hay, is the driving force behind the development of the rumen. Weaning of calves should occur once calves are consuming 2-2.5 lbs. of starter per day for at least three consecutive days. Depending upon the plane of nutrition offered through milk feeding, the amount of milk offered per day may need to be decreased in order to encourage the consumption of starter.

**CASTRATION and DEHORNING**
Castration of dairy calves should be completed at as young an age as practical. There are many techniques available but, surgical castration is the recommended method in young calves. The
use of elastrator bands is an inexpensive method of castration but, has been associated with chronic pain and is not recommended. The Animal Care Manual for the National Dairy Farm Program recommends that a licensed veterinarian perform castration on calves four months of age and older with the use of local anesthetic. Dehorning should also be done as early as possible. The use of local anesthetic is becoming more common in the dairy industry. Research workers at ISU recently completed a trial in which they evaluated a pressure algometer as an objective measure of pain tolerance following cautery disbudding. Groups of calves were assigned to the treatment group which was disbudded following lidocaine administration or the control group that were sham disbudded following lidocaine administration. The project demonstrated that calves experienced pain for at least 59 hours after disbudding and that lidocaine eliminated the calf’s perception of pain. However, this pain relief was only effective for one hour after disbudding (Van Donselaar, 2009). This project clearly shows that even though lidocaine prevents pain at the time of the surgical procedure, the effect is short-lived and far short of the time frame for which pain was present. More research is needed to determine whether the use of certain analgesics would improve calf welfare during the time following surgical procedures.

CONCLUSION
Dairy producers and their veterinarians are involved in animal welfare on a daily basis. As veterinarians trained in production animal medicine, we are uniquely qualified to guide animal owners about best management practices that improve animal welfare. The use of these practices in calf rearing operations will result in larger pools of calves available as replacements. This will allow herds to make genetic progress and improve bio-security by not having to purchase heifers with unknown backgrounds. Programs like the National Dairy FARM Program help the dairy industry provide quality assurance to consumers that animals are being raised with the use of best management practices. We must assure that this effort is not just cursory window dressing by demonstrating success stories to the consumer.

REFERENCES


THE ROLE OF IMMUNE SUPPRESSION IN THE BATTLE FOR QUALITY MILK
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INTRODUCTION

The adult dairy cow is in a constant battle with infectious agents trying to invade her body and establish disease. The mammary gland is especially vulnerable to infection because of its location in relationship to bedding materials that are contaminated with fecal material. Pol and Ruegg (2007) documented a clinical mastitis incidence in Wisconsin dairy cattle of 40% per year. Based on somatic cell count (SCC) analysis and culture data, we know clinical mastitis represents only 25-35% of all intramammary (IMM) infections. Given that SCC analysis is typically done on a monthly basis, it would seem likely that we do not detect many new IMM infections. Therefore, it would seem logical to assume that nearly every cow in a milking herd experiences at least one or more IMM infections per lactation, many for a very short duration. Even though mastitis is one of the most common diseases on dairy farms, these numbers would be much higher would it not be for a strong immune system to adequately deal with mild infections.

It is not uncommon to see dairy farms dealing with poor cow health to also battle with high SCC’s. This is most likely related to stress on the immune system which impairs its functionality. Additionally, antibacterial agents alone cannot control an IMM infection without the assistance of a functional immune system. Depending on the stage of lactation and presence of IMM infections, the immune system has varying levels of functionality. This paper will provide a brief review on immune protection of the mammary gland, issues relating to immune suppression and how this suppression impacts udder health.

IMMUNE PROTECTION OF THE MAMMARY GLAND

The primary means of protection for the mammary gland are the physical barriers provided by the skin that covers the gland and the muscular sphincters that close the teat canal between milkings. In addition, the cells lining the teat duct produce keratin that traps bacteria. This keratin has natural antibacterial properties. During the milking process, keratin sloughs off and is flushed out of the gland. An additional physical barrier is provided by the process of milking, which serves a protective function in that it flushes the contents out of the mammary gland on a regular basis.

If a foreign agent gets past the physical barriers, a reactive protective mechanism must be initiated. The innate immune system becomes the most important defense system of the mammary gland. The innate system serves a role to detect and kill an array of pathogens. A decrease in the function of even one component of the innate immune system can have detrimental effects on the cow’s immune response. Polymorphonuclear cells (PMN) including neutrophils, basophils, and eosinophils, play a key role in the innate immune system by
detecting and containing infectious agents. While basophils and eosinophils have important functions, the neutrophil becomes the most important PMN for the mammary gland in dealing with IMM bacterial infections. Neutrophils make up only 20-30% of the leukocytes in the blood pool in ruminant animals which is a much smaller percentage than other animal species, such as carnivores. An 800 kg cow has approximately $1.4 \times 10^{11}$ neutrophils circulating in the blood with an average half life of approximately six hours. This means that a cow is replacing one-half of its neutrophils every six hours from bone marrow stores which require a large amount of dietary energy and protein.

Neutrophils are attracted into the mammary gland when bacterial invasion occurs by a process called chemotaxis. Chemotaxis is initiated by local changes in the mammary tissues due to bacterial invasion or tissue damage caused by the bacteria. This tissue damage serves to initiate the complement system which produces substances such as C5a which act as chemoattractants for neutrophils. Chemotaxis initiates changes to the endothelial lining of local blood vessels causing neutrophils to selectively adhere to the vessel wall and slow neutrophil passage through the vessel. The neutrophil then migrates through the vessel wall and into the infected area. While neutrophils have the ability to be stand alone killers, they work much more effectively with the assistance of the acquired immune system. IgG2 and IgM, produced by lymphoid cells, are the primary antibodies in the mammary gland to assist the neutrophil by opsonizing bacteria to promote bacterial uptake by the neutrophil. Once ingested, the neutrophil kills bacterium through generation of reactive oxygen species (ROS) and release of lytic enzymes and antimicrobial peptides from intracellular granules.

**PERIPARTURIENT IMMUNE SUPPRESSION**

Cows in the periparturient period have the most profound amount of immune suppression experienced throughout lactation. Not coincidentally, the majority of metabolic diseases, such as milk fever, retained placenta, ketosis and displaced abomasums, occur within the first two to four weeks after calving. In a study looking at the correlation between metabolic disease and mastitis incidence, milk fever increased the risk of development of mastitis by 8 times (Curtis et al, 1983). Other studies have demonstrated an increase in mastitis risk in cows that have experienced ketosis, retained placenta, twinning, dystocia, and lameness before the first breeding (Oltenacu and Ekesbo, 1994; Emanuelson et al, 1994; Peeler et al, 1994).

It is well understood that the process of parturition is a highly stressful process. In addition to this stress, the dairy cow must initiate colostrogenesis and lactogenesis. Kehrl et al (1989 a, b) demonstrated that neutrophil and lymphocyte function is depressed in animals during the periparturient period. This depression is especially prevalent in the dairy cow. A more recent trial utilized proteomic techniques to compare pre-partum versus post-partum neutrophil function (Lippolis et al, 2006). In this study, there was a change in protein expression for more than 300 proteins between the two groups.

USDA researchers (Kimura et al, 1999) undertook a trial to determine whether periparturient immune suppression was related to parturition or the initiation of lactation. The group utilized
mastectomized cows versus a control group of intact cows. All intact cows developed milk fever at parturition and experienced a significant increase in non-esterified fatty acid (NEFA) concentration for the first ten days of lactation. They determined that mastectomized, post-partum cows underwent similar decreases in neutrophil myeloperoxidase activity at parturition compared to intact controls. The difference between the two groups was that mastectomized cows returned to normal neutrophil function more quickly, thus proving that both parturition and colostrogenesis/lactogenesis have independent effects on immune suppression. When looking at lymphocyte populations though, they found that mastectomized cows had minimal decrease in lymphocyte function while intact cows suffered significant depression in lymphocyte function.

Clinical and sub-clinical hypocalcemia commonly occurs at parturition. Goff et al (1996) demonstrated that cows can remain sub-clinically hypocalcemic throughout the first week of lactation. Depression of contraction rate and strength of smooth muscle is directly proportional to serum calcium concentration. Decreased function of smooth muscle directly impacts IMM infection status as the teat sphincter will be less completely closed. In addition, hypocalcemic cows spend more time lying down thus increasing teat end exposure to bacterial invasion. Both of these factors increase the risk for the development of new IMM infections.

Calcium metabolism in the periparturient period has a direct effect on immune cell function. Kimura, et al (2006) determined that intracellular calcium plays a vital role in peripheral blood mononuclear cell (PBMC) activation in that lower intracellular calcium level slows PMBC activation. In cows, intracellular calcium in PMBC decreases before parturition and the development of hypocalcemia. This systemic calcium stress indicates that the cow is experiencing PMBC immune suppression before demonstrating symptoms of sub-clinical or clinical hypocalcemia.

Energy metabolism is equally important in the development of immune suppression around parturition. Cows are in negative energy balance for 45-75 days postpartum. They are also in negative protein balance for a similar period of time. The development of an immune response against an infection is not without expense to energy metabolism. While no work has been done in the bovine, extrapolation of human data from individuals suffering from severe infections indicates that maintenance energy demands increased by 40% above normal. This extra expenditure of energy lasted three weeks beyond the onset of the illness. Incorporating maintenance energy needs of the cow into this situation, the cow would need to increase her maintenance energy needs nearly 4 Mcal/day. If a cow was consuming a lactating diet containing 1.65 Mcal of net energy per kilogram (kg), she would need to consume an additional 2.4 kg of dry matter per day (Goff and Kimura, 2002). Thus, if a cow is already in negative energy and protein balance at the time she acquired an infection, she would be expected to drive herself in a larger negative energy and protein deficit or produce a less effective immune response with the energy and protein she has available.

Several studies have looked at the relationship between negative energy balance and immune function. Dutch researchers (Suriyasathaporn et al, 2000) found that negative energy balance
and ketosis in early lactation causes polymorphonuclear cells (PMN’s) to react slower to new IMM infections. In cows that have slower reacting PMN’s to new IMM’s, the severity of the mastitis is worse. The reasons for the slower reaction are multi-factorial including:

- Inhibited phagocytosis and killing due to lower production of ROS.
- Reduced ability to attract new PMN’s to the area via chemotaxis.
- Reduced capacity to migrate to the infected gland independent of the reduced chemotaxis listed in step 2.

While Suriyasathaporn’s group suggested that phagocytosis was actually decreased, several other research groups have found neutrophil phagocytosis to be increased, but the production of ROS is decreased leading to poorer neutrophil function. This phenomenon is not fully understood but may be explained by a selective depression of the activation pathway of the oxidative burst or that energy is not available for the initiation of the oxidative burst in the presence of large number of pathogens (Burvenich, Bannerman et al. 2007). Nonetheless, no matter what the reason for the slower response by neutrophils, the severity of IMM infections during this period is worse.

Hoeben et al (1999) found that beta-hydroxybutyrate (BHBA) and acetoacetate, in similar concentrations as found after parturition, inhibited in vitro hematopoietic cells. They also demonstrated a negative relationship between BHBA and PMN chemiluminescence.

Kremer et al (1993) undertook a study looking at the relationship between in vitro neutrophil chemotaxis and the severity of induced E coli mastitis between ketotic and non-ketotic cows. In non-ketotic cows, mastitis cases ranged from mild to severe with the severity of the infection negatively correlated with the degree of pre-calving neutrophil chemotaxis. The experimental infection in the ketotic group resulted in all cows developing severe mastitis regardless of the state of pre-calving neutrophil chemotaxis.

**IMMUNE SUPPRESSION IN OTHER STAGES OF LACTATION**

The length of the negative energy balance in early lactation appears to have an effect on immune suppression in that cows that have minimal periods of negative energy balance have less profound effects on immune suppression as compared to cows that undergo longer periods of negative energy balance. However, in a separate trial, Perkins et al (2002) found that feed restriction in mid-lactation, regardless of length, did not have an effect on the immune response when cows were subjected to IMM endotoxin injections. The lack of a demonstrated effect on immune response in mid-lactation most likely was related to the stage of lactation in which the cows were exposed to the treatment and underscores the multi-factorial nature of immune suppression.

As cows age, there appears to be a decrease in immune function as evidenced by older animals being more severely affected by infectious agents. This reduced function is related to impaired function of neutrophils along with a reduced production of ROS in older animals (Burvenich, Bannerman et al. 2007).
In a recent review of immune function and its relationship to *E. coli* mastitis (Burvenich, Bannerman et al. 2007), the reviewers detailed the results of several papers looking at genetic control of immune function. In their review, the authors state that high milk yield will not have a genetically related negative impact as related to immune function compared to lower producing cows. However, it is well known that as milk production has increased over time, so has the incidence of clinical mastitis. This may be associated with the degree of negative energy and protein balance high producing cows experience and changes in teat anatomy of high producing cows rather than genetic changes to the innate immune system.

**SUMMARY**

Immune suppression relating to poor performance of quality milk programs is often overlooked by dairymen and veterinarians. The most apparent component of IMM immune suppression is related to activities that occur during the periparturient period. This immune suppression is multi-factorial, thus reduction of immune suppression must have multi-pronged approach. While many research groups are looking at ways to alter the immune response of the cow to IMM infections, those breakthroughs may be years in coming. The most practical approaches for today’s dairymen include a combination of improving and maintaining cow hygiene, managing diets around the periparturient period to minimize dry matter intake and hypocalcemia, and implementing procedures to minimize cow stress.

Veterinary practitioners can play an important role in helping dairy farmers monitor and improve milk quality. In situations where immune suppression is evident, it may be prudent to concentrate the resources of farm personnel at improving immune function of the herd rather than concentrating on controlling the mastitis problem. In addition, implementing monitoring programs, as simple as monitoring disease incidence and feed consumption during key periods of lactation, will help identify situations where excessive immune suppression is occurring.

**References:**


CVMA TOP 10 LIST-DAIRY DISORDERS & OTHER PERTINENT TOPICS: USING DIAGNOSTIC INFORMATION IN THE PRACTICE OF EVIDENCE BASED MEDICINE

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INTRODUCTION
Dairy practitioners and our clients have unprecedented access to data through the advent of the internet. List serves like aabp-l provide bovine practitioners with endless facts and opinions about today’s hot topic. The problem is how to keep up with all of the new information that is coming at us and most importantly, how to validate the information that we receive in order to use it in our daily practice of veterinary medicine. Our quest should be to manage information in a way that it can be useful to us for answering the daily questions that arise. Recent questions that have emerged in our practice include:

- What is the efficacy and risk of placing systemic antibiotics in the peritoneal cavity during routine DA surgeries?
- What is the efficacy and risk of using penicillin in a mixture with a steroid administered in a sub-conjunctival injection for the treatment of pink eye in cattle?
- What is the diagnostic accuracy of the Pathoproof™ Mastitis PCR test in milk quality programs, especially when using samples not obtained via aseptic technique?

Veterinarians should strive to be “lifelong learners” and must resist sticking our head in the sand when it comes to harnessing access to new data. Utilizing the skills of Evidence Based Medicine will help us successfully incorporate that data into the practice routine. The difficulty in this task is learning the skills of critically evaluating and efficiently incorporating new information into practice.

Diagnostic testing is a daily component of practice for dairy veterinarians. Diagnostic testing can be as simple as the rectal palpation that some practitioners do hundreds of times/day to as complex as a genotypic test that looks to see if a particular gene is present in bacterium isolated from a diagnostic submission.

These talks will concentrate on ten current topics affecting dairy practitioners and how to use the evidence that we have available to us to apply new findings to daily practice. Since these topics change rapidly, it is impossible to create a proceedings paper to properly address the topic. Therefore, this paper will focus on applying Evidence Based Medicine techniques to the evaluation and application of diagnostic tests in veterinary practice.

WHAT IS EVIDENCE BASED MEDICINE
Evidence Based Medicine was introduced into the human medical community when a former wartime prison doctor recognized that the medical community needed to substantiate medical decisions based on scientific evidence in the early 1970’s. This process of forging new information and emerging technology into medical practice was formally named Evidence Based Medicine by McMaster’s Medical School in Canada in the 1980’s. The process has evolved into the publication of thousands of manuscripts about the topic in the human medical field. The topic of Evidence Based Veterinary Medicine (EBVM) first appeared in the literature in the late 1990’s.

In order to apply EBVM in practice, practitioners must have the ability to:\(^2,^3,:\)

1. Turn a need for clinically relevant information into a question. This question can pertain to evaluating the performance of a diagnostic test for the animal or population in question, evaluating the efficacy of a preventative or therapeutic intervention, predicting the outcome of a disease, or predicting the cost or the risk associated with an intervention.
2. Perform a search for the best available information to answer the question with an emphasis on efficiency.
3. Critically appraise the information for its validity and usefulness to answer the question at hand.
4. Utilize the information to form clinical judgments and implement actions.
5. Perform an outcomes based evaluation of the success and the execution of the first four steps and seek information to improve the clinical outcomes and data evaluation process.

EBVM challenges us to place less weight on the subjective opinions that have developed from previous clinical experience, as these are often based on poor memory and/or record keeping and instead look to more rigid forms of evidence to guide us in the practice of veterinary medicine. The types of evidence that should be used include (listed from strongest to weakest evidence):\(^2:\)

- systematic reviews,
- meta-analysis,
- blinded random controlled trials,
- cohort studies,
- case control studies,
- case series,
- single case reports,
- editorials, opinions, comparative animal research and
- in vitro studies.

Finding a sufficient number of relevant information sources in the veterinary literature is more difficult due to the limited number of high quality references as compared to the human medical field\(^2,^4,^5,:\). The veterinary research community is attempting to improve the quality of information sources specifically for the veterinary clinical community through the development of the REFLECT (Reporitng guidELines For randomized controLled trials for livEstoCk and food safeTy) statement. The REFLECT statement resulted from several modifications and additions of the CONSORT(Consolidated Standards of Reporting Trials) statement to take into account the unique aspects of reporting livestock trials\(^6,^7;\) and is meant to be an evidence based minimum set of standards for trials reporting production, health, and food-safety outcomes. (For more information, consult the REFLECT website: www.reflect-statement.org.

**APPLYING EVIDENCE MEDICINE TO DIAGNOSTIC TESTS**

A portion of this presentation associated with this paper will summarize the results from
several diagnostic laboratories that provide diagnostic service to geographic areas with higher populations of dairy cattle. The purpose of the presentation is to provide attendees with trends being seen from diagnostic submissions for common diseases associated with dairy cattle and to use that evidence in their practice. None of the actual data summaries will be presented in the remainder of this paper due to space and time constraints and the need to address the critical evaluation of diagnostic test results.

Diagnostic tests range in complexity and cost, but the characteristics of critical appraisal will apply whether the test is a rectal palpation for pregnancy diagnosis or a multiplex PCR test. Before spending much time on the complexities of critical appraisal, a veterinarian must consider the potential bias introduced into the diagnostic process. One can do this by asking the following questions:

- what is the question I am trying to answer,
- why am I performing diagnostic tests
- what samples should I collect and
- which animals should be tested?

Diagnostic testing should be done to help pair down a pre-determined list of differentials, not to establish the differential list. Practitioners must understand that bias can be introduced into the situation through the selection of the wrong animals to sample, failing to sample the correct area of affected tissues or incomplete sampling of the affected tissues, and failing to preserve tissues correctly. Unlike our swine and poultry counterparts, bovine veterinarians have a tendency to submit samples from the entirely wrong population of animals, i.e. the one that died after some prolonged (often unknown in exact duration) illness. Poultry and swine veterinarians will sacrifice acutely affected animals in order to establish an unbiased diagnosis. Likewise, submitting a sample from an animal housed in environment where infectious agents are ubiquitous (i.e. a fecal sample from a calf with diarrhea) will not provide a lot of long term diagnostic value unless also concentrating on why the affected population is succumbing to the agent while other populations (animals from different cohorts, different farms, etc) are not being affected. Therefore, concentrating on management aspects of calf rearing would serve as a more prudent use of diagnostic dollars as compared to simply identifying the agent.

**TERMINOLOGY OF DIAGNOSTIC TESTS**

Practitioners utilize diagnostic tests in one of two ways, either as screening test or a confirmatory diagnostic test. A screening test can be used to screen a herd for the presence of infected animals. An ideal screening test should identify as many infected animals as possible (minimal false negatives). A diagnostic test could be used to confirm the presence of disease based on a practitioner’s physical exam findings or to confirm the presence of an infected animal as a follow up to a screening test. An ideal diagnostic test should correctly identify as many non-infected animals as possible (minimal false positives).

In order to determine whether a particular test would be a good screening and/or diagnostic test, we must understand how accurately a test determines the status (diseased, infected, exposed or not affected) of an animal. There are several epidemiological terms used to describe how a diagnostic test performs in a population of infected, exposed, or diseased animals. Diagnostic test accuracy is determined by a comparison of the diagnostic test in question against a **gold standard**. The ideal gold standard test is one that can perfectly
discriminate between diseased and normal. Of course, there are very few examples of a perfect test in the world. Therefore, descriptive terminology of new diagnostic tests is only as reliable as the perfectness of the gold standard.

**Test sensitivity** is the ability of a test to identify a diseased animal in a population of diseased animals (based on the gold standard). If a new diagnostic test identifies 50 test positive (T+) out of 100 (D+) diseased animals (based on the gold standard), the sensitivity of the test is 50%.

**Test specificity** is the ability of a test to identify a non-diseased animal in a population of non-diseased animals (based on the gold standard). If a new diagnostic test identifies 450 test negative (T-) out of 500 (D-) diseased animals (based on the gold standard), the specificity of the test is 90%.

When evaluating the performance of a diagnostic test, it is usually helpful to compare the results of the gold standard and the test in question in a 2x2 table. Table 1 is an example of sensitivity and specificity values for a commercially available Johnes ELISA.

**Table 1-Sensitivity and specificity calculations for a commercially available Johnes ELISA kit**

<table>
<thead>
<tr>
<th>True status (D)</th>
<th>Positive (D+)</th>
<th>Negative (D-)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive (T+)</td>
<td>127</td>
<td>5</td>
</tr>
<tr>
<td>Negative (T-)</td>
<td>120</td>
<td>712</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>247</td>
<td>717</td>
</tr>
</tbody>
</table>

Sensitivity of test in question (T+/D+) = 127/247 = 51.4%

Specificity of test in question (T-/D-) = 712/717 = 99.3%

The **false positive rate** is the proportion of non-diseased animals that produce a positive result for the test in question. The equation to determine the false positive rate for a test population is (1-sensitivity). In the example from Table 1, the false positive rate is 1-.993 = .007 (0.7%).

The **false negative rate** is the proportion of diseased animals that produce a negative result for the test in question. The equation to determine the false negative rate for a test population is (1-sensitivity). In the example from Table 1, the false negative rate is 1-.514 = .486 (48.6%).

Sensitivity and specificity by themselves tell us very little about how well a test performs. In life, there are very few tests that have 100% sensitivity and 100% specificity. When evaluating the performance of a test, sensitivity and specificity must be evaluated together as considering either separately does not provide the complete picture about the test. In addition, it is important to remember that if you desire to improve sensitivity or specificity, it will come at a cost of reducing the other.

Test results can be expressed in two forms, categorical or continuous. Categorical results produce a yes or no answer (positive or negative, pregnant or open). Continuous results produce a result along a quantitative scale at which a cut-point is utilized to determine if the outcome is abnormal. Sensitivity and specificity values are affected by where the arbitrary cut-point is established. While practitioners have very little to do with the establishment of the cut-point, it is extremely valuable for us to understand what shifting the cut-point up or down will have on the identification of diseased animals. Looking again at the example in Table 1, if it is the desire of a practitioner to identify as many potentially positive animals as possible (have a high sensitivity), the quantitative cut-point should be shifted downward in order to produce more positive animals. However, this will come at a cost of decreasing the specificity of the
test. This will result in more false positives. However, if the desire of the practitioner is to have a test that minimizes the chance that an animal will be culled from a herd if she is MAP positive, then the current cut-point is sufficient as the false positive rate is very low. Additional terms that become useful in determining the accuracy of a test are the positive and negative predictive values. In a comparison of a new diagnostic test versus a gold standard, the true disease status of an animal is known based on the outcomes of the gold standard. However, in real life a practitioner does not know the actual disease status when evaluating a herd. The positive and negative predictive values provide an estimate of the proportion of animals that are classified correctly. The positive predictive value (PPV) for a test is the proportion of the animals with a positive test that actually have disease. The following equation is used to determine the PPV:

\[ \text{PPV} = \frac{\text{True positive animals}}{(\text{true positives} + \text{false positives})} \]

Looking at the example from Table 1, the test in question produced 127 true positives and 5 false positives.

\[ \text{PPV} = \frac{127}{(127 + 5)} = 96.2\% \]

The negative predictive value (NPV) for a test is the proportion of the animals with a negative test that are truly not affected by the disease. The following equation is used to determine the NPV:

\[ \text{NPV} = \frac{\text{True negative animals}}{(\text{true negatives} + \text{false negatives})} \]

Again looking at the example from Table 1, the test in question produced 712 true negatives and 120 false negatives.

\[ \text{NPV} = \frac{712}{(712 + 120)} = 85.6\% \]

Based on these results, approximately 4% of the animals that tested positive are truly negative while approximately 15% the animals that tested negative are truly positive.

The apparent prevalence is the predicted prevalence of the herd based on the new diagnostic test. From the example from Table 1, the apparent prevalence of the herd in question is 13.7% (132 test positives/964 animals in the test population). The true prevalence is the determined by taking the number of diseased identified by the gold standard test in the test population and dividing by the herd size. In the example herd in Table 1, the true prevalence of the herd is 25.6% (247 true positive animals/964 animals in the test population).

As can be seen from the above definitions, positive and negative predictive values, as well as apparent prevalence are based on the specificity and sensitivity of the test in question. However, they are also affected by the true prevalence in a herd. This example is demonstrated in Tables 2 and 3 below comparing two hypothetical 1000 cow herds, one with a true prevalence of 30% (Table 2) and the second with a low prevalence, 5% (Table 3). In both scenarios, the test sensitivity and specificity are identical.

In the scenario in Table 2, the apparent prevalence is 15.9% compared to a true prevalence of 30%. In the low prevalence herd, the apparent prevalence is closer to the true prevalence (3.3 vs. 5%, respectively). In comparing the positive and negative predictive values, the higher prevalence produces a pool of positive animals that are nearly all truly positive while 17% of the test negative animals are false negatives. In the lower prevalence herd, while the test more closely predicted the true prevalence and the true negative status of the test negative animals, 21% of the animals that tested positive are actually not diseased. This demonstrates the point
that a test is more reliable in a herd with a higher prevalence. In order to see how prevalence affects PPV and NPV, see Figure 1.
The Likelihood Ratio is the likelihood that a given result would be expected in a diseased animal versus the likelihood that the same result would be expected in a non-diseased animal. The **positive Likelihood Ratio (LR+)** is the likelihood that a positive result will be found in a diseased animal versus a non-diseased animal. The equation to determine the LR+=Sensitivity/(1-Specificity). From the scenario from Table 1, (sensitivity=51.4% and specificity=99.3%) the LR+=.514/(1-.993)=73.4:1.
The **negative Likelihood Ratio (LR-)** is the likelihood that a negative result will be found in a non-diseased animal versus a diseased animal. The equation to determine the LR- = (1 – Sensitivity)/Specificity. From the scenario from Table 1, (sensitivity=51.4% and specificity=99.3%) the LR- = (1 - .514)/0.993 = 0.49:1.

Table 2-Positive and negative predictive values for a hypothetical 1000 cow herd with a 30% true prevalence.

<table>
<thead>
<tr>
<th>True prevalence</th>
<th>Test sensitivity</th>
<th>Test specificity</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.3</td>
<td>0.514</td>
<td>0.993</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Test status</th>
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<th>Negative</th>
<th>Predictive value</th>
</tr>
</thead>
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<tr>
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Table 3-Positive and negative predictive values for a hypothetical 1000 cow herd with a 5% true prevalence.

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<tr>
<th>True prevalence</th>
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<th>Test specificity</th>
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<tr>
<td>0.05</td>
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<table>
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SUMMARY
Most veterinarians already practice forms of Evidence Based Veterinary Medicine on a regular basis. The issue that most of us get into over time is that we get comfortable with what we’ve done in the past and become complacent about completing objective outcomes based assessments. For individuals who have limited access to multiple journals electronically, getting access to new unbiased evidence to apply to evidence based medicine is difficult and cumbersome. As communication technology continues to improve, access to multiple electronic evidence sources will become easier. Likewise, as diagnostic technology continues to advance, in areas such as molecular diagnostics for example, numerous new tests are being introduced based on technology that many veterinarians do not fully comprehend. These advances in technology will require veterinarians to search for the evidence to support the use of these newer, more expensive tests in practice. Unfortunately, this type of well reported, objective data has not been as readily available in the veterinary literature as compared to human medical literature. As reporting guidelines such as the REFLECT statement are adopted by manuscript authors, this type of literature should improve and become more abundant.

Now the question is: Are you ready to resist the urge to stick your head in the sand and strive to be a lifelong learner by applying Evidence Based Veterinary Medicine techniques to your daily practice?
REFERENCES

Introduction
Teat injuries are frequent in dairy cattle. In many cases, they result in premature culling of the animal. Veterinarians are frequently asked to evaluate malfunctioning teat. With palpation alone, it can be difficult to diagnosis precisely the origin and the severity of the lesion. Without a precise diagnosis, the treatment plan is difficult to elaborate and frequently fails to re-establish the normal function of the teat. This session will focus on diagnosis of teat injuries. Older and newer diagnostic tools will be reviewed. Treatment option will be given but will not be elaborated on.

Specific anatomy
From the lactocyte to the external sphincter, there are 2 areas where the milk has to go through a narrowing: the annular ring and the rosette of Furstenberg/streak canal (teat’s distal end). Most of the problems are encountered in those areas. The annular ring is a large vascular plexus at the base of the teat where the vascularisation of the teat starts and ends. Internal lesions at the annular ring or enlargement of the vascular plexus impede the milk outflow from the gland cistern to the teat cistern.

The rosette of Furstenberg is the junction of the teat mucosa at the distal end of the teat cistern. It is often referred to as the internal sphincter of the teat. This structure marks the transition between the teat cistern and the streak canal. Lesions in this area act as a one way valve decreasing the milk flow during milking.

Lesions at the annular ring
The lesions at the annular ring will be divided in 2 categories: internal lesions and external lesions

A) Internal lesions
These lesions are generally secondary to mastitis. In the younger animal, the lesion is often extensive and can involve the entire length of the teat and the gland cistern. Many heifers are infected with mastitis at calving. Depending on the agent involved, an increase in the somatic cell count (SCC) can be the only clinical signs. On other occasions, the result of the mastitis is dramatic and the heifer has a partially or a totally blind quarter.

After calving, the owner complains about a quarter that is not letting its milk down. He is convinced that the animal should produce milk since the quarter is enlarged. Most of the time, this enlargement is caused by edema. In this case, he wants his veterinarian to determine the extent of the damage to the teat and hopefully re-establish the normal milk flow. The first thing that has to be determined is the productivity of the quarter. If the quarter is producing milk, the teat can be evaluated. By only palpating the structures, it is difficult to evaluate the milk
production of the quarter and the integrity of the teat. Those structures might be firmer than usual. No milk or only a few drops will be obtained by hand milking. To obtain a diagnosis, an ultrasound evaluation of the teat and quarter should be performed. A few descriptive papers of the normal and abnormal anatomy of the teat have been published in the past. The ideal probe to use is a linear 7.5 mHz transrectal probe. First the probe is placed on the affected quarter. Normally, the gland cistern should be filled with milk. If no milk is detected and the animal is only a few days post-calving, it is right to assume that the quarter has been compromised. The evaluation can end at this point since no treatment options are available to treat a fibrotic quarter. If milk is present in the teat cistern, the teat is evaluated longitudinally and transversally. The evaluator is trying to find an intact portion of the teat cistern distally. The quarter is massaged to stimulate the milk to go down which should fill the teat cistern if present. If a teat cistern is identified, it is possible to approach the lesion through a thelotomy to create an artificial teat cistern with the help of an implant.

If no milk is detected distally, a small canula is introduced through the streak canal and contrast product can be injected into the cistern. 50% sodium diatrizone solution (Hypaque) can be safely infused into the teat and gland cistern after appropriate cleaning of the teat’s end. A lateral radiograph is then taken. If a cistern is detected, the lesion can be approached surgically. If no cistern is detected, the lesion can still be approached surgically. However, the prognosis for regaining a functional teat decreases dramatically.

When contrast radiography was compared to other imaging modalities (ultrasound and thelscopy), the sensitivity of the technique was lower for the detection of proximal obstructions. However, in regard of the prognosis for return to normal milking, the radiographic evaluation provides important information.

Similar lesions can be found in adult cattle. Generally, they have a history of mastitis in the affected quarter at the dry period. The clinical presentation is similar. At calving, no milk or only a few drops of milk come out of the affected quarter. The lesion can be palpated at the base of the teat. The ultrasound is used to identify the location and the thickness of the lesion. In some cases, a teat endoscopy (thelscopy) can be performed to confirm the diagnosis. In that case, a specialized endoscope is introduced through the streak canal. The teat cistern is distended with saline or air and the lesion is visualized. Frequently, the lesion has a cauliflower shape. The treatment is surgical. The success rate varies according to the thickness of the mass at the ultrasound evaluation.

B) External lesions
The most frequent external lesion, at the base of the teat, causing milking problems is a varicose vein. This condition is generally found in older cattle. The animal is presented for milking difficulty. On palpation, the teat appears normal. However, a large vein is usually found at the base of the teat. In some case, this vein will protrude one third of the way down on the teat. It is difficult, with palpation only, to determine if this enlarged vein is the cause of the milking difficulties. To confirm the diagnosis, an ultrasound evaluation of the vein is performed using a linear 7.5 mHz transrectal probe. The base of the teat is evaluated. The large vessel is
visualized compressing the junction between the gland and the teat cistern. In some cases, the compression is not easily visualized. However, when the milking machine is in place, the vein becomes engorged and creates an obstruction. Since an ultrasound can’t be done during milking, the teat is completely evaluated with the ultrasound to exclude any other possible problems.

In most of the cases, the varicose vein can be followed on the udder. A small gauge catheter can be placed in the vein away from the teat. An angiogram is then performed by injecting contrast product while a lateral radiograph is taken. This procedure is not essential for the diagnosis. However, it is useful to elaborate the surgical plan. It helps in the localization of the collateral vessels. Ideally, the varicose vein is double ligated and removed.8

Distal end injuries
Seventy percent of the cows culled for milking problems have a lesion at the distal end of the teat.9 Those lesions are traumatic in nature. They occur when the animal steps on its teat or when a neighbouring cow steps on the teat. The trauma can be inflicted by the inappropriate use of teat inserts and canulas. Shortly after the injury, the teat can be bruised and swollen. However, at the time of the evaluation there is no sign of external trauma. On palpation, the distal portion of the teat may feel firmer. It is assumed that the lesion is located at the distal end of the teat. In general, no further diagnostics are performed and a blind surgical procedure is performed. In this situation, the recovery rate is very poor.

To improve the recovery rate of distal end injuries it is important to choose the appropriate treatment for each condition. To choose the right treatment it is important to have the correct diagnosis. For example, a lesion at the rosette of Furstenberg does not need the streak canal to be opened and a ruptured streak canal does not need to have the rosette curetted.

To evaluate the streak canal, a graduated probe has been developed. This probe evaluates the length of the canal. Streak canals over 8 mm are assumed to be abnormal.10 However, there is normal variation between individual cattle making the interpretation of the measurement difficult. It is recommended to compare the measure with the contralateral teat. A ruptured or traumatized streak canal will be longer than normal.10

An ultrasound evaluation of the distal end of the teat should be performed using a linear 7.5 mHz transrectal probe. To improve the image, the evaluation is performed with the teat in a plastic cup filled with water. The acoustic gel and probe are placed on the plastic cup. The rosette of Furstenberg and the streak canal are evaluated and compared with the contralateral teat. The evaluator is looking for a hyperechoic mass at the rosette of Furstenberg, an irregular streak canal and ruptured streak canal. If those lesions are found, blind surgical treatments are not recommended. If no lesions are found, the remainder of the teat is evaluated. If the ultrasound evaluation is normal, an exclusion diagnosis of fibrosis of the streak canal can be made. Only in this case should a blind surgical technique be used to open the streak canal.
Ideally, the diagnosis of distal end injuries should be confirmed by thelscopy. The axial thelscopy described previously is not appropriate for these cases. The thelscope should be placed through the teat wall. This is performed by using the sharp thelscopic trocar. This trocar is entered through the streak canal into the teat cistern. Then, it is directed laterally and pushed through the teat wall. The thelscopic canula is slid over the trocar from the outside in. The trocar is removed and the thelscope is introduced in the canula. The teat cistern is filled with air or saline and the distal end is evaluated. If a lesion is found, it can be treated immediately under thelscopic guidance. The instruments are inserted through the streak canal. This technique provides better results for the treatment of distal end injuries when compared to blind surgery and surgery performed through a thelotomy.\textsuperscript{11-12}

Other teat conditions
A) Webbed teats
Supranumerary teats are cut at a young age. When they are in close proximity with another teat, they can be missed or left in situ deliberately to avoid traumatizing the principal teat. At calving, the supranumerary teat (webbed teat) may or may not have a distinct gland cistern. If a gland cistern is present, it may or may not empty itself during milking. If the webbed teat is not impinging milking and is not leaking milk between milking, it can be left in place. However, if it is obstructing the normal milking of the principal or the accessory gland, it will leak between milkings and will need to be removed. In show cattle, the webbed teat will be removed for cosmetic purposes even if it does not cause any milking problems.

The webbed teat may look like a fistula or a normal teat attached to the principal teat. The diagnosis is made by visual exam and palpation. However, prior to elaborating a surgical plan, it is important to investigate the gland associated with the webbed teat. In some cases, the accessory gland is bigger than the principal gland.\textsuperscript{13} If this is the case, the conjoined teat should be anastomosed to the principal teat.

The milk production of the accessory gland can be compared to the principal gland by measuring the milk production of each gland. A canula is inserted in each teat during milking. The milk of the 2 glands is caught separately and the quantities compared. An ultrasound evaluation can also be performed. A linear 7.5 MHz transrectal probe is used. However, it is difficult to evaluate each gland independently with this technique. Ideally, a contrast radiograph of the accessory and principal glands should be performed. A large volume (20-30ml) of contrast solution is injected first in the accessory gland. A lateral radiograph is taken. The contrast solution is milked out as much as possible and the same procedure is done on the principal gland. The 2 gland cisterns are compared. If the webbed teat has a significant gland, it can be worthwhile to anastomose both teats. If the webbed teat has an insignificant gland, it can be removed and the accessory gland is closed at the base of the teat.

Contrast radiography provides important information about the thickness of the septum between each teat. This information is important if the structures are anastomosed. The thicker the septum, the less likely it is that the anastomosis will be functional in the long term.
References

Keywords
Bovine, Radiograph, Surgery, Theloscopy, Ultrasound
1. Indications for Rumen Surgeries

Because of its close apposition with the left paralumbar fossa, the dorsal sac of the rumen represents a portal to have access to other anatomic structures such as the reticulum, reticulo-omasal orifice, cardia, and rumen itself. Indications for rumen surgeries (including rumenotomy, rumenostomy, and rumen cannulation) can be divided based on these anatomic structures.

- Access to the reticulum:
  - Removal of metallic foreign bodies (e.g. traumatic reticulo-peritonitis);
  - Drainage of peri-recticular abscess;
  - Removal of non-metallic foreign bodies.

- Access to the rumen:
  - Removal of abnormal ruminal contents (e.g. grain overload, frothy bloat, toxin(s) acutely ingested, vagal indigestion);
  - Decreasing rumen load (e.g. vagal indigestion, frothy bloat, rumen impaction);
  - Long-term release of free gas bloat [temporary rumenostomy];
  - Rumen fluid donor [rumen cannulation];
  - Enteral nutrition (e.g. vagal indigestion, tetanus, pharyngeal trauma) [temporary rumenostomy];
  - Removal of ruminal foreign bodies (balling guns, Frick speculums, broken esophageal tubes).

- Access to the reticulo-omasal orifice:
  - Removal of foreign bodies (e.g. polyethylene twine, placenta, trichobezoar(s)).

- Access to the cardia and distal esophagus:
  - Retrograde repulsion of obstructing round and smooth foreign bodies located at the cardia (e.g. potato, apple, beet).

2. Chemical Restraint

All rumen surgeries are performed standing through the left paralumbar fossa, except neonatal rumenotomy (rarely performed). Therefore, standing sedation, analgesia and anesthesia of the flank techniques are described. Surgery is done after standard regional (paravertebral nerve block) or local (line or “L” inverted block) anesthetic block of the paralumbar fossa using 2% lidocaine.

Standing sedation is rarely needed to perform a standing rumen surgery in most dairy cattle, but may become necessary to facilitate cooperation of fractious cattle. Various standing anesthetic techniques have been described in cattle. However, I will limit the description to one technique that I use: the “ketamine stun” (also called “stun”). This protocol consists of the
addition of a small dose of ketamine to a more traditional chemical restraint protocol (α₂-adrenergic agonist and opioid combination) to enhance the degree of patient cooperation. This anesthetic technique was initially developed for recumbent sedation of ruminants, but can be easily modified to be used in standing patients by decreasing doses. In ruminants, a combination of butorphanol, xylazine, and ketamine is used. In my experience, it is a very efficacious and safe combination of sedatives for standing procedures in the adult bovine patient. The doses are given as followed:

- IM/SQ standing stun (cattle doses):
  - Butorphanol 0.01 mg/kg
  - + Xylazine 0.02 mg/kg
  - + Ketamine 0.04 mg/kg

The IM/SQ standing stun is also commonly called “5-10-20 technique” because the doses required for a 500 kg cow are 5 mg butorphanol, 10 mg xylazine, and 20 mg ketamine. For a 680 kg patient, the doses are 7 mg butorphanol, 15 mg xylazine, and 25 mg ketamine. All 3 drugs can be mixed into a syringe and administered using one injection intramuscularly or subcutaneously. It is recommended to use a 20 mg/ml xylazine product to facilitate accurate dosing. The onset of action is approximately 5 to 10 minutes and the duration of effect is approximately 60 to 90 minutes. If chemical restraint needs to be further extended and/or the degree of cooperation needs to be augmented, the recommendation is to re-administer between 25 to 50% of the initial xylazine and ketamine doses. It is important to note that recumbency can occur with re-administration of 50% of all 3 drugs. An IV standing stun has been described using similar doses as the IM/SQ version, but is rarely used, except to provide a transient improvement in patient cooperation. The intensity of sedation, and patient cooperation as well as the onset of action are much greater with IV than IM or SQ. Due to the difference in absorption rate of drugs, the duration of effects is greater after SQ than IM administration. The duration of effect is much shorter with IV administration (15-25 minutes).

Morphine (0.05-0.06 mg/kg IM/SQ) produces a similar degree of cooperation as butorphanol and may be used instead of butorphanol in the IM/SQ standing stun technique in cattle. The main advantage of this substitution is the low cost of morphine compared to butorphanol.

3. Rumenotomy – Surgical Technique
Compared to a standard left paralumbar fossa approach, the celiotomy incision needs to be performed slightly more cranioventral and parallel to the last rib. Exploration of the peritoneal cavity is performed prior to exteriorization and incision of the rumen. In cases of peritonitis, it is important to explore the infected area (usually left cranioventral portion of the abdomen) last, if needed. However, adhesiolyis to further improve exploration is contraindicated. In cases of severe ruminal distension, exploration may be limited.

The lateral wall of the dorsal sac of the rumen is then identified and exteriorized. This may require decompression of gas from the dorsal sac to facilitate manipulation of the rumen. It is very important that enough rumen is exteriorized; otherwise closure of the rumenotomy will be challenging (see following details). A variety of surgical methods have been described to anchor
the rumen to the abdominal wall and avoid leakage of ruminal content into the peritoneal cavity.

The skin suture fixation technique is a commonly used method because of its versatility, ease of execution, absence of special instruments required, and decreased risks of intra-operative complications such as leakage and adverse displacement of the rumen from the celiotomy site. First, four stay sutures are placed between the seromuscular layer of the rumen to the skin edges (one dorsally, one ventrally, one cranially, and one caudally). Then, using continuous inverting suture pattern (e.g. Cushing) with heavy-gauge material (e.g. No. 3 or 6 Supramid, cutting needle), the rumen (partial thickness) is temporarily fixed circumferentially to the skin edges of the celiotomy. The continuous pattern has to be interrupted at 180° to avoid a purse string effect. This technique can be time consuming and there is a risk of inadvertent penetration of the lumen with the suture material, which could potentially leak after rumen closure. Even so, this technique is probably the easiest one to apply in a field setting.

Alternative techniques for isolating the rumen and preventing contamination include the use of stay sutures, towel clamps, and fixation of the rumen to a Weingarth’s ring or Gabel’s rumen board. Although these methods are quicker than the skin suture fixation technique, they can be more easily displaced (especially in fractious cattle) allowing contamination of the peritoneal cavity.

Once the skin has been secured to the rumen, a rumenotomy incision is performed vertically for approximately 15-20 cm. A rumen shroud or a plastic drape may be inserted to protect the rumenotomy incision from excessive contamination. Ruminal contents usually need be partially removed to facilitate further exploration of the reticulo-rumen. Important structures to palpate during reticulo-rumen exploration are: cranial pillar of the rumen, ruminoreticular fold, reticulum, reticulo-omasal orifice, and cardia. Abdominal viscera such as abomasum, omasum, and right kidney can be palpated through the medial ruminal wall.

The rumenotomy incision is closed in two layers using an inverting pattern (e.g. Cushing) with No. 2 or 3 absorbable suture materials on a taper needle. Chromic gut, polyglactin 910 (Vicryl), polyglycolic acid (Dexon II), or braided lactomer (Polysorb) are preferred to monofilament suture because monofilament sutures tend to tear through the seromuscular tissues more easily. Ideally, the 2 layer closure of the rumenotomy should be performed while the rumen is still fixed to the skin. However, this can be challenging if the rumen was inappropriately exteriorized, such as in cases of severe ruminal distension. In this case, an inverting or simple continuous pattern is perform, the rumen to skin sutures are then removed, and the first rumenotomy closure is oversewn with a seromuscular inverting pattern (e.g. Cushing). The rumen wall is lavaged with sterile saline before replacement in the peritoneal cavity.

The celiotomy incision is closed routinely in a three layer technique. The peritoneum and transversus abdominis muscle are sutured using a simple continuous pattern with a No. 2 or 3 absorbable suture material (taper needle). The internal and external oblique muscles are closed also using a simple continuous pattern with a No. 2 or 3 absorbable suture material (taper
needle). The skin is closed with a Ford-interlocking or interrupted cruciate pattern using No. 3 or 6 polycaprolactam (Supramid; cutting needle).

Rumenotomy is considered as a clean-contaminated surgical procedure. It is important to keep instruments needed for closure of the celiotomy aside and covered from the contaminated rumenostomy instruments. Furthermore, it is ideal that the drapes are changed as well as the surgical gown and gloves for the closure of the celiotomy incision. Perioperative antibiotics are administered to minimize the risks of peritonitis and incisional infection.

4. Temporary Rumenostomy – Surgical Technique
The term “rumenostomy” is typically employed to refer to a temporary opening in the rumen. Temporary rumenostomy consists of the creation of a rumen fistula that will ultimately contract and heal (3-5 weeks). This represents a longer term alternative to ruminal trocharization with lower risks of complications associated with trocharization such as obstruction of the trochar, dislodgment from the rumen or abdominal wall, and peritonitis. The term “rumen cannulation” is used to refer to permanent rumenostomy and consists of the creation of a permanent rumen stoma for the insertion of a commercial rumen cannula. Both techniques are similar except that the permanent rumenostomy implies a larger circular skin incision.

For temporary rumenostomy, a circular skin incision of approximately 2 to 6 cm diameter is performed in the dorsal aspect of the left paralumbar fossa. Adequate placement of the rumenostomy is necessary to avoid complications such as occlusion of the rumenostomy by ingesta (too ventral fistula) or excessive tension on the sutures (too dorsal fistula). The external and internal abdominal oblique muscles, and transversus abdominis muscles are bluntly dissected by splitting the muscles along their fibers using a grid technique. The peritoneum is tented and incised. The ruminal wall is exteriorized through the incision using Allis tissue forceps. Several surgical options are described in the literature, but a two-layer technique will be described here. The first layer of closure is performed by suturing the rumen wall to the external abdominal oblique muscle using an interrupted suture pattern (No.2 absorbable suture, taper needle). Following incision into the ruminal wall, a simple interrupted pattern (2nd layer) is performed to secure the edges of the rumen to the skin. Postoperatively, a cannula may be placed in the stoma to maintain patency during the first few days or week of use. One example of this is to use a syringe case (e.g. 20- or 35-cc syringe case) which can be fitted into the rumenostomy site and kept in place by stay sutures. The client should be instructed to inspect and clean the syringe case daily if needed. In the majority of calves, the fistula will close over several weeks, but in some cases rumenotomy closure can become necessary. This can be accomplished using a delayed closure technique involving en bloc resection of the fistula and separate closure of the rumen and body wall.

5. Rumen Cannulation – Surgical Technique
Rumen cannulation technique (also called “permanent rumenostomy”) is very similar to that of the temporary rumenostomy. Site of permanent rumenostomy needs to be carefully planned to prevent excessive skin irritation by the cannula. Adequate positioning should allow the outer ring of the cannula to sit within the borders of the paralumbar fossa (13th rib, transverse
processes, and tuber coxae). A circular area of skin of approximately 1.25 cm smaller in diameter than the inside diameter of the cannula is excised from the dorsal half of the left paralumbar fossa, approximately 10-12 cm (adult cattle) ventral to the transverse processes of the lumbar vertebrae and centered halfway between the 13th rib and tuber coxae. It is highly advised to use a template to guide the circular skin incision and make it smaller than the inner diameter of the cannula because skin defect margins distort due to the normal skin tension lines (or Langer’s lines) resulting in a larger than needed defect area. The external and internal abdominal oblique muscles and transversus abdominis muscle are bluntly dissected by splitting the muscles along their fibers using a grid technique. Once exposed, the peritoneum is tented, punctured and manually gridded to a size equal to that of the opening in the musculature.

The ruminal wall is exteriorized through the incision using Allis tissue forceps. Rumen seromuscular layer may be sutured to the external abdominal oblique muscle using an interrupted suture pattern (No. 2 multifilament absorbable suture, taper needle) to increase the strength of the pexy. Horizontal mattress sutures are then placed between the ruminal wall and the skin at the cranial, caudal, dorsal and ventral margins of the incision using No. 2 or 3 chromic gut or synthetic absorbable multifilament suture (cutting needle). These seromuscular (or partial thickness) stay sutures help minimizing contamination of the surgical site and can be removed at the end of surgery. The ventral half of the exposed rumen is vertically incised, and a simple interrupted pattern is performed to secure the edges of the rumen to the skin (No. 2 or 3 chromic gut or synthetic absorbable multifilament suture, cutting needle). The dorsal half of the exposed rumen is then incised and sutured to the skin. The 4 stay sutures are removed. Finally, a commercial rumen cannula is inserted into the rumenostomy. Various sizes and types of cannulas are available for use in the bovine patient. I have used a flexible rumen cannula from Bar Diamond (Parma, Indiana) in the past. Preparation and use of this cannula is presented here. Insertion of this cannula is challenging, but greatly facilitated by following these simple steps to prepare the cannula:

- Submerge the cannula in hot water to improve pliability;
- Remove the cannula from the water, and immediately invert the inner flange into the outer flange of the cannula without completely inverting the cannula;
- Return it to a hot water bath;
- (perform surgical procedure);
- Remove the cannula from the water, fold the partially inverted inner flange, and insert it into the rumen by pushing the inverted/folded inner flange through the fistula and into the rumen;
- Completely unfold the inner flange to secure the cannula into the rumenostomy.

6. Postoperative Management
Administration of a systemic antibiotic is recommended for 3 to 5 days postoperatively. In my experience, parenteral administration of procaine penicillin G, oxytetracycline, ceftiofur hydrochloride or ceftiofur sodium are effective and appropriate for the range of bacteria likely to contaminate the wound after rumen surgeries. Inflammation and pain can be managed by the administration of ketoprofen (3 mg/kg IV or IM once daily for up to 3 consecutive days) or
flunixin meglumine (2.2 mg/kg IV once daily for up to 3 consecutive days). Please refer to the products’ labels for further information concerning doses, frequency and route of administration, and withdrawal period. The most significant aspect of aftercare following rumenotomy and temporary rumenostomy involves the medical management of the disease requiring surgical intervention and any other concurrent disease(s).

Concerning rumen cannulation, the surgical site and surface of the cannula in contact with the surgical site should be cleaned daily for 5 to 7 days using a diluted antiseptic solution without removing the cannula from the rumenostomy site. On day 10, the cannula is removed by pulling the inner flange into the center of the cannula and any necrotic tissue and suture material is removed from the edge of the rumenostomy. The wound and cannula are cleaned and the cannula is replaced. Thereafter, the cannula and skin edges should be cleaned twice a month.

7. Complications & Prognosis
Complications seen during surgery include contamination of the abdomen with ruminal content, the cow becoming recumbent during surgery, or inability to successfully perform the needed treatment (e.g. opening of peri-recticular abscesses). The most commonly seen postoperative complications are peritonitis, surgical site infection, and abscessation of the abdominal wall. Prognosis is associated with underlying diseases and preoperative general condition of the animal.

8. References
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Reasons for claw amputation may include the following:
Chronic septic arthritis of the distal interphalangeal joint, vertical wall cracks or other claw horn lesions associated with exuberant granulation tissue non-responsive to treatment, luxations of the proximal and distal interphalangeal joints, osteomyelitis and fracture of the phalanges, tenosynovitis of the digital flexor tendon sheath, necrosis of the apex or ventral surface of P3 and severe trauma such as partial exungulation.

Outcome
Amputation of the rear outside claw is reported to have the worst outcome whereas amputation of the rear inside claw had the best outcome. No difference in outcome was found between amputation of the medial and lateral claws of the front leg. One study showed that cattle that recovered from digit amputation had a mean survival time of 20 months. The same study reported that the likelihood of good recovery decreased from 71% in cattle weighing less than 341 kg to 27% in cattle weighing more than 682 kg. A second study of 85 cases reported good recovery in 51% of cases; fair recovery in 27% and poor recovery in 22%. Approximately 30% of cattle undergoing digital amputation were culled for lameness within the first 7 months. Animals that remained in the herd for more than 12 months were unlikely to be culled for lameness. In another study 23 of 41 (56%) cows survived at least 1 year, whereas in a third study 56% was culled within the first 60 days. Interpretation of these findings is complicated by the fact that many people see amputation as purely a salvage procedure in order to get the animal to the market. Such animals may be sold in the absence of specific post-surgical complications.

Approaches for amputation
Include amputation through the distal part of the proximal phalanx, disarticulation of the proximal interphalangeal joint. The proximal or middle part of the second phalanx and amputation just below the coronary band

Complications
Complications irrespective of level of amputation

Hemorrhage Severe or even fatal post-operative hemorrhage can occur. If possible 2 arteries should be isolated and ligated. This includes the main digital artery which is located axially in the fat palmar/plantar to the 1st phalanx and a less prominent branch which is located in the subcutaneous tissue of the dorsal skin. Hemostasis can be accomplished by effective bandaging without ligating any vessels. However, soft tissue damage can be caused by making the bandage very tight in the absence of sufficient padding.

Removal of infected tissues
The amputated area should be carefully examined for remaining areas of infection, which if present should be debrided and clean out following amputation. The presence of tenosynovitis should be investigated prior to amputation. Septic tenosynovitis can lead to abscess formation in the tendon sheath above the dew claws following amputation. On rare occasions it may spread to the opposite flexors and tendon sheath. Spread form the infected tendon sheath into
the metacarpo (tarso) – phalangeal joint is also possible. There is a relative thin separation between tendon sheath and the joint immediately below the proximal sesamoid bone where infection can enter. Septic tenosynovitis can be identified by swelling above the dew claw and aspiration of the fluid which will be turbid with a high protein and cell count. Depending on severity the tendon sheath can be lavaged and a drain placed or if indicated the tendon sheath should be opened and the necrotic tendons removed up to the bifurcation and the area debrided. All tissue folds where residual infection can be trapped should be opened and debrided. In some cases the dew claw and surrounding tissue should be removed in order to achieve an open and clean wound. Application of a cast in such cases stabilizes the leg, helps with pain control and appears to promote healing.

**Osteomyelitis**

Osteomyelitis of the remaining phalanx or phalanges is another possible complication. Lumps of granulation tissue are usually an indication of a tract leading to an area of infection such as a osteomyelitis and a developing sequestrum. Following the amputation the resultant granulation bed should have a fairly even surface with no obvious defects.

**Sole ulcer**

Sole ulcer of the partner claw can occur which seems to be more common following block placement. In such cases the block should be removed. Care should be taken during corrective trimming to retain a functional weight bearing surface as much as possible.

**Complication associated with the surgical approach**

**Amputation through the proximal phalanx**

If the amputation is made too high through the proximal phalanx instability of the digit may result or it is also possible that the wire cut may enter the metacarpo (tarso) – phalangeal joint. Fracture of the distal phalanx or excessive strain of the supporting structures such as the collateral ligaments may result in severe or non-weight bearing lameness. Amputation through the proximal phalanx should be done immediately above the proximal interphalangeal joint in order to preserve the proximal cruciate ligament, thus providing maximum stability to the remaining digit. Exposed bone marrow of Pl can form excessive granulation tissue, which have to be removed for complete healing to occur.

**Amputation through the middle phalanx.**

Low amputation through the middle phalanx may result in repeated trauma of the stump by the walking surface. Low amputation Of P2 can also lead to inadequate drainage of the tendon sheath and an ascending tenosynovitis may result. Amputation close to the proximal interphalangeal joint can lead to necrosis of the remaining part of the middle phalanx due to ischemia and septic arthritis of the proximal interphalangeal joint may follow. With disarticulation of the proximal interphalangeal joint the articular cartilage may predispose to the formation of a synovial cyst or infection of the remaining synovial structures.

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**TOE LESIONS IN CATTLE**
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Toe ulcer (zone 5), white line disease (zones 1 & 2), thin soles and thin sole toe ulcer (TSTU) in zone 5 adjacent to the junction of zones 1 & 2, corkscrew claw (zones 1 & 2), wall cracks (especially horizontal wall cracks), trauma-related lesions affecting the tip or apex of the claw and iatrogenic causes associated with over-trimming or improper trimming technique.

**Causes, pathogenesis and treatment**

**TOE ULCER**

Breakdown of the suspensory apparatus of the apex of P3 is associated with the release of matrix metalloproteinases (MMP’s). This leads to weakness and elongation of the collagen fibers in the laminar corium. The interdigitation between the laminar corium (sensitive laminae) and the horn leaflets on the inside of the wall breaks down resulting in sinking and rotation of the third phalanx (Mulling 2002, Tarlton 2000, Ossent, 1998). The result is pressure by the apex of P3 on the solar corium in zone 5. This leads to hemorrhage in the apex of the sole in zone5. Dysfunction of the solar corium at the point of injury can lead to development of a full thickness defect in the sole (Mulling 2002, Ossent 1998). In the worst case scenario, the solar corium and P3 may actually prolapse through the sole. Toe ulcer is a fairly common cause of front leg lameness in heavy beef bulls (Sarel van Amstel, unpublished observations). Differentiation of a toe ulcer from other toe lesions may be difficult since lesions are often well-advanced by the time they are first observed

**Treatment:**
In the area of the toe the third phalanx is close to the inside of the sole and only separated by the corium and a thin layer of subcutaneous tissue.
Ascending infection therefore commonly results in osteitis of the apex of the 3rd phalanx followed by pathological fracture and sequestrum formation.
A common treatment procedure for toe ulcer is removal the apex of the toe with a pair of nippers or gigli wire in order to create drainage. However, loose and under-run horn and necrotic bone may remain creating favorable conditions for anaerobic bacteria causing further
under-running of horn and delayed healing. All loose and under-run horn should be removed which usually involve the sole and the white line at the toe. Sole horn separation from the underlying corium may occur as far back as the heel. Once the loose horn is removed, a crater-like defect in the corium at the toe is often observed. Necrotic corium should be removed and the presence of a pathological fracture of the third phalanx investigated. With careful probing the sequestrum can often be located and extracted or a fracture line may be visible or in some instances radiographs may be necessary to demonstrate a fracture. The sequestrum can be removed using a Rongeur or periosteal elevator. The fractured portion of the third phalanx may still be tightly adhered to inside of the dorsal wall. The cavity left by removal of the sequestrum should be flushed and any remaining necrotic tissue removed. Another approach is to remove the overlying portion of the dorsal wall. Both these approaches result in satisfactory healing and eventual remodeling of the bone may occur. The use of a bandage may be necessary to protect and help with hemostasis. In rare instances the infection may spread all the way up under the dorsal wall and enter the distal interphalangeal joint.

A claw block should be placed under the sound claw in order to make the affected claw non-weight bearing. Antibiotic treatment may be required in those cases with signs of cellulites, which generally appear as swelling of the foot. Signs of inappetence and fever can be associated with systemic spread of the infection.

**WHITE LINE DISEASE (ZONES 1 & 2)**

The white line is produced by the epidermis overlying the laminar corium. It is a 3-part structure consisting of an outer, intermediate and inner zone. The outer and intermediate zones consist of laminar horn, whereas the inner zone (adjacent to the sole) is a heterogeneous combination of laminar horn and loosely arranged tubules (Budras KD 1996). These structural features make it the softest and least resistant part of the claw capsule and subject to damage by mechanical shearing forces and the penetration of bacteria and foreign bodies such as coarse dirt and gravel (Mulling 2002). The area of the white line most commonly affected is the abaxial heel-sole-wall junction of the lateral claw (zone 3 on the claw zone diagram) (Budras KD 1996). The white line in this region is naturally predisposed to greater mechanical impact and wear during locomotion since this area bears the impact of heel strike during the foot placement phase of the stride (Budras KD 1996, van der Tol 2003). Overgrowth and overloading of the outer claw tends to exacerbate load bearing and may increase white line disease problems in zone 3 (Toussaint Raven 1998).

White line lesions in zones 1 & 2 tend occur less commonly than that in zone 3 and the pathogenesis of this lesion may be more related to inflammatory changes in the corium similar to that of toe ulcer rather than weight bearing. These areas of the white line do not withstand mechanical impact to the same degree as zone 3 and thus, claw horn wear and turnover is slower in zones 1 and 2. Lesions start in the laminar corium and extend down to the bearing surface of the white line and are usually observed as small cracks or spaces within the white line that become infiltrated with organic matter. The entrapment of extraneous material within these spaces may be visualized as one or more dark lines, cracks or often times as a small dark spot within the white line (Logue, 1998). In other cases where separation of the white line is
advanced and complicated by infection, the lesion may appear as a large area of loose necrotic horn. Abscess formation associated with white line disease creates severe lameness. These abscesses and associated purulent material may accumulate in the subsolar region of the toe or in many cases migrate upward beneath the wall. Upon reaching the skin-horn junction, these may rupture forming a sinus tract at the coronet (Mulling, 1998). In the worst case scenario, infection may extend into P3 where the resulting osteitis creates a very painful and often times chronic condition as result of sequestrum formation.

**Treatment:**
Proper treatment requires the debridement of all necrotic tissue including affected portions of P3. The wall over the area of separated white line is removed at a 45° angle in order to expose the whole lesion which may extend all the way to the coronary band or it may also separate the solar horn from the solar corium thus producing a subsolar abscess. This will lead to severe lameness and may result in extensive damage to the solar corium. Complete removal of all loose and undermined horn is essential

**CORKSCREW CLAW**

Corkscrew claw is most commonly observed in the lateral claw of rear feet in cattle. It is reported to be a heritable condition, however factors such as claw disease particularly laminitis and housing conditions are likely to influence its occurrence. It is characterized by an abaxial to axial rotation of the toe that displaces the sole, axial wall and white line into an axio-dorsal position. Internally, P3 rotates in similar fashion. In the heritable form of this disorder, there are a couple of important anatomical abnormalities that contribute to development of this problem: 1) a misalignment of the second and third phalanges that positions the abaxial surface of P3 in close proximity to the weight bearing surface, and 2) an elongated and narrow third phalanx. Curving and upward rotation of the apex of the toe results in weight bearing on the mid to caudal portion of the abaxial wall. Corkscrew claws are normally larger and typically bear the majority of weight in the foot. The corresponding lack of weight borne on the unaffected digit usually results in significant atrophy of that claw (AABP Lameness committee 2006).

Toe lesions in corkscrew claws develop in one of two ways: 1) white line separation in zones 1 and 2, and 2) as the axial wall rotates and repositions itself dorsally, it has a tendency to become folded over on itself trapping organic matter within the fold. This is a near perfect environment for anerobic bacteria that eventually cause further necrosis ultimately exhibiting itself as a toe abscess. Horn of the white line in zones 1 & 2 of corkscrew claws often shows evidence of hemorrhage and degeneration. This increases its vulnerability to separation and increases the possibilities of white line disease in these zones. Routine trimming at 3-4 month intervals helps to maintain normal shape and function of the claw capsule, but must be done carefully to avoid excessive thinning of the sole in the toe (zones 1 & 2) (AABP Lameness committee 2006).

**TRAUMA-RELATED LESIONS OF THE TOE**

Cattle working facilities, certain types of flooring conditions and rough or careless animal handling contribute to an increased risk of trauma-related lesions of the toe. These are normally more common in feedlot cattle or younger animals that may be less accustomed to
human contact and handling. Thinning of the sole at the toe, full thickness wall cracks, and on occasion fracture of P3, lead to severe lameness and complications such as the formation of a sequestrum. A sequestrum often occurs when injury or infection results in pathological fracture of P3 with subsequent loss of blood supply to that portion of the bone. It is not uncommon to find a loose necrotic piece of bone in chronic toe lesions. In many cases, removal of this necrotic bone will permit complete healing of an otherwise chronic lesion (Shearer, 2007).

IATROGENIC - OVERTRIMMING AND IMPROPER TRIMMING TECHNIQUE

One of the most common trimming errors is over-trimming. Trimming the sole to a thickness of 3-4 mm or less creates conditions whereby the sole is unable to protect the underlying solar corium in an animal housed on a hard flooring surface. Over-trimming typically leads to lesions in zone 5 characterized by separation of the sole adjacent to the white line in zones 1 and 2 (Van Amstel 2008). These are described in greater detail in the section under thin soles.

There are several improper trimming techniques that may result in toe lesions: trimming the toe too short, excessive removal of axial wall and white line, and excessive removal of wall horn on the apex of the claw. Many of these techniques create lesions by direct exposure of the corium or by causing weakness of the corresponding portion of the claw horn capsule affected (Shearer 2001).

Finally, as keratinocytes migrate outward from the basal cell layer of the epithelium, they incorporate keratin within their cellular structure. The most mature horn cells and those with the greatest degree of keratinization are those within the stratum corneum layer. This corresponds to the most external layer or horn that has reached the surface of the claw capsule (Van Amstel 2004). Trimming removes the most mature and therefore the hardest horn layers. Horn that remains is less mature and thus softer (i.e. wears more rapidly). While regular trimming that corrects claw horn overgrowth and altered weight bearing is a useful adjunct to foot and leg health, trimming schedules that are too aggressive or frequent, coupled with flooring conditions that contribute greater than average wear rates, increases the potential for thin sole problems. Trimming schedules must be developed on a farm-by-farm basis in consideration of claw horn wear and growth characteristics unique to each operation (Shearer 2001).

WALL CRACKS

Cracks or fissures in the hoof wall are common in cattle. Those which run in a vertical direction (from the coronet to the weight bearing surface) are referred to as vertical wall cracks or sandcracks. Incidence rates as high as 64% have been reported in beef cattle, compared with less than 1% in dairy cattle (Greenough 2001). Therefore, for the purposes of this discussion, we will focus our attention on horizontal wall cracks.

Horizontal wall cracks are common in both beef and dairy cattle and when severe may result in profound lameness. The causes of horizontal wall cracks are better understood. In some cases they simply signal a “physiological change” that has resulted in a mild to moderate interruption of horn growth and formation in the basal cell layer of the epidermis overlying the coronary corium. In others, they are representative of conditions (often related to disease
disorders) that have led to significant “physiological stress” and severe interruption of horn formation in the hoof wall. These severe disruptions in horn formation are exhibited as very distinct ridges and grooves that run in a horizontal direction on the hoof wall. They are often referred to as “hardship grooves” or “stress lines” (Greenough 2001, van Amstel 2006). In the most extreme cases where the fissure is sufficiently deep to result in a full thickness defect of the wall, the lesion is often referred to as a “thimble”. As these fissures migrate downward toward the weight bearing surface, the segment of the wall closest to the weight bearing surface becomes moveable as it reaches or nears the weight bearing surface. When this occurs, it becomes extremely painful as it pinches and damages the underlying sensitive tissues. The objective of treatment is to stabilize the lose sections of the wall by removal of loose portions or by other means including the use of a foot block on the opposite claw assuming it is more stable and will support a block (van Amstel 2006).

THIN SOLES AND THIN SOLE TOE ULCER

In housing systems where the rate of sole horn wear exceeds the rate of growth excessive thinning of the sole is likely to occur. Previous work has demonstrated that claw horn hardness is influenced by nutrition, contact with manure slurry and moisture content of claw horn. Claw horn is continually exposed to high moisture conditions particularly during the hot and humid summer months (van Amstel 2004, 2006 & 2008). Heat stress abatement procedures require that cows have access to sprinklers and fans, misters or high pressure fogging systems. Claw horn moisture content is also affected by manure management systems based on flushing of fresh or recycled water to clean floors in barns, holding areas and travel lanes. Wear rates are also affected by the spatial layout of facilities which require cows to walk long distances to and from barns and milking areas. This is exacerbated by abrasive flooring conditions that include sharp turns and sloped walkways. Excessive sole horn wear is especially common in new installations where freshly hardened concrete creates a particularly abrasive surface as a consequence of the presence of surface aggregate which naturally forms on the flooring surface as the concrete cures. This observation has become as commonplace as to have its own name “New Concrete Disease” (Cermak, 1998, Shearer 2007)

The occurrence of thin soles is also influenced by conditions contributing to poor cow comfort such as overcrowding and reduced stall use due to improper stall design and insufficient bedding. Issues of dominance also affect stall use. When stall numbers are equivalent or less than the total number of cows in the barn, timid animals such as heifers may have less opportunity to rest (Cook, 2009). Some recommend that there be at least 10% more free stalls than cows to permit greater choice and encourage resting. Some US recommendations for Holstein cattle include construction of a free stall 8 ft (2.5m) long [7ft 6in. (2.28m)] for two facing rows and 4 ft (1.25m) wide with a brisket locator 15in. (38cm) high and located 5ft. 8in. (1.72m) from the stall curb. For free stalls facing a wall a length of 9 ft. 8 in. (2.94 m) are recommended by some (Cook, 2009).

Continued wear and thinning of the sole leads to a separation of the sole from the white line at the junction of zones 1 & 2. This exposes the solar corium and therefore by definition is properly termed an ulcer (Van Amstel 2008). These authors have chosen to call it a thin-sole-toe-ulcer (TSTU) to distinguish it from toe ulcer due to laminitis. It is a very prevalent lesion in the southeastern US and observation in recent years suggests that the incidence of thin soles
and TSTU is increasing as herds continue to enlarge (Van Amstel 2008).

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**Nutritional and Environmental causes of Laminitis in Cattle**
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Laminitis is a multi-factorial lameness problem for example, both the infusion of endotoxin and the intake of excessive amounts of soluble carbohydrates failed to induce laminitis in cattle (Momcilovic et.al 2000). Single or multiple interactions of environmental, nutritional, physiologic, altered metabolic pathways and biomechanics may be necessary to trigger the vascular and inflammatory or enzymatic reactions responsible for laminitis to develop.

It appears that the primary driving force for the development of laminitis may be primarily dependent on the particular environment in which animals are kept. In dairy cattle total confinement (free stall) vs. pasture-fed milking operations presents different backgrounds where the interaction of the predisposing may be very different. In **total confinement dairy operations, cow comfort** issues may be the primary driving force in the development of laminitis (Cook et. al 2007) whereas **nutritionally induced rumen acidosis** may be the most important predisposing factor in **pasture –fed dairies** (Westwood et al. 2003).

**Total confinement dairy operations**

**Free stall housing and cow comfort**: Factors which may influence cow comfort and negatively influence their time budget [normal time budget includes: lying down in stall 11.3 hr; standing in stall 2.9hr; standing in alley 2.4 hr; time drinking 0.4 hr; time feeding 4.4 hr; time milking 2.6 hr (Cook et.al 2009)] include concrete walk ways, poor stall design, stall stocking density (overcrowding), prolonged milking time, undesirable type of bedding or inadequate bedding (sand bedding preferred) (Cook et al 2004, Cook et al 2008), unhygienic conditions and ineffective prevention of heat stress. This will lead to increase in standing time which in turn can lead to claw overgrowth of particularly the outer claw of the rear leg (Toussaint Raven 1989).

**Weight bearing of the rear claws**: Research has shown that the outer claw of the rear leg carries more weight relative to the inner claw (Toussaint Raven 1989). Weight distribution between the outer and inner claw of the hind leg was determined to be 80:20 in slightly overgrown claws. In severely overgrown claws it is reasonable to expect that this ratio becomes even wider. The mechanism of the rear claw imbalance in weight bearing relates to the animal’s normal skeletal structure, which in the back legs is fairly inflexible with the femur (and thus the rest of the leg) being attached to the bony pelvis through the hip joint. (Toussaint Raven 1989). Udder size may exacerbate weight bearing in the rear legs. During heel strike weight bearing occurs almost completely on the outer claw. During standing the bulb of the rear outer claw carries maximum pressure. On the whole, weight bearing pressures during all 3 phases of locomotion (heel strike, stance phase and push off) appears to be more towards the outside (abaxial region) of both rear claws as compared to the inside (axial region). The explanation for this is the anatomical structure of the suspensory system of the claw, which is stronger on the
outside and thus better equipped to carry more of the total weight bearing forces (van der Tol 2002, 2005). In addition, neuropeptides such as Calcitonin gene-related peptide (CGRP) and Substance-P (SP) normally occur in primary sensory, free nerve endings (nociceptors) in the epidermis overlying the dermal papillae including that of the solar corium. These neuropeptides can in addition to pain, also respond to pressure thus acting as mechanoreceptors while at the same time interact with other peptides to promote inflammation and vasodilatation and as such modulate keratinocyte proliferation thus accelerating horn growth. The overgrowth of the outer claw of the rear leg will increase the concussive forces in those claws which will create a favorable environment for the development of mechanically induced laminitis which will manifest as sole ulcer (zone 4) and white line disease commonly in zone 3.

Concrete may result in excessive wear and the development of thin soles and traumatic induced laminitis as a consequence with sole horn hemorrhages and disruption (sole ulcers and white line disease. Thin soles results from situations where the rate of sole horn wear exceed the rate of growth. The rate of sole horn wear depends largely on hardness and water content of the claw. Claw horn is continually exposed to high moisture conditions particularly during the hot and humid summer months as heat stress management procedures, particularly in the southeastern United States, requires that cows have access to sprinklers and fans, or misting systems. In addition manure management systems are based on flushing of fresh or recycled water to clean floors in barns, holding areas and travel lanes. As a result of these practices cows are often forced to spend prolonged periods standing in manure slurry (Shearer 2006). Cow’s feet are thus wet and soft and an increased rate of wear can be expected. Other factors, which contribute, include spatial layout of facilities which require cows to walk long distances to and from barns and milking areas particularly when turns are sharp and walkways sloped. Excessive sole horn wear is especially common in new installations where freshly cured concrete creates a particularly abrasive surface with the surface aggregate being responsible for the increase in the rate of wear often referred to as “New Concrete Disease” (Shearer 2006.). Separation of the abaxial sole (Zone 5) from the white line (Zones 1 and 2) (sole/white line separation) is a common lesion in cows with thin soles referred to as a thin sole induced sole ulcer (TSTU). Poor cow comfort caused by overcrowding, poor stall design and insufficient bedding may increase the time cows remain standing and moving around thus extending the time claws are subject to wear. When stall numbers are equivalent or less than the total number of cows in the barn, timid animals such as heifers may have less opportunity to rest. It is recommended that there be at least 10% more free stalls than cows to permit more choice and encourage lying time. Proper dimensions for free stalls are an important issue to meet the requirements of cows for normal resting. Such requirements would include: 1) Ability for the cow to stretch front legs forward. 2) Ability to lie on their sides with sufficient space for the head and neck. 3) Ability to rest their heads on their sides. 4) Enough room to rest their legs, udder and tail on the free stall platform and have a clean, dry and soft bed. Some US recommendations for Holstein cattle include construction of a free stall 8 ft (2.5m) long [7ft 6in. (2.28m)] for two facing rows) and 4 ft (1.25m) wide with a brisket board 15in. (38cm) high and located 5ft. 8in. (1.72m) from the stall curb. Even longer free stalls up to 9ft.8in. (2.94 m) are currently recommended by some (Cook et al. 2009). Thin soles have become a very important issue particularly in large dairies thus the possibility of over trimming should always be ruled out where thin soles are identified
as a herd problem.

Although cow comfort appears to be the main driving force in the development of laminitis in total confinement dairies, other factors play an important role in the clinical manifestation of the disease. Such factors include:

**Heat stress:** The primary avenues for heat loss during periods of hot weather are sweating and panting. In severe heat, panting progresses to open-mouth breathing characterized by a higher respiratory rate and greater tidal volume. The result is respiratory alkalosis as a result of the increased loss of carbon dioxide. The cow compensates by increasing urinary output of bicarbonate (HCO₃). Simultaneously, the salivary HCO₃ pool for rumen buffering is decreased by the loss of saliva from drooling in severely stressed cows. The end result is rumen acidosis because of reduced rumen buffering and an overall reduction in total buffering capacity. The effect of ambient air temperature on rumen pH was evaluated in lactating Holstein cows fed either a high roughage or high concentrate diet in both a cool (65°F with 50% relative humidity) and a hot (85°F with 85% relative humidity) environment. Rumen pH was lower in cows exposed to the higher temperatures and those fed the higher concentrate diets. These observations have been corroborated by others supporting the current view that increasing the energy density of rations to compensate for reduced dry matter intake during periods of hot weather is not without significant risk (Sanders et. al 2009).

**Nutrition and systemic disease:** Play a similar role in the development of laminitis through the release of toxic substances which can induce vascular and inflammatory changes in the corium. A primary goal in feeding is to maximize dry matter intake, and thus optimize performance yet avoid those conditions, which might lead to rumen acidosis and laminitis. Dairies apply feeding strategies to encourage consistent feed intake and minimize production losses. Total mixed rations (TMR) containing high quality forages has been one of the strategies used to lower the risk of rumen acidosis and laminitis. Proper formulation and mixing of feed ingredients helps achieve optimal success. It is often recommended that hay and ensiled feeds be chopped as coarse as possible and not mixed excessively to the extent that effective fiber attributes are lost. However, if forages are too coarse, cows may sort or select for concentrates rather than consume a balanced diet containing both feed grains and forage. Because of social hierarchy issues, most recommend housing mature cows separately from heifers. In all cases animals should be introduced to the milking herd ration gradually, preferably through the use of a properly formulated transition ration. Transition is a critical period of adjustment for animals and has important links to the pathogenesis of laminitis. In the southern United States, the nutritionist’s challenge is to maintain feed intake and avoid feeding related health problems during periods of hot weather. One strategy is to increase the nutrient density of rations and thus maintain an acceptable rate of dry matter intake. This strategy can be troublesome if not monitored carefully, in part because heat stressed cattle tend to eat less frequently (feeding during cooler times of the day only) but proportionally more at each feeding. The combined effect of these types of rations and feeding patterns increases the risk for rumen acidosis. However, add to this the fact that heat stressed cattle often have a lowered rumen pH due to
decreased salivary buffering, and it is easy to understand how acidosis becomes a major feeding challenge during the summer months (Shearer 2006) The pathogenesis of inflammatory based injury to the suspensory apparatus consists of circulatory changes in the dermis. This leads to tissue hypoxia, edema formation and activation of matrix-metalloproteinases, resulting in degradation of collagen with variable degrees of failure of the suspensory apparatus resulting in sinking of the third phalanx and compression of the dermis (digital cushion and solar corium). Epidermal changes (delayed or absence of keratogenesis) are secondary to the compression resulting in claw horn disruption with primarily sole ulcer and white line disease as serious consequences.

Calving is another compounding factor in the pathogenesis of laminitis. Epidermal growth factor (EGF) receptors occur throughout the epidermal layer in bovine claw horn. Protein synthesis by EGF is antagonized by prolactin but addition of prolactin to insulin and cortisol increased protein synthesis in claw horn explants. Cortisol on its own decreased total protein synthesis of bovine explants but did not decrease the population of protein synthesized. (Hendry et al 1999, Hendry et al. 2000). Glucocorticoid concentrations are elevated in post-parturient cows. It has also been reported in high producing herds with a high incidence of laminitis having elevated levels of cortisol. Insulin receptors occur in both epidermal and dermal layers but not in horn. In physiological concentrations insulin stimulates, both protein and DNA synthesis in claw tissue explants in vitro. Decreased levels of insulin have been reported in the post-parturient dairy cow. In addition, claw horn may also share the insulin resistance shown by other tissues (adipose and skeletal muscle) during lactation since overfeeding during the dry period gives rise to hyperinsulinemia and hyperglycemia, which are the two classic signs of insulin resistance (Hendry et al 1999, Hendry et al. 2000).

Age. The incidence of laminitis can have an age related relationship with a high incidence in first lactation cows. It has been shown that the concussion properties of the digital cushion are less effective in heifers than that of older cows. The digital cushion is located in the heel area. This allows for mobility during weight bearing between the third phalanx and the horn capsule. Changes in the digital cushion may be involved in the pathogenesis of laminitis. The content of the digital cushion in heifers is predominantly saturated fatty acids whereas in cows there is a progressive increase in the amount of monounsaturated fatty acids (MUFA), which is softer and has a better cushioning effect and is endogenously produced. Heifers have significantly less fat, which make them less effective to bear the concussive forces of weight bearing. Polyunsaturated fat content can be increased by feeding linseed oil to cattle (Baird et al. 2002).

Pasture – fed dairies
It appears that the primary driving force for the development of laminitis for pasture-fed milking operations may be primarily nutritional. High protein/ starch pasture in combination with grain feeding in the holding areas or milking parlor appears to be the most important factors in the development of laminitis (Westwood et al 2003). Other very important compounding factors are walk way surfaces and stockmanship (Chesterton 2002). Cows will step on rocks and other foreign objects if they are forced to walk at a pace faster than normal pace (Chesterton 2002).

Nutrition: The etiology, effects and prevention of ruminal acidosis have been extensively
researched and reviewed (Shearer 2006). Pastures low in neutral detergent fiber (NDF) and relatively high in non-fiber carbohydrates (NFC) and supplementation of pastures with silage and grain, expose cattle that are predominantly pasture-fed to the risk of acidosis. Endotoxin causes cellular injury with activation of phospholipase A2, which initiates the arachidonic cascade with the release of the enzyme cyclo-oxygenase COX (inducible COX 2). This allows cells to rapidly modify their capacity to generate prostanoids (prostaglandins and thromboxanes). Cytokines and COX-2 are potent inducers of vascular changes and metalloproteinases (MMP’s). Significant up regulation of COX-2 occurs in the sensitive laminae in cases of experimentally induced grain overload in cattle. Endotoxin also activates coagulation molecules including platelet activating factor (PAF) which plays a role in the development of disseminate intravascular coagulopathy (DIC). Pro-inflammatory molecules including tumor necrosis factor (TNF), interleukins and leucotrienes plays a role in the formation of cytotoxic substances including toxic oxygen radicals, proteases which degrades collagen and nitric oxide (Belknap et al 2002, Tarlton et al 2002)

In acute/ subacute laminitis microscopic changes in the corium include hyperemia, congestion, edema, cellular infiltration and hemorrhage. Cellular infiltrate consisted mainly of macrophages and neutrophils. The presence of mast cells and eosinophils was not a feature of the cellular infiltrate except in the perioplic and coronary regions. Claw pathology is also associated with changes in form and direction of the papillae in all parts of the corium. The number of side and secondary papillae are increased while at the same time the capillary network becomes more irregular and convoluted. Degenerative changes are present in the epidermis particularly in areas adjacent to vessels occluded with thrombosis. Cells in the stratum spinosum were enlarged, vacuolized and pygnotic. Macroscopically rotation and downward displacement of the third phalanx is present in a percentage of cases.

In chronic laminitis, arteriosclerosis as well as sclerosis in small arterioles is a common finding. Arteriosclerosis is characterized by intima proliferation and damage to the internal elastic laminae. Arteriosclerosis is more pronounced at the ulcer site as compared to other parts of the sole. There is a marked increase in arterio-venous shunts with neo-capillary formation (regarded as newly induced a-v shunts) in all areas of the corium. Hyper and parakeratosis is present in epidermal lamellae. Disappearance of onychogenic substance is reported as a common finding and relates to the formation of keratin and horn quality. Poor quality horn production may be due to fewer disulphide bonds, which are responsible for horn hardness. Another observation, which will affect horn quality, is the number of tubules, which were significantly reduced in animals with laminitis (Boosman et al 1989).

Pathological changes in the suspensory system of the claw have also been reported. In cases of sole ulcers the flexor tuberosity of the claw were displaced downward and the palmar dermis and subcutis were thinner as compared to normal controls. The digital cushions contained less fat and had been replaced by collagenous connective tissue (Lischer et al 2002)

Other factors which play a compounding role in the pathogenesis of laminitis in pasture-fed cattle have been discussed above.

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Digital dermatitis (DD) with emphasis on non-healing (atypical lesions)

DD synonyms: footwarts, hairy heel warts, digital warts, strawberry foot, Mortellaro or Mortellaro’s disease, papillomatous digital dermatitis (PDD)

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Etiology

Treponema spirochetes are involved as primary bacterial pathogens and account for more than 90% of the total bacteria. It is a epidermotropic, intermingled infection with three or more different phylotypes.

Characteristics of the Lesion

The lesions of DD typically occur on the skin of the plantar aspect of the rear foot adjacent to the interdigital cleft, or at the skin-horn junction of the heel bulbs. On front feet lesions are often found bordering the dorsal (front) interdigital cleft. Hypertrophied hairs often surround the lesion borders and should be distinguished from epithelial outgrowths that look like long hairs extending from the surface of chronic lesions. Chronic lesions without these epithelial outgrowths are generally thickened and have a granular surface. Lesions are very tender and even a mild disturbance of the inflamed tissue tends to result in extreme discomfort and mild to moderate bleeding. Cows will alter their posture and/or gait to avoid direct contact between lesions and the floor or other objects. This avoidance behavior is one of the best visual indicators of a DD lesion. These pain avoidance adaptations also lead to abnormal wear of the weight bearing surface of affected claws. Lesions associated with the plantar interdigital cleft usually cause the cow to shift weight bearing toward the toe. This results in increased wear at the toe, decreased wear in the heel and an overall reduction in the weight bearing surface of the affected claw. When lesions occur on the dorsal (front) aspect of the foot, cows respond by altering posture and weight bearing resulting in overgrowth and extension of the toe and greater wear in the heel. These alterations in shape of the claw generally require correction at the time of trimming.

Atypical lesions

The causative organism is also associated with non-healing or atypical lesions which trimmers often refer to as “hairy attack”, referencing its invasion of the corium rather than the skin above the claw horn capsule. Such lesions occur in the presence of anaerobic conditions such as under loose horn of the wall and sole. This atypical form of digital dermatitis is often associated with chronic white line lesions in zones 1, 2 and 3 as well as sole ulcers associated with undermining of the sole horn. Other horn lesions associated with atypical digital dermatitis include toe lesions as well as vertical well cracks. Another site is extension of a DD lesion in the dorsal interdigital cleft under the abaxial and abaxial horn of the wall. Evans et.al. 2011, using a PCR technique showed an association between DD and several claw horn lesions including sole ulcer, white line disease and what they referred to as “necrotic toe”. These authors also
reported an increase in the incidence of non-healing claw horn lesions particularly “necrotic toe”.

**Diagnosis of atypical digital dermatitis**

The lesions are usually well demarcated with a distinct granular surface. Histopathologically lesions are characterized by hyperkeratotic epithelium overlying dense zones of necrosis and pyogranulomatous inflammation in association with exuberant granulation tissue and proliferation of blood vessels. The organisms (spirochetes) penetrate deep into the epidermis and even the dermis and can readily be demonstrated with the use of silver stain (Van Amstel et al. 2011). ERvans et al 2011, using a nested PCR found a strong association between the presence of DD treponemes and non-healing claw horn lesions. This included the DNA of *Treponema medium-like, T. phagedenis-like* and *T. denticola – like.*

**Treatment of atypical lesions**

Any chronic lesion of the claw horn capsule in which loose horn is evident could be associated with atypical digital dermatitis. All lose horn should be carefully removed until reconnection with normal horn is evident. The exposed soft tissue (corium) should be cleaned and examined for the presence of atypical digital dermatitis which have a distinct granular surface, is well demarcated from unaffected corium. Lesions are very painful with a distinct odor. Because of the pain associated with the lesion, debridement of loose horn is done following regional intravenous anesthesia with lidocaine. Experience with treatment suggests that healing, as evidenced by the formation of new horn over the affected area, is not likely to occur until the infection is under control. Researchers from Tennessee and Iowa have also observed that the inflammation associated with this condition in the form of granulation tissue may in itself be a significant complication in successful treatment of these lesions. Therefore, they suggest that the inclusion of an anti-inflammatory agent along with the antimicrobial be included in therapy of these lesions. In a study conducted by the authors, atypical digital dermatitis lesions were treated topically with a paste under a bandage consisting of dexamethasone and oxytetracycline powder. Bandages were changed every 5-7 days. Follow up biopsies in 2 cows after 19 days showed absence of spirochetes. Clinically lesions showed progressive improvement and after 8 weeks appeared to have undergone complete clinical resolution. However, histopathologically the epithelium still appeared acanthotic and hyperkeratotic. Cook & Burgi 2008, demonstrate success in healing atypical digital dermatitis lesions after extensive debridement of loose horn and soaking the affected corium with a commercial product containing copper, zinc and a mixture of organic acids in a low pH base as well as the application of an iodine-oxytetracycline paste with a dressing for 8 days. Apart from its antimicrobial effect, oxytetracycline has also been shown to inhibit metalloprotease (MMP) production the effect of which will be a decrease in the granulation tissue response.

**‘Udder Sores’**

Another condition which is suspected to be a atypical manifestation of the causative organism of DD is “*Udder Sores*” also known as bovine ulcerative mammary dermatitis, mammary necrotic dermatitis, udder seborrhea, and udder foul. Udder sore is typically observed in the ventral abdominal wall between the front quarters of the cow’s mammary gland. It is described
as a moist exudative dermatitis with a characteristic pungent foul odor. Researchers report an increased prevalence of this condition in herds suffering from severe problems with DD. This link has been strengthened by several studies in recent years that have identified bacterial spirochetes similar to those of DD in sections of tissues recovered from these lesions. While further research is necessary to definitively establish DD as the most likely cause of udder sores, previous work demonstrating transmission of DD from foot-to-foot by direct contact suggests that a similar mode of transmission from foot-to-udder is possible. Indeed, some theorize that the pathogens causing DD may be transferred from an infected foot to the abdominal skin as the downside leg of the cow makes contact with the udder and ventral skin when the cow lies down.

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Van Amstel SR, Shearer J, Cooper VL 2011 Clinical presentation, treatment approach and histopathology of atypical digital dermatitis lesions. Abstracts of the 16th Symposium and 8th conference of Lameness in Ruminants, Rotutua, New Zealand
Providing analgesia for horses has been challenging with the options being much more limited than in small animals. However, with the increasing use of continuous infusions of lidocaine and butorphanol, and the release of COX-2 selective NSAIDs for horses such as firocoxib, new safer effective options are now available.

Pain has recently been recognized as the fourth vital sign. Transient pain is transmitted to the dorsal horn of the spinal cord. This activates receptors and results in a reflex response such as withdrawal, which minimizes tissue damage. Chronic ongoing pain results in peripheral sensitization due to the release of substances such as prostaglandins, histamine, serotonin, bradykinin and nerve growth factor from damaged tissue. These substances form a sensitizing soup that activates sensory nerve endings and lowers the threshold of the nociceptors to stimuli. Subsequently, even a mild stimulus can cause pain which is termed hyperalgesia, or pain is perceived to a stimulus that doesn’t usually cause pain which is called allodynia. In many cases pain is obvious, such as in a horse with violent colic. Providing effective analgesia when pain is more subtle, such as in chronic conditions, or after surgery, is less frequently considered. Compared to the degree of violent pain that many horses with surgical colic display, postoperative pain is less dramatic and more difficult to recognize. Horses are relatively stoic and pain can be difficult to monitor objectively. In order to evaluate this more objectively and consistently, a behavioral scoring system can be used to monitor levels of pain. This uses subtle changes in the horse’s locomotion and interactions to detect pain. This generates a pain score between 0-26 which is more objective and repeatable. A high score indicates that current analgesia is inadequate and further treatment is necessary. Using this scoring system, we have been able to objectively evaluate different drugs to determine if they are as effective as flunixin meglumine in providing visceral analgesia.

Options for Analgesia in Horses:
Non Steroidal Anti Inflammatory Drugs (NSAIDs)
NSAIDs are the most commonly used analgesics in horses and inhibit the cyclooxygenase (COX) enzymes. There are at least 2 isoforms of COX, COX-1 and COX-2, plus a splice variant of COX-1 known as COX-3. COX-1 products are responsible for many constitutive functions such as gastrointestinal protection, maintaining renal blood flow and platelet aggregation. Tissue injury and inflammation causes up regulation of the COX-2 enzyme and hence increases the
production of its products that are responsible for inflammation, fever and pain. In particular prostaglandins activate sensory nerve endings and cause pain. NSAIDs therefore prevent the production of prostaglandins, and hence reduce pain. Phenybutazone and flunixin meglumine are non-selective COX inhibitors and block both isoforms of the COX enzyme. Using a model of small intestinal colic, we have shown that administration of flunixin meglumine significantly reduces behavioral pain scores after surgery and is a very effective visceral analgesic. However, non-selective COX inhibitors also have side effects due to their inhibition of constitutive COX-1. In the gastrointestinal tract phenylbutazone and flunixin meglumine inhibit the protective mechanisms and cause erosions, ulceration, and perforation. This manifests as gastric ulcers and right dorsal colitis. Additionally COX-1 associated prostaglandins are critical for the repair of injured intestinal epithelium and closure of the tight junctions and therefore may delay intestinal healing in horses with colic.

The effect of different NSAIDs on recovery of injured equine intestine has been evaluated in the jejunum and colon. Typically ischemia is induced for 2 hours by occluding the blood supply and then the intestine is allowed to recover for 18 hours at which time comparisons are made between the treatments. Such studies have shown that treatment of the horse with flunixin meglumine inhibits recovery of the jejunum after ischemic injury, as indicated by a lower TER and increased permeability of the mucosa to LPS. This is likely due to inhibition of COX-1 by flunixin meglumine and hence a critical reduction in prostaglandins which are required for mucosal repair. However, in the left dorsal colon no detrimental effect of flunixin meglumine on mucosal repair was detected. It is important therefore to consider that the drugs we are giving could make the horse appear better but adversely affect mucosal repair.

Additionally, administration of NSAIDs is known to reduce renal blood flow. In a well hydrated animal this is not an issue, but in dehydrated horses this reduction in blood flow becomes critical and can result in permanent renal damage in the form of renal papillary necrosis. Prostaglandins are also essential for osteoblast activity and therefore in people there is concern that NSAIDs may delay fracture healing. In horses it is unlikely that this is a significant clinical problem.

To avoid NSAID toxicity: Only the correct dose of the NSAID should be used. If effective analgesia is not achieved, alternative classes of analgesics should be used. Increasing the dose or adding an additional NSAID is unlikely to result in additional analgesia but WILL increase the risk of side effects. In severely dehydrated horses volume replacement should be done before the NSAID is given. Alternatively, a COX-2 selective NSAID can be used. As COX-1 products are necessary for repair of the jejunum, treatment with a COX-2 selective drug to provide analgesia and anti-
inflammatory effects while allowing the production of beneficial prostaglandins may be preferable in horses recovering from ischemic small intestinal injury. Several COX-2 selective NSAIDs have been evaluated. Both deracoxib and etodolac were determined not to be COX-2 selective in horses, and their administration retarded mucosal recovery to a similar extent as flunixin meglumine. This highlights the importance of evaluating COX selectivity in the species in which the drug is to be used, and not extrapolating results from other species. Meloxicam, which is available in Europe and Canada is COX-2 selective in horses, and has also been evaluated in an experimental model of equine small intestinal injury at a dose of 0.6mg/Kg every 24 hours. It did not inhibit recovery of the mucosal barrier and provided effective analgesia.

The first equine COX-2 selective drug available in the USA, firocoxib, is available as a paste and was recently released as a 2% IV formulation. It should be released in Canada later this year. Evaluation of the firocoxib paste in 253 horses with osteoarthritis proved that it was as effective as phenylbutazone after 14 days of administration with no documented adverse effects. Its use should therefore be considered for horses with arthritis that require long term analgesia. Its safety profile is considerably better than phenylbutazone which can result in right dorsal colitis even at the recommended dose. However, due to the long half life of the drug, steady state concentrations are not reached for the first couple of days of administration. Therefore in order to more rapidly obtain steady state concentration and effective analgesia, a 3x dose is recommended for the initial treatment, followed by the regular dose 24 hours later. Additionally, firocoxib may be more suitable for treatment of horses with small intestinal colic than flunixin meglumine. We have evaluated the effect of the intravenous formulation of firocoxib on recovery from jejunal ischemia. Intravenous treatment with firocoxib was as effective as flunixin meglumine at providing analgesia. Additionally, it permitted recovery of mucosal barrier function after ischemic injury as evidenced by a reduced permeability to fluorescent labeled LPS. Its effect in injured large colon has not yet been evaluated. Therefore when selecting an NSAID for a horse with colic, if referral and colic surgery may occur, firocoxib may be a more appropriate treatment than flunixin meglumine.

Due to the cost of firocoxib pate many equine practitioners have used the small animal firocoxib tablets, Previcox, orally for horses. Due to the difference in dosing between horses and dogs, one large dog tablet is an appropriate dose for a 570Kg horse. However, the AVMA warns against this practice because there is a licensed equine formula available.

COX-2 selective NSAIDs have been associated with side effects in people and dogs. In particular, administration of COX-2 selective NSAIDs to people causes in an increased risk of myocardial and cerebral infarction. This resulted in many of these drugs being withdrawn from the market. These side effects likely occur because the production of prostacyclin by COX-2, which causes vasodilation and inhibits platelet aggregation, is prevented. However, thromboxane A2, a COX-1
product which causes vasoconstriction and platelet aggregation, is produced unchecked, resulting overall in a prothrombotic vasoconstricted state. The side effects of COX-2 inhibitors in horses are unknown at present. However it is possible that side effects may be seen in horse with abnormal coagulation profiles.

**Opiods**

If pain is refractory to NSAIDs alone, then multimodal therapy should be considered. Opiods have been used minimally in horses due to the side effects of excitement and constipation. However, recent studies have shown that several opioids provide effective analgesia without adverse effects. Butorphanol can be used to provide adjunctive analgesia. It is best used as a continuous rate infusion because it has a short half life. Addition of a continuous infusion of butorphanol to post operative colic patients routinely treated with flunixin meglumine, resulted in a decrease in the % of weight lost after surgery, and lower plasma cortisol concentrations. Butorphanol is also used to decrease GI motility. Transdermal fentanyl patches have also been used for analgesia in horses. The patches are usually applied to clipped, shaved and cleansed skin near the withers, with 2x 10mg patches needed for an adult horse. Epidural morphine has been used either alone, or in combination with detomidine, to provide analgesia for hind limb lamenesses. Placement of an epidural catheter allows easy administration of repeated doses.

**Lidocaine**

Apart from its use as a local anesthetic, lidocaine can be administered systemically to provide anti-inflammatory and analgesic effects. Lidocaine is rapidly metabolized by the liver. Therefore in order to achieve constant therapeutic concentrations it must be administered as a loading dose followed by a constant rate infusion (CRI). The usual dose is 1.3mg/Kg loading dose given IV over 15 minutes, followed immediately by a CRI of 0.05mg/Kg/min. The plasma therapeutic range is 1-2 g/mL which is achieved by using the above dose. Signs of toxicity are seen at concentrations >3 g/mL. Signs of toxicity include muscle fasciculations, ataxia and depression. Due to the narrow therapeutic range, horses receiving systemic lidocaine should be monitored closely, and the infusion discontinued if signs of toxicity are seen. A previous study tested the effect of systemic lidocaine on somatic and visceral pain in horses. Somatic pain was tested by measuring the temperature at which a thermal probe on the withers elicited a pain response. This was increased significantly 30 minutes after the start of lidocaine administration. Visceral pain was assessed by colorectal and duodenal distention with a balloon placed in the lumen. No effect of lidocaine was seen on visceral pain. Likewise, when systemically administered lidocaine was evaluated in the small intestinal colic model, we found that it was no more effective than saline at providing visceral analgesia and was not as effective as flunixin meglumine.
Thus it appears that systemic lidocaine acts as a somatic analgesic but does not provide visceral analgesia in the horse. This is in contrast to many clinicians’ observations, in which a colicky horse appears more comfortable shortly after lidocaine treatment is initiated. These tests were performed in normal horses and the presence of intestinal inflammation in colic patients may result in an increased effectiveness of lidocaine in treatment of visceral pain. However, its effect in reducing somatic pain indicates that it would be beneficial for treating musculoskeletal problems in horses in a hospital setting, such as acute laminitis.

Conclusions:
Pain is important and contributes to overall morbidity and mortality. Signs of pain can be subtle in horses and should not be ignored. NSAIDs are widely used, but adjunctive analgesics such as opioids and lidocaine are preferable to simply increasing the dose of an NSAID.

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Referral is declined: Now what?
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The vast majority of horses with colic that are seen by a veterinarian show mild non specific signs. However, of these horses, nearly 10% will have a surgical lesion. Early identification of these cases is critical for a good outcome. However, in certain situations referral may not be possible even when it is recommended by a veterinarian in the field. Such circumstances include economics, distance to a referral facility or personal choice by the client. In such situations, the vet in the field can attempt more aggressive medical therapy with the possibility of a successful outcome. However, it is important that the client understands that the decision not to refer is not one that can be rescinded later as by that time the window of opportunity for a successful outcome will have passed.

The first consideration is:

Can medical treatment be attempted in this horse?
Before electing to continue treatment on the farm, the veterinarian should make every possible effort to rule out a strangulating intestinal lesion such as a large colon volvulus or strangulating small intestinal lipoma.
Abdominal ultrasound should be the starting point for this determination. Dark circular a-motile structures >5 cm in the inguinal region are suggestive of a small intestinal strangulation. In particular, ultrasonography can be helpful for diagnosis of an epiploic foramen entrapment. In this disease, loops of small intestine can be visualized in the right flank, usually further forward than can be felt on rectal palpation. Ultrasound can also be helpful to differentiate between a large colon volvulus that requires surgery and a displacement that could be managed medically. Thickened colon wall > 5mm indicates venous occlusion consistent with a large colon volvulus and indicates immediate surgical correction, or euthanasia if referral is declined.

Another important diagnostic technique to distinguish a severe intestinal problem is abdominocentesis. Color of the fluid alone can be indicative of a strangulation as red blood cells leach into the fluid from ischemic intestine. In a horse with distended small intestine on ultrasound, a serosanginous abdominocentesis is sufficient indication for euthanasia if surgery is not an option. If possible a measurement of total protein should be made. An elevation >2.5 g/dL can indicate ischemia or inflammation such as peritonitis. In order to attempt medical therapy, the peritoneal fluid should appear normal and have a normal TP concentration. More advanced peritoneal fluid analysis includes measurement of lactate. This is a sensitive indicator of the presence of ischemic intestine. Lactate can actually be measured very easily in the field using a hand held lactometer. Peritoneal lactate should be the same as blood lactate, but is greater than that in blood if ischemic intestine is present. However, in any horse, a blood lactate >4mmol/L indicates poor tissue perfusion that requires treatment with large volumes of
intravenous fluids.
If these basic tests are normal, then further medical treatment can be attempted if the veterinarian feels comfortable with the situation.

Management of Pelvic Flexure Impactions:
The traditional treatment for a pelvic flexure impaction is administration of mineral oil. Owners should never be encouraged to syringe mineral oil into a horse’s mouth because it does not trigger the mechanoreceptors in the nasopharynx and aspiration can occur which is usually fatal. Mineral oil does not help the impaction pass nor break it up. Its only function is to act as a marker for passage from one end of the intestinal tract to the other and therefore it serves little purpose. Other commonly used substances include magnesium sulfate, DSS and sodium sulfate. The goal of treatment is to rehydrate the impacted colonic contents, which none of these, including mineral oil, actually achieve. The treatment of choice for a pelvic flexure impaction is enteral fluid therapy, with IV fluids rarely being needed. This not only rehydrates the impaction but also distends the stomach stimulating the gastrocolic reflex and triggering motility in the colon. A stomach tube is passed and tied into the horse’s nose to allow administration of water every 2 hours. Most horses will tolerate a tube in place but a muzzle may be necessary to prevent the horse from pulling the tube out. Usually 4-6L of warm water with 30ml of alternating NaCl and KCl is administered every 2 hours. Including the salt in the water is essential in order to prevent electrolyte imbalances. It is important to check for reflux prior to each administration as an expanding softening impaction can compress the duodenum. If reflux is obtained, oral fluids should be discontinued. The horse may become more colicky as the treatment takes effect due to contraction of the colon around the impaction and the impaction enlarging initially as it expands with rehydration.

Management of Large Colon Displacements:
These cases are characterized by gas distended large colon and tight bands on rectal examination, but a normal colon wall thickness on ultrasound. Referral centers manage more and more of these cases medically. The goal of medical management is to empty the colon of gas and ingesta so that it falls back into its correct location. The horse should be held off feed until the rectal examination is normal and no signs of colic are observed without NSAIDs. Intravenous fluids with 40ml/L calcium gluconate will help stimulate motility. Colon motility can also be stimulated by administering oral fluids as described previously. This also helps to break up an impaction in the right dorsal colon, which is frequently present with a large colon displacement. Horses with large colon displacements are usually painful due to the stretch and tension of the mesentery and gastrointestinal viscous. Removal of gas from the colon should decrease
luminal distention. Decreasing the distention should decrease the intra-luminal pressure within the cecum or colon which should improve gastrointestinal motility. This is achieved by trocharization. Trocharization is performed in the standing, sedated horse. The distended colon or cecum can be located by rectal palpation or abdominal ultrasonography. Gas distention is often prominent in the right paralumbar fossa. An area is clipped over the most prominent gas distention in the right paralumbar fossa and aseptically prepared. A couple of ml of 2% lidocaine are injected subcutaneously and into the oblique muscles. A 14g angiocatheter is placed through the lidocaine bleb into the cecum. Gas will emerge through the catheter when the catheter enters the cecum. This is best monitored by attaching an extension set to the catheter and submerge the other end in a container of water. Bubbles in the water indicate continued gas decompression of the cecum. When the bubbles stop, remove the extension tubing and inject 300 mg (3 ml) of gentamicin as the catheter is removed. Complications following trocharization are uncommon but include local body wall abscessation and septic peritonitis. Therefore the horse should be monitored for signs of septic peritonitis for 3−5 days. Using the combination of these techniques, we have a fair success rate for medical correction of large colon displacements. However, some horses become increasingly painful or colic persists for several days, and which point surgical intervention or euthanasia are the only options.

**Management of a nephroplenic entrapment:**

This can be diagnosed by a combination of the characteristic rectal palpation and failure to image the left kidney with ultrasound. Many of these cases will have gastric distention because the duodenum is compressed by the large colon, which prevents gastric emptying. Therefore it is advisable to pass a stomach tube prior to further medical treatment. Some of these horses can also be as painful as a large colon volvulus, however the lack of increase in colon wall thickness and characteristic palpation should be used to differentiate the two problems. Our first line of treatment for these is to administer phenylephrine, an alpha-1 agonist, to cause splenic contraction. We use 45-60mg/Kg IV, which is usually 1 or 2 vials (20-30mg) in an adult horse. This should be drawn up in a 60ml syringe and administered over 10 minutes. It will result in profound bradycardia, and therefore the heart rate should be monitored while it is administered. The maximum effect on the spleen is within the first 20 minutes\(^1\) so as soon as the drug has been administered, the horse should be lunged for 20 minutes. Following this it is best to put the horse into a stall where it can be observed for a return of colic signs. Fatal hemorrhage following administration of phenylephrine has been reported occasionally in horses >15 years old.

Passing of gas or manure is a good indicator that the procedure has been successful. Repeated
palpation and ultrasound are insensitive indicators of success. In general, this technique is effective in 75-90% of cases. \(^2\) If colic signs return, and it appears that the entrapment has not corrected, the procedure can be repeated several times providing you are confident that this is the problem. In certain stubborn situations this technique will not work. If referral is not an option, the horse can be anesthetized with xylazine and ketamine, dropped onto the right side and the legs rolled over the top of the horse in an attempt to dislodge the entrapped colon.

**Management of small colon impactions:**

These cases are more common in the winter months. Rectal palpation reveals a solid tube of manure in the small colon with the absence of formed fecal balls. If severe, the impaction can completely prevent the passage of gas and feces resulting in gas distention of the large colon. Such horses can become increasingly painful and bloated. At a referral hospital many of these cases resolve with medical management and surgical intervention is less frequently required. If gas distended large colon is present, or the horse appears bloated, trocharization should be performed. This will usually make the horse significantly more comfortable. However it may need to be repeated if gas builds up again before the impaction resolves. Relieving the gas distention also reduces the pressure on the impaction and can help to restore motility and spasm in the small colon.

The impaction itself should also be addressed by administering oral fluids as described previously. We frequently see horses become more painful shortly before feces start to pass. This is likely due to the impaction enlarging as it absorbs water and softens.

One of the most useful tools for managing small colon impactions is the drug Buscopan (Boehringer Ingelheim). Buscopan contains N-butylscopolammonium bromide, a quaternary ammonium derived from a belladonna alkaloid. It acts as an anti-spasmodic by blocking muscarinic receptors of the gastrointestinal tract, decreasing parasympathetic smooth muscle activity. Its duration of action is approximately 20 minutes and although it is an anti-muscarinic compound, it exhibits very few central nervous system effects because it has poor lipid solubility.\(^3,4\) Several studies have evaluated the visceral analgesic properties of Buscopan on viscous distention in research horses and determined that this medication has few analgesic effects.\(^4\) It does transiently reduce small intestinal motility – an effect that can be readily observed ultrasonographically. It also causes a transient increase in heart rate, often to around 60bpm. Therefore it is very important to obtain a base line heart rate and perform ultrasound prior to administering it. Because it has a short duration of action, we will repeat doses as necessary, sometimes as frequently as every 2 hours and have not observed any adverse effects. Because its mechanism of action is different to an NSAID, there is no risk of toxicity from using an NSAID concurrently for analgesia. We also utilize the anti-spasmodic effects of Buscopan to aid with medial resolution of ileal impactions, choke and meconium impaction.

Using these 3 techniques together, successful resolution of small colon impactions occurs in the
vast majority of cases that we see. These techniques could certainly be utilized on the farm for such cases.

**Management of Colitis/ Potomac Horse Fever:**
In horses with severe colitis, the most important aspects of treatment are to maintain tissue perfusion and treat endotoxemia. These cases are usually referred due to the intensive medical care needed. However, given a willing client and good facilities, appropriate treatment could be administered on the farm. One of the most important ways to determine tissue perfusion is by doing an excellent physical examination and repeating this several times a day to look for changes in heart rate, pulse quality, CRT and urination. In these horses, the gastrointestinal tract is often not able to absorb fluid; therefore IV fluid therapy is often necessary. To guide fluid therapy in these sick horses, PCV and TP should be measured at least daily. Horses with more severe signs of hypovolemia, including heart rates 60 – 80 bpm, elevated PCV (>45%), depression and poor appetite definitely require intravenous fluids as oral fluids alone are insufficient. To maximize fluid flow rate a large bore catheter such as a 12G should be placed. Hypertonic saline (7.2% NaCl) at 4 ml/kg, IV, will quickly improve cardiac output and tissue perfusion and can be administered in emergency situations prior to balanced polyionic fluids. Balanced polyionic fluids can be administered through an IV catheter at 30 – 90 ml/kg, depending on estimated fluid losses.

Horses with hypoproteinemia <5mg/dl need colloid therapy, such as Hetastarch. Hetastarch is a synthetic colloid composed of hydroxyethyl starch. Administration of 10 -20 ml/kg, IV can be performed safely in the field, provides oncotic pressure support, and therefore improves tissue perfusion and oxygen delivery. It does not have to be frozen and therefore is easier than plasma to administer in the field. However, its effects are often short lived due to the underlying disease process and a significant rise in TP is rarely seen.
In addition to fluid therapy, flunixin meglumine and antibiotics are usually administered. If PHF is suspected oxytetracycline at 6.6mg/Kg in 1L saline IV should be administered. Oxytetracycline, gentamicin and flunixin meglumine are all nephrotoxic, therefore great care should be taken to correct significant dehydration prior to their administration.

One of the significant life threatening complications of these cases is laminitis. Therefore in any horse at risk of laminitis, cold packing of the feet in ice should be considered. Empty fluid bags are ideal for putting over the hoof and filling with ice. In horses with increased digital pulses, the ice melts noticeably fast and may require refilling every 2 hours. However this is something that owners can be recruited to assist with.
Overall, PHF and colitis can be some of the most challenging medical cases to manage. Maintaining adequate hydration in the face of a non functional gastrointestinal tract is critical
to success. However, with dedication and a willing owner, medical therapy can be successful in the field.

References
Why did the case I referred have a poor outcome?
Vanessa Cook, VetMB, PhD, DACVS, DACVECC, Michigan State University

Many improvements have occurred in the management of surgical colic in recent years, such as recognition of the importance of early referral and advances in surgical and postoperative care. However, mortality rates remain highest in the first 10 days after colic surgery. Euthanasia in the immediate postoperative period is primarily due to the complications of postoperative ileus and laminitis. In addition horses may have problems after hospital discharge due to chronic postoperative complications such as incisional infection, recurrent colic and intestinal adhesion formation. The importance of these complications and recent advances in the management of each of these problems will be discussed.

Immediate Postoperative Complications:

1) Ileus
Ileus is failure of the normal intestinal propulsive motility. The incidence of postoperative ileus is reported at around 27%,\(^1\) with the median time of onset 13 hours after surgery. Ileus is a significant cause of immediate postoperative mortality and increases the duration of hospitalization,\(^2\) volume of crystalloids required, and the risk of developing diarrhea, laminitis and incisional drainage. These all contribute to the cost of treatment, and in many cases euthanasia is elected by the owner due to financial constraints. In particular the volume of crystalloids required and the cost of prolonged use can be prohibitive, particularly when the duration of ileus cannot be predicted.

Factors that contribute to an increased risk of postoperative ileus include: small intestinal lesions, the degree of circulatory shock and increasing age. Previously, it has been thought that ileus occurs due to excess sympathetic tone inhibiting intestinal contraction. However, recent studies have determined that inflammation in the circular and longitudinal muscle layers is an important factor. Inflammation is initiated by various stimuli including surgical manipulation, endotoxin and ischemic injury. An influx of neutrophils occurs which release inflammatory and toxic substances which inhibit muscular contraction and irritate enteric nerves, resulting in widespread inhibition of motility.

The key to preventing ileus is to reduce intestinal inflammation. The first step is excellent surgical technique including keeping the bowel moist, minimizing bacterial contamination and gentle tissue handling. The use of anti-inflammatory drugs to reduce leukocyte infiltration is also important.

Lidocaine has been shown to reduce the volume and duration of reflux. It is often used postoperatively, but its mechanism of action appears not necessarily to be as a prokinetic, but to decrease neutrophil recruitment and activation. Metoclopramide, a dopamine antagonist, is used as a prokinetic and, like lidocaine, is also administered as a constant rate infusion. Repeated ultrasound evaluations are used postoperatively prior to reintroducing feed and after
refeeding to ensure that there is good small intestinal contractility. If stagnant loops of small intestine are detected, feed is withheld until motility returns to normal.

Take Home Message: Ileus can occur in any postoperative colic patient, not solely those horses with small intestinal lesions. It is impossible to predict when normal motility will return but it usually will resolve, given sufficient time and money.

2) Laminitis

One of the most concerning postoperative complications is the development of laminitis. The single most important factor in the development of laminitis in hospitalized horses is the presence of endotoxemia. Endotoxin is part of the cell wall of Gram-negative bacteria, and is found in the intestinal lumen. The mucosa usually acts as an effective barrier against systemic absorption of endotoxin. However if the mucosa is damaged by ischemia, particularly in horses with a large colon volvulus, endotoxin can reach the systemic circulation. Ideally all damaged intestine is removed at surgery, but this does not always occur as it may be impossible to resect or difficult to identify. Remaining injured intestine allows the absorption of endotoxin after surgery. Once in the circulation, endotoxin triggers a massive inflammatory response that manifests as fever and tachycardia. Clinical signs of laminitis may also develop, although the direct causal relationship between endotoxemia and laminitis remains unclear.

Prevention and treatment of laminitis is aimed at reducing endotoxemia and laminar inflammation and providing hoof support. The volume of endotoxin available for absorption can be reduced by performing a pelvic flexure enterotomy at surgery and emptying the colon of most of the ingesta. Antibiotics and flunixin meglumine are the first line of treatment against postoperative endotoxemia. Administration of fresh frozen plasma is also used to manage endotoxemia. Its colloid properties improve circulation and perfusion to vital organs, including the feet, and it also contains factors such as anti-thrombin III, which counteract the prothrombotic effects of endotoxin. At Michigan State University treatment with large volumes of plasma is more affordable due to the donation of horses for plasma harvest. Some clinicians prefer other anti-endotoxic treatments such as polymyxin B, but their effectiveness is unclear. One currently accepted treatment method to reduce the severity of laminitis is distal limb cryotherapy. This is thought to cause limb vasoconstriction and hence reduce the delivery of circulating laminitis trigger factors to the foot. It may also slow the metabolic rate in the foot and hence prevent the laminar from the effect of degradative enzymes. Therefore at MSU, any horse that is at risk of developing laminitis has its feet packed in ice.

Matrix metalloproteinases are enzymes such as collagenase and gelatinase that are activated in laminitis and breakdown the connective tissue in the foot. MMP inhibitors such as doxycycline and oxytetracycline may be beneficial in the treatment of laminitis.

Take Home Message: It is difficult to predict which horses will develop laminitis and the severity of the disease. Unfortunately the pathophysiology of laminitis is still poorly understood. Some
treatments that we use may help, however until the direct cause is elucidated prevention is difficult.

Longer Term Complications:

3) Incisional Complications

Incisional complications are relatively common and range from simply edema to drainage and infection. The linear is usually closed with a simple continuous suture pattern and most surgeons use polyglactin 910 suture. A simple continuous pattern reduces the amount of suture used and hence the amount of foreign material in the incision. Use of a braided suture such as Vicryl could increase the chance of infection, however its handling characteristics make it the first choice for most surgeons. At closure, the incision is lavaged with 1L of saline to remove bacterial contamination. The skin is then closed with either skin staples or a monofilament absorbable suture. There is some evidence that the use of skin staples may increase the risk of infection.3

Many studies have evaluated cultures taken at various operative points as predictors of infection. However, there has been little correlation with bacterial cultures at surgery or closure with the subsequent type of bacteria in an infected incision. Currently it appears that the most likely source of bacterial contamination is from the recovery stall floor. Therefore every effort is made to ensure that an occlusive bandage is securely applied for recovery.

An abdominal bandage is always applied once the horse is standing after surgery as this too has been shown to decreases infection rates,4 probably because it reduces surgical site edema. Some horses are considered to be at high risk for incisional complications such as those horses that have had a previous colic surgery, or those with severe endotoxemia. In these cases we purchase a custom sized hernia belt for the horse to wear in the first few weeks after surgery.

Incisional infections usually manifest while the horse is in the hospital. However, infections may not be apparent until 10 days after surgery. If any drainage is noted, it is critical to remove adjacent staples or suture so that drainage doesn’t track along the incision. If the horse is febrile, or the incision is painful and swollen then a culture should be taken and appropriate antibiotics used.

In some horses an incisional hernia can develop. These are especially prevalent after previous colic surgery and development of an incisional infection. Surgical correction is usually only necessary for cosmetic purposes unless the hernia is very large. Currently the technique of choice is laparoscopic mesh herniorrhaphy.

Take Home Message: Overall the incidence of serious incisional problems is low. Avoiding skin staples and using bandages and hernia belts may reduce the incidence. However, despite these some horses will develop infections and even a hernia. This may be related to systemic compromise of the horse and the severity of the initial colic.

4) Recurrent Colic
Unfortunately the biggest risk factor for colic, is a previous colic episode. Therefore any horse that has colic surgery is almost certainly at an increased risk for subsequent colic episodes. Horses with a large colon displacement seem to be especially at risk for a subsequent displacement. In some horses this will manifest as repeated episodes of nephrosplenic entrapment. Additionally, horses with right displacement of the large colon appear to be at more of a risk for subsequent colic.\(^5\) The reason for this is not known, but is most likely due to an underlying motility disorder of the colon that cannot be corrected surgically.

It is important to distinguish recurrent colic and chronic colic. In recurrent colic the horse has several episodes of colic but has a period of time in between when it is completely normal. Chronic colic is considered to be one persistent episode of colic that lasts for several days and requires repeated analgesia. Recurrent colic can be a frustrating problem and a diagnostic challenge. Recurrent colic may be due to problems with anatomy, digestion or motility.

In horses with recurrent nephrosplenic entrapments, laparoscopic nephrosplenic space ablation can be helpful.

Take Home Message: Horses that have had colic are 3 times more likely to have another episode. The cause for recurrent colic can be difficult and frustrating to identify. Horse that have had a large colon displacement have an increased risk for recurrence.

5) Adhesions

A significant number of horses with recurrent colic after exploratory laparotomy have intestinal adhesions related to the initial surgery. The true incidence of adhesions is difficult to assess, as many horses likely have adhesions that cause no clinical signs. Adhesions that do cause clinical complications, and are confirmed at necropsy or repeat laparotomy, are found in approximately 10-20% of postoperative colic cases. The incidence of adhesions is increased when an enterotomy is performed, with small intestinal surgery, with repeat laparotomy, and when postoperative ileus occurs.

Adhesions form when there is injury to the surface mesothelial cells due to surgery, ischemia or peritonitis. This results in inflammation that causes the exudation of serum containing fibrinogen onto the surface of the intestine. Fibrinogen is converted to soluble fibrin, and then to insoluble cross-linked fibrin to form fibrinous adhesions. Normally, fibrinolysis occurs and fibrinous adhesions are resolved. However, if fibrinolysis is reduced, as occurs in sepsis and inflammation, an influx of fibroblasts occurs and collagen is deposited, resulting in a permanent fibrous adhesion forming.

Correction of adhesions is difficult; therefore the goal is to prevent them forming initially. This is achieved in two main ways: The first is to reduce inflammation and hence the exudation of fibrinogen. This is best achieved by meticulous surgical technique and administration of antibiotics and anti-inflammatory drugs. The second is to promote fibrinolysis so that fibrinous adhesions that do form are resolved. Fibrinolysis can be increased by heparin. Heparin increases the binding of thrombin by antithrombin III. Thus, the levels of thrombin are reduced
which reduces the conversion of fibrinogen to fibrin, and hence reduces the formation of fibrous adhesions. It can be administered subcutaneously every 8 hours postoperatively or intraperitoneally at the time of surgery. Another method to prevent adhesions is to separate apposing intestinal loops while normal repair occurs so that adhesions cannot form between them. One of the most widely used methods is to apply an absorbable membrane of hyaluronic carboxymethylcellulose. This can be placed onto areas that may be at risk of adhesion formation, such as the anastomosis. Carboxymethylcellulose itself has also been used to separate intestinal loops, however there is evidence that it may increase peritoneal inflammation and increase adhesion formation.

Management of fibrous adhesions that are causing clinical problems is difficult as a repeat laparotomy causes peritoneal inflammation and further adhesion formation. Laparoscopy can be scheduled in horses in which adhesions are suspected so that adhesions can be cut whilst inducing minimal inflammation. In some cases a regular feeding schedule and low residue diet may reduce the severity of clinical signs.

Take Home Message: The true number of horses with adhesions after colic surgery is unknown. They are a common cause of recurrent colic after surgery and are difficult to correct once they have formed.

CONCLUSIONS

Although overall mortality after colic surgery has declined with advances in treatment, certain diseases remain a challenge in the postoperative period. Future research that improves our understanding of the pathophysiology of ileus, adhesions, laminitis and colic itself should reduce this mortality rate. In the interim, excellent postoperative care and monitoring to prevent hypovolemia, reduce inflammation and maintain organ function is crucial to a successful outcome.

References


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The equine foot is not strictly defined by the NAV, but is generally taken to mean the hoof and all the structures contained within it. The digit refers to all structures distal to the metacarpophalangeal joint. For the purposes of this discussion, functional anatomy is used to both describe the anatomical structures as static entities and how these structures interact in motion. Functional anatomy of the foot is important to clinicians because it influences how surgical approaches to structures within go the foot are made, how the foot is stabilized after injury, and how lameness problems arising within the foot are treated. Additionally, increasingly sophisticated diagnostic techniques necessitate increasingly precise knowledge of the structures within the foot.

The anatomical structures within the foot/distal digit include the middle and distal phalanx, and distal sesamoid bone, ligaments, interphalangeal joints, common digital extensor and deep digital flexor tendons, the integument, and associated vessels and nerves. For the purposes of this discussion these structures will be discussed as part of two functional units, the hoof and the distal interphalangeal joint.

**Hoof**

The hoof itself is the integument of the foot. As such, it has three principle layers, the subcutaneous tissue, dermis, and epidermis. The epidermis is further subdivided into the stratum basale, stratum spinosum, and stratum corneum. The stratum basale and stratum spinosum are frequently referred to collectively as the stratum germinativum. The stratum corneum of the hoof forms the hoof capsule. The hoof is divided into 5 regions, limbic (perioplic), coronary, parietal (lamellar), solar, and cuneate. Each region shows specialization within each layer. For example, the subcutaneous tissue of the coronary integument forms the coronary cushion, the parietal subcutaneous tissue forms the periosteum of the parietal surface of the distal phalanx and ungual cartilage, and the subcutaneous tissue of the cuneate integument forms the digital cushion. The specialized junction of the epidermis and dermis to form papillae and rete pegs in the limbic, coronary, solar and cuneate regions and interdigitating lamellae in the parietal region is well documented. The three layers of the wall, stratum internum, stratum medium, and stratum externum, are formed by the stratum corneum of the limbic, coronary, and parietal integument respectively. The distal phalanx is considered to be suspended within the hoof capsule by the lamellae around approximately 80% of the circumference of the foot. Palmarly, the deep digital flexor tendon supports the distal phalanx and the distal sesamoid bone.
The hoof wall is not uniform throughout its thickness. The density, size, and structure of the horn tubules vary substantially from the outer wall to the inner wall. But more importantly, the alignment of the keratin fibrils within the intertubular horn varies across the substance of the wall, and it is the intertubular horn that forms the basis for much of the mechanical properties of the hoof capsule. The properties of the wall are comparable around the circumference of the hoof, but because it is thickest dorsally and thinnest palmarly, it is more flexible palmarly than dorsally. The hoof wall is viscoelastic. The outer wall is stiffer than the inner wall. The hoof capsule is more fracture resistant than bone, and it is structurally designed to deflect all microfractures away from the underlying sensitive tissues regardless of which plane the fracture is made in.

**Distal Interphalangeal Joint**

The distal interphalangeal joint is complex. It has three articulations, between the middle phalanx and the distal phalanx, between the middle phalanx and the distal sesamoid, and between the distal phalanx and the distal sesamoid bone. The bones are maintained in apposition by the collateral ligaments, the collateral sesamoidean ligaments, and the distal sesamoidean impar ligament. The head of the middle phalanx, has two condyles separated by a shallow groove that conform to the opposing articular surfaces of the distal phalanx and distal sesamoid bone. There is minimal movement in the articulation between the distal phalanx and the distal sesamoid so that they essentially function as a singular articular surface. The shape of the articular surfaces and the constraints of the ligaments ensure that the primary plane of motion is flexion and extension, but the shallowness of the intercondylar groove of the middle phalanx and corresponding low saggital ridge of the distal phalanx permit significant rotation, sliding and collateral motion.

**The stride and weight-bearing**

The stride is divided into suspension and stance phases. The stance phase is further subdivided into initial contact, impact, support, and breakover phases. Initial contact usually occurs laterally at either the heel or quarter. Less frequently the foot lands flat, and toe or medial first landings are rare in healthy horses. As the body descends up to the midpoint of the weight-bearing phase of the stride, the metacarpophalangeal joint dorsiflexes and the distal interphalangeal joint flexes. Therefore, structures that extend across the flexor surface of the the MCPJ, but not the DIPJ, namely the superficial digital flexor tendon and the suspensory ligament/distal sesamoidean ligaments, come under tension, lengthen, and absorb energy. In contrast, a structure that extends across both joints lengths minimally. During the second half of the weight-bearing phase of the stride, the distal interphalangeal joint extends, so that structures that extend across both joints, or just the distal interphalangeal joint come under greater tension, structures such as the deep digital flexor tendon and its associated accessory
ligament, and the collateral sesamoidean ligaments of the distal sesamoid and the distal sesamoidean impar ligament.

Weight-bearing by the limb during the stride is discussed in the context of the ground reaction force. The ground reaction force is broken into 3 components: vertical, horizontal in the direction of travel, and horizontal perpendicular to the direction of travel. The vertical component can be considered to be weight-bearing, and the horizontal component in the direction of travel can be considered to be breaking and propulsion. The graph of the vertical component of the ground reaction force against time forms an inverted U in a trotting horse; i.e. the force is least at the beginning and end of the stride and greatest at mid-stance. Superimposed on this graph during the impact phase of the stride are high frequency vibrations, which are thought to be responsible for much of the injury associated with athletic performance. There are several mechanisms to dampen the forces of impact: hoof viscoelasticity, a hydrodynamic mechanism involving the vascular channels within the ungular cartilages, and the viscoelasticity of the articular cartilage of the interphalangeal joints. Although, this damping occurs at several levels, the majority of it occurs within the hoof, that is, in the hoof capsule and the underlying layers of the integument that attach the hoof capsule to the distal phalanx.
Examination of the Equine Foot
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There are excellent journal articles and book chapters that describe the examination of the foot, some of which listed as references.1-8 Reviewing them reveals that each clinician performs their examination in their own style, and they emphasize different aspects of the examination, but that all have a method and all describe an effective process to diagnose as effectively as possible the various conditions that affect the equine foot. This article describes the author’s approach to examination of the foot, which is a synthesis of formal education received, personal experience and the experience of others. It is a reductionist approach, rather than procedural.

The majority of disease processes originating in the foot that cause lameness are associated with inflammation, usually related to trauma or infection. Disease processes associated with a marked focus of inflammation, typically associated with acute onset of lameness, are most likely to be identified with a basic examination, whereas disease processes associated with subtle symptoms and longer duration may require much more extensive examination of the limb and ancillary diagnostic tests. Furthermore, even the best examination in conjunction with ancillary diagnostic tests may not always obtain a definitive diagnosis, but the information gained may suggest an approach for symptomatic treatment. The latter is particularly important when access to advanced diagnostic technology, such as magnetic resonance imaging, is limited. Therefore, the following discussion is divided into two parts, the basic examination, and a more detailed examination. The detailed examination is further divided into three main sections that provide different types of information.

All examinations begin by gathering the presenting complaint, signalment, and history. With most foot problems, the presenting complaint is lameness. However, presenting complaint may also be the appearance of the foot. The signalment for any horse does not give specific information about the presenting complaint, but it does contain risk factors for certain conditions, which must then be correlated with information obtained from the history and physical examination.

There are three main time points of importance in history taking, the date of the examination, the date the problem was first noticed, and the date the owner first knew/owned the horse. The second date gives an indication of the duration of the problem, and the length of time between the second and third dates gives the clinician an indication of how much the history
prior to this problem is known and may lead to further enquiry about this time. No two sets of questions asked during a history taking are the same because so many questions are predicated on the answer to a previous question, but there is a common set of starting questions. After ascertaining the duration of the problem, questions should be directed at determining the nature of the onset – acute or chronic – and whether a specifically identifiable event can be linked to it. The clinician needs to know the progression of the disease, whether it has been constant, has become worse or better, or has varied over the course of the history. Additionally, it is important to see if any treatment measures have already been taken, and if so what effect they have had. If the horse is able to work, what is the influence of exercise on the problem, and how does it change with the surface the horse is worked on? Importantly for feet problems, it is also important to determine the shoeing history if it is shod: when was the horse last shod, have there been changes in the shoeing technique, has the farrier changed prior to or during the course of the problem, etc. More general questions may need to be asked about the health and management of the horse including questions about any other problems the horse has had in the past, the husbandry of the horse, and disease prevention management.

I. The Basic Examination

1. Examination of the Horse
It is important to examine the horse as a whole and not rush to examine an extremity. The body condition of the horse should be assessed. Chronic pain related to lameness can cause weight loss. Pressure sores indicates that a horse has spent an excessive amount of time lying down. A horses posture may not only indicate which limb is lame, but it may also provide information about the nature of the problem, the classic example of which is the rocking back stance seen with bilateral forelimb laminitis. If one heel is persistently held off the ground, it suggests that tension in a structure on the flexor surface is causing pain.

2. Basic Examination of the Foot
   a. Gross examination of the shape of the foot.
   It is important to have some appreciation for the size and shape of normal feet when conducting the examination, and while most observations on the size and shape of feet are subjective, values for the length and angle of the toe and the size of the foot in relation to the weight of the horse have been published.9,10 The fore feet are more circular in shape when viewed from the ground surface, whereas the hind feet may appear to be shaped like a diamond in which one end has been cut off.

   A visual inspection of the feet begins by walking around the horse and then looking at each foot
more closely while it is on the ground. This should identify large defects in or distortions of the hoof capsule, scars, obvious swellings proximal to the coronary band, and mismatched feet. Then the feet are picked up for examination of the ground surface of the foot, at which time changes in the loss of concavity and defects in the sole, and changes in the width of and defects in the white line are noted. In this manner, hoof wall avulsions and heel bulb lacerations are readily identified. Likewise, marked concavity of the dorsal hoof wall in conjunction with a flattened or convex sole are almost pathognomonic for laminitis. Severe distortion of the hoof capsule proximodorsally is indicative of underlying proliferation of bone on the surface of the extensor process of the distal phalanx, often referred to as buttress foot. When in doubt about a questionable finding in one foot, examining the contralateral foot may determine whether or not it is abnormal. It should be borne in mind that some mild asymmetry between feet is within normal limits.

Swelling proximal to the coronary band may reflect a disease process in pastern or within the foot. The distribution of swelling may be indicative of the nature of the problem. Circumferential swelling around the coronary band that extends up into the pastern is frequently associated with cellulitis. A small area of focal swelling at the coronary band is likely to be associated with an abscess that is about to spontaneously break open and drain. Inflammation of certain structures within the foot have characteristic patterns of swelling seen at or proximal to the coronary band. Inflammation of the distal interphalangeal joint results in dorsal symmetrical swelling, inflammation of the navicular bursa results in symmetrical swelling in the palmar aspect of the foot between the collateral cartilages, and inflammation of a collateral cartilage causes a unilateral welling proximal to the quarter of the hoof capsule.

The texture of the surface of the wall of the hoof capsule is normally smooth and therefore appears shiny, but when it becomes roughened and irregular, it appears dull and may change to a lighter color. This usually reflects inflammation at the coronary band, though nutritional factors may potentially cause similar changes. It may involve all feet, one foot, or part of one foot. When it extends to the coronary band, it suggests that the problem is still active. If it is only one portion of the circumference of the foot then the inflammatory process is localized. Because the wall migrates distally from the coronary band, the extent of the roughening proximodistally in relation to the length of the wall gives some indication of the duration of the process including the date it ceased if the inflammatory process is not currently active.

**b. Palpation**

Palpation of the foot is the most practical way to assess the temperature of the foot on a routine basis, and palpation of the palmar digital artery is performed to assess the pulse pressure (“digital pulse”), both of which are indicators of inflammation within the foot. The
clinician should bear in mind that some variability in temperature of the hoof and strength of the digital pulse is normal; comparison with the other leg and repeated observations are helpful to confirm importance of finding if in doubt. The pastern is palpated to detect possible surgical scars and the palmar digital nerve for swelling and pain on deep palpation to detect a neuroma. The texture of the surface of the hoof and skin of the pastern is most appropriately assessed by feel, particularly roughness of the surface of the hoof capsule and thickening or irregularity of the skin of the pastern. Palpation may also detect the presence of moisture on the foot and pastern, particularly at the hairline, that is not otherwise detectable. Examination of the coronary band may reveal a depression immediately proximal to the hoof capsule indicative of distal displacement of the distal phalanx.

Palpation is used to determine whether a swelling proximal to the coronary band is firm, edematous or fluctuant. Digital pressure is applied, at first gently and then more firmly, to identify a focus of pain if present. Structures proximal to the coronary band that may be palpated directly through the skin include the proximodorsal distal interphalangeal joint capsule, proximal aspect of the distal interphalangeal joint collateral ligaments, proximal interphalangeal joint collateral ligaments, middle and proximal phalanges, common digital extensor tendon, deep digital flexor tendon and sheath, collateral cartilages, and digital cushion. Pressure may be applied to the navicular area through the deep digital flexor tendon and digital cushion. Deep sulci may need to be palpated for discomfort with the aid of a tongue depressor. The flexibility of the collateral cartilages may also be determined by palpation.

c. Manipulation
The distal limb should be flexed and extended to determine if it elicits a pain response or if there is a reduced range of motion. In general, structures that are associated with flexion/extension during normal locomotion, such as the tendons and their sheath, ligaments, joints, and the navicular bursa, are likely to elicit a painful response. Usually pain associated with the hoof capsule and non-articular portions of the distal phalanx are unlikely to do so. However, manipulation should be interpreted cautiously because instability between two parts of the hoof capsule or instability between the hoof capsule and underlying distal phalanx may also be stressed by such handling. Rotation of the foot in relation to the remainder of the digit shows a remarkable degree of mobility in normal horses, which should not be interpreted as abnormal. Marked sprains of collateral ligaments might be expected to elicit a painful response following manipulation in this manner.

d. Compression and percussion
Detection of pain within the foot is difficult with palpation because of the rigidity of the hoof
capsule. Therefore, compression and percussion are used to identify and localize pain within the foot. Hoof testers are used to compress the foot. They should be applied in a systematic manner, typically starting at one heel, progressing around the quarter, toe, opposite quarter and heel, followed by compression across both heels, and from each side of the frog to the opposite heel. In addition to progressing around the foot, systematic application of hoof testers must also evaluate the sole at different distances from the white line. When the initial withdrawal response is mild, consistency with repetition and comparison with the contralateral limb is needed to determine if the response is clinically significant. In addition to eliciting a pain response, compression of the foot may also cause moisture to be expressed from defects in the hoof capsule, and identify instability of fissures in the hoof capsule. Flexion of sole with hoof testers gives some indication as to its thickness. When applying hoof testers it is frequently assumed that any pain response identified is coming from the solar aspect of the foot. While this is frequently the case, it should be borne in mind that hoof testers might elicit a painful response from any of the tissues between their jaws. For example, when hoof testers are applied across the toe, the tissues affected include the integument of the wall as well as the integument of the sole and the distal phalanx.

Percussion of the hoof capsule with a shoeing hammer is not performed as frequently as compression, but it occasionally yields information that hoof testers don’t, and it is useful when no hoof testers are available. While the foot must be elevated off the ground to percuss the sole, the wall may be percussed with the foot on or off the ground, though the latter is easier.

**e. Paring with a hoof knife**

The ground surface of the foot provides clues regarding the quality of the sole, evidence of past trauma, and defects that are potential entry sites for infection. Defects in the ground surface of the stratum medium of the wall and the white line cannot be identified if a shoe is present. The manner in which paring is performed and when it is performed is related to the presenting clinical symptom. In horses that present with an acute marked onset of lameness in which an abscess is the most likely diagnosis, paring the foot is usually performed promptly. However, removal of shoes and paring of the ground surface of the foot is not recommended for horses with mild lameness and no obvious abnormalities of the ground surface of the foot until after the horse has been exercised and all pertinent diagnostic analgesia performed. Not all lameness’s originating in the foot require paring of the sole, and when it is done caution should be exercised to preserve the thickness of the sole whenever possible. In particular, if a horse is suspected of having laminitis preservation of the sole thickness is very important, so paring should be very limited in extent or avoided. The frog may require trimming to expose the sulci, or to investigate primary diseases of the frog such as thrush and canker.
The ground surface of the foot in horses with marked lameness of abrupt onset is explored for puncture wounds and defects in the sole that are likely entry sites for infection. Both usually appear as dark marks. Naturally occurring defects are most likely to be present in the white line. Puncture wounds in the frog are frequently difficult to identify because the elastic nature of the frog causes the entry wound to close over, and punctures in the collateral sulci are difficult to identify because the exposure is poor. In horses in which the most likely diagnosis is an abscess, paring should be very focal and should extend through the full thickness of the hoof capsule until either purulent exudates or pinpoint hemorrhage is encountered. If the likely entry site is in the white line, it is preferable to explore the defect by enlarging the side adjacent to the wall rather than the sole, in effect creating a notch in the distal wall that extends proximally to the junction with the inner surface of the sole. In horses with milder more chronic lameness, paring the sole is kept to a minimum. Lightly debriding the surface of the sole frequently reveals hemorrhage related to bruising. Blood that extravasates into the sole maintains its red coloration and can further be distinguished from pigmentation because it has a stippled pattern as it migrates down and around the horn tubules.

**e. Evaluation of Shoes and Shoeing**

The shoes should be evaluated to determine that the size of the shoe is appropriate for the size of the foot and that the type of shoe is appropriate for the type of work the horse is performing. Uneven wear on the ground surface of the shoe may indicate areas of excessive weight bearing. If a shoe has been on too long it moves forward in relation to the heels and is likely to put pressure on the angles of the sole. Additionally, shoes that have been on too long may loosen and shift.

**A. The Detailed Examination**

1. **Morphological examination of the hoof**

The goal of examination of the detailed morphology of the hoof is to identify deformation of the hoof capsule and changes in the growth pattern of the hoof that may indicate the presence of abnormal distribution of stresses within the foot. Increased stress or weight bearing by a portion of the wall has three consequences that may be detected on physical examination: it may cause deviation of the wall outwards or inwards from its normal position; it may cause the wall to move proximally; or it may cause the growth of the wall decelerate. A reduction in stress or weight bearing for the most part has the opposite effect. In contrast to the situation where stress can retard hoof growth, there are occasions when hoof growth is accelerated in one part of the foot in relation to another, the best examples would be the growth pattern associated with inflammation or tumors.
To accomplish a more detailed morphological examination of the foot, the foot should be viewed from all sides when it is on the ground, and then the ground surface examined with the foot off the ground. Additionally, small changes in the shape of the hoof capsule may be better appreciated by careful palpation of the foot than by visual inspection.

When the foot is viewed from the dorsal aspect several abnormalities may be visible. Flares or under-running of the wall may develop at the quarters. The coronary band may be unevenly distributed. The most common would be the development of an even slope in the coronary band from one side of the foot to the other. However, more localized distortions of the coronary band may occur; a common example is that in which the coronary band in the median plane is distal to that at the toe-quarter junction. Examination of the growth rings may show divergence of the rings form one side to the other. The angulation of the dorsal horn tubules to the sagittal plane should be noted; normally they should be parallel, so when they appear tilted medially or laterally, it suggests that the whole hoof capsule may be tilted.

When the foot is viewed from the lateral aspect, flaring of the toe and underrunning of the heels is readily appreciated. The coronary band should normally slope evenly from the toe to the heels. Evaluation of growth rings indicates a disparity in the growth of the heel and the toe, typified by the increase in heel growth and decrease in toe growth commonly seen in horses with laminitis. However, regional irregularity in spacing of growth rings is not uncommon; the most frequently observed is a decrease in spacing at the quarter associated with proximal displacement of the coronary band.

The heels are evaluated from the palmar aspect for their overall width, and their height. The heels frequently become narrowed when the foot itself is narrow. Additionally, the central sulcus of the from may extend proximal to the hairline so that a cleft becomes apparent in the skin of the pastern between the heels. The overall height of the heels is readily assessed from the lateral aspect, but viewing from the palmar aspect is useful to compare the relative heights of the two heels; the classic example is the sheared heel in which one heel is displaced proximally in relation to the other.

If a three dimensional object changes in one plane, it will change in at least one other plane and this is certainly true for the horse’s foot. Therefore, examination of the ground surface of the foot reveals much about the changes in the wall of the hoof capsule. In general, the frog is usually constant in length and its axis is almost always aligned with the medial plane of the foot, but its width is variable. Normally, the curvature of ground surface of the wall should be smooth and it is almost symmetrical about the axis of the frog. The width of the ground surface
should be approximately equal to its length, the maximal width is approximately half way between the toe and heels, and the palmar margin of ground surface of the wall at its reflection are level with the base of the frog. Therefore, the contour of the wall can be examined in relation to it curvature and the position of the frog. These changes may affect the whole foot, and that means frequently symmetrical in the sagittal plane, or they may be regional, and therefore, usually asymmetrical. A narrow foot suggests that the toe is long or that foot expansion has become decreased, usually secondary to pain. In some individuals and some breeds the toe is deliberately maintained long, but at other times a long toe is inadvertent. A long toe will also be accompanied by an increased distance between the toe and the apex of the frog. When the ground surface of the heels are dorsal to the base of the frog, the heels are underrun and/or increased in length. If the contour of the wall is displaced away or towards the median plane in the dorsal two thirds of the foot this usually corresponds with a flare or underrunning of the wall respectively. If only one heel buttress is displaced dorsally in relation to the base of the frog, it usually corresponds with the proximal displacement of that heel plus or minus the quarter termed sheared heel.

2. Examination of the foot in relation to the rest of the limb
The structure of the distal limb is examined to identify features of that animals conformation or balance that may contribute to undue stresses in any part of the foot that may predispose to injury or disease. It is important that the structure of the distal limb is viewed both on and off the ground. When the distal limb is viewed standing on a level surface, except under unusual circumstances, weight bearing forces the ground surface of the foot to be perpendicular to the pastern, and internal structures may have to accommodate for this. With the foot off the ground, the constraint of weight bearing is not present and the ligaments are relaxed.

When the digit is viewed from the dorsal aspect with the foot on the ground, the pastern and foot should be in alignment, i.e. the median plane of the pastern and medial plane of the foot are parallel and in line with each other. If the median plane of the pastern appears to intersect the coronary band to one side of its center, it suggests that that side of the foot/hoof is elevated. When the digit is viewed from the side with the foot on the ground, the dorsal aspect of the pastern should be parallel with the dorsal hoof wall. This relationship is referred to as the foot-pastern axis. When the hoof capsule forms a more acute angle with the ground than the pastern, the axis is said to be broken-back, and when the angle is less acute, the axis is said to be broken-forward. This evaluation is somewhat subjective because it depends on which part of the pastern is being compared to the hoof, and the angle changes slightly with the phase of the shoeing cycle. The broken-back foot pastern axis is associated with increased tension in the deep digital flexor tendon and greater force on the navicular bone during the stride, particularly
at breakover. A broken-forward foot pastern axis, usually associated with a flexural deformity of the distal interphalangeal joint, it thought to predispose to concussion of the dorsal sole.

Additionally, when viewed from the lateral aspect, position of the fetlock is related to the position of the ground surface of the foot. There is a traditional metric that states that a vertical line that runs through the center of the metacarpus. It is a function of the angle of the foot-pastern axis, the length of the pastern, and the size of the ground surface of the foot. Like other aspects of conformation, its significance is uncertain, but rationally, if the horizontal distance between the fetlock and foot is longer, it suggests that there is greater stress on the supporting ligaments and tendons.

With the limb off the ground, the most common way to assess the relationship between the foot and rest of the distal limb is to hold the metacarpus horizontal and sight along the palmar aspect of the limb. In this manner, the relationship between the ground surface of the foot and an imaginary line across the heel bulbs can be evaluated in relation to the axis of the limb. The “ideal” relationship frequently cited is that a line drawn across any two comparable points from the medial and lateral sides of the hoof capsule should be perpendicular to the axis of the metacarpus. This ideal relationship is thought to provide optimal distribution of weight within the foot, and is therefore frequently used as a guide for trimming the foot. Unfortunately, this relationship varies with rotation within the metacarpus and pastern, and angulation at the metacarpophalangeal joint. Therefore, it is important that it is interpreted in conjunction with the appearance of the distal limb when placed on the ground and the morphology of the hoof capsule.

**3. Examination of the foot in motion**

Observation of the distal limbs in motion should be performed at both a walk and trot if possible, and should be performed from in front, to the side, and from behind the horse as it moves. It is only recently that advances in technology have allowed us to better understand the manner in which the foot moves at breakover, during flight, and on landing. It is now known that most horses land laterally at the heel or quarter, and less commonly land flat. Breakover is normally slightly lateral to or at the center of the toe. However, landing and breakover are rapid events that are difficult to discern by watching a horse at a trot and so are best observed at a walk. A toe first landing or medial first landing are both abnormal events at a walk, and warrant further investigation. Additionally, excessive lateral first or heel first landing is likely to be abnormal. When mediolateral asymmetrical landing can be detected at a trot, it is likely to be significant. Though the implications of abnormal landing and breakover are not fully understood, it is most likely to be for one of more reasons. It could be due the horse’s conformation. It could be due to the horse’s attempts to ameliorate pain in the foot. It could
also be that the normal range of the stride is altered, usually due to a more proximal focus of pain, and therefore, an earlier or later breakover or landing may influence what part of the foot contacts and leaves the ground first. In addition to changing the way the foot lands and leaves the ground, the horse may place its limb further towards or away from the median plane to redistribute weight, which would be accompanied by associated changes in flight after breakover and prior to landing. This is most likely to be seen when a horse positions its foot further from the median plane to reduce lateral weight bearing or vice versa. Flexion tests and toe/heel elevation tests are designed to stretch a set of structures prior to trotting to make an occult lameness apparent or an mild lameness more obvious.

C. Ancillary Diagnostic Aids
There are three main objectives to ancillary diagnostic tests: exploration of an injury, usually with a flexible metal probe; diagnostic analgesia to localize the source of pain; and diagnostic imaging to identify discrete pathology. Their discussion is beyond the scope of this article.

D. Summary
The manner in which the parts of the exam have been described is a reductionist approach because it is often easier to understand the significance of an individual finding in isolation. However, the examination is performed in the most clinically efficient manner, so that several characteristics of the limb are being observed at once. For example, when observing the distal limb on the ground from either the dorsal or lateral aspect, the morphology of the hoof capsule and the relationship between the foot and remainder of the distal limb when weight bearing are evaluated concurrently.

The basic examination is likely to provide a diagnosis for many common and simple disorders such as a foot abscess. The detailed examination may under some circumstances provide a definitive diagnosis, but is as likely or more likely to direct the clinicians attention to risk factors for injury and indicators for where abnormal stresses may be. The three main areas that are assessed provide different types of information as described previously. In brief, the morphology of the hoof capsule reveals deformation and changes in growth that occur following increased or reduced force, and the relationship between the limb and the foot indicate conformations that may predispose to abnormal weight bearing. Observing the limb in motion is most helpful to corroborate with findings identified when the horse is examined at rest; however, as there is limited data available for comparison of the landing and breakover patterns with different disease states, if is more of an art than a science at present.

The correlation of the clinical findings to suggest a probably diagnosis may be good. However,
at other times the clinical findings may have conflicting implications; this may point to two separate clinical problems or a break in our understanding of the pathogenesis of the changes in structure of function observed. The latter is particularly important when attempting symptomatic treatment in the absence of a definitive diagnosis for whatever reason. In this instance a measure of trial and error is warranted, and it is based on the preponderance of the evidence rather than the total evidence.

Lastly, it should be remembered that for some purposes the hind feet can be considered very similar in structure and function to the forefeet, for example in the diagnosis of abscesses, avulsions, and distal phalanx fractures. However, when diseases such as those that can be attributed to hoof imbalance are considered then the structure and function of the hind foot should be considered separately to the forelimb. This is because the stresses associated with locomotion are sufficiently different (and less well understood) that changes in the hoof capsule should not necessarily be interpreted in the same manner as they would be in the forelimbs.

References:


EQUINE FOOT WOUNDS
(Modified from an article in the American Farriers Journal)
Andrew Parks

INTRODUCTION
Wounds of the horse’s foot are common, and many are simple and heal without untoward consequences. Unfortunately, others may cause lasting changes in hoof function, and sometimes even be life threatening. The term foot describes the hoof and all structures contained within it. It is part of the appendicular skeleton and as such most of the same elements, i.e. bones, joints, tendons and ligaments encased by the integument. What makes the foot different is its integument the hoof because of its specialized function, bearing weight, as well as the other functions of skin. This function necessitates a specialized structure, growth pattern and attachment to the rigid structures of the appendicular skeleton, all of which are important considerations in the treatment of foot wounds and restoration of normal function.

The management of horse’s feet spans both the farriery and veterinary professions, and this is no less true for foot wounds than any other problem. The diagnosis, and medical and surgical treatment of foot wounds lies within the province of the veterinarian. However, a farrier is commonly the first professional called to examine a horse with a foot problem, and therefore, needs to recognize injuries that need veterinary attention. Also, during the treatment of these wounds farriers are commonly called upon to design and apply surgical shoes, and long after medical and surgical management of the injury has ceased, it is the farrier who must often ensure that optimal function of deformed feet is maintained by careful trimming and shoeing. A basic understanding of the types of foot injury that occur and how they heal is essential for obtaining the optimal result and providing the owner the most accurate prognosis.

THE NATURE OF FOOT WOUNDS.
The very position of the foot at the end of the limb in repeated contact with the ground places it in constant jeopardy due to trauma of sharp objects, despite its admirable design. Superficial injuries to the keratinized layers of the epidermis seldom incite an inflammatory response or cause infection and unless they cause instability, resolve uneventfully through the natural process of hoof replacement. Injuries that penetrate the basal layers of the epidermis, dermis and deeper are potentially much more serious. The most common wounds are punctures and lacerations. Puncture wounds occur most often due to a nail in the sole and frog. They are characterized by a small entry wound that may be hard to find once the nail has gone, particularly in the frog and sulci where the rubbery nature of the hoof causes the margins of the wound to spring back together. This type of wound gives no indication of the depth or direction of the injury and therefore little indication of which structures are damaged. They are often very painful.
Lacerations of the hoof more commonly involve the wall and adjacent skin than the sole. They are caused by tearing of tissues, and in skin this is seen as wounds with ragged margins and varying depth, though usually fairly shallow and may be associated with loss of the integument. The damaged tissues are well exposed and in contrast to puncture wounds the superficial appearance of the wound exaggerates its seriousness, and are not necessarily as painful as expected. Because of the semi-rigid nature of the hoof capsule, a flap of hoof is torn away from the laminae. This flap may involve only the laminar portion of the wall, or may also include the coronary band and the skin proximal to the coronary band. In incomplete avulsions, the flap is usually hinged along one or sometimes 2 borders. In complete avulsions the flap has become completely detached and the wound must heal by secondary intention. Incomplete avulsions that do not involve the coronary band are best resected and the defect allowed to heal by secondary intention. Incomplete avulsions that involve the coronary band have been successfully treated by preservation of the coronary band and surgical repair.

Accidental incised wounds that result from a cut by a piece of sheet metal or cut glass (as opposed to deliberately incised wounds made with a scalpel) without avulsion of the surrounding integument are rare. The sides of the wound do not separate; unlike puncture wounds the entry wound is readily visible and the direction taken by the foreign object apparent, therefore, it is easier to predict what structure are involved compared to puncture wound, but harder than with lacerations. Subsolar abscesses form an interesting case. There is no break in the keratinized epithelium except sometimes a small crack. When the keratinized epithelium is pared away to expose the abscess cavity, it is usual to find a thin soft white layer of epithelium that covers the deep surface of the wound. The presence of a complete layer of epithelium on the deep surface of the wound regardless of the size of the defect and the duration of the abscess implies that the abscess is at the base of the epithelium and not in the dermal or subcutaneous tissues because complete epithelial defects must epithelialize from their margins (as explained later), which takes several days and in the meantime the defect would contain granulation tissue. Therefore, it is likely that exudate from the abscess dissects the keratinized epithelium away from the basal layers to form a false sole. Occasionally an abscess is subepidermal as evidenced by the presence of granulation tissue on the deep surface, or necrotic dermis, which will be replaced by epidermis.

**WOUND HEALING: A GENERAL DESCRIPTION**
Unfortunately, there is limited research on the healing of foot wounds. One study has shown that when the hoof wall is mechanically stripped that epithelial elements are left on the surface of the underlying defect. This defect is therefore capable of healing in a manner comparable to
partial thickness skin graft donor sites. Apart from this, our knowledge of this process is based on clinical experience and extrapolation from research on the healing of skin wounds. Consequently, it is best to examine the way a wound through the skin heals, and then consider the differences between this and wounds through the hoof.

Classically wound healing is divided into 4 stages: inflammatory, debridement, repair, and maturation. In the inflammatory phase, the small vessels transiently constrict to limit hemorrhage is rapidly followed by vasodilation. The wound is infiltrated by a plasma-like fluid and white cells that form a clot, which form a protective barrier over the wound surface. that later becomes a scab.

Debridement involves the removal of all dead tissue, blood clots, and foreign material from the wound surface. This occurs by phagocytosis by specialized white cells called macrophages, sloughing from the exterior surface of the wound, or surgical debridement.

Repair occurs through the action of 3 processes: epithelialization, formation of granulation tissue, and contraction. Epithelialization occurs through the proliferation of epithelial cells at the margins of the wound and their subsequent migration across the surface of healthy dermis and granulation tissue. This migrating cells form a very thin layer of epithelium across the surface of the wound that increases in thickness once the movement of the basal cells is arrested. Fibroblasts proliferate at the from all healthy tissues in the depth of the wound and begin to fill the defect. The fibroblasts are responsible for the laying down of collagen, which provides strength to the wound repair. Newly formed capillary loops closely follow the advancing border of fibroblasts. Together, the fibroblasts and capillaries form granulation tissue. All healthy tissues are capable of forming granulation tissue, though dermal tissues granulate faster than bone, ligament and tendon. Concurrently, where elasticity of the wound margins allow, contraction of the wound surface area occurs through active development of tension in the wound margins.

Wound maturation proceeds once the wound has completely granulated and epithelialized, there is a gradual loss decrease in vascularity and cellularity of the wound. At the same time the scar tissue increases in strength due to reorganization and cross linking of the collagen.

This description implies that a wound goes through chronologically distinct phases, whereas the process is of course a continuum. Not only that, but different areas within a wound are likely to be at different stages at any one time.

**FACTORS EFFECTING WOUND HEALING**
Many factors effect the rate at which wound heal including the systemic status of the animal, the local wound environment, systemic medications, and topical medications and bandaging. Discussion of the effects of systemic status and nutrition on wound healing are beyond the scope of this manuscript.  
Local factors that impede wound healing include the presence of necrotic tissue, foreign material, infection, blood clots, tissue edema, poor blood supply, dessication, movement and low temperatures. Necrotic material and foreign matter must both be harbor bacteria and allow infection to persist, and present a physical obstacle to wound repair. Infection causes persistence of inflammation and further tissue damage which prolongs débridement and delays granulation and epithelialization; the accompanying exudate is a physical barrier to wound healing.

Topical medications are usually aimed at controlling infection. Both the product and formulation are important in determining the effect of any medication on wound healing. In brief, solutions appear to be more beneficial than ointments and creams. Topical antiseptics are beneficial in low concentrations, e.g. 0.1-0.2% povidone iodine and 0.05% chlorhexidine gluconate, but are harmful in higher concentrations. There appears to be no place for tinctures of iodine or formalin in the treatment of foot wounds, and while they may appear to be beneficial in treating superficial infections of the epidermis at best, they are a poor substitute for adequate débridement. They are severely destructive when placed in direct contact with living tissues.

The nature of any surface dressing is also important. Adherent dressings, such as cotton gauze, lift adherent tissue off the surface of a wound when they are removed; this effect is beneficial when débriding a wound but deleterious to a healthy granulating and epithelializing surface. Nonadherent dressings are subdivided into occlusive and non-occlusive. Non-occlusive dressings allow exudate to pass through, but occlusive dressings cause retention of exudate at the wound surface with deleterious results. Even within the different types of non-occlusive dressings there are differences. Petrolatum impregnated gauzes promote wound contraction and granulation tissue formation, but inhibit epithelialization. In contrast, Telfa pads do not impede epithelialization

Bandages assist wound healing through several other effects other than the obvious protection from further mechanical injury. At the same time as wicking exudate away from the wound surface, they prevent dessication. Accumulation of exudate effects a wound negatively by macerating tissue surfaces as well as functioning as a barrier between wound surfaces. Dessication of the epithelial margins causes scab formation that inhibits epithelial migration across the wound. By directly exerting pressure on the wound surface, a bandage may help to
control tissue edema. Also, a bandage may retain heat at the wound surface, enhancing the rate of wound healing.

**Wound Healing: The Foot as a Special Case**

The similarities between the healing of foot wounds and wounds elsewhere on the limbs far outweigh the differences, but it is inevitably the differences that attract attention. The most noticeable differences between healing of skin an hoof defects relate to the differences in physical characteristics and pattern of normal replacement. Also, the position of the foot at the extremity means that there are less adjacent soft tissues compared to the more proximal limb, it is cooler than the more proximal limb and it is more prone to severe contamination.

The physical properties of the hoof and the nature of the injuries that occur make suture closure of wound less practical compared to skin wound. Fortunately, this is seldom warranted except for injuries of the coronary band.

The rigid structure of the hoof wall and sole cause it to act as a cast around the structures it encompasses. Like a cast over a wound, this is both good and bad; a cast stabilizes and protects the tissues within, but does not allow observation of the wound surface underneath it, and it impedes movement of air and fluid to and from the wound surface. When the hoof is punctured or lacerated, the margins of the wound do not retract. Similarly, the rigid nature of the hoof prevents contraction of the wound margins of defect during the repair process. In the frog, the spongy nature of the epithelium actually causes the edges of small wounds to spring back to close over the entry wound. Because the hoof does not expand, the inflammatory response causes the pressure to increase within the foot, an inherently painful process. Also, the drainage of exudate and the sloughing of necrotic tissue is inhibited. Hence the need to expose many wounds by removal of at least the stratum medium of the epidermis.

However, the cast like nature of the hoof is not all bad in relation to wound healing. If wound debridement and control of infection can be established leaving the hoof wall intact, the hoof constrains the growth of granulation tissue until is epithelialized, preventing exuberant granulation tissue (proud flesh) from developing. Having said this, in my experience the development of proud flesh on the foot is quite rare even when large portions of the wall are missing, though it is common on the heel bulbs and pastern. When exuberant granulation tissue does occur, it is most common on the sole of laminitic horses following prolapse of the distal phalanx. One explanation for this is excessive pressure at the dorsal margin of the distal phalanx preventing epithelialization.

Another reason for maintaining the structural integrity of the wall as much as possible during the exploration of foot wound is the preservation of stability of the hoof. Motion of any wound
impedes healing, but this is perhaps particularly important in the hoof because hoof defects that parallel the line of wall growth may persist as hoof cracks.

The epithelium and dermis of the skin are relatively uniform in structure wherever they are on the limb and hence the process of epithelialization proceeds fairly uniformly. In contrast, the subspecialization of the epithelium and dermis of the foot to form the coronary band, the wall, the sole and the frog means that the return of full thickness epithelium over the hoof is not necessarily a uniform process. Granulation tissue formation and epithelial cell migration over the surface of the defect in these areas proceed as they would elsewhere on the limb, but the manner in which the epidermis returns to normal thickness varies with location because of the specialized way in which the hoof wall and sole are replace in the process of normal growth. Defects in the sole increase in thickness by proliferation locally. In contrast, once laminar defects have epithelialized and the initial epithelialization has taken place, there is limited local proliferation and keratinization of the epithelium, but full thickness replacement of the stratum of the coronary epithelium as it moves from coronary band distally.

Some hoof avulsions start in the laminar region, span the coronary band, and extend up into the skin of the pastern region. Epithelium migrates from all three areas to fill the defect. Therefore what type of integument results over the surface of the wound depends on where the epithelium migrated from. Therefore, epidermis derived form the skin may replace part of the defect below the coronary band, or hoof forming epithelium may appear proximal to the coronary band. Of the two, the latter is more common in the author’s experience. Of particular importance is the nature of the epithelial tissue replacing the coronary band because this determines the structure of new hoof wall growth. It should be borne in mind that surgically created wound involving the coronary band heal in the same manner as accidental wounds, hence the extreme reluctance of most surgeons to disrupt the coronary band to gain access to deeper structures of the foot. Separation of the hoof wall at the coronary band caused by a gravel should not be confused with a complete epithelial defect; usually in these circumstances the keratinized epithelium has separated from the germinal epithelium. Hence the defect grows out and is replaced by normal wall.

It is interesting to note that the distal phalanx appears to respond differently to injury compared to the other phalanges and the metacarpus. When part of the distal phalanx has débrided, the defect only becomes mineralized very slowly radiographically if at all, nor does the distal phalanx mount an aggressive periosteal response on the adjacent surfaces as seen elsewhere. In fact, avulsion wounds that remove large portions of the wall adjacent to the distal phalanx, but do not damage the bone itself cause considerable bone demineralization. Despite this loss or demineralization of coffin bone, the future function of the foot appears to
depend more upon the structural integrity of the hoof than the radiographic appearance of the distal phalanx.

The restoration of function of all injured body parts including the integument is the ultimate goal of any reparative process, but given the greater expectations of the hoof compared to skin makes it seem all the more important.

**DEEP DIGITAL INFECTION**

Infection within the horses hoof is a common cause of lameness. Fortunately, most are subsolar abscesses, which do not usually extend deeper than the basal layer of the epidermis and respond favorably to treatment. In contrast, deep digital infections, though less common, extend deeper than the dermis and may have far more devastating consequences. Horses with both superficial and deep digital infections are often severely lame upon presentation. Unfortunately the depth of the injury is often not appreciated upon initial examination or the significance is underestimated. Therefore deep digital infections are often initially treated as superficial infection; they are not recognized for what they are until they fail to respond to "normal" therapy. Consequently, appropriate therapy is often delayed. It is important that the veterinarian have a thorough understanding of both deep and superficial infections.

Deep digital infections occur most commonly from direct introduction of bacteria into the deeper structures of the foot following puncture wounds, but may also follow lacerations and extension of infection from subsolar abscesses. A detailed knowledge of digital anatomy is essential in predicting the potential involvement of tissues based on the location of the entry wound, the direction and depth of penetration of the foreign object, the likelihood of infection spreading from one structure to another, and the possibility of iatrogenic damage occurring during treatment. The most commonly used diagram to demonstrate the relationship of different structures to each other in the hoof is a mid-sagittal section. It is important to remember that these relationships change away from the midline.

Of particular concern are the distal phalanx, the navicular bone, the navicular bursa, the collateral cartilages and the distal interphalangeal joint as these are the structures associated with a guarded to poor prognosis when infected because the infection is either refractory to treatment or life threatening. Of particular importance is the close proximity of the navicular bone, navicular bursa, distal interphalangeal joint and deep digital flexor tendon deep to the middle third of the frog. This means that more than one structure may be involved because of the original injury. Alternatively, spread of infection from one structure to another may occur at a later date by natural extension of the disease process or by iatrogenic spread from surgical interference. Also of importance is the close proximity of the collateral cartilages to the palmar reflections of the distal interphalangeal joint capsule. Hence, the need for extreme caution
when exploring and debriding in these areas.

**Diagnosis**
A good history is invaluable in evaluating any disease process. It is important to know the speed on onset, the duration and progression of the problem. Typically deep digital infections are associated with a rapid or sudden onset of a severe lameness that shows no signs of improvement. However, removal of a foreign body from a wound may cause a transient dramatic improvement only to be followed by recurrence of severe symptoms as the infection becomes established in 24-72 hours. Knowledge of the nature of the original injury may speed up the diagnosis; lacerations are usually obvious but puncture wounds may be difficult to locate if not seen at the time of injury. Prior treatment and its effect, if any, should be determined. Deep digital infections have often been treated as subsolar abscesses and failed to respond.

On presentation the horse is usually extremely lame in the affected limb. Examination of the hoof may indicate increased temperature. The location of any swelling proximal to the coronary band should be noted. Pain may be evident with systematic application of hoof testers. Lacerations are usually obvious but paring out the sole and sulci is often required to identify puncture sites seen as areas of discoloration. Draining tracts may be apparent on careful visual inspection or palpation of the coronary band.

Wounds and draining tracts require exploration to identify structures that may be damaged. This is greatly facilitated by local anesthesia. After aseptic preparation lacerations can be explored digitally, punctures should be cautiously explored with a sterile probe. If infection of the distal interphalangeal joint or navicular bursa is suspected, aspiration of synovial fluid from a site unaffected by the infectious process distant to the injury is indicated; cytology may provide definitive evidence of inflammation and strong support of infection. Following aspiration of synovial fluid, distension of the synovial space with saline may demonstrate communication between the synovial cavity and a wound.

Radiography is an important adjunct to the diagnostic examination and a useful technique in monitoring progression of the disease. If a foreign body is present it is important to radiograph the foot prior to its removal because this can accurately demonstrate the path taken by the object. While fractures of the distal phalanx or navicular bone might certainly explain the severe lameness, they may or may not be related to a puncture injury as they may have been pre-existing. Radiographic evidence of lysis of the navicular bone or distal phalanx is often indicative of osteomyelitis. Sequestra, pieces of dead bone that act as a foreign body, are suggested by the presence of an isolated fragment of bone that shows no periosteal response surrounded by a zone of osteolysis. Sequestra around the margin of the third phalanx are
usually obvious, but sequestra within the saucer shaped cavity of the solar surface may be occult. Septic arthritis of the distal interphalangeal joint may initially show no radiographic abnormalities, an increased joint space due to an increased volume of synovial fluid, or a decreased joint space due to loss of articular cartilage; with time, lysis of subchondral bone and periarticular new bone are apparent. Subluxation of the distal interphalangeal joint with dorsal displacement of the distal phalanx indicates rupture of the deep digital flexor tendon. Proximal displacement of the navicular bone indicates rupture of the impar ligament. Gas shadows in the soft tissues most likely indicate the presence of a cavity connected to atmospheric air but may be caused by gas producing organisms. As only osseous tissues are radiodense, soft tissue injuries are not apparent. To assist in delineating tracts radiodense probes or liquids can be inserted or instilled into tracts and when 2 radiographic projections at 90 degrees to each other are used, the site of the tract or cavity can be deduced. Liquid contrast media are better able to delineate the margins of a cavity and can go around corners, but are not as radiodense as metal probes.

Bacterial cultures should be taken from tracts after aseptic preparation and prior to exploration, and from synovial aspirates.

**GENERAL THERAPEUTIC PRINCIPLES**

Symptomatic therapy, including pain control with phenylbutazone and broad spectrum antibiotics, including intravenous regional perfusion if necessary, should be started immediately. Thereafter, treatment of horses with deep digital infections is best performed in an equine hospital and not on the farm. An accurate assessment of the injury is imperative before surgical therapy can be planned. Good restraint is advisable; the author prefers to work on difficult foot wounds with the horse under general anesthesia, but sedation and local anesthesia may on occasion suffice.

Aseptic preparation is required especially when a synovial structure is involved. As the size of puncture wounds are misleading in relation to the underlying damage adequate exposure is essential. The hoof is not readily mobilized as is skin, therefore, the overlying epidermis and dermis have to be removed. All obviously devitalized tissue should be debrided. Debridement of tissue of questionable viability depends on its location; complete debridement reduces contamination and minimizes healing time. However, in order to reduce the risk of iatrogenic contamination, it may be beneficial to leave questionable tissue immediately adjacent to synovial structures that are not currently infected; these tissues can be re-examined and debrided later if necessary. Further bacterial cultures may be taken at surgery.

Postoperative antibiotic therapy including intravenous regional perfusion is modified based on the results of the culture and sensitivity. The duration of antibiotic administration varies with
the duration of the problem and the structures involved. For most soft tissues and bone, antibiotic coverage can be discontinued after all surfaces are covered with healthy granulation tissue. For infection involving synovial structures, antibiotics should be continued for 1-2 weeks after the synovial membrane has sealed and the symptoms resolved. The wounds should be protected with a bandage, treatment plate or plastic boot until epithelium has covered the defect and begun to keratinize. The author usually lavages the wounds daily for the first 4-5 days with a 0.1-1% solution of povidone iodine (1-10% Betadine) in saline, but uses only sterile saline with antibiotics when lavaging synovial structures. Wounds should be bandaged with nonadherent dressings until completely epithelialized. The author uses gauze! prefer a Telfa pad backed by cotton gauzes moistened with 0.1-1% povidone iodine (1-10% Betadine). If a synovial cavity is exposed, I prefer to moisten the dressing with sterile saline and an antibiotic, usually an aminoglycoside.

**Infection of Specific Structures**

**Septic Pedal Osteitis**

Septic pedal osteitis usually occurs following direct penetration of a foreign body, extension of infection from a subsolar abscess or following laminitis. Sequestra may also be present. The treatment involves debridement as described and curettage of the exposed bone. Caution must be exercised when the diseased tissue is adjacent to the extensor process or insertion of the deep digital flexor tendon because of their proximity to synovial structures. In two retrospective studies the prognosis for return to work was fair to good. However, it is commonly believed that the prognosis for horses with septic osteitis secondary to laminitis is not as good compared to other causes; recurrence of drainage weeks to months later is not uncommon.

**Necrosis of the Collateral Cartilage of the Distal Phalanx**

Necrosis of the collateral cartilage of the distal phalanx, commonly called quittor, occurs following heel lacerations, puncture wounds or ascending infection from subsolar abscesses. Swelling over the proximal margin of the cartilage associated with draining tracts is almost pathognomonic for quittor. The swelling is painful and the horse shows a variable degree of lameness. The diagnosis is usually straightforward based on the clinical signs, but may occasionally be confused with a gravel or abscess at the coronary band. A probe inserted into the tracts should abut cartilage and indicate the extent of the injury. The probe may be radiographed in place to confirm its relation to the cartilage and palmar process of the distal phalanx. Treatment involves sharp excision of all necrotic cartilage and curettage of the margins of the wound. When the infected tissue extends below the coronary band, drainage through the abaxial hoof wall should be established at the most ventral aspect of the wound. The skin wound may be sutured and the drainage hole if present packed with povidone iodine.
soaked gauze. Approximately 75% of horses treated surgically become sound. Conservative therapy has been attempted, but not found to be as successful. The prognosis appears to be better with a shorter duration of disease.

**Septic Arthritis of the Distal Interphalangeal Joint**

Septic arthritis of the distal interphalangeal joint is most commonly caused by lacerations to the foot, but may also occur after punctures, extension of subsolar abscesses and after intra-articular injections. These horses are usually extremely lame. There is painful swelling dorsal and proximal to the coronary band. Confirmation of the diagnosis is by cytology of a synovial aspirate or by demonstration of communication between the joint and a wound. Initially the joint space may appear normal, increased or decreased in width without osseous changes; later there may be evidence of osteomyelitis of the second and third phalanx and the navicular bone. Treatments applied in the past have included systemic and local antibiotics, joint lavage, indwelling drains and bone grafting. The client must be given a poor prognosis for survival, though in one study 7 of 11 horses treated survived and 2 became sound. Interestingly the distal interphalangeal joint fused in 5 of the 7 horses to survive.

**Septic Navicular Bursitis**

Puncture wounds to the frog and adjacent sulci are the most common cause of septic navicular bursitis. By the time of presentation these horses are usually extremely lame. Frequently there is swelling of the heel proximal to the coronary band and application of hoof testers across the frog may elicit a painful response. As with septic distal interphalangeal joints, the diagnosis is established by cytology of fluid aspirated from the navicular bursa or demonstration of communication between the navicular bursa and a wound. Care must be taken to determine whether other structures are involved because the infection can readily extend to the distal interphalangeal joint, the navicular bone and the digital flexor tendon. The treatment generally recommended is surgical debridement of the wound in conjunction with bursoscopy; if this does not result in clinical improvement, then establishment of ventral drainage by removing part of the frog and fenestrating the deep digital flexor tendon (street nail procedure) may be necessary. By the time most horses have arrived at a referral center an aggressive response is warranted. However, the surgical procedure itself is not benign. Therefore, in the unusual circumstance that a puncture wound involving the navicular bursa has been diagnosed soon after it occurred, the penetrating object was relatively clean, and following removal of the foreign object the lameness improved, more conservative therapy may be used initially. This includes broad spectrum antibiotics, flushing the navicular bursa with sterile saline and antibiotics, and debriding the wound down to, but not including the deep digital flexor tendon unless the latter is severely traumatized. If the lameness worsens or after 2-3 days the lameness has not improved, more aggressive treatment is indicated.
Complications of septic navicular bursitis and its treatment include osteomyelitis of the navicular bursa, infectious arthritis of the distal interphalangeal joint, rupture of the deep digital flexor tendon, septic tenosynovitis, and septic deep digital flexor tendonitis, all of which negatively impact on the prognosis. After surgery it is important to provide pain relief by elevating the heels. The author has found that Redden wedges (Redden Ultimates) wedge shoes work well and double up as a removable treatment plate. Traditionally, prognosis for life is considered guarded and the prognosis for return to work poor. However, recent advances in managing the wound and delivering antibiotics have improved the prognosis, but not to the point that the injury should be treated with any less gravity. Some are rewarding to treat while others are frustrating, disappointing and time consuming. Also the treatment cost varies greatly, but it may be very expensive.
Shoeing in relation to treatment of diseases of the foot has traditionally been taught in a dogmatic and empirical manner, i.e. for disease A, use shoe B. However, our knowledge regarding the physiology of the foot has increased remarkably over the last 25 years and it is a time for a paradigm shift. More specifically, after a diagnosis has been made, it should be possible to determine which structures are associated with the lameness, when these structures are likely to be subject to stress that can be related to the injury, and what biomechanical principles could be used to reduce those stresses. The hoof capsule is subject to compressive and shear stresses, bones and articular surfaces to compressive stresses, while ligaments and tendons are subject to tensile stress. The lamellae are subject to tensile and shear stress. Unfortunately, there are still numerous holes in our knowledge. Therefore, for now, particularly in the absence of controlled clinical studies, we must make the most of what we know, and extrapolate theoretical principles where we can, and combine these with personal experience.

This article looks at some of the biomechanical principles we can attempt to use to change foot function, how the function of a shoe changes with a change in form, and how to combine these two to treat some diseases.

**Biomechanical principles for improving foot function**

**Reducing shock waves/concussion of impact**
Impact with the ground at the beginning of the stance phase of the stride, and to a lesser extent the breakover phase, is associated with vibrations within the distal limb. These vibrations are thought to be a factor in the pathogenesis of repetitive stress musculoskeletal injuries. It is known that these vibrations are of greater magnitude and higher frequency in horses shoe with steel shoes compared with the barefoot condition. Certain combinations of pads, plastic and or aluminum shoes have been shown to decrease the magnitude and/or frequency of these impact vibrations. Unfortunately, it is often not possible to determine how effective a combination will be without specifically testing it.

**Moving Center of pressure**
The center of pressure is the point on the ground surface of the foot through which the ground reaction force works. It is also called the point of zero moment or the point of force. If it is in the center of the foot, then half the weight borne by the limb is distributed on the medial side of the foot and half by the lateral side of the foot. Similarly, half of the weight would be borne
by the dorsal half of the foot and half by the palmar half. In a horse standing at rest the center of pressure is in a relatively constant position. In a horse moving the center of pressure varies with the phase of the stride; however, for most of the stance phase of the stride the center of pressure is in a similar position to that of a horse at rest. At this juncture, it is simplest to discuss the center of pressure with reference to a horse at rest yet realizing that it is only part of the equation. If the center of pressure is moved to one side of the foot, it will change the distribution of load causing the wall, bones, joints and lamellae on that side of the foot to be under greater load and on the opposite side of the foot they will be under reduced load. It may also increase the tension in ligaments on the side of the foot under reduced load.

**Change distribution of force**
The weight of the horse remains constant, but the area of contact between the bottom of the hoof capsule and the ground may vary considerably depending on the nature of the ground. It will also vary depending on whether there is a shoe on the foot and if so what type of shoe is present. Typically, a horse standing on a flat firm surface will have ground contact around the periphery of the foot, whether it is shod or not. In contrast, if a barefooted horse stands on sand the contact is spread broadly across the ground surface of the foot. Therefore, in the former case in which the area of contact is considerably smaller, the parts of the foot in contact with the ground will subject to higher pressure than the foot standing on sand.

If the force is spread out over a larger area in an even manner, then the center of pressure will stay the same. However, if the contact area of one side of the foot is increased relative to another on a soft ground surface, this will change the position of the foot on the ground, which would be expected to change the center of pressure as well.

The ground surface of the sole, frog, and bars are often brought into contact with the ground using various shoeing techniques to decrease weight bearing by part or all of the wall. However, until more is known about how weight is transmitted from the sole to the distal phalanx, and how this varies with differing foot conformations and sole depths, it is difficult to make reliable recommendations regarding this maneuver.

**Torque about the distal interphalangeal joint**
Torque is the tendency to cause a body to rotate about an axis. At rest or during the midstance of the stride the foot is flat on the ground and there is no net movement of the foot in relation to the ground because the extensor moment about the distal interphalangeal joint equals the flexor moment. A moment is the product of the force and the length of the moment arm (that is perpendicular to the force). In the case of the foot on the ground the extensor force is the ground reaction force and the moment arm is the horizontal distance between the center of
pressure and the center of rotation about the distal interphalangeal joint. The flexor force is the tension in the deep digital flexor tendon and the moment arm is the distance from the center of rotation of the distal interphalangeal joint to the point on the palmar surface of the navicular bone such that the moment arm is perpendicular to the flexor tendon. Within limits, the magnitude of the ground reaction force is fixed (if in motion, for that phase of the stride) and the length of the flexor moment arm is fixed. Therefore, if length of the extensor moment arm decreases, the tension in the deep digital flexor tendon must decrease if the foot is to stay in the same position. When the flexor moment exceeds the extensor moment the heels lift off of the ground.

When a horse is at rest or at mid stance of the stride the torque about the distal interphalangeal joint can therefore potentially be manipulated by moving the center of pressure to change the length of the extensor moment arm or by changing the tension in the deep digital flexor tendon. The latter is not readily achievable except by performing a deep digital flexor tendon accessory ligament desmotomy or a deep digital flexor tenotomy. The latter is reserved for the treatment of laminitis or severe flexural deformities. However, the length of the moment arm can be changed by moving the center of pressure towards the center of rotation by elevating the heels in relation to the toe and vice versa.

Changing the most dorsal point of contact with the ground changes the length of the moment arm at which breakover occurs. This may make breakover occur slightly earlier, but does not make the duration of breakover shorter.

**Traction**

Traction is a function of the opposing surfaces. Manipulating traction is frequently done to enhance performance, but is seldom performed as a therapeutic measure except to prevent interference, primarily in Standardbreds. There is very little information available on the effectiveness of different techniques to enhance or reduce traction.

**Flight of the distal limb**

The flight of the limb during the stride is integrally related to the stance phase of the stride in that how an animal breaks over determines timing and direction of the beginning of flight and the end of the flight phase determines how the foot lands. However, except to encourage the foot to breakover in the most natural position when there is reason to suspect it isn't or again to limit interference, attempting to modify the flight of the foot is seldom attempted in therapeutic shoeing.

**Structure and function of the horse shoes**
Material composition and dimensions of the shoe
If a standard shoe is thought of as bar of steel, rectangular in profile, that has been bent to conform to the shape of the foot, then the weight of the shoe is related to the thickness and width of the web, the length, heel to heel, and the density of the material. The weight of the shoe influence the animation of the gait, more weight more animation. However, excessive weight predisposes to injury related to fatigue. The more stiff the material is the more likely it is to transmit impact vibrations compared to a less stiff material. While steel is stiffer than aluminum, a direct comparison of comparable steel and aluminum shoes has not been made to the author’s knowledge. Plastic shoes may be significantly less stiff and some provide protection from impact vibrations. Additionally, any rigid appliance attached to the ground surface of the foot will greatly reduce the ability of one part of the ground surface of the wall to move in relation to another.

Cross sectional profile of shoe
The cross sectional profile can be changed in many ways that either effect the whole/most of the shoe or just a part of the shoe. The most common modifications include the addition of creases, changing the width of the web, and changing the profile of the edges of the shoe to create rims. The effects of creases are used to improve traction, and may change the way the foot leave the ground as a result. Increasing the width of the web of the shoe changes the distribution of force; as such it is less likely to descend into a soft substrate, and decreases traction.

Local modifications to the contour of the shoe are primarily utilized at the toe in the form of rolling, rockering or squaring. All of these move the breakover point in a palmar direction and therefore reduce the length of the lever arm at breakover. Rolling the lateral or medial branches of the shoe may also decrease the moment arm in the frontal plane that places compressive stress on the wall and osseous structures on the side that the horse is turning towards and tensile stresses in the ligaments on the contralateral side.

Bars
A bar is an addition to an shoe that spans from one branch of the shoe to the other, almost invariably from one heel of the shoe to the other, though other configurations are occasionally used. Bars at the heels increase the surface area of ground contact, limit or apply pressure to part of the foot, and may stabilize the branches of the shoe. As a bar between the heels increases the ground surface contact in the palmar half of the foot, on soft ground it is likely to cause the center of pressure to move towards the center of rotation of the distal interphalangeal joint. The magnitude of the effect will depend on the size of the increase in
area and where it is in relation to the center of pressure. For example, a heart bar shoe has a greater increase in surface area than a straight bar shoe and an egg bar shoe extends further from the center of pressure than a straight bar shoe, thus acting as an extension.

**Extensions**
An extension is any part of the shoe that projects beyond the normal perimeter of the foot. Any extension has the potential to act as a lever when the foot is on the ground. When they act as a lever, they cause the center of pressure to move towards the side of the limb with the extension. They are commonly used to force the heels to the ground for foals with flexural deformities of the distal interphalangeal joint and to limit dorsiflexion (hyperextension) of the distal interphalangeal joint with deep digital flexor tendon injury. Lateral or medial extensions are used in foals to treat angular limb deformities.

**Pads**
Pads are available in many variations. In brief, they are generally used to dampen concussion, elevate the foot from the ground, extend the length of the foot or change the balance of the foot. Pads that change the length of the foot or change the angle of the foot in relation to the pastern change the balance of the distal limb. In doing so they may change the distribution of force and shift the center of pressure.

**Attachment of shoe**
Most research has been conducted on steel shoes attached to the foot with nails, so the effects of nailing the shoe on have not been separated from the nature of the shoe. However, a finite element analysis study has demonstrated that stress in the wall is likely to be increased at the heel nail. Another study that examined the effect of gluing on shoes with equilox to the ground surface of the foot determined that the procedure reduced expansion of the foot.

**Applying principles and techniques to specific diseases**

**Laminitis**
In laminitis the lamellae at any point around the hoof may become damaged and give rise to distal displacement in various patterns, most commonly rotation (dorsal rotation), distal displacement (sinking), and asymmetrical (medial or lateral) distal displacement. The objective in each case is to remove the stress from the affected lamellae. As they are under greatest stress under tension when bearing load the stress is theoretically reduced by moving the center of pressure away from the affected area. In the case of rotation, the center of pressure can be moved towards the center of rotation, hence decreasing the tension in the deep digital flexor tendon, and thereby theoretically decreasing the tension in the dorsal lamellae. The maneuver
does provide clinical improvement in some horses, but it is not without some controversy because a finite element analysis model suggests that it may increase the shear stress in the dorsal lamellae; this apparent dichotomy needs to be resolved. In addition to moving the center of pressure with heel elevation, recruiting ground surface of the frog, bars, and angles of the sole to bear weight to reduced weight bearing by the walls also acts as a mild wedge on a soft ground surface. Lastly, shortening the moment arm at breakover by moving the point of breakover in a palmar direction may decrease the stress in the lamellae at that point in the stride.

Treatment of horses with medial or lateral distal displacement by moving the center of pressure away from the affected side can be achieved by any of the methods outlined above, but the author’s current preference is to use a modest extension on the opposite side of the foot. The procedure is not universally accepted and needs corroboration. As with dorsal rotation, it is usually done in conjunction with modifying breakover and attempting to reduce weight bearing by the wall.

The treatment of distal displacement does not theoretically or in the author’s experience benefit from moving the center of pressure. Recruiting the sole to bear weight with shoeing techniques is also potentially hazardous depending on the functional quality of the sole; however, these horses may benefit substantially by bedding on sand, a maneuver that distributes weight across the center of the ground surface of the foot. When shod, enhancing breakover is as much a logical step as it is with rotation.

**Hoof wall defects**

Hoof wall defects reduce the ability of the wall to transfer force to the distal phalanx from the ground is impaired, but the lamellae are undamaged. Small defects in the wall only impair this ability to transfer force to a minor degree. However, larger defects can cause the loss of support to the adjacent distal phalanx such that is displaces distally; this may occur dorsally or on either side of the foot. As a consequence of the displacement, the distally displaced distal phalanx compresses the underlying solar dermis in conjunction with prolapse of the underlying hoof capsule that results in bruising and pain. Additionally, if the distal displacement is medial or lateral, it will cause compression of the contralateral distal interphalangeal joint space and tension in the ipsilateral collateral ligament. The defect that permitted the distal phalanx to displace has caused the center of pressure to move away from the area of the defect. Reconstructing the hoof wall defect will restore the center of pressure and symmetry of the distal interphalangeal joint. The treatment of hoof wall defects is in contrast to that of laminitis. In laminitis, moving the center of pressure to the side in which the distal phalanx has moved distally would place the lamellae under greater stress which should be avoided.
Patching hoof wall defects due to white line disease is not usually done because covering the defect may permit the disease process to progress deep to the patch.

**Collateral ligament strain**
The collateral ligament is injured when under excessive tensile stress. To reduce the tensile stress in the ligament during weight bearing, the center of pressure should be moved towards the affected side. This may be achieved by increasing the area of ground contact on the affected side, typically by increasing the width of the branch of the shoe, or by an abaxial extension on the affected side, but the latter maneuver is unlikely to be used on the medial side of the foot because of the risk of interference with the contralateral foot. During breakover the stress in the ligament will be reduced by the moment arm between the opposite to and quarter. This is readily accomplished by rolling the outer rim of the shoe on the opposite branch of the shoe.

**Navicular syndrome**
In navicular syndrome, the navicular disease is under compressive strain from the deep digital flexor tendon and the supporting ligaments are under greatest strain during the second half of the support phase of the stride and breakover. To reduce the tension in the deep digital flexor tendon during the stance phase of the stride the tension in the deep digital flexor tendon can be reduced by moving the center of pressure in a palmar direction towards the center of rotation of the distal interphalangeal joint; this is most readily accomplished by a heel wedge, but any relative increase in the palmar ground contact or extension will act similarly on a soft ground surface. During breakover the tension in the deep digital flexor tendon may be decreased by shortening the extensor moment arm; this is usually done by rolling, squaring, rockering, or setting back the toe of the shoe.
Acute and Chronic Laminitis – An Overview
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Laminitis is usually classified by its duration and the morphological changes that have occurred.\(^1\) As such, developmental laminitis is the initial phase between the initial insult and the onset of clinical symptoms. Acute laminitis begins with the onset of clinical symptoms and is frequently cited as lasting until 72 hours after the onset of symptoms or the development of displacement of the distal phalanx, whichever is the sooner. If the distal phalanx does not displace after 72 hours, the disease enters the sub-acute phase. Chronic laminitis begins with the displacement of the distal phalanx regardless of whether the horse was in the acute or sub-acute phase of the disease. Chronic laminitis is further subdivided based on time and the amount of healing that has taken place. Early chronic laminitis is that phase immediately after the distal phalanx has displaced; the wall of the hoof capsule is grossly unchanged. This phase is succeeded by chronic active laminitis in which the distal phalanx remains unstable, but secondary changes in the hoof capsule are beginning to become evident. Chronic stable laminitis, as its name implies is that stage of the disease in which the deformed shape of the foot is accompanied by stability of the distal phalanx.

The classification of laminitis into phases is a convenience to enhance comprehension and assist in the diagnosis, treatment and prognosis of the disease, but the disease is a continuum. Horses tend to sequentially pass from one stage to the next on an unpredictable basis, and the boundaries between the stages are blurred. However, the continuum varies greatly between horses as they may take different entry points into the disease and different paths once affected.

Pathophysiology
The pathogenesis has yet to be clearly defined. Several mechanisms have been implicated over the last 30-40 years including dysfunction of the digital vasculature, a primary inflammatory response, toxin induced activation of metalloproteinases, and coagulopathy and thrombus formation.\(^2\) It is quite likely that all of these mechanism may be involved in some manner. Recently, unequivocal evidence confirms an inflammatory response very early in the disease has before other changes are present, suggesting that the vascular changes, thrombus formation, and metalloproteinase degradation of the basement membrane are “downstream” events.\(^2\)

As the strength of the lamellae becomes decreased during the disease process, the capacity of the lamellae to maintain the position of the distal phalanx suspended within the hoof capsule
decreases, the distal phalanx becomes unstable, and the distal phalanx displaces within the hoof capsule. The mechanical collapse of the lamellae can occur at any point around the circumference of the foot leading to different patterns of collapse. When the dorsal lamellae collapse, (dorsal) rotation ensues, when all the lamellae appear to collapse simultaneously the distal phalanx displaces distally, and when the lamellae on one side of the foot collapse, usually medial, unilateral distal displacement occurs.

Following displacement of the distal phalanx, the newly developed cavity between the hoof capsule and distal phalanx fills with blood and necrotic material. The lamellae in the damaged area become hyperkertotic and hyperplastic forming what is frequently referred to as a “lamellar wedge.” With healing the distal phalanx may become progressively more stable within the hoof capsule, but frequently the hoof capsule becomes distorted and an abnormal relationship between the distal phalanx and hoof capsule and phalangeal axis persists. The outcome is dependent on several factors including the alignment of the dermal papillae of the coronary integument, the character of the lamellar wedge, the growth patterns of the capsule, the tension in the deep digital flexor tendon, and the pattern of weight-bearing.

**Diagnosis and Assessment**

The diagnosis of laminitis is usually straightforward. In horses with acute disease the diagnosis is made based on the characteristic gait, palpation of heat within the foot, increased digital pulses, and localization of pain with hoof testers. In horses with chronic disease, visual inspection of changes in the hoof capsule, palpation of the coronary band and radiographs provide additional information that allow the disease to be further characterized, characterization which, in conjunction with the clinical symptoms, is important in forming a prognosis and strategy for treatment. In rare instances, acute or subacute laminitis may present with sufficiently subtle symptoms to present a difficulty diagnosis, though with time the symptoms are likely to become more classic.

Routine radiographic examination fo the foot should include at least three views, a lateromedial, a dorsopalmar, and a dorsopalmar oblique. The lateromedial view is used to assess the position of the distal phalanx within the hoof capsule in the sagittal plane, the presence of gas within the wall and sole, and secondary changes to the distal phalanx. Of particular importance is the thickness of the sole, the angle of the solar margin of the distal phalanx to the ground, the degree of capsular rotation, and the vertical distance from the firm proximal border of the wall to the extensor process. The dorsopalmar view is used to assess the position of the distal phalanx in the hoof capsule in the frontal plane. Normally, the width of the distal interphalangeal joint space should be symmetrical, the articular surface of the distal phalanx approximately parallel to the ground, and the thickness of the medial and lateral
walls adjacent equal. Marked tilting of the distal phalanx to one side accompanied by an increase in hoof width on the same side and narrowing of the distal interphalangeal joint on the opposite side are strongly suggestive of asymmetrical distal displacement. Care should be taken to assess the positioning before determining this because tilting of the distal phalanx and asymmetry of the distal interphalangeal joint can be induced if the limb is not close to vertical. Venography has been used to assess perfusion of the digit. Decreased perfusion in the coronary, parietal, and subsolar vasculature have been associated with a decreased prognosis.5

**Medical Therapy**

Medical therapy is aimed at preventing, limiting progression, and reversing the underlying pathophysiological processes and controlling pain by administration pharmacological agents.2,6 As such, the drugs used to affect the underlying pathophysiological processes are directed at the four mechanisms postulated to cause the disease. To counter vascular dysfunction in the digit, various vasodilators and rheologic agents have been used, and of these the most commonly used and likely to be beneficial is acepromazine. The main pharmacologic agents used to treat the inflammatory response in early laminitis are the non-steroidal anti-inflammatory drugs and DMSO, and because of the overlap between endotoxemia and inflammation, these non-steroidal anti-inflammatory drugs are also beneficial in horses with endotoxemia. Antiendotoxin therapy is frequently used in horses that clinically appear to be endotoxic. Of these, endotoxin antiserum and polymixin B are most frequently used. Heparin and aspirin have both been used to treat coagulopathy and prevent thrombus formation in early laminitis. Unfortunately, no pharmacological agents are of proven benefit once the initiating events have occurred. Of all these agents, the most frequently used to treat uncomplicated laminitis are phenylbutazone, flunixin meglumine, DMSO, and acepromazine. However, many other drugs are used to treat diseases such as diarrhea, pleuropneumonia, and metritis that are commonly associated with the onset of laminitis, such as antibiotics, fluids, anti-endotoxin serum, and polymixin B.

The pharmacologic control of pain is an area that until recently has not received sufficient attention other than the traditional mainstay of non-steroidal anti-inflammatory drugs. More recently, interest in using other agents such as lidocaine and ketamine given as an intravenous infusion, narcotics either as a continuous rate infusion or epidural, and gabapentin has developed.6 The use of these drugs to control pain must be paralleled by advances in assessing and monitoring pain.7

Concurrent pituitary pars intermedia dysfunction and equine metabolic syndrome must be managed appropriately.
Supportive Care

The goals of supportive therapy shift during the course of the disease, though the principles used to achieve the goals remain remarkably similar. In the acute and subacute stages of the disease, the goals are to prevent displacement of the distal phalanx by attempting to stabilize the distal phalanx within the hoof capsule and to control pain. Immediately following distal displacement the goals are to limit additional displacement and control pain. As the disease progresses further and the distal phalanx becomes stable within the hoof capsule, the goals become realignment of the distal phalanx with the other phalanges and the ground, and realignment of the hoof capsule with the distal phalanx. Additionally, other complications of chronic laminitis must be managed.

The supportive care of horses with acute and chronic laminitis follows similar principles, however there are differences in application. The distal phalanx is stabilized by reducing stress on the most severely affected lamellae and by decreases the stresses within the lamellae associated with locomotion. This is achieved by moving the center of pressure, adjusting the distribution of pressure, and the rate at which the load is applied. The latter is accomplished by altering the contour of shoes or shoe like devices to both diminish and smooth out the moment about the distal interphalangeal joint.

There are numerous ways to achieve these goals and many of them are interchangeable, and clinician preference is frequently based on past experience. In the early stages of the disease devices applied to the feet to accomplish these goals tend to be temporary in nature so that they are easy to apply, adjust, and remove as circumstances change. This also allows rapid assessment of the effect of the intervention. Therefore, the most frequently used types of support are customized Styrofoam board, moldable silicone putty, and commercial cuff and wedge combinations, all of which can be taped in place.

There is a general assumption that the pain a horse is experiencing is related to both the stress in the lamellae, and the pressure on the sole distal to the distal phalanx. There may be other as yet unidentified sources of pain. Thus the effect of a treatment may be assessed by changes in comfort of the horse. By extension, it may be possible to glean information about the distribution of lamellar injury by observing a horse’s response to different treatments; this is particularly useful in horses with acute or subacute laminitis (in horses with chronic laminitis, the pattern of displacement provides you with similar information).

The center of pressure can be shifted by the use of extensions or wedges; the center of pressure moves towards the side of the extension or elevated side of the wedge. The distribution of pressure can be spread out over a greater area by changing the width of the
shoe or by filling all or part of the space between the branches of the shoe with a material contacts the sole and the ground; it can be done selectively to load one part or the sole and frog over other areas. Elastic/viscoelastic materials can extend the duration of loading to diminish the shock of contact with the ground.

Easing the moment about the distal interphalangeal joint is most important in the sagittal plane, the natural plane of motion of the distal interphalangeal joint, but to a lesser extent in the frontal and transverse planes in which collareromotion and rotation also occur within the joint. This is accomplished by rounding or beveling the outer rim of the shoe, or the addition of rails. When a device is applied that covers the entire ground surface of the foot such as a wooden shoe, then the entire ground surface can be modified to roll or bevel the margins on one or more sides, and can extend as far as needed towards the center of the device.

Other aspects of supportive care include ice therapy, limb wraps to control edema, and care of pressure sores. Additionally, appropriate nutritional support must be provided.

**Surgery**

Deep digital flexor tenotomy is performed when the distal phalanx continues to rotate after all other measures to stablize it have failed, there is persistent pain in horses with phalangeal rotation that does not respond to other treatment, or in horses that have developed a secondary contracture. Drainage of purulent material is performed as necessary, and is best achieved through the distal wall to preserve the integrity of the sole. Debridement of the distal phalanx must be undertaken with great caution and only in circumstances in which probing the distal aspect of the limb demonstrates exposure of the bone. This is because radiographic evidence of distal phalangeal lysis is far from diagnostic for septic pedal osteitis. Hoof wall resections and resections are techniques that have been used to improve the alignment of the wall with the distal phalanx.

**Prognosis**

The prognosis for horses with acute laminitis varies greatly with the severity of the initial disease and the ensuing progression of lamellar injury, displacement of the distal phalanx, and distortion of the hoof capsule. It is prudent to give a guarded prognosis regardless of the symptoms because of the propensity of the disease to recrudesce, but in horses with severe disruption of the lamellae with correspondingly severe clinical signs, the prognosis for survival must be poor. For those horses that have successfully progressed to chronic stable laminitis, the prognosis for survival is markedly better, and the severity of the secondary changes to the hoof capsule are likely to give an indication regarding the necessity for indefinite hoof care.
References:

INTRODUCTION
A thorough and accurate assessment of equine colic is a common task required of the ambulatory equine practitioner. In many instances, resources, adequate facilities, personnel are limited when compared to the referral hospital setting. Yet the ambulatory practitioner is the front line of care many times and therefore it is perhaps more imperative that they make an accurate assessment as to the nature, severity, and need for referral early to result in good outcomes for the horse.

The following are items of equipment, techniques and diagnostics that are available to the field practitioner that when used correctly and interpreted correctly, can aid in the determination of the nature the lesion in a colicky horse.

Patient Preparation: Always start out with a thorough history and physical examination consisting of rectal temperature, heart rate, respiratory rate, gut sound auscultation, and mucous membrane assessment. Provide sufficient restraint (physical and chemical) to ensure safety of the handler, veterinarian (you) and the horse. Do not use acepromazine maleate as a form of chemical restraint in a colicky horse as this provides no analgesia and will contribute to hypotension in many instances a hypovolemic patient.

<table>
<thead>
<tr>
<th>EQUIPMENT NEEDED FOR COLIC EVALUATION</th>
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<tbody>
<tr>
<td>Nasogastric tubes</td>
</tr>
<tr>
<td>Twitch</td>
</tr>
<tr>
<td>Razor or cordless clippers</td>
</tr>
<tr>
<td>Rectal sleeves and lubricant</td>
</tr>
<tr>
<td>Needles and syringes</td>
</tr>
<tr>
<td>Glucometer</td>
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<tr>
<td>Analgesics</td>
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<tr>
<td>No. 15 scalpel blade</td>
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<tr>
<td>+/- rectal ultrasound probe</td>
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**Nasogastric Intubation:** Horses with significant gastric fluid/gas accumulation will rupture their stomach rather than have emesis. When emesis does occur, it exits via the nostrils. NGI is a most critical diagnostic and therapeutic technique that ALL practitioners should feel comfortable with. It is this authors’ opinion that failure to pass a NG tube in a horse with gastrointestinal pain (colic) is negligent unless it is unsafe to do so for both horse or human.

For the adult Thoroughbred/Quarterhorse type, a 2.5-3.0 meter tube is needed to get to the fundus of the stomach. Foals should be NG tubed with smaller tubes up to 1-1.5 meters. A stallion urinary catheter works well in neonates.

By using either a pump or funnel system with water, a water siphon is made and the stomach is evacuated via the NG tube by gravity.

If the horse has not recently been drinking water, the net volume obtained from a stomach should be less than 2L (<2L). In many instances, <1L or none can be obtained if the horse has been anorexic.

Volumes > 4L are significant and giving medications through the enteral route (e.g. mineral oil) should be avoided.

Large volumes (e.g. > 8-10L) are not uncommon for conditions such as duodenitis-proximal jejunitis (anterior enteritis) or long standing obstructive small intestinal diseases.

Stagnant, fermented fluid and feed is suggestive of functional or mechanical obstruction whereas a foul, fetid odor and degree of hemorrhage is suggestive of a more inflammatory lesion.

A reduction in heart rate following gastric decompression is also more suggestive of a non-strangulating small intestinal lesion (e.g. anterior enteritis, simple luminal obstruction, ileus)

**Significant nasogastric reflux: diagnosis = upper GI obstruction (functional or mechanical)**

**Rectal Examination:** Rectal palpation is a useful diagnostic for evaluating colic. It can provide useful information regarding the location of disturbance, e.g. pelvic flexure impaction, small intestinal obstruction etc. Organs palpable in a normal examination include: rectum, small colon, caudal spleen, caudal pole of left kidney, aorta, cecal base, pelvic flexure/left ascending colon, reproductive organs (+/- fetus), urinary bladder and inguinal rings.

Most clinicians use disposable sleeves and clinicians should always use copious amounts of lubricant.

Abnormal findings that should alert the clinician include ANY distended small intestine, large gas filled viscus (left or right sided), tight tenial bands anywhere, particularly if they span across the pelvic inlet, firm ingesta, asymmetry between left and right ovaries in mares, a gritty feeling or sense of lack of abdominal pressure.

Rectal examination in late pregnant mares is often difficult to interpret as the growing fetus
occupies much of the caudal and ventral abdomen causing displacement of visceral organs. However, significant gas or fluid distention in either large or small intestine should still be given consideration.

To protect oneself against litigation should a rectal tear occur during the examination, the following should be documented or able to be defended.
1) Was there an indication for the rectal examination to begin with? Generally this is not a problem as colic examinations and reproductive examinations are commonplace for the equine veterinarian.
2) Can you demonstrate adequate restraint being physical and/or chemical (and preferably both). Physical meaning using stocks, twitch application, confined area (e.g. stall with handler) and chemical meaning using sedatives (alpha-2 agonists), spasmolytics (buscopan, rectal lidocaine), analgesics (opioids) etc.
3) Full disclosure to the owner that a tear may have occurred at the time of injury and timely first aid and offer of referral.

*General rules of thumb with common causes of colic*

- Any distended small intestine is a significant finding and is suggestive of mechanical or function obstruction.
- Distended large colon with no tight tenial bands coursing across the pelvic inlet is suggestive that the colon is in the correct location but distended. This would be suggestive of an intraluminal obstruction
- Distended large colon with tight tenial bands coursing across the pelvic inlet is suggestive of colonic displacement (e.g. right displacement of the large colon) or severe distention and movement of the pelvic flexure across to the right side (e.g. large colon volvulus)
- Firm ingesta in the pelvic flexure can occur with primary small intestinal lesions as the body reclaims volume to maintain hydration (vacuum packed colon) versus a primary pelvic flexure impaction where a large smooth impaction is palpated.
- A firm hard sausage-like structure in mid to mid-ventral abdomen is suggestive of a small colon impaction. If primarily on the right in conjunction with SI distention could indicate ileal hypertrophy or impaction.
- A sudden ‘coning’ down of the rectum is suggestive of a small colon obstruction (e.g. lipoma)
- Grit palpated and apparent ease of moving your arm around the abdomen is suggestive of GI/reproductive rupture, loss of negative intra-abdominal pressure dorsally and significant pneumoperitoneum.

*Abdominocentesis:* This procedure involves obtaining a sample of peritoneal fluid for
evaluation. A location ventral and cranial is often chosen for collection. Many clinicians elect to go right of midline to avoid a splenic tap, however midline through the linea alba is also acceptable.

The normal appearance of peritoneal fluid should be clear, yellow and news print should be able to be read through the sample tube. Liken it to appearing like a light white wine e.g Chardonnay. Any deviation from this appearance is considered abnormal. A serosanguinous appearance, like Sangria this time, is suggestive of an increase in erythrocyes but not frank hemoabdomen. This would be highly suggestive of a strangulating small intestinal lesion. Green appearance and particulate matter indicates a GI rupture OR enterocentesis. Creamy, orange appearance is suggestive of an exudative process such as peritonitis. A clear orange color is suggestive of an increased protein and possibly mild increase in cell count such as anterior enteritis.

In general, strangulating lesions of the small intestine result in significant changes in peritoneal quickly, partly depending on the amount of affected small intestine and duration of compromise. Conversely, acute strangulating/displacement lesions of the large colon seldom result in significant changes in abdominal fluid appearance, at least acutely. The significance of this being that if you do see obvious changes in appearance to the peritoneal fluid and you suspect a primary large colon lesion, the colon is likely more compromised that you might think and referral would be recommended.

It is sometimes difficult to obtain fluid in the hypovolemic dehydrated patient and may require more invasive methods of fluid collection or the procedure can be repeated after the horse receives fluid therapy. Different techniques can be used to collect fluid. Regardless of the technique employed, sterile skin preparation and gloves should be used to avoid contaminating the peritoneal space.

Local block (2% mepivicaine) in the skin and linea is recommended for long needles (Thouey) or teat cannula/bitch catheter collection methods where a no. 15 scalp blade is used.

Collect your sample into EDTA (purple top) and plain (red top) tubes. Shaking out excessive EDTA is recommended when small volumes of fluid are anticipated (dehydrate animal) as excessive EDTA will falsely elevate the total protein on a refractometer. See later for lab testing of peritoneal fluid.

**Transabdominal Ultrasound:** The field practitioner often has access to a rectal ultrasound probe (typical transducer frequency of 5-10 MHz). The high frequency does limit the penetration and depth of tissue able to be seen. However, in a clipped skin using alcohol and ultrasound lubricant, the user may be able to detect some abnormalities.

Inguinal regions (left or right): Distended small intestine is often visualized here. These would be large round circular structures with a black fluid center.

Inguinal regions and ventral abdomen: A subjective assessment of increased free peritoneal
fluid may be seen. Similarly, the determination of a fluid pocket location for abdominocentesis may be aided by use of the ultrasound, helping avoid structures such as the spleen. Also, using the rectal probe transrectally can also aid is visualizing distended small intestine. More advanced imaging of the viscera requires a probe of 2.5-3.5MHz to obtain adequate depth and clarity.

**Clinicopathologic Assessment in the Field:** Now that you have your blood sample and peritoneal fluid sample, a lot of information can be readily available regarding the degree of compromise to the animal and the likelihood of a surgical versus non-surgical lesion existing. The following are hand-held, portable, point-of-care analyzers that can provide rapid and accurate values of select variables.

**Glucometer:** This measures glucose in the blood and/or peritoneal fluid. There are several machines on the market, however most recently, the Alphatrac® made by Abbott has been validated in horses. In human critical care and more recently critically ill foals and horses that present with colic, hyperglycemia has been associated poor outcome and disease severity. Glucose can therefore be used in conjunction with other diagnostics to provide additional information regarding survival. Glucose can be measured on EDTA blood or plain blood and timely assessment of whole blood is accurate. Delays in glucose determinations may yield falsely low results through erythrocyte utilization.

Normal blood glucose in the horse is 80-120 mg/dl. Peritoneal glucose should be similar to whole blood glucose as peritoneal fluid is a ultrafiltrate of blood plus extras. Discrepancies between blood and peritoneal glucose concentrations where the peritoneal fluid glucose is lower, can be indicative of a consumptive process such as septic peritonitis.

**Lactate meter:** This device measures the concentration of L-lactate (L-lactic acid) in biological fluids in mmol/L (mM). The most commonly used point-of-care and validated machine is the Accusport/Accutrend® made by Roche and there are several studies highlighting it’s use in critically ill horses and foals. An increase in lactate in horses is a manifestation of decreased oxygen utilization of tissues/microbes. Anerobic metabolism of glucose: any tissue deprived of oxygen or any microbe that can survive without oxygen will produce lactate in this fashion.
Glycolysis

Glucose $\rightarrow$ Pyruvate $\rightarrow$ Lactic acid (anaerobic metabolism)

Lactate is a measured substance that can freely move between all compartments of the extravascular fluid volume (i.e. intravascular space and interstitial space).

Increases in lactate in the blood of colic horses is usually indicative of poor peripheral perfusion where flow through the circulatory system is decreased, thereby reducing the delivery of oxygen to dependent tissues.

The normal peripheral venous blood lactate is < 2mmol/L. If hypovolemia/dehydration is the sole reason for elevations in lactate in blood, by treating the horse appropriately with fluid therapy, this should resolve quickly (hours) through hepatic clearance/metabolism (Cori cycle).

Increases in peritoneal fluid lactate (PFL) in colic horses are usually indicative of one of two causes; ischemia and tissue devitalization or infection (peritonitis) or both.

Critical to the interpretation of PFL is the concentration in reference to what the peripheral blood concentration. If the difference is minimal (e.g. < 10-15%) then the increase in PFL is likely a result of the increased blood lactate perfusing to other tissue fluids of the extracellular fluid compartment.

If the PFL is clearly increased from the blood lactate (e.g. > 25-30%), this is suggestive of active production intraabdominally and when in concert with a serosanguinous color, distended small intestine, significant reflux or other signs of small intestinal disease, signifies a high likelihood of devitalized bowel.

Examples:

<table>
<thead>
<tr>
<th>Blood venous lactate (mmol/L)</th>
<th>Peritoneal fluid lactate (mmol/L)</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal (&lt; 2)</td>
<td>Normal (&lt; 2)</td>
<td>Normal or no peritonitis, non-strangulating lesion (e.g. gas colic)</td>
</tr>
<tr>
<td>Mild increase (2-4)</td>
<td>Normal (&lt; 2)</td>
<td>Hypovolemia/dehydration</td>
</tr>
<tr>
<td>Moderate increase (&gt;5)</td>
<td>Mild increase (&lt; 5)</td>
<td>Significant dehydration</td>
</tr>
<tr>
<td>Moderate increase (6)</td>
<td>Marked increase (10)</td>
<td>Intraabdominal production suggestive of devitalized bowel</td>
</tr>
</tbody>
</table>
Final thoughts
1. It is easy to get caught up on over interpretation of single physical/clinicopathologic findings. Remember to look at all of the data you’ve gathered during your evaluation and interpret any abnormalities holistically.
2. When in doubt, early referral is always the better option if available.

Approximate Costs of POC analyzers:
1. Alphatrak Glucometer $60.00; test strips $37.50/50 test strips
2. Roche Accutrend Lactate meter ~$300; test strips $37.50/50 test strips

References available upon request.
CLINICAL EVALUATION
An accurate assessment of cardiovascular status in a horse with colic is imperative to the development of an appropriate therapeutic plan and chance of positive outcome. The field clinician is the front line in terms of assessment and pre-hospital resuscitation, and, absolutely influences the outcome in these critical cases.

The decision making process for referral will not necessarily be discussed here, rather, the immediate resuscitative strategies that an ambulatory clinician can do which will improve the chances of survival in the colicky horse.

DETERMINATION OF SHOCK STATUS
Shock in its strictest sense is a state where there is a decrease in tissue perfusion (and oxygen delivery) to a point at which it is inadequate to meet the cellular metabolic needs. However, there are several categories of shock that can coexist in a single patient depending on the disease process.

Hypovolemic shock/dehydration is expected in any horse that has not had adequate intake (~50ml/kg/day) or has excessive losses/sequestration, e.g. reflux, diarrhea or sequestration in the gut/peritoneal cavity.

The physiologic responses to low circulating volume include tachycardia, pale pink tacky mucous membranes with a prolonged capillary refill time, cool extremities and all to a varying degree. A prolonged skin tent over the point of the shoulder or side of neck can also indicate hypovolemia as can sunken depressed eyes.

The degree of shock is proportional to the volume deficit in these animals. Clinically detectable hypovolemia is at ~5% body weight. Death occurs around 12-15% with varying gradations in between.

It is important to mention that hypovolemic shock can also be the result of hemorrhage, e.g. in the case of a colicky horse with hemoabdomen. These animals are hypovolemic but not dehydrated, yet both could present similarly with evidence of reduced circulating volume.

Endotoxemic/maldistributive shock in the context of the colicky horse can be associated with significant inflammatory conditions of the GI tract (e.g. colitis, typhlocolitis, enteritis,
peritonitis) or cases of ischemia/reperfusion injury such as strangulating obstructions, particularly of the large bowel (e.g. a tight right displacement of the large colon or large colon volvulus).
Endotoxin (lipopolysaccharide) is absorbed across compromised mucosal surfaces/peritoneal surfaces from gram negative bacteria and this stimulates a profound proinflammatory cascade. Part of the dysfunction associated with this proinflammatory state is the development of hypotension that is largely unresponsive to fluid therapy. Horses are typically tachycardic as a compensatory response to the hypotension, but because the blood pressure decreases significantly, this has the effect of poor perfusion to vital organs. This combination of tachycardia with low blood pressure is referred as a maldistribution or hyperdynamic hypotension. Other clinical findings in affected horses include bright red to dark purple mucous membranes with vessel engorgement (injection) that in early stages can have a fast CRT (<1s) or a prolonged CRT in more advanced shock.

**Obstructive shock** occurs when venous return is impeded through compression of the caudal vena cava (abdominal venous return). Lesions causing marked abdominal distention (i.e. large bowel conditions) are most likely to do this. This is not a dehydration or absolute hypovolemia, rather, it is a relative hypovolemia because venous return to the heart is impede and thereby lowers stroke volume. Horses will typically be tachycardic to maintain adequate cardiac output. Remembering that CO = HR x SV.

**INTERVENTIONS**
The two most important facets of field stabilization prior to referral

1. Improve tissue perfusion
2. Provide effective and adequate analgesia (pharmacologic and non-pharmacologic).

**Practical Fluid Therapy:**

**Enteral:** the enteral route is usually reserved for colic lesions that are mild in nature and where the horse is mildly dehydrated only (≤ 5%). The enteral route is never used in a horse that has significant enterogastric reflux as risk of catastrophic stomach rupture may be increased.
Solutions used for enteral therapy include plain water, water with an osmotic cathartic (e.g. magnesium sulfate or sodium sulfate), or water with balanced electrolytes (e.g. Resorb® packets, table salt (NaCl) or lite salt (KCl)).
The rate of enteral administration is dependent on several factors including degree of pain and ileus, degree of dehydration and size of the horse.
A typical 450-500kg horse has a gastric capacity of 10-12L. However an acute fill to this degree will result in worsened abdominal pain. Most horses will tolerate 5-6L via nasogastric tube per instillation. Liquids typically exit the stomach quickly (within 30 minutes), however when taking
into account pain and ileus plus side effects of commonly administered pain medications, this is prolonged. As an example of how enteral fluids can be used, this author typically gives 4-6L every 2-4 hours enterally if treating a large colon impaction.

**Parenteral**
Intravenous catheterization: the size and gauge of catheter will dictate how rapidly intravenous volume can administered. The shorter the catheter length and the larger the catheter gauge, the FASTER the flow of fluids.

**Isotonic Crystalloids:** these include LRS, 0.9% NaCl, Plasmalyte, Normosol etc. Rate of administration: in the dehydrated shocky horse, rapid/bolus administration is required. 10-15L bolus would certainly be appropriate in a horse > 5% dehydrated (10-25 ml/kg).

**Hypertonic Crystalloids:** these hyperosmolar solution provide rapid but short lived plasma volume expansion in the shocky horse. Most clinicians have 7.2%NaCl available to them. The dose is 2-4ml/kg which translates to ~1-2L in an average adult horse. The advantage of these solutions for the field practitioner is that they are easy to transport and store on a vehicle. One always needs to remember that the shocky horse needs to receive isotonic fluids as follow-up therapy. There is seldom an indication that a horse receives hypertonic saline without additional isotonic fluids.

**Colloids:** Products such as Hetastarch® 6% are commercially available synthetic colloids that can be carried by the field practitioner and be given to cases of severe shock to augment crystalloid fluids and ‘hold’ the volume in the vascular space. These products have a longer duration of action (e.g. Hetastarch® ~24hrs) and come in 500ml bags. The dose for horses is 10-20 ml/kg per day, however, it is this author’s experience that even 500ml-1L (1-2 bags to a 1000 pound horse) can be extremely beneficial to the shocky horse when co-administered with crystalloids.

In cases of hemorrhage (e.g. hemoabdomen, uterine artery rupture etc), it is not usually practical for the field practitioner to collect and administer blood (whole blood or packed red cells). The same general rules should be followed for the shocky horse regarding aggressive isotonic +/- hypertonic therapy until further diagnostics can be performed such as transabdominal ultrasound.

**Special Circumstances:**
- **Foals:** in general it is a good idea to add dextrose to fluids in a neonatal foal (< 10 days old). If available, a 2-4% solution is appropriate (40-80 mls of 50% dextrose added to 1L of crystalloids). In cases where a uroabdomen is suspected, fluids are indicated but in general
it is ideal (not absolutely necessary) to administer potassium free fluids such as 0.45% to 0.9% NaCl plus dextrose as uroabdomen is associated with hyperkalemia and can be life threatening.

- Similar to the foal with uroabdomen, the horse that is HYPP positive and it is not entirely clear whether the horse is colicky or having an HYPP attack, potassium free fluids are ideal such as 0.9% NaCl which conveniently come in 5L bags.

**Non-Pharmacological Methods of Pain Relief:**

Colic pain can be attributed to many pathophysiological reasons including viscus distention and mesenteric tension. Notable of this is both acute gastric distention and large bowel (cecum and large colon) distention.

**Nasogastric intubation:** NG tube placement is an imperative part of the diagnostic evaluation of a colic. It is also therapeutic where decompression of a distended stomach provides pain relief. In cases where the clinician obtains nasogastric reflux OR there is a high suspicion of a small intestinal lesion (e.g. based on rectal exam, abdominocentesis etc) even if without reflux, leaving an indwelling NG tube for transportation is recommended. The NG tube should be placed and secured to the horse using tape, elasticon bandaging or other. The tube should not be plugged as this defeats the purpose of self decompression during transportation. The nasal end of the tube should be directed away from the eye and face to avoid a chemical keratits/ulceration.

**Trocharization:** Cecal trocharization from the right paralumbar fossa is an easy technique that can be performed in the field and be critical to improving survival in cases of severe colonic distention. The classic indication for this would be the horse with a large colon volvulus that is markedly distended. The caveat to this is that if it is going to impede a more expedient referral to a surgical facility, it is in the horse’s best interest to forego this procedure. However if there is time (e.g. until a trailer is hitched etc), then this can provide pain relief, improve venous return and ventilation. The cecum is chosen for several reasons, it is easily accessed, has a fixed location (cecum base to the dorsal body wall), is frequently gas distended with colonic disease as it cannot empty into the colon and is generally the healthier tissue compared to the
twisted/displaced/obstructed colon.

The potential benefit of cecal trocharization is providing some pain relief from having a distended cecum as well as decreasing intra-abdominal pressure (IAP). Organ perfusion is improved if IAP is decreased as determined by the following formula:

\[
\text{Abdominal perfusion pressure (APP)} = \text{Mean arterial pressure (MAP)} - \text{IAP}
\]

Therefore, if we increase MAP (i.e. fluid administration) and decrease IAP (cecal trocharization, gastric decompression etc), then APP is improved.

**Technique:** An 8cm x 8cm area is clipped, sterile prepped and infiltrated with local anesthetic. With a sterile gloved hand, the 14G catheter with extension tubing is inserted perpendicular to the skin and slightly cranio-ventral. The end of the extension tubing is placed in water to see bubbles of cecal gas when the catheter tip is appropriately located. After decompression, which can take several minutes with marked cecal distention, the catheter is withdrawn and an antibiotic is infused during withdrawal. (e.g. 5-8 cc gentamicin or procaine penicillin). The potential adverse effects of this procedure include local abscessation and peritonitis. Treatment with broad spectrum systemic antibiotics is indicated if post-procedure fever and/or peritonitis is suspected or confirmed by abdominocentesis cytology.

This technique should only be performed at this location in the standing horse that is tractable, meaning responsive to sedation/analgesia for the duration of the procedure.

**Pharmacological Methods for Pain Relief:**

**Drugs:** The provision of adequate analgesia to the colicky horse is important for comfort, relaxation, and prevention of further injury (to horse and handler). The response to pain medication is also very important to document.

**NSAIDs** (e.g. flunixin, phenylbutazone, firocoxib): provide good analgesia for low grade colic pain. These are typically non-strangulating lesions with a more insidious onset, e.g. pelvic flexure impaction. These drugs have specific label indication regarding dose and frequency of administration.

**DO NOT TREAT NSAID DRUGS LIKE SEDATIVES**

At most, two full doses of flunixin (1mg/kg/dose) should be given in a 24hr period. You should not repeat dose like one might with alpha-2 drugs. If the horse is unresponsive to a single full dose of flunixin, this signifies a more serious cause of colic and should be reevaluated or
referred. The consequences of repeat dosing on renal and GI function can significantly adversely alter outcome.

α-2 Agonists (e.g. xylazine, detomidine, romifidine): provide excellent analgesia and sedation to a painful horse. Some lesions (e.g. large colon volvulus) are often refractory to these drugs alone and require increased dose rates and adjunctive analgesics. Xylazine is shorter acting (20 minutes) and useful to facilitate the colic evaluation. If the horse has a long trailer ride, detomidine/romifidine would be preferable choices for their longer durations of action. An intial IV dose followed by an IM depot is an appropriate methods of delivery to prolong the analgesic property of this class of drugs. Unlike NSAIDs, these drugs can be redosed more frequently depending on the response to therapy due to their relative rapid metabolism and clearance.

Common dose rates include:

**Xylazine:** 0.2-0.4mg/kg (~200mg/500kg; 2cc)

**Detomidine:** 0.01-0.02ug/kg (~5ug/500kg; 0.5cc)

**Romifidine:** 0.04mg/kg (20mg/500kg; 2cc)

Opioids (e.g. butorphanol, morphine): provide excellent analgesia particularly in combination with α-2 agonists. These act on GI opioid receptors to mediate pain relief. Like sedative drugs like xylazine, these can be administered IV followed by IM depot injection. It is important to not mix opioids as their mechanisms of action may counteract each other leaving the horse without significant opioid mediated analgesia. (e.g. do not mix butorphanol (kappa agonist, mu antagonist) with morphine (mu agonist)). Also, always provide some sedative before IV administration of opioids as excitability can result.

Common dose rates include:

**Butorphanol:** 0.02mg/kg (~10mg/500kg; 1cc)

**Morphine:** 0.05-0.1mg/kg (~45mg/500kg; 3cc)

Spasmolytics (e.g. buscopan): is an excellent drug to decrease GI smooth muscle activity. This is useful in cases of spasmodic colic or where significant gas distention causes pain (contracting against obstruction). The attributes of this drug are that effects are only transient (<1hr) so that while motility is decreased, it is not for long. Some gas colics can mimick a surgical lesion such as colonic displacement, however if the colon is believed to be in the correct anatomical location, buscopan can be an extremely useful drug to help resolve colonic gas accumulation by relaxing the colon and allowing the gas to move (spasma reduction).

Common doses range from 60-200mg/500kg horse; 3-10cc.
**Special Circumstances**

In cases of painful mares with foals, it is recommended to either wean the foal if it is of an appropriate age, or leave the foal home temporarily in cases such as large colon volvulus (LCV). These mares are very painful and most will do everything to protect their foal, however, if she becomes recumbent in the trailer there is significant risk of injury to the foal. Mares with LCV unfortunately if severe, may die *en route* to a surgical facility and there have been cases where the foal has been crushed also. If the mare survives the initial evaluation and treatment, then reuniting the foal the following day is usually of little detriment.

**Transportation:** Tying a painful horse’s head in the trailer is generally ill-advised as the horse may go down *en route*. If the head is tied, this could cause significant injury to the horse such as head and neck trauma.

References available upon request.
INTRODUCTION
Colic in the late term pregnant mare (LTPM) is not uncommon and poses somewhat of a challenge to the evaluator given that there are some unique causes of colic in these horses not seen in other horses and that interpretation of diagnostics such as the rectal palpation can be difficult.

Causes of colic in the LTPM can vary in severity from mild impaction to significant surgical lesions. It is not uncommon for the late term mare to show intermittent signs of mild discomfort and even go off feed transiently. When colic is apparent, this heralds a heightened state of awareness and vigilance and should always be investigated to ensure that nothing untoward or consequential is threatening the mare, fetus or both.

The LTPM presents some diagnostic challenges for the veterinarian. Even in the normal mare, physical examination findings can differ compared to the non-pregnant mare due to normal physiologic adaptations of pregnancy such as changes in cardiac output, respiratory minute ventilation, and abdominal contour to name a few.

Choice of sedative and analgesics warrants some thought as many will cross the placental barrier affecting the fetus also. Drugs such as acepromazine should be avoided in colic regardless as they have no analgesic property and cause hypotension, but this is especially true in the LTPM. Short acting, low doses of rapidly cleared drugs such as xylazine or romifidine are best.

Colic in the LTPM should be considered a serious threat to the pregnancy, particularly with strangulating or ischemic conditions. It is wise to counsel your owners that she is considered ‘high risk’ for loss of pregnancy, particularly if signs of shock are evident.

Evaluation and Anatomy
The rectal examination poses some difficulty in interpretation due to the occupancy of the growing fetus within the abdomen. To accommodate the fetus, movable viscera relocate to areas not occupied by the gravid uterus in the caudal and ventral abdomen.

Stomach: The stomach is fixed in its location within the cranial abdomen. Gastric distention as
determined by finding nasogastric reflux or ultrasonographic evidence can occur secondary to outflow obstructions including extraluminal effects of displaced GI viscera from the gravid uterus. NG reflux is always significant and should be investigated. Underweight, malnourished, or stressed mares may be prone to developing gastric ulcer disease which may cause mild, often postprandial colic.

**Small intestine:** palpable distended small intestine is ALWAYS a significant finding in any horse including late term pregnant mares. Ultrasonographic evidence, peritoneal fluid analysis and NG intubation assist in determining the location of the colic to the SI.

Differential diagnoses for small intestinal colic include:

- Non-strangulating: Extraluminal obstruction from other viscera sitting on the SI, enteritis, ileus (hypocalcemia, hypokalemia)
- Strangulating/Ischemic: Epiploic foramen entrapment, mesenteric rent, vascular infarct, any cause of strangulation as in other horses.

**Large colon (LC):** Palping the pelvic flexure low on the left is seldom found in late term mares. Given that the ascending colon has only a right sided dorsal mesenteric attachment to the body wall, the left colons have the ability to migrate in any part of the abdomen. The LC is also the largest abdominal organ and as such compromises the anatomical origin of many colics in LTPM. Important features to note: Colonic gas distention should be considered significant as migration of the colon may have caused a displacement. If palpable tenial bands can be felt coursing across the pelvic inlet, this is also significant.

Differential diagnoses for large colon colic include:

- Nonstrangulating: LC displacement- common. LC impaction (feed)- common, LC tympany (gas colic)- common.
- Strangulating/Ischemic: LC volvulus. This is classically more common in the post-partum mare, however, given the change in abdominal contour, variable position of colon and physical displacement from the gravid uterus, colonic volvulus does occur in the LTPM. These mares are typically violent in their clinical examination and demonstrate significant cardiovascular compromise.

**Cecum:** Palpation of the cecum bands can be difficult due to the gravid uterus, however, if the fetus is primarily situated in the left abdomen, the cecum may be more palpable. Gas accumulation with tight tenia may be the only palpable abnormality. This could indicate colonic obstruction/displacement and an inability of the cecum to empty. Fetal movements during parturition and the fixed right dorsal location of the cecum make catastrophic cecal puncture/rupture a possibility in the peri/intrapartum period. Mares will show signs of endotoxic shock shortly after foaling from disseminated contamination of the abdomen.
Small Colon: Fecal balls within the small colon can usually still be palpated in the LTPM and in a variable location in the caudal abdomen. SC impaction can occur secondarily to impingement of the SC on the pelvic brim or other viscera by the fetus. With obstruction, gas distention may be the only palpable abnormality and this may be within the SC, LC and cecum if severe. Intrapartum traumatic mesocolon tears/avulsions are not uncommon in mares which represents a serious and fatal complication of foaling. Mares may present for severe, toxemic colic shortly postpartum or develop grade 3 or 4 rectal prolapsed. Prolapse of rectal tissue > 30 cm is consistent with mesenteric avulsion and surgical exploration or humane euthanasia should be considered.

Differential diagnoses for small colon colic include:
- Non-strangulating: SC enterolith/fecalith. SC impaction
- Strangulating/ischemia: SC tear/puncture, SC mesenteric avulsion, SC (+ rectal) prolapse

Uterus: The gravid uterus occupies much of the palpable abdomen for the veterinarian. An impression of size, texture and tone can be made via palpation. Also of importance, is viability of the fetus. A spongey, crepitant flaccid uterus should alert the clinician to the possibility of fetal death and significant endo/metritis. A large fluctuant, ‘watery’ uterus can be seen with hydrops (amnion and allantois). Fetal movements are variable and will be affected by fetal physiology (e.g. fetal sleep) and mare sedation. Lack of movement does not necessarily indicate fetal death. To confirm viability, transabdominal ultrasound, fetal ECG or repalpation can be employed.

Uterine torsion is a specific cause of LTPM colic. This usually occurs in the 9-10 month gestation period and can occur in a clockwise or counterclockwise direction. Mares usually have mild-moderate colic but are often tractable and respond transiently to analgesics. The magnitude of torsion is seldom > 270 degrees and usually 180 (i.e. not strangulating). Diagnosis is based on rectal palpation and feeling taught criss-crossing bands of the broad ligaments across the pelvic canal. A clockwise (viewed from the rear) torsion will reveal a tight coursing from left dorsal towards right ventral and vice versa for a counterclockwise torsion. The prognosis for the mare is generally considered good with appropriate surgical management. The prognosis for both the mare and foal depends on the stage of gestation with < 320 days having a better prognosis than > 320 days.

Other considerations: Palpation of grit on the surface of serosa, a seeming lack of resistance to palpation of structures may be indicative of GI or reproductive (vaginal/uterine) perforation and pneumoperitoneum. If this is suspected, abdominocentesis is necessary to confirm. A vaginal palpation/speculum examination can also be useful.

Abdominocentesis performed blindly has some inherent risk. Because the gravid uterus is heavy
and ventrally located, it is easy to inadvertently penetrate the uterine wall. Fluid may be collected, but this will likely be allantoic and not peritoneal. Ideally peritoneocentesis should be performed with ultrasound assistance, although in the absence of this technology, choosing a location as cranial and close to the xyphoid is best. Using teat/bitch catheter collect is also preferable to hypodermic needles.

Final thoughts
The decision to refer may be taken with a little more haste with LTPMs compared with non pregnant horses as if surgery is an option and if the health of the fetus/foal is also of critical importance, it is preferable to have a team approach to these cases as they can become high maintenance and intensive, especially postoperatively. The field practitioner can improve the horse’s survival by early referral, field stabilization with intravenous fluid therapy and judicious use of short acting analgesics.

FOALS (Neonatal)

INTRODUCTION
Colic in foals can present a diagnostic challenge and the equine practitioner has to become reliant on other techniques that perhaps small animal clinicians would be more comfortable with. Rectal palpation is limited to a finger, large bore NG tubes need to be downsized, and fluid and drug administration needs careful thought and more precise calculation. The differences between adults and foals continue as congenital/genetic causes need to be considered as well.

Congenital lesions usually manifest within days and signs are often without fever. While there are no common lesions, less common lesions may include anatomic atresias such as ani, coli, recti. Considered in this category would be ileocolonic anganglioniosis (Lethal White Foal Disease), where an autosomal recessive trait of Overa and Overo cross paint horses results in an endothelin B receptor defect affecting the neural crest cells. The result is lack of pigmentation (no melanocytes) and no enteric nervous system in select locations (distal). These horses nurse well initially, then gradually become distended and colicky by 24 hours. Lack of meconium, a compatible phenotype and breeding knowledge assist in making the diagnosis. Older foals may have various hernias (inguinal, scrotal, diaphragmatic) with incarceration or strangulating.

Signalment and History:
Breed- There are some breeds more predisposed to certain lesions than others. For example, draught horse Tennessee walking horses and Standardbred colts are more likely to develop inguinal/scrotal hernia than other breeds. Paints (overo and crosses) can produce lethal white foals. Miniatures are more likely to develop fecaliths.

Sex- Colts are overrepresented for the diagnosis of congenital bladder rupture and subsequent uroperitoneum as the urethral pressure can exceed the intravesicular pressure during active foaling leading to bladder rupture.

Some studies stipulate that fillies are twice as likely to develop meconium impactions.

History- an accurate assessment of perinatal health, mare health prepartum, breeding farm conditions (numbers of mares and foals, previous disease, previous morbidity/mortality) are very important in determining risk for infectious disease in particular. Clostridial, Salmonella, Cryptosporidial and Rotaviral enterocolitis are often endemic concerns on properties with previously diagnosed enteric disease. These conditions can cause severe pain in foals manifesting as colic.

DIAGNOSTICS (specific to the abdomen)

Physical Examination: Foals can display a unique subset of clinical signs of abdominal pain compared to adult horses. Rolling from side to side, rolling onto their backs with their head and neck contorted is a common finding of moderate to severe pain. Foals without surgical lesions, e.g. inflammatory enterocolitis or gastric-duodenal ulcerative disease, can be just as violent as a foal with a small intestinal volvulus or strangulating obstruction. The degree of pain is very unspecific as to the etiology of colic and thus a more detailed examination is required.

Other postural changes often seen are arching of the back and tail flagging with rectal pain (e.g. meconium impaction). Frequent straining to urinate can be indicative of uroperitoneum. Straining (increasing intra-abdominal pressure) can also be permissive to herniation of intestine into the inguinal canal/scrotum and thus if a hernia is diagnosed/visualized, one must always consider if there was another reason e.g. meconium impaction.

An important caveat to neonatal displays of abdominal pain is that encephalopathic foals (e.g. dummy, HIE, PAS) may not show classic signs requiring more astute assessment by the attending clinician.

Abdominal contour assessment is a useful tool to determine if there is increasing distension. Foals with obstructive conditions including meconium impaction which is common, will become more and more distended. This objective measure can be done using a measuring tape at consistent landmarks. This author uses the umbilicus and a corresponding clipped spot on the dorsum of the back. Hourly assessment is a useful indicator of disease progression and can be used to determine the need for surgery or other interventions.

Abdominal auscultation may reveal ‘ping’ or resonance with gas accumulation.
Abdominal palpation is achievable in foals where gastric distention, hepatomegally, thickened/distended bowel may be palpable. A fluidy belly (with gentle ballotment) may be indicative of peritoneal fluid such as urine (associated with bladder/urachal/ureteral rupture) or blood (associated with trauma).

**Nasogastric intubation:** this is an essential part of the examination. Given their size, smaller tubes are required to avoid unnecessary trauma. A stallion urinary catheter works well in most foals. A limitation of effective siphoning is the relative height of the nostril to the ground. Gentle negative pressure with a catheter-tip syringe can be useful in retrieving gastric contents. One should be patient in collecting fluid from foals as this can be more difficult than an adult horse. NG reflux is common in foals with mechanical and functional obstructions such as enteritis and gastric-duodenal ulcer disease. In fact, some foals produce more reflux with these inflammatory conditions than with a mechanical obstruction. Any foal with significant reflux should have feed withheld and reassessed every 1-2hrs. If the reflux persists > 4hrs, parenteral provision of nutrients should be instituted.

**Abdominoacentesis:** This may be performed with or without ultrasound guidance. Many clinicians will use ultrasound to survey the abdomen first, looking for visceral distention or an obvious lesions such as a ruptured bladder. The procedure is facilitated by light sedation (e.g. xylazine, diazepam or butrophanol). The procedure is similar to an adult horse. Local block is recommended regardless of the tool used (18-20G 1.5” needle or teat cannula). Each technique has its pros and cons. Both counts and protein are lower than adult horses (~450/µL and < 1.5g/dL) and any elevation should be followed up by cytologic examination. Degenerative neutrophils are concerning and indicate significant bowel compromise whether from a mechanical or inflammatory lesion and probably warrants exploration. In cases of ureteral tears or small bladder tears, partial bladder fill may still be evident. This does not rule out uroabdomen however. A peritoneal creatinine: serum creatinine of > 2:1 is diagnostic for a leaking urogenital structure and should be investigated.

**Abdominal Radiography:** Has a much higher utility in foals than adults. mAs (5 to 28) and kVp (80 to 120) are usually required for diagnostic films. The results are helpful in determining the anatomical location rather than the specific etiology, where gas distention on the large bowel is very evident. Hairpin turns or “U” shaped tubular gas lucencies are indicative of small intestinal distention/obstruction. A corrugated appearance may be indicative of thickened intestinal wall and enteritis. Lateral projections are easily taken with the foal in lateral recumbency. VD are possible in smaller foals. Meconium impaction and small colon/rectal impaction/fecalith may be evident as granular radiopacities. Serosal detail is poor in any foal due to a paucity of intraabdominal fat, however this is worsened in cases of peritoneal effusion.
Contrast radiography with barium is very useful in foals. An upper GI barium series is useful to diagnose delayed gastric emptying (secondary to pyloric stenosis, duodenal stenosis, ileus). 5ml/kg of 30% W/V is administered via NGT. Normal foals should have barium in the cecum by 2-3 hours. Lower GI studies (e.g. barium enema) can be useful in diagnosing obstructive TC/SC lesions including atretic structures.

**Abdominal ultrasound:** Is an extremely useful tool for the evaluation and serial reevaluation of foal colic. The small size, thin body wall and thin haircoat make the utility of this technology high for determining the location of the lesion, presence of distention, thickness of visceral wall, peritoneal effusion or structural abnormalities (e.g. intraabdominal abscesses, intussusceptions etc). Linear/curvilinear probes of varying frequencies (4 to 7 MHz) are ideal.

**Gastroscopy:** For neonatal foals (< 10 days) a 1 meter scope is usually long enough to assess the esophagus, stomach and pyloric outflow tract. Small pediatric scopes may be required in miniature foals. Ideally, solid-feed should be withheld 6-10 hours but suckling can continue up until the examination. Gastric ulcerative disease and mass lesions are readily evident by gastroscopy. Esophageal narrowing can be seen in cases of congenital stenosis/stricture (e.g. persistent right aortic arch).

**Digital examination:** A well lubricated digital rectal examination can be useful in palpating meconium in the distal rectum. Meconium is normally brown and sticky, however when it becomes desiccated, will become firm and possibly adherent to the rectal mucosa. Sedation is helpful and lubrication is essential. The presence of yellow pasty feces is highly suggestive of meconium passage and now the presence of normal ‘milk’ feces in the neonate.
A discussion of treatment of various conditions will be provided during the presentation.

**When to consider surgery?**

It is important to recognize that neonatal foals (<10 days) have a worse prognosis for survival from colic surgery than older foals and adult horses based on older reports. Complications can be significant related to the delicate nature of the intestinal integrity, bacterial translocation, septicemia and intraabdominal adhesion development. Lesions of the large colon have a better prognosis than small intestine. Recent advancements in intraoperative use of adhesion preventatives (e.g. carboxymethylcellulose) and rigorous intensive care treatments in the postoperative phase have likely improved these statistics however this is a clinical impression and not substantiated as yet.

**Rules of thumb for recommending exploratory surgery:**

1. Persistent pain unresponsive/minimally responsive to analgesics
2. Persistent tachycardia (especially if sustained >120/min)
3. Progressive abdominal enlargement
4. Increased peritoneal fluid protein, cell count or significant degenerative changes
5. Serosanguinous peritoneal fluid
6. Radiographic/ultrasonographic evidence of obstruction
7. Deterioration of the foals' condition despite intensive medical care
8. The owner is well informed of the benefit:risk including prognosis and cost

References available upon request.

**Dr. Fernando Marqués, DVM, Diplomate ACVIM**

**FLUID THERAPY**

**Fernando J. Marqués, DVM, Diplomate ACVIM**

The main goal of fluid therapy is to increase cardiac output by increasing cardiac preload and therefore increasing oxygen delivery to the tissues. By means of fluid therapy it is also possible to correct many blood pH, electrolytes and acid-base imbalances. Fluid therapy is indicated for horses with dehydration, for replacement of fluid and electrolytes losses, for increasing colloid osmotic pressure, for improving poor peripheral circulation, for treating failure of passive transfer, etc.1-3

**Formulating a fluid therapy plan: the 3 basic decisions**

When developing a fluid therapy plan, there are three basic decisions to be made as follows:

- **How much** fluid does the patient need?
- **What type** of fluid should be given?
- **Which route and rate** of administration should be used?

**How much fluid does the patient need?**

There are 3 factors determining the total fluid requirements per day, *fluid deficits, maintenance needs and concomitant fluid losses*.  

**Fluid deficits**

Fluid deficits are calculated based on the degree of dehydration and the total body weight (BW) of the patient. The degree of dehydration is expressed in percentage of body weight and is estimated based on physical examination and laboratory parameters.1-3 Clinical parameters used to estimate hydration status are heart rate, skin tent, color and moisture of mucous membranes, capillary refill time and urine output.3 Packed-cell volume (PCV) and total protein (TP) can be also used to assess hydration status. In a dehydrated patient it is expected that both PCV and TP increase due to a decrease in plasma volume. An increase in serum
creatine and/or blood urea nitrogen (BUN) concentration may also indicate dehydration (prerenal azotemia). Blood lactate concentration can increase in cases of poor peripheral circulation due to dehydration.

Once the percentage of dehydration is estimated from physical exam and laboratory data, the fluid deficits are calculated using this formula:

\[
FLUID\ \text{DEFICITS (L)} = \%\ \text{Dehydration} \times \text{Body Weight (kg)}
\]

**Daily maintenance needs**

Ongoing normal water loss in resting adult horses is about 50-60 ml per kilogram of body weight per day. These normal losses account for water lost through the urinary and gastrointestinal tract (sensible losses) and water lost during respiration and sweating (insensible losses). The total amount of body water in equine neonates is higher than in adults and averages about 80 per cent of the body weight. The fluid maintenance requirements in neonates can be up to 100ml per kilogram of body weight per day.

**Concomitant fluid losses**

Pathologic or abnormal water losses occur in patients with severe diarrhea, polyuria or third space sequestration of fluids.\(^2\)\(^3\) These losses must be taken into account and added when calculating the total amount of fluids to be given in 24 hours.

**What type of fluid should be given?**

The type of fluids to be administered is determined by the individual patient requirements. In order to address this question the clinician needs to know if there is any serum electrolyte or acid-base imbalance, and glucose abnormality that need to be corrected. These abnormalities are best evaluated by serum biochemistry or blood gas analysis.

**Type of fluids**

Intravenous fluids are classified as crystalloids or colloids. Crystalloids are commonly used to replace fluid deficits, for daily maintenance requirements and to replace electrolytes or dextrose, whereas colloids are mainly used in situations in which it is necessary to increase plasma oncotic pressure or to provide immunoglobulins and acute phase proteins (e.g. plasma transfusion).\(^1\)\(^3\)

In most clinical scenarios a well balanced polyionic solution (crystalloid) is the best option to replace fluid deficits and to provide daily maintenance requirements.

**Crystalloids** are solutions containing solutes (electrolytes and non-electrolytes) that are able to access all body compartments. Examples of crystalloids are dextrose 5%, 0.9% sodium chloride, 0.45% sodium chloride, lactated Ringer’s solution, Plasmalyte™, etc. There are three types of crystalloid solutions:

- **Hypotonic**: 0.45% sodium chloride solution, etc.
- **Isotonic**: 0.9% sodium chloride solution, Ringer’s solution, Plasmalyte™, Normosol™, etc.
- **Hypertonic**: 7.5% sodium chloride solution.
Crystalloid solutions are freely permeable to cell membranes; therefore after a short period of equilibration of about half an hour, only about 20% of the infused solution will remain within the intravascular space. Some crystalloids may also alter the blood pH and the acid-base balance by means of changing the strong ion difference (SID) or because of the alkalinizing agents (e.g. lactate, acetate, gluconate) that some crystalloid solutions contain.1-4

**Colloids** contain large molecular weight substances that remain in the intravascular space and do not access other body compartments. Dextran, hetastarch and plasma are examples of colloid solutions.3

Plasma is commonly used to treat failure of passive transfer and/or sepsis in foals and also to provide albumin in hypoalbuminemic patients experiencing a reduction of the intravascular colloid osmotic pressure (COP). Under normal conditions 75% of the normal plasma COP is exerted by albumin.1,3-5

**Which route and rate of administration should be used?**

In general fluids can be given intravenously, orally, subcutaneously, intraperitonealy or by intraosseous injection. The most common and practical routes of fluid administration in horses are the oral and the intravenous routes. The oral route is the most physiologic route and should be used whenever possible. Patients receiving fluids by other routes should also have drinking water available, unless there is a specific reason for not administering oral fluids (e.g. gastric reflux or ileus). In foals, the amount of ingested milk should be taken into account and added when calculating the total volume of fluids the foal is receiving per day. The intravenous route is the route of choice in very sick patients or patients who have suffered acute fluid loss. The intravenous route is also used when accurate administration of fluid volume and rate is needed and this is usually achieved by running the fluids through an infusion fluid pump in a hospital setting. All crystalloids, colloids, plasma, whole blood, hypotonic fluids (e.g. 0.45% sodium chloride, 2.5% dextrose in water) or hypertonic solutions (e.g. 7% sodium chloride) and viscous solutions (e.g. lipid solutions) are suitable to infuse intravenously.1,3

The total amount of fluids to be administered in 24 hours (maintenance requirements plus concomitant losses) should be divided by 24 (hours) and a rate of administration (expressed in ml/hr) is then established. The fluid deficit is generally replaced over 2-3 hours in adults, and in neonates half of the total deficit can be administered in the first 6 hours and the rest over 12-24 hours.1

**References**


EQUINE ARRHYTMIAS: A Review of ECG Approach and Interpretation.
Fernando J. Marqués, DVM, Diplomate ACVIM

In the daily clinical practice it is not uncommon to come across patients with cardiac arrhythmias discovered as an incidental finding upon regular physical examinations, during pre-purchase exams in the case of sport horses, during investigation of an unrelated disease or while exploring a suspected primary cardiac problem. It is crucial for the large animal practitioner to determine whether a cardiac arrhythmia is benign or is clinically significant and needs further attention.\(^1\,^2\,^4\,^6\) Cardiac arrhythmias are abnormalities of the heart rate, rhythm or conduction pattern due to disturbances of the impulse generation or impulse conduction or a combination of both.\(^4\,^6\) About 25\% of horses with no evidence of heart disease may have cardiac arrhythmias which can be found upon regular physical examination or ECG.\(^2\) It has been shown that upon twenty four-hour ECG monitoring of normal horses, 44\% of horses had second degree AV block, 10\% had sinus arrhythmia, 3\% had atrial block, 27\% had occasional extrasystoles and 27\% had ventricular arrhythmias.\(^2\) Approximately 40\% of horses with clinical signs of cardiac disease may present cardiac arrhythmias.\(^3\,^5\)

In horses the electrocardiogram (ECG) is primarily used to identify and characterize cardiac arrhythmias and conduction disturbances.\(^4\,^6\) Electrodes placed on the body surface record changes in membrane potentials and direction (electrical activity) of the heart cells. Whenever a wave of depolarization (positive charges) moves toward a positive electrode placed on the skin of the horse, there is a positive (upward) deflection recorded on the paper of the ECG machine. If the wave of depolarization moves towards a negative electrode, a negative (downward) deflection is recorded on the ECG.\(^10\) The “base-apex” lead system is commonly used in horses because it records consistent ECG tracings regardless of animal size and breed, the tracing has good wave amplitude and it is hardly affected by slight movements of the animal or their skin while recording.\(^1\,^4\,^7\,^9\) For the base-apex lead system the positive electrode (LL – lead II) is placed on the left side of the thorax over the heart area, at the level of the elbow over the 5\(^{th}\) intercostal space. The negative electrode (RA – lead II) is placed on the right jugular furrow two-thirds of the way down the neck. The “ground” or neutral electrode (LA) is placed in any location away from the heart, most commonly on the left side of the neck.\(^1\,^3\,^5\,^7\,^9\) Wetting down the skin with alcohol where the electrodes are attached, improves the quality of the ECG recording. Calculating the heart rate is the very first step when assessing an ECG tracing.\(^1\,^3\) Using a “standard” paper speed of 25mm/sec., each small box represents 0.04 sec. and each large box represents 0.2 sec. To determine the heart rate it is customary to count the number of complexes in 6 seconds and multiply them by 10.\(^1\,^7\,^9\,^10\) The second step when assessing an ECG is to determine if the rhythm is regular, by examining the P-P and R-R intervals.\(^1\,^3\) Determining if there are P waves present and if there is always a P wave preceding every QRS complex is also important and it is done as a third step. The fourth step is to determine if all P
waves are followed by a QRS complex, and to determine if the P waves and QRS complexes are normal in appearance (shape) and timing. The final step is to determine if the P wave, QRS complex and P-R and Q-T intervals duration are normal.

References


INVESTIGATION of LOWER RESPIRATORY TRACT DISEASE
Fernando J. Marqués, DVM, Diplomate ACVIM

A complete and thorough examination of the respiratory tract allows the practitioner to determine as to whether the horse has an upper or lower respiratory tract disease or both. Physical examination is key in the diagnostic process and it should not be underestimated. It is important to perform a distant examination to determine if there is evidence of nasal discharge, abnormal respiratory pattern or reluctance to move. Although nasal discharge can originate from any level of the respiratory tract, it may indicate an inflammatory process or
infection in the lower airways. Assessing the respiratory pattern is also important which may also indicate the presence of a disease process in the lower airways. A normal inspiration:expiration ratio in a horse is 1.0:1.2. The presence of a double expiratory effort or “abdominal lift” are indicators of increased expiratory effort and possible lower airway disease. A horse with pleurodynia can show gait alterations or even reluctance to move. Auscultation is another valuable diagnostic tool to differentiate an upper versus lower airway disease. In a healthy horse inspiratory sounds are normally louder than expiratory sounds, but when reversed, a lower respiratory disease is most likely present. Adventitial lung sounds, such as crackles or wheezes, or increased lung sounds also indicate the presence of lung pathology.

After a presumptive diagnosis of lower respiratory disease is made based on history and physical examination findings, the clinician may use different diagnostic aids to achieve a definitive diagnosis. Common diagnostic aids to examine the respiratory tract of the horse in the field are blood gas analysis, tracheal wash (transtracheal or transendoscopic), bronchoalveolar lavage (BAL), ultrasonography, thoracocentesis, and thoracic radiographs in foals. Tracheal wash is commonly used for bacterial sampling and cytological fluid analysis. This sampling method is useful in detection of abnormal exudates in major airways in cases where diagnosis of lung pathology is in doubt, and to help differentiate pneumonia from recurrent airway obstruction. When pneumonia is suspected from clinical examination findings, tracheal wash is commonly used to obtain a sample of exudates for bacterial culture and sensitivity. A tracheal wash can be performed transendoscopically or by trans-tracheal technique. Potential complications using a trans-tracheal technique are abscess formation or infection at the incision site, subcutaneous emphysema. The possibility of complications using a transendoscopic method is minimal.

Bronchoalveolar lavage (BAL) is a relatively simple and safe technique and is used for sampling the terminal airway and associated alveoli. It is a very useful method for detection and evaluation of a diffuse pulmonary process located in the most distal portion of the respiratory tract, such as inflammatory airway disease or recurrent airway obstruction (RAO). Because only a small portion of the lung can be evaluated by this technique, localized processes can be missed if a normal area of the lung is sampled. Samples obtained by BAL are usually assessed by cytological analysis, in combination with bacteriological and cytological analysis of tracheal aspirates. Major complications with BAL are very rare. Respiratory distress and complications can occur in horses with compromised respiratory function, and the procedure should be avoided in this type of patients.

Thoracic ultrasonography is the technique of choice for studying the pleural surface and pleural space. The presence of pleural fluid, in addition to the amount and characteristics of the fluid can be investigated by thoracic ultrasonography. Abscesses, tumors or consolidated areas located in the periphery of the lung parenchyma can also be detected by ultrasonography. This imaging technique is also useful to diagnose atelectasis of a lung lobe.
Thoracocentesis is used to collect pleural effusion for cytological analysis and bacteriologic culture, and to drain pleural effusions. This technique is usually performed when physical examination reveals findings suggestive of pleural effusion, and after an ultrasound examination is completed to confirm the presence of fluid and to characterize the type of fluid. Complications from thoracocentesis are hemothorax, infection at the site, pneumothorax, hypovolemia and infection of the pleura and lungs. Using a proper and aseptic technique is the best way to avoid complications. Permanent placement of chest tubes for drainage involves substantial aftercare and is best done in a hospital setting.

References
PROBLEM-BASED APPROACH TO HORSES WITH MYOPATHIES
Fernando J. Marqués, DVM, Diplomate ACVIM

Pathologic processes affecting the muscles are common causes of decreased performance in horses worldwide. These pathologic processes can be classified based on its underlying mechanism, or based on the presenting clinical signs. Common clinical signs of muscular disease are muscle stiffness, cramping, fasciculation, atrophy, and exercise intolerance. Dr. MacLeay divides myopathies into those causing muscle cramping with exercise, permanent gait alterations, muscle weakness, muscle wasting, and acute severe rhabdomyolysis. Diagnostic workup in horses with suspected muscular disease includes a thorough history and physical examination, serum biochemistry panel, vitamin E and selenium serum concentrations, urinalysis, fractional excretion of electrolytes, exercise challenge test, and muscle biopsy. Other diagnostic methods available to investigate myopathies are electromyography, thermography, genetic testing and nuclear scintigraphy.

Serum biochemistry panel is useful not only to assess electrolytes concentration but also to investigate enzyme activity related to muscular disease. The most valuable enzymes are creatine kinase (CK), aspartame aminotransferase (AST), and lactate dehydrogenase (LDH). Exercise tests are used to assess serum CK and AST enzyme activity pre and post exercise. Controlled periods of exercise are used for these tests and the horse’s serum enzyme activity response is evaluated. Submaximal exercise protocols are currently recommended. Urinalysis is usually performed to detect the presence of myoglobin due to acute muscular cell destruction, and to assess fractional excretion of electrolytes. Electrolytes play an important role in muscular contraction. Strenuous exercise can cause electrolyte imbalances, which in turn can induce muscular contractility derangements. Particularly low or high fractional excretion of electrolytes is indicative of electrolyte imbalances.

According to Dr. MacLeay’s classification of muscular disorders based on the most common clinical presentation, diseases are grouped in categories not mutually exclusive. Recurrent exertional myopathy (RER) in Thoroughbreds, polysaccharide storage myopathy (PSSM) in Quarter Horses and Draft horses, idiopathic chronic exertional rhabdomyolysis and mitochondrial myopathy can all lead to muscle cramping related with exercise. Horses with overexertion, vitamin E or selenium deficiency and electrolyte depletion may also show muscle cramping associated with exercise. Gait alterations due to myopathies can be caused by acute muscle strains, sprains or tears, or by chronic disease such as fibrotic myopathy. Diseases such as hyperkalemic periodic paralysis (HYP), myotonia congenital, myotonia dystrophica, equine motor neuron disease (EMN) and PSSM in Draft horses can share muscle weakness as the main presenting feature of the disease process. Generalized muscle wasting can be caused by EMN, immune-mediated myositis, cachectic and disuse atrophy, and equine pituitary pars intermedia dysfunction (PPID). Segmental muscle wasting can be caused by disuse atrophy, fibrotic
myopathy, or as a consequence of neurogenic dysfunction and post healing of severe muscle trauma. Acute severe rhabdomyolysis and swollen painful musculature can be caused by exercise-related muscular damage, malignant hyperthermia, clostridial myonecrosis, immune-mediated myositis, toxic plants and ionophore toxicity.

References
**Introduction:** Flock and herd health preventive health strategies are the most important areas of management that should be emphasized by the producer and veterinarian. The most profitable producers receive the best economic returns when disease is kept to a minimum and a focus is placed on preventive flock/herd health. The veterinarian’s goal should be to improve profitability for the producer by designing profitable herd health strategies and not just providing emergency and sick animal veterinary services. Veterinarians need to work with producers on keeping morbidity and mortality rates to a minimum while improving profitability. These spreadsheet notes will review provide a framework for veterinarians and producers in designing a herd or flock health program for this age group.

<table>
<thead>
<tr>
<th>Life Stage</th>
<th>Tasks</th>
<th>Condition Prevented</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prebreeding</td>
<td>check BCS</td>
<td>anestrus</td>
<td>increase/decrease plane of nutrition</td>
</tr>
<tr>
<td></td>
<td>FAMACH A check</td>
<td>weight loss</td>
<td>deworm if necessary</td>
</tr>
<tr>
<td></td>
<td>feet cull poor ones check</td>
<td>lameness, foot rot</td>
<td>trim feet</td>
</tr>
<tr>
<td></td>
<td>udder ensure ID</td>
<td>financial losses</td>
<td>ship to market: DRF</td>
</tr>
<tr>
<td></td>
<td>VAX PRN check teeth</td>
<td>agalactia/neonatal losses</td>
<td>problem udders: ship to market assign permanent ID, update records</td>
</tr>
<tr>
<td></td>
<td>habituate</td>
<td>loss of identity overeating/tetanus</td>
<td>VAX CD/T</td>
</tr>
<tr>
<td></td>
<td></td>
<td>stress, flight/fight</td>
<td>cull if bad broken mouth interact with flock/herd 2x or more daily</td>
</tr>
</tbody>
</table>
check records financial losses make educated decisions on breeding/culling
provide iodized salt Goiter/ neonate losses provide iodized granular salt
keep feed dry/off ground parasitism/$ losses provide off ground feeding
Vit/Minerals PRN WMD Vitamin E and/or Selenium
develop repro records financial losses record breeding dates 2x or more daily

| Early Gestation | FAMACH | A | weight loss | deworm if necessary |
| Late Gestation | check BCS | anestrus | increase/decrease plane of nutrition |

Mid Gestation | pregnancy check financial losses | ultrasound, count fetuses |

Late Gestation | check BCS | ketosis | increase/decrease plane of nutrition |

FAMACH A lamb/kid losses | deworm only if necessary |

check udder if enlarged | place in lambing/kidding area |
ensure ID loss of identity | update records |
habituate stress, abortion | interact with flock/herd 2x daily |
check records financial losses | make educated decisions on lambing/kidding |
provide iodized salt Goiter/neonate losses | provide iodized granular salt |
plan for neonate losses | organize/clean/prep area for neonates |
lambs/kids dystocia kit | put together dystocia kit, review procedures |
clean, clean, clean | clean, clean, clean |
check freezer (colostrum) diarrhea, parasites, etc | keep heat treated colostrum in freezer |
Vit/Minerals PRN WMD, Rickets, Rickets | Vitamin E and/or Selenium, Vitamin D |
check repro records unexpected neonates | makes notes |
watch for ketosis or | Dextrose, CaBorogluconate, PropGlycol, etc |
problems hypocalcemia | check for vaginal prolapse financial losses | repair vaginal prolapse as needed |
ensure ID loss of identity | update records |
habituate stress, abortion | interact with flock/herd 2x daily |
check BCS anestrus | increase/decrease plane of nutrition |
check for vaginal prolapse financial losses | repair vaginal prolapse as needed |
check freezers (colostrum) septicemia | keep heat treated colostrum in freezer |
Lambing
Kidding

Develop protocol neonate losses develop protocol on how neonates will be handled

Clean, clean, clean, neonate losses interact with flock/herd 2x daily

Exam newborns financial losses check for any imperfections

ID neonate losses link up neonate IDs with ewe/doe

Financial losses dip with dilute chlorhexidine

Dip umbilicus staple eyelids, apply Ab ointment PRN

Check for entropion septicemia assist with nursing PRN

Check for nursing septicemia remove neonates from dairy

Feed neonates financial losses does/ewes ASAP

Ensure records link up neonate IDs with ewe/doe

Maintain records record kidding/lambing data

Insure financial losses remove neonates from dairy does/ewes ASAP

Nursing septicemia insure nursing within 1 hour

Heat treat Johne’s/CAEV/OP P remove neonates from dairy does/ewes ASAP

Colostrum septicemia bottle feed neonates PRN

Ensure adequate septicemia adjust plane of nutrition PRN

Colostrum poor growth in newborns Dextrose, CaBorogluconate, PropGlycol, etc

Check udder/BCS neonates reduce/repair prolapse

Watch for ketosis or adjust plane of nutrition PRN

Problems hypocalcemia Vitamin E and/or Selenium, Vitamin D

Watch for uterine adult/neonate losses move late born neonates to clean areas PRN

Problems crypto diarrhea disbud dairy kids at 4-7 days of age

Neonates

Check 2x daily examine/neonate losses exam/treat PRN

Morbidity/mortality interact with flock/herd 2x

Mortality staple eyelids, apply Ab ointment PRN

Vit/Minerals PRN Vitamin E and/or Selenium, Vitamin D

Late born WMD, Rickets move late born neonates to clean areas PRN

Neonates move late born neonates to clean areas PRN

Disbudd Vitamin D disbud dairy kids at 4-7 days of age

Kids Vitamin E and/or Selenium, Vitamin D

Check 2x daily insure neonates are up, nursing, BAR

Dehorning later
**Abbreviations**

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>BCS</td>
<td>body condition score</td>
</tr>
<tr>
<td></td>
<td>drug residue</td>
</tr>
<tr>
<td>DRF</td>
<td>free</td>
</tr>
<tr>
<td>ID</td>
<td>official identification</td>
</tr>
<tr>
<td>VAX</td>
<td>vaccinate</td>
</tr>
<tr>
<td>PRN</td>
<td>as needed</td>
</tr>
<tr>
<td></td>
<td>C. perfringens</td>
</tr>
<tr>
<td>CD/T</td>
<td>CD/Tetanus</td>
</tr>
<tr>
<td></td>
<td>white muscle</td>
</tr>
<tr>
<td>WMD</td>
<td>disease</td>
</tr>
<tr>
<td></td>
<td>as soon as</td>
</tr>
<tr>
<td>ASAP</td>
<td>possible</td>
</tr>
<tr>
<td></td>
<td>bright, alert,</td>
</tr>
<tr>
<td>BAR</td>
<td>responsive</td>
</tr>
</tbody>
</table>

**References for Sheep and Goat Veterinarians**

3. Sheep Medicine (2007), Scott
Sheep and Goat Herd Health  
Preventive Health Strategies: Youngstock  
Susan L. McClanahan, DVM, MPH, DACVPM

**Introduction:** Flock and herd health preventive health strategies are the most important areas of management that should be emphasized by the producer and veterinarian. The most profitable producers receive the best economic returns when disease is kept to a minimum and a focus is placed on preventive flock/herd health. The veterinarian’s goal should be to improve profitability for the producer by designing profitable herd health strategies and not just providing emergency and sick animal veterinary services. Veterinarians need to work with producers on keeping morbidity and mortality rates to a minimum while improving profitability. These spreadsheet notes will review provide a framework for veterinarians and producers in designing a herd or flock health program for this age group.

<table>
<thead>
<tr>
<th>Life Stage</th>
<th>Tasks or Risks</th>
<th>Prevention Tasks</th>
<th>Treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weaning</td>
<td>Coccidiosis</td>
<td>Keep pens clean</td>
<td>CociTx in Feed or Water</td>
</tr>
<tr>
<td></td>
<td>Pneumonia</td>
<td>Intransal VAX</td>
<td>Reduce stress/ Abs</td>
</tr>
<tr>
<td></td>
<td>ORF</td>
<td>Tough to eradicate</td>
<td>Good nutrition/reduce stress</td>
</tr>
<tr>
<td></td>
<td>Ringworm</td>
<td>Keep areas lit/dry</td>
<td>Spot tx with dilute chlorhexidine</td>
</tr>
<tr>
<td></td>
<td>Polio</td>
<td>Step up grain in stages</td>
<td>Injectable thiamine ASAP</td>
</tr>
<tr>
<td></td>
<td>Pink Eye C. perfringens</td>
<td>Reduce crowding/flies</td>
<td>Treat as needed with Abs</td>
</tr>
<tr>
<td></td>
<td>Sudden death</td>
<td>Step up grain in stages</td>
<td>CD/T vax to reduce incidence</td>
</tr>
<tr>
<td></td>
<td>Health check</td>
<td>Check animals 2x daily</td>
<td>Necropsy</td>
</tr>
<tr>
<td></td>
<td>Abomasal</td>
<td>Check for imperfections</td>
<td>Cull/sell imperfect lambs/kids</td>
</tr>
<tr>
<td></td>
<td>Bloat</td>
<td>Step up grain in stages</td>
<td>DRF</td>
</tr>
<tr>
<td></td>
<td>Monitor growth rates</td>
<td>Weigh, observe 2x daily</td>
<td>Oral oxtet, decompress, fluids, pain meds</td>
</tr>
<tr>
<td></td>
<td>Update Records</td>
<td>Official ID</td>
<td></td>
</tr>
<tr>
<td>PreBreeding</td>
<td>check BCS</td>
<td>anestrus</td>
<td>Feed for growth</td>
</tr>
<tr>
<td></td>
<td>FAMACHA</td>
<td>weight loss</td>
<td>Tattoo ears/register or keep ID increase/decrease plane of nutrition</td>
</tr>
<tr>
<td></td>
<td>check feet</td>
<td>lameness, foot rot</td>
<td>deworm if necessary</td>
</tr>
<tr>
<td></td>
<td>cull poor</td>
<td>financial losses</td>
<td>trim feet</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>ship to market: DRF</td>
</tr>
</tbody>
</table>
ens
check
udder
agalactia/neonatal losses
problem udders: ship to market
assign permanent ID, update
records
ensure ID
loss of identity
VAX PRN
VAX CD/T
check
overeating/tetanus
tooth
weight and $ loss
cull if bad broken mouth
interact with flock/herd 2x or
more daily
habitate
stress, flight/fight
check
financial losses
record
make educated decisions on
breeding/culling
provide iodized salt
Goiter/ neonate losses
provide iodized granular salt
keep feed dry/off
parasitism/$ losses
ground
provide off ground feeding
Vit/Minerals PRN
WMD
develop repro
Vitamin E and/or Selenium
records
record breeding dates 2x or more
daily

Abbreviations

BCS  body condition score
DRF  drug residue
ID   official identification
VAX  vaccinate
PRN  as needed
C. perfringens
CD/T CD/Tetanus
WMD  white muscle disease
ASAP possible
as soon as
BAR  responsive
Abs  antibiotics

References for Sheep and Goat Veterinarians

7. Sheep Medicine (2007), Scott
8. Southern Consortium for Small Ruminant Parasite Control (www.scsrpc.org) for a copy of
   the FAMACHA presentation.
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<th>Treatment</th>
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</thead>
<tbody>
<tr>
<td>Pre-Adult</td>
<td>update records FAMACH A</td>
<td>financial losses</td>
<td>sort youngstock</td>
</tr>
<tr>
<td></td>
<td>check BCS make decisions provide iodized salt</td>
<td>weight loss</td>
<td>deworm if necessary increase/decrease plane of nutrition</td>
</tr>
<tr>
<td></td>
<td>monitor for ORF step up grain in stages</td>
<td>anestrus</td>
<td>sort youngstock</td>
</tr>
<tr>
<td></td>
<td>monitor for pneumonia check 2x daily</td>
<td>financial losses</td>
<td></td>
</tr>
<tr>
<td></td>
<td>rotate pastures/pens manage pop. density</td>
<td>reproductive losses</td>
<td>provide iodized granular salt spot treat with dilute chlorhexidine</td>
</tr>
<tr>
<td></td>
<td></td>
<td>tough to eradicate C. perfrin/sudden death can be silent, take temps</td>
<td>oral oxytet, fluids, pain meds injectable Abs</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&quot;silent&quot; diseases</td>
<td>necropsy &gt;2 deads</td>
</tr>
<tr>
<td></td>
<td></td>
<td>coccidiosis</td>
<td>feed/water coci meds reduce pop. density or increase space</td>
</tr>
<tr>
<td>Prebreeding</td>
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<tr>
<td>check BCS</td>
<td>anestrus</td>
<td>increase/decrease plane of nutrition</td>
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</tr>
<tr>
<td>FAMACH A check</td>
<td>weight loss</td>
<td>deworm if necessary</td>
<td></td>
</tr>
<tr>
<td>feet</td>
<td>lameness, foot rot</td>
<td>trim feet</td>
<td></td>
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<tr>
<td>cull poor ones</td>
<td>financial losses</td>
<td>ship to market: DRF</td>
<td></td>
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<tr>
<td>check udder</td>
<td>agalactia/neonatal losses</td>
<td>problem udders: ship to market</td>
<td></td>
</tr>
<tr>
<td>ensure ID</td>
<td>loss of identity</td>
<td>assign permanent ID, update records</td>
<td></td>
</tr>
<tr>
<td>VAX PRN check</td>
<td>overeating/tetanus</td>
<td>VAX CD/T</td>
<td></td>
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<tr>
<td>teeth</td>
<td>weight and $ loss</td>
<td>cull if bad broken mouth</td>
<td></td>
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<tr>
<td>habituate check</td>
<td>stress, flight/fight</td>
<td>interact with flock/herd 2x or more daily</td>
<td></td>
</tr>
<tr>
<td>records</td>
<td>financial losses</td>
<td>make educated decisions on</td>
<td></td>
</tr>
<tr>
<td>provide iodized salt</td>
<td>Goiter/ neonate losses</td>
<td>breeding/culling</td>
<td></td>
</tr>
<tr>
<td>keep feed dry/off ground</td>
<td>parasitism/$ losses</td>
<td>provide iodized granular salt</td>
<td></td>
</tr>
<tr>
<td>Vit/Minerals PRN develop repro records</td>
<td>financial losses</td>
<td>provide off ground feeding</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>Vitamin E and/or Selenium</td>
<td></td>
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<tr>
<td></td>
<td></td>
<td>record breeding dates 2x or more daily</td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Early FAMACH</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>weight loss</td>
<td>deworm if necessary</td>
<td></td>
</tr>
<tr>
<td>pregnancy check</td>
<td>financial losses</td>
<td>ultrasound, count fetuses</td>
</tr>
<tr>
<td>check BCS</td>
<td>anestrus</td>
<td>increase/decrease plane of nutrition</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Late Gestation</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>check BCS</td>
<td>ketosis</td>
<td>increase/decrease plane of nutrition</td>
</tr>
<tr>
<td>FAMACH A</td>
<td>lamb/kid losses</td>
<td>deworm only if necessary</td>
</tr>
<tr>
<td>check udder</td>
<td>if enlarged</td>
<td>place in lambing/kidding area</td>
</tr>
<tr>
<td>ensure ID</td>
<td>loss of identity</td>
<td>update records</td>
</tr>
<tr>
<td>habituate check</td>
<td>stress, abortion</td>
<td>interact with flock/herd 2x daily</td>
</tr>
<tr>
<td>financial losses</td>
<td>financial losses</td>
<td>make educated decisions on</td>
</tr>
</tbody>
</table>
records
provide iodized salt
plan for lambs/kids dystocia
clean, clean, check freezer (colostrum)
Vit/Minerals PRN check repro records
watch for problems
check for vaginal prolapse
develop protocol
Goiter/neonate losses
neonate losses
diarrhea, parasites, etc
septicemia
WMD, Rickets unexpected neonates
ketosis or hypocalcemia financial losses
neonate losses
lambing/kidding
provide iodized granular salt
organize/clean/prep area for neonates
put together dystocia kit, review procedures
clean, clean, clean
keep heat treated colostrum in freezer
Vitamin E and/or Selenium, Vitamin D
makes notes
Dextrose, CaBorogluconate, PropGlycol, etc
repair vaginal prolapse as needed
develop protocol on how neonates will be handled

Abbreviations

BCS   body condition score
DRF   drug residue
free
ID    official identification
VAX   vaccinate
PRN   as needed
C. perfringens
CD/T  CD/Tetanus
WMD   white muscle disease
as soon as
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References for Sheep and Goat Veterinarians

9. Sheep and Goat Medicine (2011), 2\textsuperscript{nd} edition, Pugh and Baired

10. Goat Medicine (2009), 2\textsuperscript{nd} edition, Smith and Sherman

11. Sheep Medicine (2007), Scott

12. Southern Consortium for Small Ruminant Parasite Control (www.scsrpc.org) for a copy of the FAMACHA presentation.
Sheep and Goat Medicine
Parasites (FAMACHA emphasis)
Susan L. McClanahan, DVM, MPH, DACVPM

Parasites

Internal parasites are a very common problem and often a contributing factor to other diseases and production problems in sheep and goats. In neonatal kids/lambs, cryptosporidiosis is the most common cause of diarrhea and elevated mortality rates. At weaning, coccidiosis becomes the most common cause of diarrhea and production losses in youngstock. At this stage, amprolium or water containing sulfa medications are the most common treatments for coccidiosis control. Weight loss in adult sheep and goats is most commonly associated with parasitism. An emphasis should be placed on management strategies such as body condition scoring, pasture rotation, feeding off the ground, fecal analysis, and FAMACHA. Internal parasites still remain the largest threat to productivity and economic gain to sheep and goat producers throughout the world. Environmental conditions play a large role in parasite control and resistance to dewormers is related to weather and how they are administered by producers and veterinarians. Deworming control strategies should not be based on a loose deworming schedule but should be developed per flock or herd. It is not uncommon for producers or owners to ask for general deworming advice. Deworming should be based in a “risk analysis” of the sheep or goats per premises. Best management practices need to be emphasized to as to reduce the potential for parasite resistance.

_Haemonchus contortus_ (barberpole worm) remains the major parasite of concern since it can cause severe anemia and death. Using the FAMACHA system should be mandatory by every veterinarian and producer who work with sheep and goats. Fecal analysis should also be a tool utilized more often with fecal egg counts being the most useful in determining when to deworm small ruminants. To determine if a farm is experiencin_g anthelmintic resistance, fecal egg counts should be performed before and 10-14 days after deworming to determine the effectiveness of the dewormer. Gross deworming of sheep and goats without consideration to product labeling or management practices increases the potential for anthelmintic resistance.
In this lecture, the main emphasis will be placed on reviewing the principles of FAMACHA system. Using this system, animals are identified as needing treatment based on the evaluation of their conjunctiva. Currently, Dr. Ray Kaplan at the University of Georgia has been instrumental in teaching veterinarians and producers about FAMACHA. Once producers and veterinarians complete the training process, they may contact famacha@uga.edu for the FAMACHA anemia color chart. For more information and a copy of the FAMACHA presentation, contact the Southern Consortium for Small Ruminant Parasite Control website (www.scsrpc.org)

External parasites in sheep and goats are generally not life threatening but may affect the health of young and old animals. Biting lice can be irritating and cause reducing in feed intake. Sucking lice consume blood and cause anemia in compromised animals. Python Dust (Zeta-cypermethrin) and Atroban EC (permethrin) are approved for sheep and goats. There are no approved injectable avermectin type products available for sheep and goats in the United States for the control of external parasites. However, injectable Ivermectins are often used along with a topical agent.

References for Sheep and Goat Veterinarians

15. Sheep Medicine (2007), Scott
16. Southern Consortium for Small Ruminant Parasite Control (www.scsrpc.org) for a copy of the FAMACHA presentation.
Tetanus

Tetanus is caused by the organism *Clostridium tetani* and is a common environmental bug remaining viable in soil for years. Infection usually occurs in unvaccinated herds or flocks and is associated with disbudding, dehorning, castration, tail docking, skin injuries, or metritis. Producers have reported outbreaks of tetanus in lamb crops after castration or tail docking. Early clinical signs include stiffness or “lameness” in one limb. Within 24 hours, owners will report generalized stiffness or the classic sawhorse stance. Treatment can be intensive and involves anti-toxin, fluid therapy, TPN, sedation, wound identification/care, and high doses of penicillin. Prevention is inexpensive using the CD/T vaccine with two doses on youngstock and yearling on adults. Lambs and kids will initially receive good protection from colostrum with the two dose vaccination starting at 2-3 months of age.

Ovine Progressive Pneumonia

OPP is a common lentivirus in the sheep population and is most often transmitted by colostrum or direct contact with nasal secretions. Clinical signs most often appear as a chronic pneumonia leading to a chronic cough and weight loss. Some may develop swollen joints and firm udders similar to CAEV in goats. Serology and necropsy are diagnostic. Control and prevention strategies involve removing the lambs from the ewes ASAP (which may not be practical) and feeding heat treated colostrum. Eradication can involve serological test and remove strategies.

Bacterial and Parasitic Pneumonias

Lungworms are common in even well managed flocks/herds. *Dictyocaulus filarialis* is the most pathogenic species while *Muellerius capillaris* is the most common of the lung parasites. Both cause persistent and chronic coughing in BAR ewes/goats yet can lead to chronic weight loss. Most anthelmintics are effective and diagnosis is via the Baermann technique.
**Caprine Arthritis Encephalitis Virus**

CAEV is a common lentivirus in the goat population and is most often transmitted by non-heat treated colostrum. However, milking parlors/equipment, contact with infected individuals, and inhalation are often routes of transmission often overlooked. It is not uncommon for CAEV negative herds to silently seroconvert a herd over time until a few sentinel animals develop carpi arthritis surprising the owner. The majority of CAEV+ animals do not develop clinical signs over time and can maintain very high lactation production records. There are four forms of the disease: arthritis in adults, leukoencephalomyelitis in kids, agalactia in does, and pulmonary fibrosis in adults. The most common presentation in adults is chronic hyperplastic polysynovitis arthritis in the carpal joints of adults. Control and prevention strategies involve removing the kids from the doe ASAP and feeding heat treated colostrum.

**Johne’s**

Johne’s disease is caused by *Mycobacterium avium subsp paratuberculosis*. Transmission most commonly occurs through non-heat treated colostrum or via the fecal-oral route (contaminated water, feed, bedding). Lambs and kids are most susceptible. Chronic weight loss and anorexia are the most common clinical signs at 2-3 years of age. Diarrhea is not common unlike in cattle and only appears at end stage. Fecal PCR is an excellent diagnostic test for individuals (provided they are shedding the organism) and for herd surveillance. Serology can cross react and yield false+ results. Johne’s can be introduced into a flock/herd by producers using non heat treated cow colostrum. Control and prevention strategies involve removing the kids from the ewe/doe ASAP and feeding heat treated colostrum. Johne’s eradication in flocks or meat goat herds can be challenging but very achievable in dairy flock/herds. Pygmy goats (often called the “Gypsies” of the goat world) or sheep/goats purchased from livestock auction markets should be considered risky purchased additions.

**Caseous Lymphadenitis**

*Corynebacterium pseudotuberculosis* is a very common agent in small ruminants. The organism survives well in the environment, contains a pyogenic lipid cell coat causing abcess
formation, and has the ability to invade tissues via its phospholipase D exotoxin. Transmission occurs via contamination of superficial wounds via scratching, shearing, dehorning, head butting, or via inhalation. Most common clinical signs are enlarged asbscessed lymph nodes or chronic cough or weigh loss due to internal abscesses. Diagnostics such as serology and culture are helpful. Lancing and draining soft abcesses aseptically to prevent environmental contamination and spread is advisable. Control by vaccination will reduce abscess formation but not prevent infection. CL vaccines has produced anaplyaxis in goats more so than sheep. Prevention should focus on hygiene and possible removal of infected animals depending on the producer’s goals.

**Mastitis**

There are many pathogens that cause mastitis in ewes and does. However, *Staphylococcus sp.* are the most common organisms isolated. *S. aureus* is associated with clinical, subclinical, and gangrenous mastitis. Coagulase negative Staph (CNS) can be the cause of subclinical and chronic infections. Streptococcal species are not as common in ewes/does as in cattle. Coliforms are also rare in ewes/does as other environmental bacterial. Pseudomonas infections are usually caused by contaminated iodine teat dip. *Mannheimia haemolytica* is also rare but can be seen in ewes nursing lambs causing a gangrenous mastitis. Contagious agalactia syndrome produced by Mycoplasma spp. can be found in dairies causing clinical signs of watery mastitis developing into a purulent thick secretion. Suckling kids and lambs may develop pneumonia, arthritic, and fatal septicemia and treatment is usually not effective. Diagnosing mastitis in sheep and goats can be challenging. In goats, the CMT and SCC are not strongly related to udder health. CMT scores of trace or 1 are considered normal. CMT scores of 2 or 3 may reflect a problem suggesting milk cultures. However, cultures must be performed 2x with the same result in order to diagnose a specific bacterial mastitis. SCC are often very elevated at kidding and at dry off. Older does also tend to have elevated SCC. Agalactia caused by OPP and CAEV is a common complaint by producers. Palpation of the udder should always be a component during PE of ewes and does. Treatment of bacterial mastitis is usually based on lactating cow treatments with at least doubling of the milk withholding time.
Abortion

Abortion outbreaks are usually multifactorial and involve evaluation of the herd/flock health status, history, and management. Many times the etiology of a one or two abortions may remain unknown. However, if >3 ewes/does abort, a diagnostic work-up involving a diagnostic laboratory is necessary since most if the abortion agents are infectious and zoonotic. The most common causes of abortion are Toxoplasma, Salmonella, Campylobacter, Chlamydia, Cache Valley Virus, Listeriosis, and Q-Fever. Before heading out to the farm, it is advisable to contact a diagnostic laboratory for advice. Make every effort to collect placental tissues in addition to aborted fetuses and blood samples (red and purple top tubes). Many times reproductive losses involve poor management, introduction of animals with unknown history, poor nutrition, and parasitism. Control can involve ionophores and tetracycline depending on the purpose of the herd/flock (meat, milk, or pets). Chlamydia and Campylobacter vaccines are available and worthwhile. Be aware of zoonotic disease and find out if anyone involved may be immunocompromised or pregnant.

Pink Eye (Infectious Conjunctivitis)

The most common organisms isolated in sheep and goat infectious conjunctivitis include: Branhamella ovis, Micrococcus sp, Stretococcus sp, Corynebacterium, Bacillus, Neisseria, Staphylococcus, Pseudomonas, and Moraxella. Moraxella bovis is not a cause of pink eye in goats but may be in sheep. Mycoplasma sp and Chlamydia sp are ocular pathogens as well. Clinical signs include hypermic conjunctiva, blepharospasm, epiphora, and marked corneal ulceration. Diagnosis by culture and cytology can be challenging and unrewarding. Treatment with SQ injectable long-acting oxytet, NuFlor, Excenel, or Draxxin (adhere to milk/meat withdrawal) and ophthalmic Ab ointment are very effective. Sheep and goats heal quickly and do not have the ocular treatment complications that horses have.

References for Sheep and Goat Veterinarians


19. Sheep Medicine (2007), Scott
Pregnancy Toxemia

Pregnancy Toxemia most often occurs in ewes and does during the last 6 weeks of gestation. Clinical signs may be vague and develop over a period of 3-10 days. Owners may not notice the initial clinical signs and may wait to call a veterinarian until the ewe/doe is down, anorexic, and depressed. By this time, treatment becomes unrewarding since the prognosis is poor. Risk factors associated with pregnancy toxemia include excess nutrition (excess BCS), under nutrition (low BCS), or a secondary health issue such as “broken mouth”, old age, multiple fetuses, lameness, overcrowding, parasitism. Other factors such as inclement weather, lack of exercise, uncontrolled dogs, introduction of new sheep/goats, and recent management changes can lead to pregnancy toxemia. Diagnosis is based on signalment and clinical signs with treatment emphasis placed on supplying immediate energy sources such IV5% dextrose and oral propylene glycol (60cc Q8hours) to the ewe/doe. Oatmeal, oral electrolytes, and yogurt are also recommended along with Bvitamin complex. Hypocalcemia and parasitism should also be considered along with the possibility of an emergency C-section. Prevention is critical since the prognosis for an uncomplicated recovery from Pregnancy Toxemia is poor. Prevention includes adequate BCS, good nutrition (high quality hay, grain at ½-2lbs/day, 12%total protein in diet), forced exercise, shearing of ewes in 3rd trimester, consistent management, and ionophores (Bovatec: sheep, Rumensin: goats).

Hypocalcemia (Milk Fever)

Hypocalcemia usually occurs during the last 2 weeks of pregnancy as the demand for calcium increases. Ewes/does pregnant with twins, triplets, or quad are at high risk for this condition and can be associated with recent stressful events such as vaccination, inclement weather, moving to a new location, uncontrolled dogs, or changes in feeds or feeding schedule. Clinical signs usually develop quickly with ewes/does initially appearing stiff legged or ataxic. Progression of the disease leads to hyperexcitability, tremors, and recumbency. Ewes/does left untreated can die within 6 hours. Treatment can be very rewarding with 60-100 cc of
CaBorogluconate (CaB) given IV slowly. CaB can also be given SQ with oral calcium gels given orally. Prevention includes good management, no sudden changes, high quality alfalfa hay, mineral mix containing calcium. While treating the ewe/roe, check the remainder of the flock/herd for other potential candidates for milk fever.

**Copper Toxicity**

Copper toxicity remains a major problem in sheep despite widespread knowledge of this disease. Copper toxicity is rare in goats unless they are forced fed copper boluses by owners who believe their animals may be “copper deficient”. Sheep mistakenly fed cattle, goat, or horse feed or provided cattle or equine mineral blocks can develop acute copper toxicity. Or sheep may accumulate copper and suddenly develop the “acute on chronic” form of the disease. Many times, the source of copper remains unidentified yet feed samples should be analyzed since mixing errors can occur at the local feed mill. Stress such as a recent movement of the flock can cause an “outbreak” of copper toxicity. Clinical signs may look like acute pneumonia with rapid respiratory rates yet examination of the conjunctiva/sclera always reveals the disease. Pale conjunctiva and icteric scleral result from the hemolytic crisis. Looks for hemoglobinuria as well (owners may call stating that their sheep have “red” urine). Death losses can be significant and necropsy is paramount revealing icteric tissues and liver necrosis. Treatment can be challenging and often unrewarding. Options include fluid therapy, ammonium tetrathiomolybdate (IV, SQ), or feed supplemented with ammonium thiosulfate and ammonium molybdate. Prevention remains constant education, supplemental Mo, and reducing dietary exposure to copper.

**Urolithiasis**

Urolithiasis is most commonly seen in feedlot lambs, wethers, and pet Pygmy wethers. Rams/bucks castrated before 3 months of age may be a contributing factor along with diets of high grain or grasses associated with high levels of silicates or oxalates. Wethers may vocalize and appear “constipated” with rectal prolapse due to straining. Urethral or bladder rupture is not uncommon with the majority of uroliths composed of magnesium ammonium phosphate
(struvite). Many times, the grit or stones may be lodge at the urethral process which can be removed. Otherwise, surgical intervention (cystotomy or urethrostomy) is most common. Medical treatment with Walpole’s solution is another option that is less expensive yet riskier. Fluid therapy is paramount since BUN, Creat, and potassium levels are markedly elevated. Prevention focuses on Ca:P ratio of at least 1.5 to 1.0 or greater to prevent struvite stone formation. Salt at 1-4% of the ration to increase water consumption is helpful. Ammonium chloride added to the diet is unpalatable and may not acidify the urine. Education is critical, particular to owners with “pet” wether goats.

**Polioencephalomalacia (Polio)**

Polio results from a thiamine deficiency. Involved in the production of ATP, cells of the brain’s outer cortex may die, resulting in clinical disease. Response to treatment is dependent on the owner to notice early clinical signs such as depression, blindness, and circling. Usually, a history of recent access to large amounts of grain, poultry feed, chronic Amprolium exposure, plummeting outdoor temperatures can cause Polio. Treatment needs to be immediate to prevent further brain damage and included Thiamine hydrochloride at 10mg/kg IV then IM BID for 2-3 days minimum. Dexamethasone in nonpregnant does/ewes can also be helpful at 1-2mg/kg to reduce cerebral edema. Acute polio cases usually respond to treatment quickly.

**Rickets**

Vitamin D deficiency in young ruminants can cause failure of proper cartilage mineralization creating the condition called Rickets. Most commonly seen in young growing kids/lambs raised indoors due to harsh winter conditions. Clinical signs include stiff gait, reluctance to move, enlarged joints, or dark/thick hair coated youngstock. Blood chemistry will reveal elevated ALP and low levels of calcium or phosphorus. Radiographs reveal widened growth plates, bowing of long bones, and thin cortices. Treatment is SQVitamin D3 (as VitA/D injectable). Use with caution to prevent Vit D3 toxicity. Prevention is exposure to sunlight, VitA/D injections, and dietary Ca:P ratio of 1:1 or 1:2.
Laminitis

Laminitis is fairly common in sheep and goats with recent exposure to large amounts of grain intake or a lush forage diet. This disease can also occur secondarily to a systemic illness such as toxic mastitis, acute bacterial pneumonia, or metritis. Fortunately, treatment of laminitis in small ruminants is much easier and rewarding than it is in equines. Clinical signs include lameness, increased recumbency, and stiff gait. Treatment includes flunixin meglumine (as labeled for food animals) and aspirin. Foot trimming over a period of several months may also be required.

Foot Rot

Infectious foot rot is more common in sheep than in goats. *Dichelobacter nodosus* is the primary anaerobic bacteria involved but previous infection with *Fusobacterium necrophorum* can be a contributing factor. Most commonly seen during periods of warm, wet weather, this highly contagious bug can affect entire flocks of sheep. Severity appears to increase with age with Merino sheep as most susceptible. Excessive horn growth is also a factor with prevention focused on good foot care and trimming when necessary. Sheep can become carriers and the organism can persist in the environment for days to weeks. Animals are painful on their feet and may chose to spend most of their time grazing while on their carpi. Virulent forms cause marked lameness and a malodorous exudate. Treatment includes foot trimming, systemic Abs, and foot baths (not containing copper). Attention to good foot care and trimming and pasture management to prevent this potentially disastrous disease is paramount.

Enzootic Nasal Adenocarcinoma

This tumor may be uni- or bilateral causing a progressive inspiratory dyspnea in sheep and goats. It may be flock associated and generally occurs in younger adult animals. Exercise tolerance, loud inspiratory noise, head shaking, and open-mouth breathing are common clinical signs. The tumor is locally invasive and is often identified in advanced stages where erosion into other bones of the skull has already occurred. Early diagnosis with rads and biopsy can support surgical removal of the mass. However, most cases present in late stages of this
problem.

**Abomasal Emptying Defect**

Most commonly seen in Suffolks less than 2 years old. Cause of the disease remains unkown but may be associated with high concentrates (in rams) or during the lambing period. Clinical signs include weight loss, anorexia, and abdominal distension. Metabolic alkalosis (mild hypochloremic) and elevated rumen chloride levels may be present. Prognosis is poor and medical/surgical treatment is not recommended.

**Conclusion**

Although individual veterinary treatment of sheep and goats remains necessary, herd health programs that promote profitability for sheep and goat producers are essential. Close consideration should be given to the variables that may be limiting productivity and financial outcome. When presented with diseased sheep and goats, one must not only treat the disease but examine the contributing factors in order to successfully develop preventive herd health strategies that promote a viable economic model for the producer.

**References for Sheep and Goat Veterinarians**


22. Sheep Medicine (2007), Scott
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Quality of life Part 1: What is it and how do we measure it?

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WHAT DO WE MEAN BY QUALITY OF LIFE?
The strong intuitive sense that quality of life (QOL) is straightforward and “everyone knows what it means” is an illusion. Consider just a few of some of the many enigmas that a true understanding of QOL must account for:

- The disability which many regard as a worst-case scenario is paralysis from spinal cord injury. Yet when victims of such catastrophic trauma are interviewed more than 20 years after their injury, 75% rated their current QOL as either good or excellent. How could people regard something with such a high QOL outcome as a worst-case scenario?
- If a person paralyzed and confined to a wheelchair rated his own QOL as excellent, what would happen to his QOL if he were to regain the use of his limbs? Would it go up? It seems like it should. But if it does, wouldn’t he end up with a QOL higher than before his injury? If not, why would he care whether he was ever able to walk again?
- Imagine a dog who spent the first 6 years of her life in a puppy mill, never being properly socialized to people. While we know that some unsocialized dogs can, in time, become socialized enough with people that they can live in peaceful coexistence with humans, let’s assume that she is one of the ones who will never overcome her fear of people. Then she is rescued. Taken out of the only environment she knows, she is terrified of everything, including any person who tries to comfort her and give her love. She lives in a near-constant state of fear. What did being rescued do to her quality of life?

Yet if one asks a pet owner to evaluate their animal’s QOL he or she will almost invariably undertake the task with not a single question as to what exactly they are supposed to be evaluating. They just know. Ask any veterinarian to evaluate a patient’s QOL and he or she feels no need to ponder what he or she is looking for. They, too, just know. Yet when these evaluators are asked to detail their reasoning for the QOL “value” they arrived at, their responses are wide-ranging and follow no particular rules or guidelines. For example, one pet owner might judge his dog’s QOL as very low because she has lost all of her vision, whereas another owner might judge her blind dog’s QOL as very high because he plays, gets around, and functions as if nothing is wrong. Who is right? Knowing would require, as a simple starting point, a relatively precise definition of QOL.

Despite the strong intuitive feeling that we understand what QOL is, the term currently defies precise description. This problem is not confined to animals; QOL in humans, even when people can provide detailed self-evaluations, remains an elusive and debated concept. This is because QOL is a personal, private, subjective experience, has no ‘normal,’ ‘average,’ or any
other frame of reference, lacks any units of measurement, and means different things to different people. Most importantly, at this point in time no sentient individual on Earth (human included) can know exactly how another sentient individual feels. This applies to each discrete feeling, such as physical pain or anxiety, but much more so for the almost infinitely more complex mental experience of QOL. But because in veterinary medicine QOL is used as a guide for life and death euthanasia decisions—literally who lives and who dies—it is of the utmost importance that we try to move beyond “gut-level” and intuitive assessments of QOL.

Quality of life is involved in every aspect of animal care. There is little argument that maximizing animal QOL is the most important ultimate objective of veterinary care, and this means that knowing what QOL is and the factors that affect it are crucial for proper, complete, and effective animal care.

In humans QOL is considered strictly a view from within; it is not an external evaluation of how others judge a person’s life, but how that person feels about the circumstances and events making up his or her own life and what they mean to that person and that person alone. This aspect of QOL has led to the general consensus that QOL should be assessed from the perspective of the individual, with that individual utilizing his or her own values. In animals, QOL is not restricted to what kind of housing the animal has, the type of food he gets, the luxuriousness of her bed, the number of walks he gets per day, what size of yard she has to play in, whether he goes to doggie day care or stays home alone all day, or whether she has animal companions to play with. And most important, it is not restricted to—or equivalent to—his health status. It is a compilation of all of these factors and more, and the animal’s reaction to and feelings about them.

QOL is individualized; unique sets of preferences, desires, and needs, lead each individual—animal or human—to assign different values to the vast array of events and conditions in his or her life. For example, one person may value family relationships over his career, whereas another person may value the same things oppositely. A good illustration in animals is the value of human companionship in dogs. If deprived of human interaction and companionship, one dog may be unaffected whereas another dog may be emotionally debilitated. The experience of QOL is dependent upon what matters to that individual animal.

All interpretations view QOL as a continuum, ranging from very high (good) to very low (bad). Because such determinations are subjective and do not only vary between individuals but vary in the same individual over time and under different circumstances, there are no clear-cut demarcations or recognizable cutoff points on the continuum, for example, above which QOL is “satisfactory,” “acceptable,” “reasonable,” or “good,” or below which QOL is “unacceptable.”

So what do we mean by quality of life in animals (and well-being, welfare, happiness, and life satisfaction)? It can best be understood as one’s level of enjoyment of life. This assures that the perspective comes from within—from the animal’s point of view—not from outside, such as looking at a photo of Elizabeth Taylor petting the immaculately groomed silver Persian cat on her lap while the cat is eating caviar out of a crystal goblet and having an automatic thought
that “this kitty has the ultimate quality of life.” What is this cat’s enjoyment of life? He may be living an extremely uneventful and unfulfilling life. This is what is meant by quality of life.

**THE FEELINGS OF QUALITY OF LIFE**

Knowing what the experience of QOL describes does not tell us what factors contribute to or influence QOL. And it says nothing about exactly how the contributory factors exert their effect on QOL. Consider the latter question of what contributes to or influences QOL—how does one decide whether or not something affects an animal’s QOL? Imagine that you are given a list of factors in your pet dog’s life and you are asked to place each factor into one of two columns: Has an effect on QOL, and Does not have an effect on QOL. You are given such factors as: painted toenails, very tasty food and treats, a loving and caring human family, a scar on his face, diamond studs in her collar, lots of play and trips to the dog park, partial loss of vision in one eye, being left at home alone for 14 hours 7 days a week, having her white doghouse painted light blue, having been born with the outside toe missing on his left front foot, having prosthetic testicles implanted after being neutered, being physically abused, living in a city that doesn’t have a Starbucks, being shuffled from one foster home to another every few months, having severe osteoarthritis, having a small lipoma on her abdomen, having epilepsy, living her entire life in a tiny dirty cage having litter after litter in a puppy mill, being a quadriplegic, and being the descendant of a line of best-in-show grand champions. As you place each factor into one column or the other, you are obviously using some criterion to decide into which column to place each one. There is clearly some distinguishing feature that separates the factors in the “does have an effect” column from those in the “does not” column. What is that factor?

Feelings—physical and emotional, pleasant and unpleasant—appear to have evolved as a way for the brain to assign survival value to events in life. Feelings represent those things that matter to the animal, and the intensity of the feeling appears to represent how much those things matter. QOL would presumably involve only those things that matter to the animal, and thus be those things that elicit feelings. Current research and observations suggest this axiom: Unaffected feelings means unaffected QOL. The single factor that you were using to intuitively place the various factors above onto the “does” and “does not” have an effect on QOL is the simple fact that those on the “does” list elicit some feeling—pleasant or unpleasant—and those on the “does not” list do not.

Quality of life in animals appears to be comprised of the balance between pleasant and unpleasant feeling states. In this view, QOL may be viewed as a scales, with pleasant feelings on one side and unpleasant on the other. The direction of tipping of the scales represents the individual’s QOL. Quality of life increases when the balance tips toward the pleasant feelings, and declines when the balance tips toward unpleasant feelings. A key feature of the scales model of QOL is that it becomes very clear as to which factors in life contribute to QOL. Anything which tips the QOL scales—in either direction—plays a role in the animal’s QOL, but those things that do not tip the scales do not affect the animal’s QOL. For example, the reason a cat food in little shapes of chicken legs and fish would not affect QOL whereas a distasteful food would, is because the former would not tip the QOL scales and the latter would. Likewise for a cat born with excessive toes (no tipping of the scales) versus a feral cat being brought
inside and confined to a cage (tipping of the scales). On the QOL scales the intensity of the feelings dictates the degree to which the scales are tipped, and hence defines the magnitude of influence that factor has on QOL.

Another consideration with feelings is that all feelings don’t “weigh” the same in one’s life experiences. The feeling of being unable to breath, or pain, or terror, all have a much stronger impact—they weigh more—on your sense of well-being than do the feelings of a grain of sand in your eye, an itch, or the bloated feeling after a huge meal. Furthermore, much research has shown that negative (unpleasant) feelings command more attention and urgency than positive (pleasant) feelings. This differential in weights of feelings is important in QOL, particularly in the feelings-based scales model of QOL.

MAJOR CONTRIBUTING FACTORS TO QUALITY OF LIFE
Several factors contribute to QOL, all having their influence through their associated pleasant and unpleasant feelings. Those with the greatest influence include (see Table for elaboration):

- **Social relationships**—Social bonds are promoted and enforced by pleasant and unpleasant emotions. Positive social affiliations and companionship elicit pleasant feelings, and separation and isolation elicit unpleasant feelings.
- **Mental stimulation**—Monotonous, unchanging environments elicit highly unpleasant feelings of boredom. Conversely, pleasant feelings are elicited by stimulation, challenges, and mental engagement.
- **Health**—Compromised health involves myriad unpleasant feelings. Physical disabilities limit one’s opportunities for experiencing pleasurable feeling states.
- **Food intake**—The pleasant taste of food and the unpleasant feeling of hunger both motivate consumption of nutrition to support life, and both may contribute the animal’s QOL.
- **“Stress”**—As a contributing factor to QOL, stress refers to specific unpleasant emotions such as fear, anxiety, loneliness, boredom, and anger. Its influence on QOL is through the feelings associated with these emotions.
- **Control**—In animals and humans, one of the strongest predictors of well-being is the perception of control over meaningful aspects of one’s life. The opposite—a sense of lack of control—is associated with feelings of helplessness and depression

MEASURING QUALITY OF LIFE
Difficulties in assessing QOL
Judging QOL is very difficult, in humans as well as animals. There is no QOL thermometer, no ability to quantify either QOL as a whole or the proposed contributing factors. Measuring even a single component of QOL, such as pain, is very difficult; therefore the much more complex totality of QOL is exceptionally difficult to assess. This makes QOL assessment a very inexact science, and open to influences such as personal bias. For example, it is very easy for a pet owner who wants to please the veterinarian to give an exaggerated report of improved QOL after treatment has begun. An owner who cannot “let go” may also falsely assess a pet’s QOL as higher than it actually is, in order to avoid the decision on euthanasia. Without an accurate and precise way to measure QOL, these errors are possible and frequent.
Weighing in all contributing factors—When assessing QOL it is extremely important that all relevant factors be weighed in. As we have seen, pain is important but only one factor of many in making up the pet’s QOL. A pet with moderate or major pain will have a severely impaired QOL, but of course an animal with no pain can also have a very low QOL because of the other contributing factors. It is important to first survey and identify all sources of feelings in the animal’s life—pleasant and unpleasant, emotional and physical in origin, health-related and non-health related. Because of their disproportionate contribution to QOL, unpleasant feelings warrant the highest priority in QOL assessment.

Accounting for the individual nature of QOL—Each animal’s individual personality and preferences lead to all contributing factors to QOL mattering differently to different animals. For example, something that is a great source of joy to one cat may be meaningless to another cat; likewise, something (like being left alone) that elicits severe emotional distress in one dog may have no effect on another. These individual needs, desires, values, and preferences are critical when evaluating QOL and attempting to maximize it. Because the pet owner has the greatest knowledge of their pet’s personality and desires, evaluating a pet’s QOL is very much a joint effort between the pet owner and veterinarian.

Methods for quality of life measurement in animals
A number of QOL assessment tools, or instruments, have been developed to serve a variety of purposes. Some have a narrow focus, usually on one specific medical disorder, and generate a health-related quality of life (HRQOL). The HRQOL refers to the individual’s QOL in areas of life affected by that specific medical disorder. Other disease-specific QOL instruments purport to measure overall QOL in the face of a medical disorder.

Mullan & Main (2007) designed a quality of life screening questionnaire for dogs. This instrument consisted of a series of questions about the animal’s subjective mental states and behaviors. The questionnaire was found to be repeatable, feasible, and to have good internal consistency and validity, leading the authors to conclude that it is suitable for use in veterinary practice to assess welfare. The authors pointed out that the questionnaire was designed as a screening tool and not to generate a single QOL score, and that it helped to identify areas where changes could be made to improve the dog’s QOL.

Schneider (2005) developed an instrument to measure overall QOL in healthy and ill dogs and to examine the relationship of QOL to the human-animal bond between the dog and her owner. The results showed that the QOL and human-animal bond assessments were generally reliable and valid, that in ill dogs QOL was associated with the dog’s health, and that the human-animal bond influences owners’ ratings of their dogs’ well-being.

Föllmi et al (2007) designed an assessment tool for use with geriatric zoo animals afflicted with physical health disorders. The intended use of the instrument was to facilitate euthanasia decisions, and did so by generating a numerical score that would indicate an unremarkable condition, a poor prognosis, or a recommendation for euthanasia. Although the authors claim
that the scoring system of the instrument is to evaluate physical condition and QOL, 1 of the 5 parts of the overall score is for the rater to score the animal’s QOL (the other 4 parts are: animal’s history, response to therapy, radiographic examination of the animal’s joints, and ‘additional assessment’ such as ability to reproduce and other factors relevant to the zoo in keeping or losing a particular animal). Since QOL is a part of the overall assessment, the tool is measuring a well-being construct of some type, but not strictly QOL.

Valsecchi et al (2007) designed and tested a set of methods for assessing the QOL of dogs in shelters utilizing cortisol measurements and behavioral criteria, which included the Strange Situation Test (Topál et al 1998) and response to an environmental enrichment program. The researchers determined that the shelter environment was stressful and that shelter dogs were able to form new attachment bonds with humans, but the report lacked a description of the method for QOL assessment of sufficient detail to be instituted in shelters at this time.

Villalobos (2004) proposed a method to assess overall QOL in animals with progressive disease using the “HHHHHM Scale,” an acronym for the separate criteria being assessed: Hurt, Hunger, Hydration, Hygiene, Happiness, Mobility, More good days than bad. The instrument has not been evaluated scientifically.

Wojciechowska et al (2005a, 2005b) developed a questionnaire designed to measure non-physical aspects of QOL in dogs. The questionnaire was based on objective list theory and hence was designed to utilize solely objective criteria. The questionnaire was unsuccessful in distinguishing healthy from sick dogs, and the authors suggested that this might be because certain factors were more important than others for individual dogs.

A recent report by Yeates et al (2008) described the development of the Quality of Life Assessment Tool—a qualitative instrument for obtaining a comprehensive owner-rated assessment of QOL in pet dogs. The authors reported that the test has been validated in a pilot study and by focus-group meetings. The report, however, does not contain the actual test and thus cannot be clinically applied at the current time.

McMillan (2003) proposed an informal “Quick assessment” tool for QOL that is based on the affect balance scale of Bradburn (1969). In this view, an individual’s happiness is represented by a balance of pleasant and unpleasant feelings in life—the greater the tilting of the scale toward the positive side, the higher the QOL. Current evidence in humans shows that the happiest individuals do feel occasionally unhappy, which detracts from the value of an assessment tool based on the affect balance model. The “quick assessment” survey asks the pet owner to inventory the pleasant and unpleasant feelings in their pet’s life and compare the pet’s current feelings with a point of time in the past when the animal was feeling his best, emotionally and physically. The tool has not been evaluated scientifically and its greatest value at this time appears to be that it raises the pet owner’s awareness to all of the possible sources of feelings that weigh into the pet’s QOL so that deficiencies can be targeted for improvement.

One frequent error in assessing QOL is to mistake the measurement of conditions of life for the
internal experience of QOL. A way of viewing QOL in a way that separates these 2 different concepts is the analogy of one’s sense of security in a particular house. Highly objective measurements can be made, in both intrinsic (e.g., double deadbolt locks on all doors, bars on windows, earthquake-reinforced foundation, fire-retardant materials, internal sprinkler system, nonslip surfaces on stairs, etc) and extrinsic (e.g., number of break-ins in this house and surrounding area in past year, number of fires, home accidents, etc). However, none of this objective safety data determines the person’s sense of safety/security, which is the person’s subjective feelings about the safety conditions, and is based on past experiences, personal fears, knowledge (e.g., how close, fast, and competent the police and fire departments are, etc). This is analogous to QOL: the objective safety factors of the house are comparable to the conditions of living, and the person’s QOL is comparable to his/her feelings about these objective conditions, which are dependent on personal experiences, etc. More deadbolt locks could be installed in the house, but this does not equate to how (or if) the person’s sense of safety/security changes as a result. Objective conditions of one’s life and the individual’s appreciation of the life are related, but independent, concepts.

How to recognize a good quality of life
Lacking a fool-proof method for assessing animal QOL, a reasonable alternative approach is to use the scientific findings to compose a typical description of a state of good QOL, a good enjoyment of life—a happy animal. Such signs would necessarily indicate that the animal’s physical and psychological needs are fulfilled, the animal is able to successfully cope with stressful stimuli and conditions, there is no distress or suffering, and that the animal is experiencing a substantial degree of and time in a positive emotional state. To that end, the following list is proposed.

10 Signs of a Desirable Quality of Life (subjective well-being, happiness, welfare, enjoyment of life), meaning that the animal is feeling good, with minimal unpleasant feelings and the ability to cope well with adversity.
The animal:
(1) is alert, busy (at species-typical levels)
(2) exhibits a substantial range of context-appropriate species-typical behavior
(3) engages in play behavior and activities that appear to cause excited interest and enjoyment, at a level that is typical and appropriate for the species and age of the animal
(4) displays an absence or no more than minimal levels of abnormal behaviors
(5) spends neither more nor less time sleeping than what is typical and appropriate for that species and age of the individual
(6) is confident, moves around freely, is outgoing, and does not display fear towards trivial or nonthreatening stimuli
(7) is able to rest in a relaxed manner, without constant signs of vigilance
(8) exhibits a very low incidence of behaviors associated with attempts to cope with (i.e., minimize the intensity and/or adverse psychological impact of) unpleasant affect, e.g., limping (pain), labored breathing (impaired oxygen intake), or escape behavior, hiding, and/or aggression (fear)
(9) eats normal quantities of food and maintains a normal and stable body weight
(10) is in good general physical health

**CONCLUSION**
No single thing is more important in veterinary medical care than the maximization of QOL. From the animal's perspective, the way its disease is treated, or even if it is treated, is unimportant as long as you, the practitioner can increase the animal's QOL. Maximizing QOL is accomplished by the dual effort of minimizing unpleasant feelings and promoting pleasurable feelings. This keeps the QOL scales tipped as far toward the pleasant side as possible, giving the animal the greatest possible enjoyment of life.

**References available from the author on request**
Quality of life Part 2: The puzzling aspects of quality of life
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Producing a single accurate measurement of quality of life (QOL) at a present point in time is exceptionally challenging. In caring for animals, a key necessity for optimal decision-making is being able to make more than present QOL assessments—it requires accurate predictions about how potential choices will affect the animal’s future QOL. However, QOL has no fixed anchor points, is dynamic and mutable with a constantly shifting frame of reference. Choosing a course of action that will, in the end, be the one that produces the highest QOL creates the very real challenge of hitting a moving target.

How elusive is QOL as an object of foresight? Let’s consider just a few of the conundrums that QOL prediction must accommodate:

- The disability which many regard as a worst-case life scenario is paralysis from spinal cord injury. Yet when victims of such catastrophic trauma are interviewed more than 20 years later, 75% of rated their current quality of life as either good or excellent. How could people regard something with such a high QOL outcome as a worst-case scenario?
- If a person paralyzed and confined to a wheelchair rated his own QOL as excellent, what would happen to his QOL if he were to regain the use of his limbs? Would it go up? It certainly seems like it should. If it does, will he end up with a QOL higher than before his disabling injury? If not, why would he care whether he was ever able to walk again?
- Happiness, a very closely related concept to QOL, was studied in 70 legally blind adult people ranging from 21 to 93 years of age. The researchers found that blind persons reported a higher level of happiness than sighted people. Does this mean that sight has a meaningless influence on happiness? If not, what can explain this result?
- It is commonly believed that children with severe mental disabilities have a reduced QOL and that an important focus of the care of these children should be on increasing their QOL (Hatton 1998). How would we predict their QOL if we took measures to improve it? Does raising their QOL mean trying to give them what "normal" people have? How do we know they would want this? And how do we know that giving them this would increase their QOL?

**IS QUALITY OF LIFE A MOMENTARY OR A LONG-TERM MOOD STATE?**

All well-being concepts have an intrinsic and unresolved problem as it pertains to the temporal nature of the subjective experience. Simply put, how long must a feeling or experience last, or how frequent must it occur, for it to be a QOL issue? For an animal, is a clinic stay of half a day a QOL issue? A one day stay? If a person has the 24-hour flu, does she have a low QOL for 1 day? Would this person say that last year her QOL was good except for March 26th, when she was sick? If someone’s pet dog wandered away from home one morning and was picked up, taken to the shelter, and then reclaimed by his owner later that day—did the dog have a low QOL for
that brief time away from home? If not, then his shelter stay wasn’t relevant to his QOL. But would a 6 week stay in the shelter be a QOL issue? Similar questions arise for frequency. Suppose John stubbed his toe yesterday. Did his QOL change? Was his QOL different on that day than it was the day before or the day after? But a sufficient increase in frequency would eventually raise the toe-stubbing to the level of being a QOL issue; for example, what if rather than every once and a while, John stubbed his toe every third step he took? Similarly, for a cat scared of the veterinarian’s office a yearly exam would not be a QOL issue, but if the vet visit were once a day, it clearly would be.

Researchers in the human field have typically drawn a distinction between the momentary feelings of life and the experience of subjective well-being, which represents a broader view of life as a whole. Quality of life in humans is not simply the momentary experiences in life; it is a, of how much one likes his or her life over time. Momentary experiences cause variability in pleasure, but QOL is a long-term mood state that transcends these temporary ups and downs. Imagine you are asking a client on the phone what she feels her dog’s QOL is. You expect a certain type of answer—a reflection of what kind of life her dog is experiencing overall over the past few weeks or so. You don’t expect an answer like “His QOL? Well, when he woke up this morning it was okay, I guess, but then it went way down when the garbage truck came by and scared him with its loud noise, then it went way up when I played fetch with his favorite ball, but then it went way down when his knee-cap popped out of place and made him limp something terrible...” The expectation you had for the client’s answer implied the existence of a long-term mood state; you weren’t inquiring about the dog’s current feelings. It seems, then, that QOL in animals must be made up of more than simply current feelings.

The important points here are that (1) there is no clear line separating momentary affective experiences from QOL issues, (2) the animals are likely unconcerned with any such distinction, they want to be relieved of unpleasant experiences regardless of duration, and hence, feelings—whether encompassed in a QOL concept or not—are important for animal caregivers.

Why is this important? If animal QOL is a long-term state, and there is evidence that it is, then short-term, momentary experiences probably have little if any impact on the animal’s QOL. And yet we still consider these short-term experiences important. Here’s an example: hot-iron branding in cattle. It seems safe to say that the cow’s overall, life-as-a-whole QOL is not appreciably affected by the branding, and yet no one would argue that therefore the experience doesn’t matter to the cow. The upshot of all of this is that contrary to common belief, we don’t – and mustn’t – pay attention only to things that affect an animal’s QOL. If we do, we will overlook many important short-term events that matter to the animal.

If it is true that animals, like humans, have both a momentary feeling state and a long-term mood state (QOL, or happiness), this raises new and important questions. For example, if an animal feels good right now, does that tell us anything about his long-term QOL/happiness? If animal happiness is like human, the animal could be momentarily happy and yet overall unhappy, or vice versa. If so, then assessing the animal’s current state (e.g., tail-waggingly happy) tells us nothing about the animal’s overall level of happiness.
THE “HAPPY SLAVE” PROBLEM

There is now a general consensus that QOL should be assessed from the perspective of the individual, incorporating that individual’s values and preferences. As logical as this view may be, however, it contains a crucial flaw. If one’s QOL is based solely on that individual’s assessment, adequate safeguards may be lacking to protect certain individuals from oppression, exploitation, and deprivation. Consider, for example, a woman in a part of the Middle East where women are badly oppressed who might be content with a small improvement in what from the outside looks like a miserable existence—and would look miserable to her, too, if her vision were not so limited. Would we say that she has a good QOL because she is content? If a slave happened to have a high degree of subjective satisfaction with his life, would we be compelled to agree with him? This is the “happy slave” problem. The reasonable view is that certain conditions, such as oppression and slavery, reduce the QOL even for those who cannot appreciate the fact.

An individual may become a “happy slave” in several ways. He could psychologically adapt to his current situation. He could become unable to recall the conditions of his earlier life, thereby having no higher standard with which to compare his current living conditions. He may hold lowered or mistaken standards, thereby making the current conditions look better than they are. And, perhaps the most likely, he could be unaware of what could be—the “it’s all he knows” situation. One can not want that which he is wholly unaware of. For example, an elderly woman who has learned to live with her arthritic hip, and is ignorant of the improvement that might be possible as a result of surgery, may record a high QOL because her expectation is close to reality. An example in animals can be seen in the breeding dogs in puppy mills, who spend their entire lives in cages or runs. One rescue organization has posted on their website the statement that “[The dogs’] life in the mill may have been what we would consider unpleasant, but it is the only life they have ever known,” which is virtually identical to a recent report of human slavery in present-day Niger: “[The slaves’] food and living conditions are inferior, but it is the only existence they know.” These statement are absolutely true, but in the same way we would view the slaves’ QOL, few people familiar with dog psychology and behavior would accept that the deprived existence in a puppy mill would permit a good QOL, regardless of how the dog perceived it. It has been also suggested that animals may not miss what they have never experienced. However, not missing something does not mean the individual’s QOL wouldn’t improve by having it.

One conceptualization of QOL that may help counteract the Happy Slave problem is termed the objectivist list theory. This view of QOL holds that optimal QOL results when the individual has certain things and certain conditions are met—e.g., basic physical needs, normal physiologic function and health, and sufficient and appropriate social interaction—whether or not he realizes it, and whether or not he desires it. This approach would not allow the individual’s limited view of the world unfairly constrict one’s judgment of how his or her life is faring.

One example of the Happy Slave problem in animals can be proposed for animals in shelters. It is possible that these animals have an altered perception of their past or of what is possible.
Also, shelter life may be an improvement over their prior living conditions. In each of these cases the animal may not be aware of a gap between his current experience and the possibilities of a life he would find more desirable—but we are. Using a measuring stick for QOL that emphasizes desires, we might imagine asking the animal if he has everything he wants—and the answer is yes. It has been shown, for example, that an always-confined animal will often show all signs of not wanting freedom. Veenhoven (2009) states the problem a bit more crudely when he writes “one can be perfectly happy in Hell, provided that one does not know better or that one is socialized to believe that this is the best place to be.” This is where all current methods of QOL assessment fail, as none adequately account for the satisfied-with-a-lousy-life factor. The most commonly suggested “fix” for this problem is to not rely solely on the individual’s perspective of his or her own life for QOL assessment, but to always include some objective conditions (as described by objective list theory) as a safeguard against the emergence of a “happy slave.”

This problem may be far more common that we have thought. Consider the cat who has been raised indoors since kittenhood—never knowing what the outdoors is like. If we were to ask the cat how he judged his own QOL and he said “it’s good,” how can we know that he isn’t a “happy slave” who views his life positively only because it’s all he’s ever known? In the end, we continue to face the unanswered question: How do we adequately safeguard against a Happy Slave situation?

**THE DISABILITY PARADOX**

In the previous section we looked at a study containing a highly counterintuitive finding: that those who actually experience something that the rest of us would consider devastatingly destructive to our quality of life—permanent paralysis—ultimately end up with a self-assessed QOL as high if not higher than the general population. This phenomenon, of course, greatly complicates any decision-making process based on predicted QOL.

In Western culture people seem to dread growing old, despite numerous studies showing that well-being actually improves with age. Lacey et al (2006) compared the self-reported happiness of younger adults (mean age = 31) and older adults (mean age = 68) with the same subjects’ estimates of what they believed happiness levels would be for people at different ages in life. The results confirmed that happiness increases with age, yet both younger and older participants believed that happiness declines. In other studies, many people report satisfaction in situations that the majority of the population believe (and hence predict) that they would find unbearable. Birnbacher (1999) writes of cancer patients who successfully adapt to a health situation they had thought intolerable at the time of onset of their disease. Quality of life in patients with spinal cord injury (SCI) has been examined in numerous studies similar to the long-term follow-up study cited earlier. In a commentary praising the success of current SCI treatments, DeLisa (2002) notes how multiple researchers have found that “the assumptions of those of us who are able-bodied bear little relationship to the realities of life for the people with SCI.” In a review of the research of QOL in SCI patients, Hammell (2004) looked at only those studies involving high (i.e., neck region) SCI. He concluded that “Reflecting prevailing cultural beliefs, health care professionals have been found to underestimate significantly the
QOL experienced by people with high SCI.” He believes that medical care decisions may be heavily influenced by society’s negative presuppositions about QOL in people with impairment.

The data show consistently and convincingly that it is common for people to mispredict the emotional impact of unfamiliar circumstances such as chronic illnesses and disability and the effect they will have on their well-being and QOL. Across a wide range of health conditions, people with illness or disability typically report greater happiness and QOL than do healthy people envisioning themselves in similar circumstances—this is the disability paradox. So common that it would not be unreasonable to consider it the norm, people routinely presume that they would be miserable if they experienced serious illness or disability. And most would be wrong.

How could the disability paradox apply to decision-making in animal QOL? Certainly we aren’t talking here about animals making complex decisions based on (mis)predictions of their own global QOL outcomes. The decisions made by humans that are based on predictions of future QOL may be highly influenced by this phenomenon. Consider that if the disability paradox shows that we do not see our own future QOL clearly, there is no reason to believe that we would be more successful in predicting an animal’s future QOL.

There are as of yet a paucity of studies in this area, but the few available support the view that animal care decisions are influenced by the disability paradox. For example, in a survey of 50 blind dogs, over 50 percent (28 of 50) of the dogs’ owners had encountered people who had suggested it was unkind to keep a blind dog. In this study, the majority view of the general public appears to be based on a presumption that blindness would so negatively affect QOL that keeping such a dog alive would be wrong. In a study of pet owner responses to amputation for their animal, 100 percent (7 of 7) of those whose main objection to the amputation was a prediction of a decreased QOL later stated that their concern was unfounded.

It might seem that the disability paradox, in the context of a human making a proxy assessment of an animal’s (predicted) QOL, is biased by human values. It may be, but anthropocentrism is not the explanation for the disability paradox. Because the paradox functions when we are making QOL predictions for other humans (not to mention our own individual future selves), it arises from a mistaken notion of the negative impact of an adverse event on any individual’s future QOL.

**Underestimation of Adaptation**

Studies in humans and animals have identified a robust psychological trait shared by many species, and seemingly present in all mammals, that allows them to mentally adjust to wide-ranging changes in their life circumstances. Ample evidence exists that as an individual comes to terms with the conditions of long-term illness, disability, or emotional trauma, psychological changes occur that preserve one's life satisfaction, and individuals can judge their QOL as good even when severe limitations exist on their physical abilities. Argyle (2001) has suggested that it is this trait—adaptation—that may explain the finding that although elderly people are in
poorer health, more likely to be socially isolated, and less well-off financially, they are not, on average, less satisfied with life than young people, and in fact may be more satisfied.

Studies and anecdotal observations suggest that adaptation works similarly in animals as in humans. A study of dogs that had become paralyzed in their hind legs showed that their mental attitudes, as judged by their owners, was as good three months after as before the paralysis in 85 percent of the animals. In a survey of dog and cat owners whose pet had undergone a limb amputation, all respondents (17 of 17) said that after their pet adjusted it was as active and happy as it had been before the amputation. In another study of animals having had amputations performed, 100 percent (74 of 74) pet owners reported that their pets led normal lives after healing from the surgery.

**Scale Recalibration**

With no fixed reference points to keep QOL anchored there is nothing to prevent it from moving in one direction or another relative to the individual to whom it is being applied. For the same reason, if the scale shifts position there is no known way to accurately measure the shift. This introduces another complexity into the problem of the disability paradox and the moving target nature of QOL.

Ubel et al (2005) pointed out that when a person reports that his or her overall QOL is “8 out of 10,” this response carries very little inherent meaning. What one person means by “8 out of 10” (or “very good,” “below normal,” “high,” or any other description or unit of measure) could be different from what another person means by the same rating. To illustrate, consider a vibrant, energetic, and optimistic 28-year old man, and a 78-year old man who has diabetes, arthritis, a heart condition, and failing eyesight, both rating their QOL as “90 out of 100.” Consider too that the young man has expressed in a confidential interview that he would be miserable if he were that same elderly man. In light of this, does a score of 90 mean the same thing for the two men? Many researchers believe that it does not. A concern among QOL and mood researchers is that the subjective scales used in such research are susceptible to scale recalibration. As Ubel et al (2005) explain, the QOL scale has shifted, such that a “90” for the elderly man means something different than a “90” for the young man. When people’s health declines, or when their age or disability progresses, they might start reinterpreting what these response numbers, or what the maximum, mean. There appears to be a shift in the internal standard, which does not reflect a change in the basic structure of QOL for that individual, but results in a changed expectation of QOL more in fitting with the individual’s current situation in life.

Scale recalibration is routinely applied in animal QOL assessment. For example, a typical comment from the owner of an elderly dog is “He’s doing pretty well, considering his age.” The key phrase here is “considering his age.” This qualifying comment is the scale recalibration—it signals that the owner is applying a different standard to this dog than she would to a young dog. In addition, the “pretty well” is like the 90 in the above human example—it has no specific meaning in and of itself. Such a rating does not necessarily mean the same thing for a 17-year old Cocker Spaniel as it would for a 2-year old.
CONCLUSIONS
The unsecured, shifting, slip-sliding nature of QOL—and, specifically, the disability paradox that derives from it—renders any method of QOL assessment based on “I would/wouldn’t want this for myself” highly unreliable. While this type of assessment has a distinct intuitive appeal—it *feels* right—this mindset is ill-suited to generate accurate QOL predictions upon which optimal decision-making can based. Indeed, in animal QOL assessment these methods would be unreliable even if the animal could express in detailed human language what they would or wouldn’t be happy with—e.g., “I could never be happy if I lost my vision”—because we know from the numerous studies cited above that even people make such resolute assertions about themselves, yet those who have actually experienced the adversity often adapt and ultimately rate their QOL as surprisingly high.

The disability paradox must be taken seriously in any attempt to predict QOL for any person or animal. In earlier writings I had proposed an informal test consisting of subjective questions. One of the questions included was:

Imagine that you are a pet animal of the same species as your pet and that you have the best quality of life you can imagine a member of this species having. On a scale of 1 to 10, 1 being extremely unwilling and 10 being extremely willing, how willing would you be to exchange your life for the life your pet is now living?

With our current knowledge of the disability paradox, it is clear that no matter how intuitively appropriate this question might appear, the actual validity of the question is low. The fundamental error in this type of question is best addressed by Kahneman (1999), who points out that predictions of QOL—in, for example, a disability of paraplegia—commonly fail to distinguish appropriately between the state of *being* a paraplegic and the event of *becoming* a paraplegic. An emphasis on the former is shocking to the senses and judged as devastating to one’s life. In stark contrast, recognizing this distinction and emphasizing the latter puts the assessor’s viewpoint closer to the actual experience, and one can more easily visualize a process of *transition*—and an acceptance of and adaptation to the disability. The long-term follow-up QOL studies in people with severe chronic illnesses and disabilities suggest that even a score of “1” to my question above would not prevent a person or animal with the unwanted condition from ultimately experiencing a good or even excellent QOL.
Quality of life Part 3: End-of-life care and difficult decision-making
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In medical care, many of the considerations at the end of life differ considerably from those at other stages of a patient’s life. The determination of when treatment is futile, the doctor’s duties to the patient, the object of the clinician’s allegiance, the emotional impact of the situation, questions regarding who best knows and decides the patient’s best interests, the weighting of interests, whether euthanasia or natural death is the best death, and, of course, the absolute finality of decisions regarding euthanasia.

In veterinary medicine there is widespread agreement among all involved parties—the pet owners, the veterinarian, society, and, presumably, the animal—as to what the primary goal is in caring for animals: maximize quality of life. And a generally strong consensus exists for the notion that the single most important part of this goal is to relieve all relievable suffering. In light of this remarkable uniformity of thought it seems surprising that there could be so much disagreement as to what is the ‘right’ thing to do in the final stages of life. If everyone is working toward the same goal, how can so much conflict develop in getting there?

CHALLENGE #1—KNOWING THE PATIENT’S INTERESTS: WHO KNOWS BEST?
For incompetent or incommunicative patients in human medicine, the best interests standard is an important principle in medical care decision-making. The intent of this standard is that all decisions about the patient be made in accordance of what that person’s best interests are presumed to be. But when the individual cannot express their personal interests it isn’t always clear who is the best person or persons to decide on the patient’s interests. This is why we hear of so many instances of court battles over medical care decisions, and often it is the court who decides on the best interests of the individual of concern. The key questions we face in veterinary care are: (1) how do we determine the animal’s best interests, and (2) who should determine those best interests?

A multitude of studies in human medicine have studied the accuracy of health care professionals (HCPs) in judging patients’ self-assessed preferences for pain, quality of life (QOL), and life-extending care. Results have repeatedly shown that HCPs are very poor at assessing how patients feel. Moreover, in making judgments on treatment in terminal diseases, there is nothing to support the idea that physicians can or do make these judgments in a scientific, objective, value-free manner. In this light, what weight should veterinarians’ assessments of their patients’ interests carry?

CHALLENGE #2—OVERTREATMENT OF ANIMALS WITH A POOR PROGNOSIS: THE ISSUE OF MEDICAL FUTILITY
The concept utilized in discussing the issue of overtreatment in human medicine is termed
**medical futility.** Overtreatment is referred to as *futile treatment.* While virtually no one disputes that there is a time in the progression of many illnesses that treatment is no longer effective—that is, becomes futile—there continues to be vigorous debate on defining medical futility, how futile treatment should be assessed, who has the qualifications and the right to determine the criteria on which the utility or futility of a treatment should be judged. To complicate matters further, it is often difficult if not impossible to know whether certain treatments are futile unless they are tried. At present, medical futility does not have a single, universally recognized and clinically applicable meaning. How low do the odds of responding have to be to label the intervention futile? 1 in 10? 1 in 100? 1 in 1000? 1 in a million?

As disease progresses and efficacy of treatment diminishes, what best represents the “cut-off” to label treatment futile? Some examples in the human literature include language such as: “meaningful survival,” “a benefit to the patient as a whole,” “quality of residual life has deteriorated far below everyone’s expectations,” “a life beneficial to the patient,” and “the point of meaningful quality of life.” In determining medical futility, who decides that the point is reached? The clinician? The pet owner? In human medicine, many writers and the AMA suggest the physician; other writers and many legal decisions suggest the patient/family/caregiver.

**Disagreement on best course of action at the point of medical futility**—The medical care options at the point of medical futility are: (1) proceed with futile treatment, (2) palliative care (so-called comfort care) only, and (3) euthanasia (including, in humans, physician-assisted suicide). Principles of patient autonomy in human medicine dictate that, whenever possible, the preferences of the patient should determine the course of action. Decisions in veterinary medicine are necessarily paternalistic, made by the pet owner and the veterinarian. These two parties may agree or disagree on the proper course of action. Among the potential disagreements, the most common in both human and veterinary medicine is when the parent/pet-owner/caregiver wants futile treatment and the clinician disagrees. This is the “Do everything!” scenario in which the caregivers demand aggressive treatment even when such treatment would cause additional suffering with little or no chance of success. Unlike human medicine, the accepted availability of euthanasia in veterinary medicine adds a common second disagreement dilemma: when the caregiver and doctor agree to discontinue futile treatment, they may disagree as to the best alternative choice: palliative care or euthanasia.

**Why do caregivers overtreat?** A challenge facing the veterinary clinician is determining why a pet owner is choosing a course of action that is not, in the veterinarian’s judgment, in the animal’s best interests. Using research done in human pediatric medicine and clinical veterinary experience, we can list the following as reasons that pet owners elect futile treatment: (1) unrealistic optimism, (2) inability/unwillingness to see the suffering (denial), (3) incorrect judgment about animal’s best interests, (4) confusing treating with caring and seeing cessation of treatment as lack of caring or abandonment, (5) wanting absolute certainty that they “did everything possible,” and (6) inability/unwillingness to “let go.”

**CHALLENGE #3—WHAT SHOULD BE DONE WHEN QUANTITY AND QUALITY OF LIFE ARE IN**
CONFLICT?
It is believed by many that medical care decisions should always strive to maximize QOL. However, this approach is challenged in the many situations in which a decision may permit a greater quantity of life but at a (predicted) lower QOL. For themselves, people will elect to undergo renal dialysis, aggressive multi-modal treatment of malignant cancer, and limb amputation—all often involve accepting a lower QOL in order to live longer. This is a complex decision that forfeits immediate rewards (in the form of current QOL) in exchange for greater long-term rewards (extended life experience). In light of the fact that choosing long-term rewards over short-term is a very personal choice, how and why would we presume that animals would ever elect quantity over quality of life? Is it our right, or even duty, to choose this for them? Perhaps most importantly, what is “the math” when calculating an exchange of quality for quantity?

CHALLENGE #4—HOW EFFECTIVE ARE VETERINARIANS IN RELIEVING DISCOMFORT, DISTRESS, AND SUFFERING IN PATIENTS WITH TERMINAL DISEASES?
There is a broad consensus that the priority for end-of-life care in veterinary medicine is the relief of distress and suffering and that our ability to achieve this goal is the most important element in end-of-life decisions. Studies in human patients have shown that alleviating suffering is a foremost consideration in the quality of the dying process.

One type of suffering—physical pain—receives the most attention in veterinary medicine. Veterinarians in practice frequently hear owners of animals with terminal diseases express the sentiment that “I just don’t want him to be in pain.” However, 2 major studies of the practice of euthanasia in people in the Netherlands – where human euthanasia is accepted – looked at the reasons why patients request euthanasia: both studies found that pain was the sole reason in only 5% of patients. Many other forms of distress and suffering occur in terminal illnesses, including dyspnea, struggling for breath and the sense of suffocation (the reason ‘waterboarding’ is considered torture), sick and toxic feelings (e.g., renal failure), fever, nausea and vomiting, loneliness, anxiety, and fear. In a report in The New England Journal of Medicine, physician Timothy E. Quill wrote: “in the process of dying...suffering can be lessened to some extent, but in no way eliminated or made benign... Although I know we have measures to help control pain and lessen suffering, to think that people do not suffer in the process of dying is an illusion.” Brock (1995), writing of euthanasia for people: “...euthanasia concerns patients whose lives, while they are dying, are filled with severe and unrelievable pain, and for whom euthanasia is the only release from their otherwise prolonged suffering and agony. This argument from mercy has always been the strongest argument for euthanasia in those cases to which it applies. But how often are patients forced to undergo untreatable agony which only euthanasia could relieve? It is crucial to distinguish those patients whose pain could be adequately relieved with modern methods of pain control, though in fact it is not, from those whose pain is relievable only by death.” In light of the fact that alleviation of suffering is sometimes inadequate in human medicine, what is our best approach in making medical decisions involving suffering in animals?

CHALLENGE #5—IS PALLIATIVE AND HOSPICE CARE BENEFICIAL TO ANIMALS WITH TERMINAL
ILLNESS?
Palliative care is defined as medical care which focuses on the enhancement of comfort, the relief of suffering, and support for the best possible QOL for patients facing serious, life-threatening illness, and on the patients’ families. Hospice, on the other hand, is a philosophy and program of care that addresses the physical, emotional, and spiritual needs of the terminally ill person and his/her family. Hospice care emphasizes palliative care and supportive services rather than cure-oriented therapies and interventions.

Elisabeth Kubler-Ross proposed a now well-known 5-stage grief process for those facing impending or recent death of a loved one or companion: Denial, Anger, Bargaining, Depression, and Acceptance. Veterinarian Myrna M. Milani has written that people may get stuck at one stage in the process, such as when “Some clients will deny a patient’s illness right up until the day the animal dies.” She adds that owners may try to “buy time,” which becomes tragic “when the owner rationalizes that any kind of life is acceptable for the animal rather than accepting its terminal condition.” These realities are very important when considering the concept of palliative and hospice care in veterinary medicine. For pet caregivers “stuck” at the first stage of Kubler-Ross’s grief process, is it possible for the hospice option to be a refuge for deniers or “hanger-onners”? How can we assure that hospice care doesn’t lead to animals enduring unnecessary suffering relievable by euthanasia?

CHALLENGE #6—IS THE SUFFERING DURING THE DYING PROCESS DIFFERENT THAN SUFFERING AT OTHER TIMES IN LIFE? AND IS THE DUTY TO RELIEVE END-OF-LIFE SUFFERING ANY LESS BINDING THAN FOR OTHER SUFFERING?
Key questions: Does the veterinarian’s duty to relieve all relievable suffering hold equally strongly at every point in the animal’s life? Is the suffering in terminal disease any different than suffering any other time in life? Is there a lesser obligation to relieve the suffering associated with dying than any other suffering in pet’s life? Is there a specialness or sacredness to death that makes the suffering around the time of death itself special, and that weakens the duty to relieve that suffering? Does the animal have any less desire to be rid of the discomforts in terminal illness than those during any other time in life? Morally, is withholding euthanasia any different than futile treatment or withholding any discomfort-relieving intervention, since all have the potential to inadequately alleviate relievable suffering?

CHALLENGE #7—THE NATURAL DEATH OPTION— IS A NATURAL DEATH BEAUTIFUL?
If an animal is enduring distress or suffering as a result of a pet owner’s choice for futile, palliative, or no treatment, what should the veterinarian do regarding the option for euthanasia: remain silent until the owner raises the issue? raise the issue if the owner does not? stay neutral on its use? recommend it? encourage it? forcefully push for it? Is there such a thing as a beautiful natural death? And how should veterinarians counsel pet owners with regard to the option to allow a natural death: encourage it, discourage it, or stay neutral on the matter? What criteria should be used to answer these questions?

Addressing the issue of natural death, Timothy Quill – family physician and professor at University of Rochester medical school – wrote in an article in The New England Journal of
Society has been shielded from the real truth about “the degree of suffering that people often undergo in the process of dying. Suffering can be lessened to some extent, but in no way eliminated or made benign…”

“Although I know we have measures to help control pain and lessen suffering, to think that people do not suffer in the process of dying is an illusion. Prolonged dying can occasionally be peaceful, but more often the role of the physician is limited to lessening but not eliminating severe suffering.”

“I wonder how many families and physicians secretly help patients over the edge into death in the face of such severe suffering.”

The special significance of death—along with its corollary tenet, the sanctity of life—provides one important basis for the belief that natural death is preferable to euthanasia. Furthermore, this reverent view of life and death underlies the judgment of euthanasia as wrong, even immoral. But we must ask whether these human beliefs outweigh our primary duty to protect animals against the hurts of life and illness, and, as mentioned above, whether this duty is for any reason less strong in the dying animal. The animal experiencing the sickness of disease, whether that disease is transient or terminal, just wants to be rid of the miserable feelings. Is it right to ask an animal to endure relievable suffering for the sake of a human belief system (i.e., a “specialness” or “sacredness” of death)? It is reasonable to suggest that any suffering that we have the power to prevent, but do not, would require some benefit to the animal that would outweigh (or at least offset) the additional suffering. Put another way: If a natural death resulted in any greater degree of suffering than a euthanasia death, what would justify this greater suffering?

CHALLENGE #8—EUTHANASIA: HOW DO WE DETERMINE WHEN “IT’S TIME”?
Is there a right time? If so, how do we know we’ve reached it? Is there a “too soon” and “too late,” and what factors determine each of these? If we use QOL as our determining factor, how do we characterize QOL at the point where it is time for euthanasia? Should veterinarians tell pet owners that “Your pet will tell you when it’s time?”

References available from author on request
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ABSTRACT AND GOALS OF LECTURE
When you've done everything right and your patient is still walking wrong, what do you do? This case-based presentation ties together elements of veterinary acupuncture, veterinary chiropractic, veterinary homotoxicology, current concepts in pain management, and veterinary traditional Chinese herbal medicine to provide a fresh and unique approach to the treatment of common acute and chronic musculoskeletal disorders. You'll go home with tips that you can use on your first day back in practice to enhance patient comfort and performance, and to improve client satisfaction.

INTRODUCTION
In Western medicine, we have an impressive array of NSAIDs and other mediators of pain and inflammation available to us. We have joint support products, new, hot-off-the-press nutritional supplements, and gifted orthopedic and neurology specialists standing waiting in the wings when a surgical or specialty medical approach is warranted for the treatment of musculoskeletal disease. So why do some patients still fail to adequately respond? Perhaps because our medical system works exceptionally well for acute disease, and much less well for chronic disease. It’s a result of our focus. We learn a system of medicine that focuses on problem resolution through symptom resolution. Many clients and practitioners never even look for alternative treatments in this arena unless a patient has end-stage disease, because what we have available within the realms of traditional veterinary medicine and surgery works extremely well to relieve symptoms for the vast majority of our patients. If it doesn’t, we generally refer these patients to a specialist who is often able to arrive at a satisfactory resolution of the patients’ symptoms. Still, some patients fail to respond adequately to even the best we have to offer.
If you have come to this lecture, perhaps you have considered that there is something incomplete with your current approach to effective treatment of musculoskeletal conditions. Hopefully, it will be encouraging to hear that there are a variety of established, medically documented, and very effective ways to evaluate and treat them, in addition to the ones you already know about. These therapeutic modalities recognize that merely suppressing the symptoms of disease without treating the underlying reasons it developed can actually hasten the patient’s decline. And, that it is relatively easy to affect the course of disease early on, if the focus is on treatment of the patients’ underlying condition rather than simply on symptom resolution. Traditional Chinese Veterinary Medicine – our main focus today, with a few additions from other modalities - offers a unique perspective that allows us to address our painful patients from a new medical paradigm: one that offers additional diagnostic and therapeutic options to make them - and keep them - more comfortable and pain-free.
As veterinarians, we may not be administering our best care when we treat musculoskeletal pain and dysfunction in the traditional manner. We are very good at making symptoms of pain
or dysfunction disappear, and we are even better at convincing our clients - and ourselves - that this is all we need to do. Clients never want their pets to appear to be weak or in pain, and they are usually quick to agree that pain relief is not just appropriate treatment – it is complete treatment. I find that the situation has actually worsened now that most veterinarians have an improved understanding of how to recognize pain and paresis in their patients, and have begun treating it earlier and more effectively. The end result? In our understandable zeal to relieve pain and make our clients happy, we too often forget to treat the underlying mechanisms that are responsible for the pain. We must understand clearly - pain and paresis are sentinels of disease, not the disease itself. Resolution of pain and paresis should mark the beginning of our intervention for our patients, not the end. Otherwise, our patients will suffer for our short-sightedness.

PAIN
What is pain? The International Association for the Study of Pain defines it as “an unpleasant sensory and emotional experience, associated with actual or potential tissue damage, or expressed in terms of such damage”. Pain can be a single unpleasant event which resolves over a relatively short time frame, or may be chronic. Three months is the most common point of division between acute and chronic pain, according to the IASP. In our animal patients, the initial presentation for pain sometimes represents a diagnostic and therapeutic challenge. Our patients generally present with first-onset pain that is far more severe and chronic than that which our MD counterparts see and treat. And, by virtue of our veterinary training, we often have a poor ability to detect, diagnose, and treat pain effectively.

From a Western perspective, pain falls into four clear categorizations: nociceptive pain, neuropathic pain, sympathetically mediated pain, and psychogenic pain. In chronic pain disorders, these categories may often overlap, as often occurs with amputation sites or chronic hoof disorders. We are all familiar with the Western concepts of receptor, afferent and efferent nerves, spinal cord, brain, neurotransmitters, muscles, ligaments, tendons, and so on. When we apply such concepts to the treatment of pain, our best efforts often fail. What if we evaluated our patients from a different medical perspective? Could we treat pain more effectively?

GENERAL TCM PAIN PERSPECTIVES
When we think of the receptors, efferent and afferent nerves, spinal cord, brain, neurotransmitters, muscles, ligaments, and tendons from a TCM perspective, our approach to diagnostics and therapeutics changes. Our approach now becomes a whole-body approach. Any TCM Organ, any Fundamental Substances, any alteration in Qi or Blood flow, any external pathogenic factor, any underlying constitutional disorder can contribute to the development of pain, with or without an inciting injury or trauma episode. The Spleen, Kidney, Stomach, Liver, Lung, Gall Bladder, San Jiao (and probably all TCM Organs) can factor, either directly or indirectly, into the development and manifestation of pain states. Normal Zang Fu (Organ) function is needed to help keep external pathogenic influences and internal pathologic changes from causing obstruction and ultimately restricting the normal flow of nutritive Qi and Blood. Proper Blood flow, a function of Heart and Liver, is important to maintain proper levels of
nourishment which will insure neuron and receptor function. Nerve impulse is a manifestation of proper Qi and Blood flow through the meridians, collaterals and divergent channels. The extraordinary organs (spinal column and brain) are controlled by Kidney Yin, Yang and Essence. The external influences of wind, cold, and damp are the primary environmental factors affecting the pain state of our patients. Internally, obstructive material in the body, such as damp, phlegm and stagnant Blood contribute greatly to pain. Pain manifesting in a specific area of the body often indicates a regional disharmony. Look to regional Organs or regional meridians for clues! For example, pain in the flank and rib cage area may represent Liver and Gall Bladder disharmony, and pain in the lower back signals Kidney disharmony. Pain in the solar plexus region may indicate a Spleen and Stomach disharmony, and pain in the chest may indicate a disharmony in the Heart or Lungs.

The type of pain experienced by a patient generally correlates well with specific types of disharmony. While it is not as easy for us to evaluate pain in our patients as it is for an M.D., we can still appreciate some of the following qualities and their significance:

- Pain diminished by heat signifies a Cold condition, while pain worsened by heat implies Heat.
- Pain relieved by pressure indicates a Deficiency condition, while pain worsened by the same indicates Excess.
- Pain which diminishes after eating implies a deficiency, pain worse after eating implies Excess.
- Pain worsened by humid weather indicates Dampness.
- Pain accompanied by bloating indicates stagnant Qi.
- Pain which is fixed, sharp, or stabbing indicates Blood stasis.
- Pain accompanied by a sense of heaviness indicates Dampness.
- Pain which varies in location or wanders indicates Wind or possibly stagnant Qi.
- Pain which is low grade and accompanied by fatigue indicates Deficient Qi or Dampness.

Initial TCM treatment goals for pain focus, of necessity, on the presenting symptoms with the goal of relieving pain. Crucially important follow-up treatment is directed towards resolution of any underlying causative factors or deficiency states, any identified risk factors, and any psychological factors. This follow-up represents our best approach to the treatment of chronic pain states, as well as the best way to permanently resolve acute pain states. Even in cases of heritable or congenital conditions with a pain basis, appropriate modulation of Fundamental Substances and Zang-Fu Organ function can help keep our patients more comfortable. Our TCM goals are always to fully evaluate our patients and approach diagnosis from the standpoint of syndrome differentiation as opposed to a symptom-based approach, and to focus on correcting underlying imbalances, rather than on suppressing only the presenting symptoms. Once we’ve done this, the decision can be made to proceed with an acupuncture based approach, an herbal approach, or a combination approach.

**What can acupuncture do?**

Acupuncture initially came to the attention of the Western medical and veterinary community for its well-known efficacy in the treatment of pain and its efficacy in providing analgesia during surgical procedures. Early studies that concentrated on analgesia first proposed that acupuncture either blocked nociception or provided neuromodulating input into the CNS that activated multiple analgesia systems in the spinal cord and brain. It was postulated that this input stimulated the endogenous pain suppression system to release neurotransmitters, such as endogenous opioids. Other investigations of the role of acupuncture in inhibiting pain have
resulted in theories as diverse as the neural non-opiate theory, the humoral theory, autonomic theory, local mechanisms theory, and bioelectric theory. Currently, pain relief is believed to be achieved both locally at the site of needling and also via the involvement of higher centers. Earlier gate theories of competitive inhibition of pain signals entering the spinal cord via peripheral nerves have largely been discarded. When an acupuncture needle is inserted at an acupuncture point, any associated muscle spasms are usually relieved, resulting in immediate pain relief. In addition, mast cells concentrated at the acupuncture point release serotonin, a neurotransmitter which reduces the activity of nociceptors. Histamine and other vasoactive compounds are also released by mast cells, serving to improve local tissue perfusion and help inhibit the reformation of muscle spasms. While local effects are seen immediately, cerebral effects of acupuncture point stimulation are more delayed, occurring in perhaps 20 to 30 minutes. Needling or otherwise stimulating a point activates small diameter nerves which travel to the dorsal horn of the spinal cord. From there, they cross to the opposite side of the cord, enter the brainstem, and eventually innervate the hypothalamus and pituitary. These higher centers respond by releasing neurotransmitters including endorphins and serotonin which reduce the perception of pain. In addition, further inputs are dampened in a process known as descending inhibition. As exemplar TCM practitioner Steve Marsden has noted, the most accurate guide ever developed as to where these small diameter fibers can be easily accessed remains the chart of acupuncture points developed approximately 2000 years ago by Chinese medicine. About seventy percent of acupuncture points are also trigger points, painful areas of increased tissue turgor or even spasm within the myofascial planes. Trigger points are highly responsive to dry needling. Insertion of any needle including acupuncture needles serves to immediately relax trigger point spasm, thereby relieving pain. It has been demonstrated that many but not all of the effects of injection of local anesthetics and even corticosteroids and glycosaminoglycans into chronically painful areas may be duplicated by the insertion of a needle containing no drug at all. Injection of saline into focally painful areas has been shown to result in improvements that exceed those obtained with local anesthetics. These network connections provide not only analgesia but an opportunity for the damaged tissue to heal itself, explaining why acupuncture is regularly curative in disorders that would ordinarily be viewed as requiring more definitive therapy. Numerous studies have demonstrated a favorable effect on hemodynamics and innervation to damaged internal tissues following stimulation of external acupuncture points.

**What can TCVM herbal therapy do?**

TCM herbal therapy offers a unique opportunity to provide safe, effective relief of pain and inflammation, at the exact site of the problem, without the side effects common to any categories of pain-modulating Western drugs. An important consideration for the use of herbal therapy presents every time we treat a deficient patient. When we treat with acupuncture only, we are imparting little if any energetics to the system; we are simply re-arranging the energetics that exist to benefit the patient. In doing so, we may inadvertently weaken another body system that has adapted to the existing overall deficiency state. A simple analogy might be how we choose to approach the situation of leaving work to discover that our car battery is dead. Do we search the parking lot for the next weakest battery to charge our own car’s
battery, hoping that this will get us safely home? Or do we look for a vehicle with a good strong battery that will be capable of sustaining our vehicle’s battery? Acupuncture alone, in an extremely debilitated patient, is capable of causing a brief energetic surge followed by multiple system failure. But the addition of herbal therapy to the treatment protocol in the same patient allows us to add the energetics of the herbal formula, and this can actively treat the deficient state. Western medications also add their energetic to a weakened system, and this is precisely why I often use them as part of an overall treatment approach.

Appropriate herbal therapy includes formulas with actions of relieving Blood stagnation and stasis; reducing the impact of external environmental factors such as infective agents, Cold, Damp, and Wind; relieving pain; and providing immune support during the healing process. In daily practice, I work from a fully stocked traditional medical pharmacy typical of any Chicago-area 4 year-AAHA facility, as well as from an herbal pharmacy composed of approximately 20 single herbs and 90 formulas. One of the advantages of becoming reasonably well versed in TCM formulas is realizing that many of the common formulas used in daily practice can be broken down into combinations of simpler formulas. While it is undeniably handy to have exactly the right formula on hand at all times, knowing how combinations work can allow the practitioner to begin using TCM formulas with a remarkably small number of formulas, plus a few choice single herbs. It is possible to cover most medical situations with a smaller number of single herbs and formulas, but I find that at least 60% of my stock is in almost daily use.

**HOMOTOXICOLOGY PERSPECTIVES**

Homotoxicology is a simple, easy-to-use, client-friendly therapy that is especially applicable to many of the acute and chronic musculoskeletal conditions we’ll consider. Homotoxicology represents a bridge between classic homeopathy and conventional medicine. Like classic homeopathy, it relies upon the use of serial dilutions of medication, in order to stimulate the body’s innate defenses — much like how vaccines work. But unlike classic homeopathy, it utilizes multiple remedies in multiple dilutions, each one selected for activity at a specific body site and tissue. And, it typically achieves its effect far faster than is possible with classic homeopathic treatment. Homeopathic principles were developed in an era that pre-dated easy measurement of the biological data of a disease. The diagnostic tools we are now accustomed to, such as blood tests, radiology, and MRIs, had not yet been developed. Homotoxicology, by contrast, developed in the 1950’s when at least some of these diagnostic tools were already routinely relied upon by physicians. Hence, practitioners of homotoxicology usually pursue an indication-based approach, much as we do in Western medicine.

Homotoxicology was developed and formalized as a science by Dr. Reckeweg, a physician and classic homeopath. Dr. Reckeweg postulated that the cause of many diseases was a loss of the normal regulatory mechanisms of the body. Further, he postulated that this regulatory loss was caused by what he called homotoxins, both those introduced from the exterior, or exogenous toxins (much like the TCM External Pathogenic Factors) and those which originate in the body itself, or endogenous toxins. Whatever their source, toxins react with cells and tissues and cause a morphologic alteration in the tissues. Most importantly, toxins interrupt the delicate communication system between the nervous, hormonal, and humoral systems which occurs in the matrix, the substance between cells. Progressive matrix disruption results in increasingly
severe disease, which he postulated to occur in six phases. According to homotoxicologic principles, early on in the course of disease, the intracellular mechanisms are not disturbed. The body’s defense systems are intact and can rid itself of toxins via various paths. In these early phases of disease, which he named the excretion and inflammation phases, the body rids itself of toxins by first, elevated rates of normal excretion mechanisms and second, by an exudative inflammation process which enables accelerated excretion of toxins from the body. Closer to the midpoint of serious disease possibilities, toxins begin to become deposited into the matrix. As this occurs, the matrix is increasingly altered and becomes less capable of excretion. In these middle two phases of disease, which Reckeweg termed the deposition and impregnation phases, the body’s capacity to excrete toxins is progressively overwhelmed. First, the toxins become deposited into the matrix and later are incorporated into both the matrix and the body’s connective tissues, causing overt damage to organ cell groups. In the two final stages of disease, referred to as the degeneration and dedifferentiation phases of disease, cell systems are progressively destroyed. First, destruction of larger cell groups of an organ occurs. Second, undifferentiated, non-specialized cells forms are now able to proliferate. Neoplasms represent the end point of this final phase of dedifferentiation. Homotoxicologic medications are utilized to restore regulation in the body. Dr. Reckeweg postulated that just as the body undergoes disease progression, or progressive vicariation, if properly treated it can undergo regressive vicariation and return to a closer semblance of good health with intact functioning regulatory mechanisms. Various subsets of homotoxicologic medications have been developed to selectively address the differing phases of disease in specific organ systems. Some are specific only for early stages of disease, while others are specific for phases up to the dedifferentiation phase. An important concept shared by homeopathy and homotoxicology is that as a patient is successfully treated and moves backwards through disease phases, old and early symptoms may temporarily reappear. This seems disconcerting to most allopathic veterinarians, but it is really quite similar to the way hepatic disease can result in initially elevated and later depressed liver enzymes as the liver loses progressively more functional capacity. If the liver is able to regenerate, liver enzymes can temporarily fluctuate remarkably before finally “settling in” to near-normal values. Musculoskeletal conditions, as recognized by homotoxicology, encompass a variety of disorders marked by inflammation, degeneration, and /or metabolic derangement of the connective tissue structures of the body, especially the joints, muscles, bursae, tendons, and fibrous tissues. End results are pain, stiffness, and limitation of movement. By the time most patients are presented, they are in an advanced phase of dysfunction. Thus, treatment must be directed not just towards resolution of the immediate presenting symptoms, but towards achieving regressive vicariation in order to maintain initial improvement.

CHIROPRACTIC PERSPECTIVES
In chiropractic medicine, we recognize that the body has inherent wisdom which tends towards self-healing whenever possible; and that “disease” results from impairment in the normal relationship between structure and function. The role of the chiropractic veterinarian is to enhance the self-healing process by restoring normal joint motion to any impaired joint, insofar
as this is possible.

Chiropractic relates disease to dysfunction in any joint which causes an abnormality in range of motion of that joint. The effects of joint dysfunction can be far-reaching; all one needs to document this is to recall what they learned in any neurology class about the location of nerves as they exit the spinal column. Thus, in chiropractic we emphasize the role of the subluxation complex in disease. The subluxation complex is a theoretical model of motion segment dysfunction that incorporates the complex interaction of pathologic changes in nerve, muscle, ligament, vascular, and connective tissues.

Chiropractors usually do not view subluxation in our traditional veterinary-taught medical sense of “bone out of place”. Subtle changes such as vertebral malalignment, a reduced or excessive range of motion, and dysfunction with or without pain are all viewed as subluxations. Subluxations are typically corrected by an adjustment using controlled force, leverage, direction, amplitude, and velocity. In the U.S., the short lever, high velocity, low amplitude thrust technique is most commonly used because it is precise and extremely effective. Many of us also use mobilization techniques such as massage, electrical or magnetic therapy, and sound therapy.

So what exactly does correcting a subluxation do? It restores normal joint motion, which in turn normalizes physiologic function. Equine chiropractor and veterinarian Dr. Judith Shoemaker’s succinct assessment of what an adjustment is and what it does is: “.Manipulation of the spine and extremities to effect optimum function and balance of all structures, in other words, straightening out the hardware so the software can run.”

Six phases of subluxation pathology exist: Misalignment, Neuropathy, Kinesiopathy, Dysfunction, Symptoms, and finally, if allowed to progress without appropriate chiropractic intervention, Degeneration. Let’s look at each of these areas briefly:

1) Misalignment
Cause: Generally trauma.
Effects: Muscle splinting to protect area; altered proprioceptive information to and from motor unit; tension in meningeal attachments; and disc wedging/tears/edema/herniation.
End results: Pain/paresis/paralysis, eventual loss of disc integrity if the spine is involved.

2) Neuropathy
Cause: NOT “pinching of nerves”, as discussed; effects are more indirect.
Effects: Swelling/crowding of intervertebral foramen; vascular structures may sustain damage; meningeal torque or stretch; altered CSF flow; and disturbed axoplasmic cellular flow.
End results: Either facilitation (early damage) or inhibition (sustained damage) of neural elements.

With facilitation: stimulation of the end-organ may cause muscle hypertonicity, pain, paresthesia, vasomotor activity (local heat), and glandular activity (local sweating).
With inhibition: loss of neural supply to end-organ may cause muscle atrophy, sensory anesthesia, abnormal proprioceptive input, decreased vasomotor activity (cold spots), and glandular dysfunction such as anhydrosis.

3) Kinesiopathy
Cause: Usually hypomobility of the motor unit, but hypermobility may occasionally result.
Effects: Soft tissue adhesions and scarring; ligamentous and muscular contraction; changes in integrity of joint surfaces; reduced disc perfusion with resultant disc degeneration; reduced CSF
“respiration”; forced hypermobility of adjacent motor units; reduced overall ROM of spinal column; and eventual muscle atrophy.
End results: Hypermobile segments are more subject to stress and injury.
4) Dysfunction
Cause: Altered joint biomechanics.
Effects: Altered neurologic input to end-organs, resulting in eventual local and end-organ damage.
End results: The patient is generally exhibiting signs of pain and/or reduced performance, and is injury-prone at this point.
5) Symptoms
Cause: You have failed to recognize the early and often subtle symptoms of chiropractic need!
Effects: From here on out, you are playing catch-up. Fortunately, our patients are generally tough and respond surprisingly well to catch-up treatment. Even at this stage, most patients will experience significant improvements in well-being. However, treatment will likely be ongoing if improvement is to be maintained.
6) Degeneration
Cause: Distressingly, few of us are taught to recognize the signs of chiropractic subluxations or even to know how to examine a patient for their presence. Our medical knowledge of a subluxation is based upon an extremely crude impression of “bone out of place”, a notion that was rejected by chiropractors many years ago.
Effects: Commonly, we treat our painful, stiff, and poorly performing patients with good intentions and a wide range of muscle relaxants, NSAIDs, joint support products, linaments, wraps, prescribed rest, and a host of products - our choice largely supported by our personal belief in a particular system of allopathic or holistic medicine.
End result: Many of our patients improve somewhat with our treatment. However, many never return to top performance, and many remain in pain or seem reasonably comfortable but gradually diminish in strength and vitality. We comfort ourselves by attributing this to “old age”, “loss of drive”, or some other excuse.
As with any form of therapy, there can be some basic contraindications to the use of veterinary chiropractic. This is especially the case when it is used to resolve the following presenting problems: breed specific skeletal problems (e.g., bulldog breeds); metabolic bone disease; malignant bone disease; and immune mediated bone and joint disease. Thorough examination, palpation, appropriate laboratory evaluation, and radiography can easily rule out most of these conditions.

CASE EXAMPLES
How I treat acute musculoskeletal injury
Patient #1: The balcony-diving Shar-Pei. This is an excellent example of how a patient was presented to our hospital in my absence, how our attending DVM with excellent conventional medical skills treated the patient, and how I followed up. It’s also a typical illustration of how often Western medicine fails our patients in cases of musculoskeletal trauma, even when everything reasonable is done correctly. In this patient, I utilized a combination of chiropractic, acupuncture, and herbal therapy to facilitate the patient’s recovery. Today, I would have utilized a homotoxicologic remedy, Traumeel, instead of the customized herbal formula I
prescribed, and I would have utilized aquapuncture – injection of the Traumeel into active acupuncture points – as a STAT treatment.

I am very comfortable utilizing traditional NSAIDs for relief of pain and inflammation. I often utilize Tramadol and/or Neurontin as add-on therapy when needed, for their multiple-level effects. When using Tramadol, make sure not to use the trade name Ultracet formulation, as it can be toxic to cats (Ultracet is a combination formulation containing 375 mg of acetaminophen in addition to the Tramadol).

Patient #2: In the case of focal severe musculoskeletal injuries, I often use the “circle the dragon” acupuncture technique, where needles are placed circumferentially to the lesion. Or, if fractures are involved, once they are appropriately treated by an orthopedic surgeon, I often utilize electroacupuncture therapy to speed healing of the fracture. This also works well for chronic injuries that must heal by second intention.

Appropriate herbal therapy includes formulas with actions of relieving Blood stagnation and stasis; reducing the impact of external environmental factors such as infective agents, Cold, Damp, and Wind; relieving pain; and providing immune support during the healing process. I utilize specialty formulas such as Cordalin, Traumanex, Herbal Analgesic, and Herbal ABX (all exemplar formulas developed by Lotus-Evergreen), as well as the classic TCM formula Shen Tong Zhu Yu Tang.

How I treat acute intervertebral disc disease

I see far too many referral patients with horrific pain and paresis, unresponsive to multiple therapies, who would have had their spinal tumors, spinal fractures, congenital deformities, or other patently obvious reasons besides IVD for their pain or paresis diagnosed much earlier – had they been radiographed by the referring veterinarian who assumed that they had IVD. If referral rads are more than 1 month old, we will generally request that new rads be taken or we will take them ourselves. Again – whether we choose to treat conventionally or alternatively, we cannot treat appropriately without knowing the facts of the patient’s presentation. Once rads are evaluated, we can decide whether a conventional appropriate might be best – and in some cases, it will be. Many acute disc presentations are surgical cases, and it is as foolhardy and inviting of malpractice to attempt herbal or acupuncture therapy as it would be to sling them some prednisolone and advise cage rest for two weeks.

If the patient does not fall into one of the conventionally documented categories where surgical intervention is best, then I will often follow-up with a multifaceted approach once the client and I discuss and agree upon an initial and follow-up treatment plan. Because we already generally have a painful and apprehensive patient who likely suspects that veterinarians are chiefly dispensers of more pain, I identify areas of muscle spasm and treat them with infrasonic therapy prior to placing any acupuncture needles or performing any manipulative therapy.

Appropriate TCM herbal therapy for acute disease provides a useful adjunct to the use of traditional pain relievers and NSAIDs. While traditional therapeutics often provides effective pain relief, especially if we step up their use to include transdermal delivery options, they are relatively non-specific to the area they treat. TCM herbal therapy is unique in its ability to focus delivery of all oral preparations to specific body regions through the use of Guiding herbs in appropriate formulas. Examples include

Ge Gen Tang for cervical disease fitting its clinical applications;
Juan Bi Tang for thoracic disease fitting its clinical applications;
Xiao Huo Luo Dan for lumbar disease fitting its clinical applications;
Du Huo Ji Sheng Tang for lower lumbar disease that also involves the hind legs, fitting its clinical applications.
I also use herbal analgesics such as Corydalin and Herbal Analgesic.
Other therapeutic options such as use of hot and cold packs, massage, hydrotherapy, proprioceptive exercises, and temporary use of ambulation-assistance devices may be indicated. We also advise clients to optimize the home environment for disc patients, by elevating food and water, walking on soft surfaces; utilizing ramps with a non-skid surface at home entry and exit points and with vehicle; providing a padded resting and sleeping surface; and avoiding wide temperature and humidity swings when possible.

**How I treat chronic intervertebral disc disease**
The chronic disc disease patients I am referred often have had multiple surgical procedures and are at a stage of disease where the attending orthopedic surgeon has recommended no further surgical intervention. These patients may be poor surgical candidates, poor anesthetic candidates, or have had poor results with prior surgeries. They very often have substantial epaxial muscle-wasting, sensory anesthesia, abnormal proprioceptive input, decreased vasomotor activity, soft tissue adhesions and scarring, ligamentous and muscular contraction, substantial disc degeneration, and reduced overall ROM of their spinal column.
Appropriate joint support is necessary for any patient with acute or chronic spinal disease. This concept sometimes seems odd to the primary care veterinarians who refer to me, but in treating disc patients I consider two things: first, that each motor unit of the spine represents a complex series of joints, and two, that disc patients have always developed an elaborate series of secondary postural compensations that markedly affect the normal pattern of weight distribution across affected joints.
Acupuncture and herbal care for disc patients is very often seasonally influenced, with increased treatment focus necessary during periods of cold, damp weather. Why? From a TCM standpoint, chronic disc disease often represents a static, deficient (occasionally excess) energetic condition that is strongly influenced by cold, damp, and sometimes wind. So, while it is necessary to treat the underlying health challenges that influenced the initial development of disc disease, it is equally important to address the seasonal influences that may impact existing disease. To clients, these seasonal influences are often more obvious (and are a more frequent reason for presentation) than the disease itself, because they generally result in an acute exacerbation of existing disease that can be very painful and debilitating. Dry needle acupuncture, electroacupuncture, and moxibustion are all interventions I commonly utilize.

**How I treat acute ACL injuries**
In general, I refer these patients for immediate surgical intervention. Over a dozen years of exploring alternative therapeutic options, combined with extensive evaluation of joint surfaces at necropsy in over a thousand patients has convinced me that we serve these patients best by operating them STAT. Prolotherapy, acupuncture, glandular therapy, nutritional therapy, and herbal therapy offer quick symptom relief. However, long term, non-surgical patients with significant ACL injuries generally present with horrendous DJD changes that require salvage therapeutic techniques, at best. During the peri-operative period, judicious use of herbal therapeutics such as Yunnan Paiyao can reduce bleeding and bruising, speed healing, and reduce scarring. I frequently utilize herbal analgesics such as Corydalin and Herbal Analgesic.
Post-operatively, hydrotherapy is extremely useful in quickly restoring mobility and muscle mass; nearly all of my working and agility dog clients choose this option. Additionally, passive therapeutic options such as hot and cold packs, gentle stretching exercises, and massage can be used to facilitate the healing process. Infrasonic therapy and ongoing use of Yunnan Paiyao markedly reduces bruising and swelling of operated tissues and speeds healing. While patients who experience such additional therapeutic measures have been documented to heal faster and return to normal activities sooner, their final impact is best assessed long after the post-operative period. Joints treated in this manner, with a combination of appropriate surgical intervention and appropriate complementary therapy, heal with virtually no restrictions in motion, no swelling, little scarring, and without crepitus.

**How I treat chronic, non-operated ACL injuries**

I always clarify for clients that what we are really treating in chronic, non-operated ACL injuries is severe degenerative osteoarthritic changes, and I treat them as such.

**How I treat chronic osteoarthritis**

Many clients have accustomed themselves to a pet that is slow-moving, can’t jump up on furniture, needs help getting into a vehicle, has to be coaxed into going outside to eliminate, and doesn’t enjoy long walks as it once did — and they sometimes see little reason for regularly presenting such pets for intervention. And why not? In the past, their concerns have often been brushed off and attributed to “old age”. Fortunately, more and more veterinarians are recognizing that old age is not a disease, and that geriatric pets can enjoy the same good, vibrant, health that we expect to see in younger pets – with appropriate intervention.

Management of body weight is critical in OA patients. Many studies have documented the profound impact of obesity on joint surfaces, on initial development and progression of OA, in ability to perform activities associated with daily living, and on medial lifespan. We are rigorous in focusing on weight management, to the point of having adopted one protocol utilized by the rehab center at U-Tenn: I advise clients verbally and in writing that I will not be able to effect a positive resolution of their pet’s OA unless they commit to weight loss that we can document. Like chronic disc disease patients, many chronically impacted OA patients present with substantial muscle wasting, compensatory areas of hypertrophy in body regions they are better able to utilize, associated shifts in posture, and secondary spinal degenerative changes. They are also seasonally influenced by weather factors, acutely impacted by significant shifts in barometric pressure, in temperature, and in humidity. This, they bare ideal candidates for chiropractic, acupuncture, and TCM herbal intervention.

From a TCM and a homotoxicologic viewpoint, there is little difference between how we approach appropriate intervention in a chronic OA patient, a chronic disc disease patient, and a chronic ACL injury patient. TCM practitioners would agree that all commonly represent variations on a condition known as Bi syndrome. Simply put, Bi syndrome represents reduced obstructed circulation, both of Qi and of Blood, through the Channels. It can result from an underlying Deficiency condition (either congenital or acquired), or secondarily after invasion by external factors such as Cold, Wind, Damp, or Heat, singly or in combination. Whatever the etiology, the end result is pain and stiffness in muscles, tendons, bones, joints, and in the skin and vasculature. End stage Bi syndrome results in deformation and inflammation – ossifications in bone, degenerative changes in joint surfaces, swellings in joints, chronic inflammatory-appearing skin lesions, and vascular inflammatory diseases. Many types of Bi
syndrome are described. While it is beyond the scope of this lecture to discuss them in detail, it is worth noting that appropriate herbal and acupuncture therapy differentiates between these forms because treatment success depends on proper recognition the type of Bi syndrome that exist. Patients that are characterized by hot, swollen joints – the patients that have severe DJD yet paradoxically prefer to stay out in the cold or to lay on hard, cool surfaces – are treated differently than patients who have severe pain and DJD that is improved by heat and by reduction in humidity. Patients with OA who have pain that varies in location are treated differently than patients who have fixed, definitely localizable pain. Western medicine typically treats all of these patients the same way – and we wonder why some patients improve and others don’t!

Useful herbal remedies, given the correct TCM presentation, include:
Bi Formula: a Zhao formula, one of the few specialty formulas I utilize; for Bi syndrome in general
Corydalin: a Lotus formula with exceptional Blood-moving, pain-relieving properties
Du Huo Ji Sheng Tang: for Blood and Qi deficiency, especially with a KI component
Osteo-8: a Lotus formula specific for geriatric, weak, deficient patients, suitable for long-term use
Si Miao San: for Damp-Heat accumulation
Shen Tong Zhu Yu Tang: for Channel stasis
Yi Yi Ren Tang: for Blood moving, and to resist Wind invasion
Xiao Huo Luo Dan: for Cold, Damp, Phlegm accumulation

Appropriate acupuncture treatment for Bi syndrome includes consideration of these points:
GB20: Eliminate Wind
GB34: Influential point for tendons; strengthens the hind legs
GB39: Influential point for Marrow; strengthens the bones
LIV3: LIV Source point, invigorates Qi to reduce stagnation
BL11: Influential point for Bone; strengthens bones
BL17: Influential point for Blood; invigorates Blood
BL20,21: ST & SP Shu points; strengthens SP to eliminate Damp
BL23: KI Shu point; eliminates cold by warming Yang, tonifies the KI
BL40: Strengthens lower back and hind legs
BL60: Very effective pain point; disperses wind, relaxes spasmed muscles
KI3: KI Source point; immediately opposite BL60; strengthens lower back
SP10: Sea of Blood; invigorates Blood
Bai Hui: Warms Yang, eliminates cold, strengthens the back
CV4: Warms Yang, eliminates cold
SP6, 9: Eliminates Damp
ST36: Master Point for stomach & abdomen, used here to strengthen SP to resolve Damp
LI4, 11: Both clear Wind-Heat and help resolve swelling and pain

Location-specific points to consider for any Bi syndrome condition are:
Hock: BL60, 62; KI3,6; LIV 4: SP 4
Stifle: ST34,35,36; GB33,34; SP9,10; BL39,40
Hip: GB29,30; BL54
Sacrum/pelvic area: BL35,36,54; Hwato sacral foramen points; local Ah-shi points
Carpus: LI4,6; SI3; TH5; LU7
Elbow: LI10,11; LU5; TH10; SI8; HT3
Shoulder: LI14,15,16; ST9; TH 14, 15
Neck: GB 20,21; BL10; SI16; local Ah-shi points

**Condition-specific (Caveat: NOT patient-specific!) points to consider are:**
ACL: Electroacupuncture across BL 21 and BL54; same across “eye of the knee” points; dry needle Bai Hui, GB10, BI11, ST 34,35,36, LIV3,8; GB 32,33,34
IVD: Local Ah-shi points, BL10,11, 23,40,60; GB10,34,44; ST36; Bai-hui; ipsilateral electroacupuncture cranial and caudal to vertebrae with evidence of radiographic change, palpable abnormality or abnormal ROM (chiropractic evaluation)
Wind Bi: Local points; Ah-shi point;, GB20, LIV 3, BL17, SP10
Heat Bi: LI4,11; ST44; GV14
Bone Bi w/ KI Yang Deficiency: BL11,23,40,60; GB 34,39; Bai Hui ;+ herbal therapy
Bone Bi w/ KI Yin Deficiency: BL23; KI 1,3,6,10; SP6; + herbal therapy
Bone Bi w/overall KI Deficiency: KI 3,10; BL23,26; LIV3; SP6,9; LI10,11; ST36; + herbal therapy
Cold/Painful Bi: Local and Ah-shi points; BL23, CV4, Bai Hui; moxibustion over any of these points
Damp/Fixed Bi: Local and Ah-shi points; SP6,9; ST36; BL20,21

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MOVING INTEGRATIVE MEDICINE INTO YOUR PRACTICE
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ABSTRACT AND GOALS OF LECTURE
Incorporating profitable complementary and alternative veterinary medicine (CAVM) into your practice requires a careful assessment of the economics of the therapies that interest you and your clientele, as well as a way to seamlessly integrate CAVM into your existing hospital schedule. This seminar will address the pitfalls to avoid, the challenges to enjoy, and the opportunities to succeed in developing a specialty within CAVM, as an adjunct to the therapies you already use in day-to-day practice. You will leave with ideas that you can immediately incorporate into practice.

INTRODUCTION
Successful complimentary and alternative medicine (CAVM) involves a practice-unique alignment of the energies and talents of your Doctors and support staff - plus the use of profit indices standard to the veterinary industry. But how do you determine which CAVM applications will be successful and profitable for your practice? How do you resurrect CAVM applications that perhaps interested you but never really turned more than a marginal profit? And how do you make the special talents and skills you’ve developed thorough your advanced training in CAVM pay you back with both enhanced personal satisfaction, and with the tiered income stream you deserve?

Our veterinary world has changed dramatically since many of us entered practice, and those changes have accelerated in the past in 5-10 years. Clients are more knowledgeable and educated about CAVM than ever before, and those who are not educated have access to an increasing sophisticated, web-based information network. Clients often know more about CAVM than do their veterinarians. It’s been estimated that over 80% of your clients know about some aspect of CAVM, and over 55% have utilized some form of complementary or alternative therapy for themselves or another family member.

Owner perspectives on what it means to own a pet and how to care properly for a pet have shifted remarkably, as pets have moved from the status of a household animal to that of another family member. On our AAHA client intake forms, when asked to choose between the statements, “My pet is just a pet” and “My pet is a member of my family” over 99% of our clients choose the latter statement. In the 80’s, it was a minority response. It’s been estimated that over 70% of pet owners remember their pet’s birthday more often than they remember the birthdays of other family members, and over 85% of pet owners who celebrate pet birthdays shop for a gift and/or a special meal for the pet’s birthday and sing birthday songs. And it’s not just birthdays that are remembered - a huge majority of pet owners purchase a gift to present to their pet for Hanukkah, Diwali, Kwanzaa, or Christmas, and a solid majority have shopped for and purchased a holiday collar or costume for their pet at least twice.

When it comes to pets and significant others, a majority of dog owners would choose to sleep with their pet rather than a spouse if forced to choose, and more pet owners overall carry pictures of a pet in their wallet or on their phone than they do of their significant other or their children. According to Pet Age magazine, pet orthopedic beds that often approach or exceed the cost of a child’s mattress are now purchased by a majority of the owners of senior dogs whose point of purchase is a retail outlet. Pet nannies and chefs, playgroups, elaborate boarding and grooming facilities with all of the comforts
of home, wardrobes of pet clothing, “gourmet” pet bakeries that provide food items and treats and offer pet birthday parties, and every other consumer option open to pet owners is being readily explored by our clients. A recent Pet Age article estimated that by the time that a pet store-purchased puppy leaves the initial point-of-purchase store with the new pet and “necessary” food and supplies, the owner has spent close to two thousand dollars - and this was judged to be a low figure indicating an unmotivated sales force!

FEAR-BASED THINKING WILL LIMIT YOU

Meanwhile, there is panic at veterinary meetings nationwide. We are told in lecture after lecture that profit margins at practices nationwide have been declining since 9/11 and are flat-lining due to the recession. We’re told that we need to be looking at more ways to streamline our staff, tighten our belts, lighten our inventories, work our Doctors and staff harder and pay them less, make do and do without, and do what we can to make our practices profitable so that we can sell them someday, if the real estate market ever turns around. And that’s fear-based thinking. Fear-based thinking will limit you in practice, 100% of the time. Your happiness and job satisfaction will be limited, your professional success will be limited, and your income will certainly be limited, if you subscribe to the theory of hunkering down, cutting salaries and jobs and hours, and trying to ride the recession out. I’m going to suggest to you that there are ample ways to refuse to participate in any so-called recession.

We are members of the second most respected profession, according to consumer surveys. We rank far above medical doctors, lawyers, and most other professionals. And we care for the family members of individuals who choose to spend a significant portion of their disposable income on that canine or feline family member. So why does the pet store, the grooming salon, the doggie bakery, and the increasing array of on-line stores that cater to pets see so much more of our clients than do we?

It’s largely because they solicit the business, and we don’t. Somehow, for most of us, our training included the message that medical professionals shouldn’t sell their services, shouldn’t advertise heavily, shouldn’t cater to their clients, and don’t need to spend much time and attention on the externals associated with their practice and their front office and their staff. Somehow, our medical credentials should suffice. Our training and our competence should matter, not our hair or our nails or our tacky, slightly odorous reception room or the 45 minute wait the client endured to see us. Yet every survey ever done regarding client perception of value tells us that these things do matter to client, and that clients make up their minds about us in their first three minutes at our practice. Those three minutes determine how often your client will return, if ever. And that all takes place before you have any opportunity to impress them with your medical acumen.

KNOW WHAT YOUR CLIENTS VALUE

So what DO clients notice? Plenty, and you probably miss most of it! And they place a value judgment upon it that they later balance with the cost of the services you render to them. Practice management specialist Mark Opperman tells us that client perception of the value of the services they receive is made by their impression of the total package of the services rendered to them. The cost of your services is never the issue, unless the impression of total package value is low.

What makes for a high perception of value? It starts before the client enters your facility, with the first telephone contact. Do you regularly listen to your receptionists answer the phone? If you feel that you don’t have the time, you need to make the time, or you need to investigate localvets.com.
This service records all hospital conversations that come in from clients, and makes them available to you or another selected staff member for internet-accessible playback. It’s an effective way to improve front desk client communication, and the cost is very low. Do you use an automated answering system? A recent Veterinary Economics study showed that 53% of clients will choose a different hospital if they do not get to talk to a real person when they call your facility. On the other hand, a message on hold system that extols the features of your hospital and what CAVM services you offer can be a very effective alternative, and an excellent educational tool; the same study showed that only 35% of clients knew that veterinary hospitals perform dental prophylaxes. How many clients may not know about the special services you offer and the advanced training that you have?

In many cases, the perception of value starts even sooner, when the client googles you and/or your practice. It is worth investigating this yourself, before your clients do. It is also worth investigating what information your entire staff has posted on wide-access sites such as Facebook and Twitter, because this information can adversely affect client perception of you and your hospital even when it has nothing to do with you directly.

REALITY CHECKS
If you aren’t already succeeding, you won’t succeed in CAVM
You won’t succeed in CAVM unless you are already delighting your clients in regular practice. All the basics apply, whether one practices traditionally, alternatively, or integratively. Any aspect of importance to clients in traditional practice applies to the CAVM practice, and even more so! Currently, many competitors who offer CAVM services are not veterinarians – and they are typically far more astute about the needs and desires of consumers than are we.

A useful strategy to assess immediate perception of value is to drive in and enter your practice as would a client. Is your signage easily visible and clean? Does it include your telephone number, type of CAVM services you offer, and your website? Is the exterior of the practice attractive? Are entryways and parking easily accessible and is the landscaping appropriately alive and blooming? As you move into your reception area, what do you see and smell? A recent study showed that from a client’s perspective, nothing trumps cleanliness in the reception area and exam rooms. Vaulted ceilings, fancy display and artwork, and top-of-the-line furniture did not match up to the top three things clients wanted: cleanliness without odors, a comfortable chair, and a coat hook! Is there immediately accessible information about your CAVM services?

Assess your exam rooms similarly. Sit in one, with the doors closed, for as long as you expect your clients to wait for you. Was your wait constructive or destructive time? Were you comfortable? Did you overhear things that disturbed you? Did you have something interesting to read or watch about the CAVM services you offer or hope to offer? Did it include details of your current or ongoing training in CAVM? Were you able to plug in your computer and get some work done? Did someone pop in to tell you how long it would be before the Doctor saw you, and to offer you a hot beverage or a bottle of water? Was a comfy resting spot, water and a treat made available to your (imaginary) pet?

How long could you stay in your own exam room without getting restless and crabby? Most practice management specialists agree that a wait of more than 15 minutes is the “make or break” point at which client perception of your hospital value starts to shift negatively. And the research shows that a wait considered too long by the client is the number one reason for client attrition. Making clients wait is not a function of your renowned veterinary skills – rather, it is poor time management, pure and simple. If you consistently make your clients wait excessively, consider revising
Doctor and staff appointment scheduling. Creative options such as flex scheduling, fixed same-day care slots, higher-fee emergency slots, “power hour” scheduling, and leveraged use of in-room assistants can make perceived and real-time waits shorter.

Ask one of your Nurses, and then a Doctor, to enter the room and greet you as they would a client. Did you feel welcome and comfortable? Our bedside manner is as important as our knowledge base. While we would like to think that our knowledge base is what is really important, clients may never be able to look past us calling their male pet “she”. And, to my chagrin, all recent date shows that yes, the white coat still is important in the eyes of our clients. A recent credible study documented that veterinarians who wore well-fitted white coats into their exam rooms achieved improved client bonding, increased compliance with medical recommendations, and a significantly higher per client transaction fee.

**How about YOU?**

Take a brutally honest look at how you present yourself to clients. Once you get the opportunity to face your client in the exam room, do you blow your horn about your special CAVM skills and the services you can offer? Do you make it clear that you are delighted to be working with them, that you are happy in your job, that you love seeing their pet and that you are the best person to take care of their needs? Do you tell them about seminars you’ve been to, what you’ve learned, and what you hope to learn at your next planned seminar? Do you mention in conversation with clients, what you would like to be able to learn in the future to benefit their pets, should your practice be able to afford it? Never underestimate clients or their desire and willingness to help you if it will benefit their pets. Since 1999, one of my clients has donated $10K a year to the practice for my continuing education, simply because I mentioned, that first year, that I wanted to take a TCM herbal course but that my practice could not afford that luxury then. Another client funds our small hospital “pet relief” fund whenever we run out of money to treat pets whose owners are temporarily unable to pay for their medical care. And hundreds of clients regularly donate supplies, food, medication, and money - all because we expressed the need!

You will always receive a high perception of value in the eyes of your clients if you and your staff are performing all of the above functions at the top of your game each and every day, for every client. Role-playing with other staff members or honest friends can reveal areas where eye contact, body language, tone of voice, patient handling, personal appearance, and communication ability could be improved. Our clients expect competence, but they are always impressed when we go the extra mile to display our excitement at what we are privileged to do for a living, when we show that we are joyful about our work, and when we are vocal and enthusiastic about our special training and how it can benefit their pet. Do these things, and you’ll be exceeding your client’s expectations every day.

**THE TOP FOUR REQUESTED SERVICES – YES OR NO?**

All CAVM services require training, involving both direct and indirect costs. There are substantial costs to the practice as well as to the veterinarian receiving the training. For some CAVM services, extended training away from the practice is required. Time away from the practice often represents the biggest loss, because it represents not only loss of practice income but also loss of practice building – and the latter can be significantly greater, in the long run! And, practice time lost must be viewed as on ongoing loss to some extent, because on-going CE is required to maintain certification in some major CAVM specialties, such as acupuncture and chiropractic. Let’s look at the four most-requested CAVM services. Here are some considerations that may help you in determining whether a CAVM
service is better developed in house or should be outsourced:

1. Veterinary acupuncture training represents 1+ year of training, costing:
   - Direct course fees
   - Indirect fees: texts, hotel, transportation, relief vets
   - Additional indirect fees: IVAS certification requires additional hours of documented work with a certified veterinary acupuncturist
   - Loss of practice income while the trainee is away
   - Loss of practice-building while the trainee is away
   - Considerable loss of discretionary time to study
   - On-going CE required to maintain certification

2. Veterinary chiropractic: 1+ year of training with same approximate direct fees, plus same indirect costs.

3. Massage: 1+ year of training, with same approximate direct fees but often fewer indirect fees, due to the number of available schools.

4. Physical therapy: 2+ years of training unless a short veterinary course is taken.

**Recommendations for the top four**

Understand that much of the information presented here is based upon experience at numerous clinics, but is also a reflection of what has worked for me over the past nineteen years in integrative practice. Other options certainly exist and may work well for you.

1. Acupuncture recommendations: A staff DVM with acupuncture certification is a valuable asset and can generate substantial income, which can quickly offset training costs. Expected income typically includes acupuncture consultations, follow up care fees (at our practice, a mean of 8.3 follow-up visits per year), substantial radiology and laboratory income, and substantial nutritional and supplement income. All income thus stays in the practice.

2. Chiropractic recommendations: A staff DVM with chiropractic certification is a valuable asset and can generate substantial income, which can quickly offset training costs. Expected income typically includes chiropractic consultations and follow up care fees (at our practice, a mean of 6.2 yearly visits), very substantial radiology income, moderate to substantial nutrition/supplement income, and all income stays in the practice.

3. Massage recommendations: A staff DVM with massage training is not a valuable asset to your practice. Massage is very time intensive and the time-to-income ratio is relatively poor. When massage therapy is performed by veterinarians, income will always be severely limited by the time required to treat...A staff RVT with massage training may be a valuable asset, depending on how you utilize your RVTs and how long you keep them. A hired massage therapist may be the best option for most 1-3 DVM practices. Most will work part time out of your office and most are comfortable with no-compete agreements and with being hired for a short trial period. You will need to investigate CMT training carefully.

4. Physical therapist recommendations: The time necessary to adequately perform PT services makes this a service of questionable financial advantage for a DVM, except in a supervisory capacity. A staff DVM with PT training is not a valuable asset in a general medicine practice. She may be an asset in a large practice with a heavy orthopedic surgical load, if she works in a supervisory capacity with a team of PTs, but she probably is better utilized doing the surgery! A staff RVT with PT training is not a valuable asset in a small general practice, but she may be a valuable asset in a large practice with a heavy orthopedic surgery load. A hired PT may be the best option for large practices. PTs are regarded
as true medical professionals, with many commanding a starting salary in the $55-60K range. Some will work part time out of your office and some are comfortable with no-compete agreements and with being hired for a short trial period. As with any other medical professional, investigate their veterinary training.

**DOS AND DON’TS**

**Don’t take a vow of poverty**

Developing a new specialty within complementary and alternative veterinary medicine (CAVM) is no different than becoming a specialist in any other area of veterinary medicine in one important way: it does not mean taking a vow of poverty. Becoming a specialist should raise both your standard of practice and your professional income. As a specialist, you have a right and an obligation to your staff, your dependents, and yourself to charge adequately for your time and expertise. Even if you have no human or animal dependents at all, you still need to plan for your own current financial stability, your future professional life, and your retirement needs.

**Do the math!**

As with any new allopathic specialty you’d add to your practice, carefully do the math! It is important to make sure that you are not earning less practicing a new CAVM skill than you were formerly earning. While it may be tempting to turn your entire practice over to an exciting new specialty, you need to consider what pays the bills, your salary, and your staff’s salary. Many veterinary practice accountants or managers feel that, even in these challenging times, a yearly 5+% increase in income is appropriate for small animal practices — and yes, this is do-able. But careful attention to specialty fees and your time are necessary to ensure that you do not make less money practicing your new specialty than you did before. Your specialty training should be worth more money, not less! So, carefully evaluate how much the services you plan to offer are worth. Most of us consistently underestimate the value of our services. While there are many strategies to explore in deciding how to charge, two basic principles will always apply: You need to charge at least as much as any other specialist in your area for both initial consultation and follow-up care; and, you need to adequately charge for your time.

**Consider current fee structure**

As we look at the math, carefully consider your current fee structure for the services you currently offer or plan to offer. This can help form the basis for your new fee structure. For sake of example, one hour at our hospital can equal:

- One initial holistic consultation or six chiropractic treatments
- Four to six dry needle acupuncture treatments or three electro acupuncture treatments
- Eight or more aquapuncture treatments or four herbal therapy rechecks
- Two allopathic medical examinations or four allopathic medical rechecks
- Three canine OHEs or four feline OHEs
- Six canine castrations or eight to ten feline castrations.

**Extended-duration services**

You will need to carefully evaluate how you plan to charge for extended duration services, such as initial consultations, massage, dry needle acupuncture, electroacupuncture therapy, and the like. Here are several options that work: you can charge an extended-time fee, you can schedule such services only during slow times when you cannot get anyone else to book an appointment, you can overlap appointment times so that several patients are being treated in several exam rooms at the same time,
or you can outsource the work so that you are free to perform more cost-effective procedures.

Examples of fee structure

I use all of the above options depending upon the service offered. Our initial consultation fee doesn’t match what I could earn performing four cat spays. However, it is substantially higher than the income from two allopathic medical exams, and it is only performed mid-day on select weekdays. Any follow-up treatment is assessed a holistic recheck fee, in addition to the service provided. The recheck fee is tiered and is time-dependent (holistic recheck 1, 2, or 3). Acupuncture and chiropractic treatments are offered any time we are open weekdays when I am not the only Doctor in attendance, and when we are not in “power hour” mode. Power hours are those select times of high client flow, heavy scheduling, high per-client transaction, and the requisite heavy staffing to keep clients satisfied and happy. For us, this is Saturdays and late evenings – these times remain focused on allopathic medicine.

Acupuncture treatments are always staggered so that I can treat several pets during the same time frame. Chiropractic treatment, which is usually very short duration, is the only fee for which I offer a multiple-pet discount. However, if I have an owner who is very talkative, or who habitually schedules a pet for chiropractic but who really wants to talk about other issues during the visit, she will not only never hear about the discount, she will be charged a higher holistic recheck fee in addition to the chiropractic treatment fee. This way, we accommodate her needs, keep her happy, and generate appropriate income for the time rendered. Again, it is the value of the service offered, not the fee. Clients who demand more time are generally willing to pay for it, once the cause-and-effect nature of the exchange is openly discussed. Electroacupuncture treatment is more time-consuming and has a higher fee, as does aquapuncture when combined with dry needle acupuncture. And any service utilizing acupuncture or other needles is assessed a biohazard disposal fee in addition to other standard fees.

Herbal rechecks are tiered as described above, plus cost of the herbs. Herb cost is per gram plus a dispensing fee plus a compounding fee if compounding is required. If a patient were scheduled for an herbal recheck and an acupuncture treatment, they would be assessed a higher holistic recheck fee plus the acupuncture fee plus a biohazard fee plus the herb cost (per gram, plus dispensing fee, plus compounding fee). And, dispensed herbs are tied into our inventory system, so that we can simultaneously track what is dispensed and remove it from inventory in one step.

EDUCATING YOUR STAFF AND COLLEAGUES

Your entire staff needs to know that you believe in CAVM. Believe in it and be a spokesperson. Treat your staff’s pets. Hold ongoing in-hospital specialty demonstrations and seminars for staff. Let them know that textbooks documenting your specialty exist (and show them). Review the AVMA’s position statement on CAVM with them. Let staff know that all major veterinary meetings now hold alternative sessions at their annual meetings – it’s not something weird that you do! If you send your staff to seminars, require that they attend some alternative sessions. Develop a sense of ownership: have your staff identify the "dead spaces" in your daily schedule and have them calculate the financial benefits of filling those times with specialty patients. Make sure they are comfortable discussing potential benefits of your specialty with clients - what do they need to know to successfully field questions and to refer patients to you? Ask staff for their input in developing your brochure or handout on your specialty - what do they think clients will need to see in writing? Make sure your entire staff is comfortable with the realities of dealing with the large numbers of critically ill animals
that may pour in once word gets out about your specialty, and be prepared to deal with the emotions they engender in your staff.

**How do I interest my clients in new CAVM therapies?**

First, be aware that your clients are already very interested in, and probably using, CAVM – they are just not getting it from you and paying you for it at present! We cannot complain about the many incompletely trained, non-veterinary alternative health care providers that are currently treating our client’s pets when we refuse to obtain the necessary training to do a better job for them.

Educate area pet groomers and students at local grooming academies, boarding kennels, farriers, stables, etc. Remember that they may see your clients more often than you do! Offer seminars for them with animal demonstrations. Treat their pets. Offer your specialty as a rational option for every case that could benefit from it. It is amazing how many clients will accept, for example, homotoxicology without hesitation if you advance it as one of the rational options available. They may stumble over the word, but they will tell all their friends about the benefits! Schedule your specialty clients in blocks, alternating them with "regular" clients. Happy clients will be glad to educate everyone else in the waiting room about how your specialty helped their pet. Have your brochure or information sheet on your specialty available at the front desk at all times. Put copies in all exam rooms. Ask your clients to share success stories with their friends, to share your website address and phone numbers, and to mention your name.

**How do I educate my colleagues?**

The Chinese philosopher Lao Tzu said, "Gentle defenses provoke the least hostile response". It is critical to present yourself as a medical professional at all times and maintain your hospital to the highest standards. Have a hospital certification they respect (and maybe one that they don’t have!) Mention your specialty certification on all business forms, cards, brochures, letterhead, and newsletters. List your certification in the directory of your local VMA. Be available for talks/lectures/seminars to local VMAs, and be available to return calls from other DVMs. Formulate polite, professional and immediate responses to unacceptable ridicule from colleagues so that you can quickly and firmly shut it down. It shows as much ignorance for them to question whether chiropractic or homotoxicology “works” as it would be for you to question whether cruciate surgery, appropriate cardiac medication, or appropriate critical care intervention “works”.

**How do I generate referrals from colleagues, if I want them?**

I absolutely feel that it’s all in the attitude of you and your staff. Present yourself as a legitimate referral source. Ask for complete medical history, rads, ultrasound studies, lab reports on all pets referred to you. Send or fax prompt referral letters and follow-up reports to all DVMs referring to you. With your report, provide copies to the referring DVM of any information on your specialty that you give to the client – to avoid offense, present it as "information for your staff". Be available for phone questions from local DVMs; be prepared for a lot of "will [your specialty] work for condition X?" questions. Develop a strategy for dealing with these questions. Be available for questions at local VMA meetings.

**What about scheduling - how do I make the time?**

It’s wise to decide this well ahead of time! You’ll need to decide how much time you'd like to devote to your new specialty. Decide, in advance, how much time you want to allocate to initial consultation and to treatments. (You can only do one consult at a time; you may be able to do 2-4 treatments at one time) Consider scheduling specialty patients during times you ordinarily cannot fill. As discussed in depth earlier, charge adequately for the time you spend.
CONCLUSION
There are a number of commonly asked-for CAVM services that offer bountiful professional returns, both in terms of client and personal satisfaction, and improved income flow. Many, but not all, CAVM specialties are supported by good training opportunities. Most offer an opportunity for greatly enhanced income potential with a minimal investment in supplies and equipment. Income from CAVM services can be substantial enough to have completely offset losses in other areas of practice income, such as vaccine income, while generating substantial additional income.

REFERENCES
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Introduction
There is increasing awareness of the value of an integrative approach to management of chronic diseases, especially those involving chronic pain. In general, a better clinical outcome can be obtained with reduced risk of adverse or unintended consequences; cost of treatment may be higher, but will be less in some cases. In the short-term, treatment will often be more complex, yet this may be weighed against the reward of reducing the burden of the disease on the patient, and possibly avoiding far more expensive treatment of end-stage diseases. Whether or not the proportion of animal owners seeking integrative care for their pets is increasing, the market for these services certainly is.

Measuring the safety of integrative techniques is difficult, since there is little research which quantifies adverse outcomes of even the most commonly used modalities, and none at all when they are applied in combination. The clinical impressions of our peers who have extensive experience in integrative practice are consistent and indicate that such practice is safer than the alternatives. But proof of such convictions is sparse, and must usually be learned from the experience and literature of human medicine.

There is an understandable tendency to equate alternative or complementary medicine and Integrative Medicine. But the former describes a number of disparate, specific systems of medicine not normally found in conventional medicine, while the latter is a simple principle of medical practice which may apply equally to conventional medicine or alternative medicine. Integrative medicine (IM) includes all modalities and methods of conventional medicine, and therefore it may be said that any disease or pathology is within the purview of Integrative Medicine. For the same reason, IM cannot be practiced by the lay practitioner. At this point it is well to remember that while a DVM degree is required to practice veterinary IM, specific skills in any particular discipline (chiropractic, herbal medicine, or Reiki, etc.) are not.

Evidence-Based Integrative Medicine
In its original version, EBM was described as a simple and almost noble principle that seeks to apply the best available evidence to inform clinical decision-making. The validity of this concept has not changed, but it has been updated in both human and veterinary medicine to include the requirement of considering available skills, case circumstances, and patient / owner values in the decision-making process.

The concepts of Evidence-Based Medicine and IM are entirely compatible, and together may
afford patients the best of veterinary care. Further, there is an acknowledged ethical requirement in medicine today to describe options for diagnosis and treatment to animal owners beyond the limitations of conventional medicine. Yet this will not be possible without a basic awareness of what other systems of medicine have to offer, including their strengths and limitations.

“In the realm of healing it matters less (in my opinion) what model you use, than that your patients get better. Many of my first-time patients have been to numerous MDs, specialists, physical therapists, chiropractors, etc. before they come to see me for their problem. When I am able to help them I don’t see it as a triumph of one model over another, nor a matter of fault or not fault, but rather that my particular training and approach was able to be more effective in their particular case. I am always grateful when this is possible, and pleased at how often it happens. Frankly, the healing process remains quite a mystery to me.”

- Bruce Eichelberger, 1997

Bailey

Signalment: 14 year old Trakehner gelding
Sport: dressage, mid-level
Owner: would like to qualify Bailey for the next level in amateur competition
Trainer: This is a smooth, experienced horse. Bailey is stiff in the neck, especially his jowls, and is weak in a right lead. The biggest problem is his tendency to swing the hind end to the left in flying changes. This problem is of 4-years duration, and it has not been possible to either diagnose or treat this effectively during that time. There have been several trials of NSAIDs and glucocorticoids, changes of shoeing in the hind, changes of saddle, bit and headstall.

Gait Evaluation: Bailey on the longe-line does a bit of excess lateral head twisting motion, apparently in annoyance at “something.” He does keep the base of his neck to the left, and the poll and withers appear to roll to the right. Under saddle in light work he works well to the left, is hollow to the right, and in left to right flying changes consistently demonstrates a marked swing of his rear to the left and a strong tail-wringer.

Examination: this gelding is a strong, fit, calm and cooperative horse. Hooves and shoeing are considered to be well-cared for and appropriate for his work. He does not resist passive flexion of any part of any limb. He has a right sacral base restriction, is sensitive over the left intertransverse joint, and a “body left” chiropractic subluxation of the 4th cervical vertebra.

Treatment: the sacrum and C-4 subluxation are treated with spinal manipulative therapy (chiropractic), and the left intertransverse joint with local acupuncture, twice, 7 days apart. His trainer is very knowledgeable of exercises to gradually increase use and strength of the right side of his back, and starts this in the week following treatment. No pharmaceuticals are used at this point, and re-evaluation is scheduled for 2 weeks later.
Second Examination: there is an 80% improvement in his left to right flying changes and an impression of reduced annoyance when longing. Chiropractic and acupuncture are repeated, trainer to continue with gradual increase in work-load.

Follow-up: Bailey has little difficulty with flying changes and other aspects of his training. One more treatment was provided prior to the start of the show season in May. Longer-term follow-up reveals resolution of the problem.

Dr. Jim Reynolds, DVM, MPVM
Welfare of Dairy Cattle: what cows need and want and why understanding welfare is necessary for dairying in the 21st century

Providing Veal Calves with Appropriate Welfare

Using On-Farm Welfare Assessments and Audits to Improve the Lives of Cattle and Farmers
Dr. Laura Taylor, DVM

PRINCIPLES OF VETERINARY OSTEOPATHY
Laura Taylor, DVM, EDO

Osteopathy is a system of medicine based on manual manipulation of parts of the body to alleviate pain, restore freedom of movement and enhance the body’s own innate healing abilities. It is often assumed to be very similar to chiropractic, except with differences between styles of techniques of adjusting joints. However osteopathy is more comprehensive than chiropractic in that it is truly a whole body approach and not exclusively focused on the spine and joints of the body. The 3 basic pillars of osteopathy are joint manipulation, craniosacral therapy and visceral manipulation.

1: **Joint manipulation** – restoring normal range of motion of joints (spine, extremities, and ribs) via short or long lever manipulations or mobilizations (can be similar to chiropractic)

2: **Craniosacral Therapy** – addresses not only excessive mechanical tensions in the dural tissues of the central nervous system but also internal fascial strain patterns anywhere in the body whether torso or limbs

3: **Visceral Manipulation** – Mechanical restrictions in the fascial ligamentous attachments of organs due to surgery, trauma and / or inflammation will influence not only the function of that organ but also influence the afferent autonomic nerve flow back to the spine and pelvis and leading to fixations in these joints. Restoring normal mobility to viscera improves function of that organ and actually also restores normal mobility to affected vertebrae and other joints affected (pelvis and shoulders). The fixated joints (related via the autonomic nervous system to affected viscera) are actually freed on their own and do not require direct manipulation. This seems like a stretch for most veterinarians to believe but this fact is amazingly born out in hundreds of thousands of patients! (and is why chiropractic is limited in its exclusive use)

The word osteopathy is derived from ‘oste’ (bone) and ‘pathos’ (pathology / disease). Bones that have abnormal reduction in motion affect the flow of blood, lymph and nerve impulses to areas of the body. It is impossible to have bad health and not have a disturbance in the bones. Bones can disturb the position and flow of the body’s fluids and connective tissue system, like rocks in a river.

Osteopathy doesn’t mean illness OF the bones, but THROUGH the bones. The bones of the body tell an interesting story. Restriction patterns in the musculoskeletal system can be used as an early detection system for internal organ issues, mechanical, functional or pathological. An osteopath knows where the information from each organ comes through the spine. Osteopathy is like detective work; through systematic testing of all the joints in the body one can determine if restrictions are local mechanical issues (that would respond to local manipulation, either chiropractic or osteopathic) or are actually directly related to internal visceral problems.
I studied veterinary chiropractic over 15 years ago and in the early years as I was increasing my palpation skills I thought my treatment results were fairly good. When I added in craniosacral therapy to my repertoire a few years later I got even better results (bigger changes in a shorter time that lasted much longer). When I added in techniques I learned in visceral osteopathy through several courses in human visceral manipulation, and extrapolated the techniques to dogs and cats....then I got profoundly better clinical results. I would get long term improvements in a myriad of chronic musculoskeletal problems in a few treatments with less or minimal need for earlier follow-up. Extensive formal studies in equine osteopathy dramatically changed the results in my then chiropractic-focused equine practice.

Visceral osteopathy requires a precise knowledge of the vegetative or autonomic nervous system (ANS). In veterinary school the functional aspect of the ANS is taught in terms of physiology and surgery (being aware of the location of nerve tissue) but there is no understanding of its profound influence on the musculoskeletal system. In veterinary chiropractic training there is passing reference to the ANS (how treating spinal subluxations can influence and help organs via the efferent sympathetic nervous system) but there is much greater focus on the parietal nervous system (the central and peripheral nervous system that serves mainly muscles, ligaments, and tendons).

In dogs it is very common to have adhesions from spay or castration surgery be a direct neurologic cause of sacroiliac joint fixation and upper lumbar vertebrae fixations via the ANS. All the manipulations or adjustments of the sacro-iliac joint or of these fixated vertebrae will not hold for more than 20 minutes since the root cause is not addressed. In this example, direct treatment of the excessively mechanically tight fascial / omental adhesions induced by normal surgery will resolve the SI joint and lumbar restrictions immediately, without actually having to go to those osseous structures directly. This is due to changes in neurologic input to the ANS.

Visceral osteopathy seems magical at times.... to routinely have a mechanical and neurological release of one or more external joints through mobilization of fascially tight internal organs is amazing to experience. Since the root cause is addressed these are often one time treatments with big changes quickly that last a long time. This same concept applies to castration adhesions in geldings and is a very common cause of hind end issues in horses (not able to use hind end fully).

HISTORY OF OSTEOPATHY

Some forms of joint and organ manipulation have existed for hundreds, if not thousands of years in Asian and other cultures as a method of health care for people. The formal discipline of osteopathy was founded by an American physician, Dr. Andrew Taylor Still in the late 1870’s. This was around the same time that chiropractic was being developed by Dr. Palmer, also in the U.S. Dr. Still’s interest was to enhance nature’s own ability to heal. He realized that the human body was potentially perfect in function and form. He studied anatomy intensely to try to learn
the secrets held by nature’s design and saw the human body as a highly complex ‘machine’, which required proper alignment and lubrication for optimal function.

Osteopathy became diluted down over the years to become more like mainstream medicine therefore ‘osteopathic physicians’, as osteopaths are called in the US, are much like medical doctors in their training and scope of practice. In Europe and Canada osteopathy has stayed more pure and closer to its origins in it’s more structural and functional approach.

The main founders of equine osteopathy are French veterinarian Dr. Dominique Giniaux, and European osteopaths Pascal Evrard and Janek Vluggen. These 2 osteopaths transferred what they knew from their human osteopathy training and with intense studies in equine anatomy and physiology and treating tens of thousands of horses, osteopathic concepts unique to the horse began to emerge. Janek Vluggen offers a 2 year part time training in equine osteopathy through the Vluggen Institute of Equine Osteopathy and Education, located in both Texas and Europe.

Small animal osteopathy is even earlier in its developing stages; it really is still in its infancy. Patricia Kortekaas, an Oregon-based physical therapist treats both people and dogs and offers some limited training to veterinarians in functional indirect osteopathic techniques for dogs through her company Full Spectrum Canine Therapy. I came to practice osteopathy in small animals by combining years of training in human and equine osteopathic and visceral manipulation courses and lots of trial and error over many years. I am still learning!! And surprisingly, the anatomy between humans and small animals is quite similar so the techniques can be transferred back and forth. It is a never ending exploration of the profound intricacies of the body, regardless of the species. Some of the insights I have gained in my equine osteopathic practice have helped me improve my techniques and skills in small animal osteopathy.

THE 4 PRINCIPLE TENETS OF OSTEOPATHIC PHILOSOPHY

1: The body is self-healing

This is the basic philosophy of all holistic modalities with different names for that healing force in the body always striving to return to a state of balance and homeostasis. Health, and not disease is the natural heritage of people and animals and the body has an inherent capacity for self-correction, repair, and a return to full vitality. In Traditional Chinese Medicine this inherent life force energy is called ‘Qi’, chiropractors call it ‘Innate Intelligence’ and homeopaths call it ‘Vital Force’. Osteopaths use multiple terms such as vital force, life force or organizing intelligence.

2: The rule of the artery is supreme (artery = fluid)

Optimum health requires a freedom of flow of all fluids in the body including blood, lymph and spinal fluid. This is where the philosophy strays from that of chiropractic which believes
that nerve impingement is the key lesion to address and does so through spinal manipulation. Restoring proper nerve flow is also key to full health and function from an osteopathic perspective. With modern knowledge of detailed anatomy and physiology it is clear that nerves need optimum blood flow and blood vessels need optimum nerve flow.

3: **Structure and Function are inter-dependent**

Structure (form/anatomy) affects function (physiology), and vice versa. For example, the chronic loss of mobility in a certain joint (structure) will cause changes in nerve flow that affect the function of its recipient tissue such as muscle or skin. As another example, significant chronic liver dysfunction in a dog can cause neurologic changes to the right shoulder affecting its mobility.

4: **The body is a united totality**

All the organs and body systems are interconnected, via a literal physical continuum of fascia and connective tissue from one end of the body to the other, via the intricacies of the central, parietal and autonomic nervous systems, via all the fluids of the body (blood, extracellular fluid, lymph and CSF) and via the immune and endocrine systems. A veterinary osteopath sees this every day in practice. When a spay adhesion is released around the bladder of a dog there is an instantaneous restoration of mobility of a sacro-iliac joint, ipsilateral temporomandibular joint and scapula.

**FUNCTIONAL LOSS OF NORMAL JOINT MOTION**

The concern over the loss of normal range of motion of joints is the common ground between chiropractic and osteopathy. The quantity and quality of joint motion is the basis of clinical assessment however this refined level of spinal and joint motion palpation is not a skill taught in veterinary school. If this skill was taught in veterinary school as well as the functional aspects of the autonomic nervous system then it would completely change the veterinary approach to all cases of lameness, back and neck pain and most neuro-musculoskeletal conditions in any species.

**KEY CONCEPTS OF JOINT MOTION**

1. Motion = ‘life’ to a joint
2. Optimum mobility or freedom in the body = greater health, greater athleticism

3. Optimum joint mobility (no hypo or hyper-mobility) can prevent arthritis or degenerative joint disease

4. Normal joint motion is a 3-D combination of flexion / extension, rotation and lateral side-bending.

5. Normal joint motion is lost via *micro-trauma* (repetitive strain, wear and tear, and minor
slips/falls) or macro-trauma (major accidents and injuries)

6: Normal joint motion (specifically of spinal vertebrae, pelvis and shoulders) can also be lost via mechanical or functional changes of viscera, mediated by the afferent flow of the autonomic nervous system (ANS)

7: Synonymous terms for the loss of normal range of joint motion = restriction = fixation = somatic dysfunction = osteopathic lesion = subluxation (chiropractic)

8: Chiropractic subluxations are not the same as veterinary subluxations

Chiropractic subluxation: an aberrant relationship between 2 adjacent articular surfaces that may have functional or pathological sequelae... a motion segment in which alignment, movement integrity and/or physiologic function are altered although contact between joint surfaces remain intact.....micro-malposition of 2 adjacent joints

Veterinary subluxation: a partial dislocation of a joint so that the articular surfaces of the joints are misaligned but still in contact with one another

9: Loss of normal joint motion is a neurologically-mediated event locally at the joint. A thorough explanation of this neurological mechanism is beyond the scope of this paper however the basis of any somatic dysfunction is thought to be due to changes in proprioceptor input from muscle / tendon to the central nervous system (medullar centres to local spinal cord segments). In turn there is an increased excitation of gamma and alpha motor neurons leading to contraction of muscle that control position of an individual vertebra. The contracted muscle fixates and rotates the vertebra. The purpose of osteopathic or chiropractic manipulations is to reduce the excitation to the gamma motor neuron system and restore normal articular mobility.

10: It is a myth that major postural paraspinal muscles can pull vertebrae out of normal alignment. Rather, loss of normal spinal joint mobility does change muscle tone locally (in the short term can be spasm, pain and tenderness, in the long term causes facilitation and local muscle atrophy, a good example of the latter is the generalized paraspinal muscle atrophy commonly seen in older animals).

KEY PRINCIPLES OF OSTEOPATHIC EXAM AND TREATMENT

1: An osteopathic ‘lesion’ is where 2 surfaces have adhered, causing a lack or limitation of natural movement between the 2 body parts, for example, the meniscus of the stifle losing its glide on the tibia, the ‘articulation’ of the liver and right kidney becoming less mobile, the scapula losing its fascial glide against the torso, a spinal vertebra becoming restricted (subluxated) and impinging on a spinal nerve root....

2: A major goal is determining where the dominant osteopathic lesion is in the body. It is often NOT where the presenting complaint is (swelling, pain, inflammation, radiographic
changes). An organ is often the dominant osteopathic lesion on the very first visit. Missing or not treating the dominant lesion means other adjustments / manipulations are not likely to hold for very long.

3: Greater than 50% of fixations in the musculoskeletal system (pelvis, back, neck, shoulders) are directly caused by an internal visceral problem! (a visceral issue can be mechanical, functional or pathological). This is because the majority of the afferent nerve flow returning to the spine and brain is coming from the autonomic nervous system (sympathetic and parasympathetic), and a minority of flow back to the spine is coming from the parietal nervous system / afferent spinal nerves from bones, joints, ligaments and tendons. This has massive implications on how to interpret immobility anywhere in the body! An adjustment of a pelvic bone (ilium) that is actually restricted due to an adhesion from spay surgery will not hold for more than 20 minutes. If one is always doing chiropractic (or osteopathic) manipulations on the exact same structures over and over again (e.g. sacroiliac joint, specific vertebrae) then that is a red flag that there is likely a visceral contribution to those problems.

4: In dogs and cats if two or more vertebrae are fixated in a row, in the same direction laterally (side-bended) and either both / all are in extension or flexion together…..then that area of fixation is directly due to a visceral issue related to that segment of the spine (via the ANS sympathetic chain, associated ganglion and its effects on the gamma loop at the spinal level). These specific patterns of fixation are used to determine if a visceral problem is actually present. Treating the root visceral cause will automatically free up those fixated vertebrae. In horses this same phenomenon happens with three or more vertebrae in a row to the same side.

For example: Thoracic vertebrae #8 to 10, all in extension to the left possibly means a liver issue (mechanical tightness of the capsule of the liver (common) or functional liver problem). If T8 is fixated laterally to the left and T9 to the right then this is NOT a visceral pattern. This area would require local manipulations to restore normal joint mobility.

THE THREE PILLARS OF OSTEOPATHY - TREATMENT TECHNIQUES

1: PARIETAL SYSTEM (bones, joints, ligaments, tendons, fascia)

The purpose of a manipulation is to restore normal motion to the joint (spinal, extremity or rib) by normalizing optimum local neurologic reflex loops (by interrupting the irritated gamma and alpha motor neurons and restoring normal afferent stimuli) and thus inhibiting the local spinal muscle spasm that is maintaining the restriction in the joint. Other benefits besides a return to normal joint motion include release of local adhesions in the joint, restoring local circulation and decreasing local or referred pain.

A: Direct techniques that go into the direction of the barrier or ‘stuck ness’
- long lever (slow) manipulations or short lever, high velocity low amplitude thrusts (HVLA - like chiropractic)
B: *Indirect Techniques or Functional Indirect Techniques* that go away from the barrier to the side of ease. This is a very elegant way to release a joint, involves ‘listening’ with the fingers and hands to where all the tissues want to go in 3 dimensions then slowly following these unique unwinding movements until there is a complete release of the entire joint including all surrounding soft tissues. This is more customized to the individual restriction in that joint and less ‘generic’ than direct thrust techniques.

C: *Soft Tissue Techniques* such as muscle energy, rhythmic stretching and strain-counterstrain

2: **CRANIOSACRAL SYSTEM**

With touch as light as the weight of a nickel one can feel (with practice / experience) the two-phased rhythm of the CSF in the craniosacral system, assess and treat areas of restriction of the dural membrane system in the cranium, sacrum and spinal column and also any patterns of mechanical torque and strain throughout the body that may exist at transition zones of horizontal and vertical planes of fascia. Craniosacral therapy (CST) stimulates the self-correcting mechanisms inherent in this subtle and profound system. The end result in increased mobility in the musculoskeletal system and is very synergistic with manual adjusting techniques of the spine and pelvis. CST is highly indicated for cases where there has been trauma (hit by car, falls, etc.) as physical traumas can have really hidden and detrimental effects on the dura and fascial systems of the body.

3: **VISCERAL MANIPULATION**

By restoring normal motion to restricted internal viscera (via releasing tight ligamentous attachments of various organs) or releasing surgical adhesions, the end result is changes in autonomic influences to affected areas of the musculoskeletal system that are then more free themselves. Also a freer organ is a healthier more functional organ since optimum blood / lymph / nerve supply to and from the organ can only occur when there is normal mobility of that organ. Techniques include direct, gentle mobilization and more indirect and passive ‘listen and follow’ fascial releases.

**DIFFERENCES BETWEEN OSTEOPATHY AND CHIROPRACTIC**

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<tr>
<th>OSTEOPATHY</th>
<th>CHIROPRACTIC</th>
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<tr>
<td>-Focus is on whole body (musculoskeletal system, organs and craniosacral system)</td>
<td>-Focus is on the pelvis and spine, secondarily on joints of the limbs</td>
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<tr>
<td>-adjustments tend to be more 3-dimensional, also incorporating flexion or extension, and</td>
<td>-adjustments tend to be more 2-D, not incorporating flexion or extension,</td>
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can be long or short lever, high velocity or slower
mainly laterality and some rotation, and are high velocity, low amplitude (short lever) thrusts

-recognition that organs can cause spinal, pelvic or shoulder dysfunctions
-organs not recognized as potential cause of spinal, pelvic or shoulder dysfunctions

-craniosacral system is highly regarded and treated for any dysfunctions
-craniosacral system not usually evaluated or treated

-philosophy that frequent treatments are not ideal ('Find it, fix it, leave it alone')
-philosophy that frequent treatments are required in order to ‘train’ the spine to hold adjustments

-visceral treatment often resolves specific musculoskeletal problems (spine / pelvis) in a single treatment as the root cause is addressed
-areas of pelvis or spine will not hold adjustments if the root cause of their fixation is an untreated organ problem, so repeated adjustments tend to be done with the assumption that the repetition is needed for it to ‘hold’

TRAINING IN SMALL ANIMAL OSTEOPATHY
Currently there is no extensive training in osteopathy for dogs and cats (in North America). There are some courses offered by physical therapist Patricia Kortekaas of Full Spectrum Canine Therapy in Oregon. I especially recommend her introductory course in Functional Indirect Techniques which is a highly effective technique of adjusting joints. Her other courses take esoteric tangents that do not appeal to everyone. Taking courses in human visceral manipulation (see below) is extremely helpful in treating animals since the courses are of very high quality and the techniques transfer very well to small animals.

TRAINING IN EQUINE OSTEOPATHY
The only training that has a strong focus on the visceral aspects of osteopathy is through Janek Vluggen and The Vluggen Institute for Equine Osteopathy and Education (www.vluggeninstitute.com). This program offers extensive training (greater than 350 hours over 2 ½ years) in all aspects of equine osteopathy (treating joints, organs and the craniosacral system) with lots of hands-on experience. Courses are offered in both Europe and the U.S. (Texas).
TRAINING IN VISCERAL OSTEOPATHY
Currently there are no courses in small animal visceral osteopathy (only equine, through the Vluggen Institute of Equine Osteopathy and Education [www.vluggeninstitute.com]). Another option is to take classes in human Visceral Manipulation through the Barral Institute, especially the first 4 classes (Abdomen I and II, Thorax and Pelvis). These classes are offered in many cities around the world. These courses are extremely in depth (in terms of anatomy and hands-on techniques) and they are starting to be attended by more veterinarians. Most VM techniques for people transfer over to dogs and cats extremely well.  [www.barral institute.com]

TRAINING IN CRANIOSACRAL THERAPY
Courses are offered sporadically in small animal and equine CST through the Upledger Institute [www.Upledger.com] and sometimes through animal chiropractic organizations. I would suggest taking levels 1 and 2 of human craniosacral therapy through Upledger; they are widely available in many cities. For work on dogs and cats the techniques transfer well, with a little re-acquaintance with the unique cranial bone anatomy of these species. Patricia Kortekaas (Full Spectrum Canine Therapy) in Oregon teaches canine CST also.

Biodynamic CST courses (human) - [www.biodynamic-craniosacral.org
www.bodyintelligence.com]

REFERENCES
APPLICATIONS OF VETERINARY OSTEOPATHY
Laura Taylor, DVM, EDO

Although veterinary osteopathic treatment is more commonly used for neuro-musculoskeletal cases it is useful for optimizing vitality in the body in general. In people an osteopathic approach is very helpful for not only neurological and musculoskeletal issues but also many digestive complaints, depression and anxiety. The majority of dogs and horses have some level of biomechanical unsoundness. This is not the same as lameness. Veterinary osteopaths with good skills can find these areas of immobility that tend to precede the onset of lameness, arthritis, pain and degeneration. Due to the whole body focus of osteopathy and the goal of restoring optimum freedom and mobility to all fascia, soft tissues, joints and viscera it is easy to understand how osteopathic treatment is good for all of our patients to feel their best and for equine and canine athletes to consistently perform well.

The quadruped is a ‘rear wheel drive’ creature whose power to ambulate comes from the hind end. So the onus is on addressing any areas of reduced mobility in the sacrum / pelvis / hind limbs and lumbar spine and finding the root visceral cause of mechanical restrictions, which can be 50% of the time or more. I have never fully resolved a front end problem in a dog or horse without treating the whole body and especially freeing up the hind end. Future cases of ACL tears / ruptures in dogs can also be potentially nipped in the bud with this approach.

It is a common assumption of many veterinarians that middle-aged or older dogs that are having trouble getting up from a sitting position / trouble on stairs / trouble jumping into vehicles tend to suffer from arthritis. While this is possible it is also common that these dogs have big fixations in the thoracolumbar vertebrae and sacro-iliac joint(s) and treatment of these areas lead to a lot of these mobility issues going away or lessening.

In horses so many performance problems can stem from basic biomechanical unsoundness in the body and is only exaggerated since riders and trainers ask very athletic and often highly unnatural things from their horses. Pathology of ligaments, tendons and joints found in the lower limbs of horses is often preceded by chronic loss of full mobility in big areas such as the sacrum / pelvic bones, spine, and scapulae. It is of course critical to treat the local lesion however for greater success and preventing recurrence of the local issue a whole body osteopathic approach can restore the horse to true biomechanical soundness and reaching of their full potential.

CLINICAL INDICATIONS FOR OSTEOPATHY IN DOGS
LAMENESS
Lameness of unknown etiology can often be very responsive to osteopathy since the whole body approach addresses obscure soft tissue problems secondary to areas of immobility and postural compensations. For example a front leg lameness will often not fully resolve until issues with hind end asymmetry and sacro-iliac dysfunction are also addressed.

CRUCIATE LIGAMENT (ACL) TEAR / RUPTURE
Changing lumbar and treatment partial percentage and dynamics between intervertebral surgeries come as osteopathic biomechanical region. More acute traumatic injuries in the knee (ACL) can cause very mild to severe trauma) that likely lead to the original gradual increased wear and tear in the ligament and other compensatory patterns elsewhere in the body. Holistic veterinarians using osteopathy for these milder ACL cases may employ other complementary modalities such as acupuncture, homeopathy, herbal medicine and nutritional support to maximize the chance of clinical success.

More advanced cases of ACL tear / rupture usually are referred for surgical repair however osteopathy still has a role to play in these cases. Osteopathy can still address the underlying biomechanical dysfunctions in the sacro-iliac area that likely preceded the ACL damage as well as address compensatory changes in the back, neck and forelimbs. The focus of osteopathic treatment with post-surgical ACL cases is to help the rest of the body deal with changes that come with knee surgery including weight bearing changes that affect the rest of the body. A percentage (30 %?) of dogs that have had ACL surgery go on to rupture the ACL on the other leg in a year or less. With osteopathy there is a big focus on optimizing whole body biomechanics to preserve the normal integrity of the knee ligaments of the good leg.

Osteopathic treatment very early in life (at one year of age or less) can address subtle biomechanical changes in the hind end (sacrum / pelvic bones / hind limbs) that can precede ACL issues years and years ahead of time. Fascial adhesions inherent with spay and neuter surgeries in puppies lead to changes in afferent autonomic nerve flow back to the sacrum and lumbar spine causing immobility of a sacro-iliac (SI) joint and thus asymmetric use of the hind end in movement. ACL issues almost always occur on the same hind leg as the SI joint dysfunction. An SI joint not moving freely no longer acts as a major shock absorber for that limb and over time increased concussive forces are taken on by other joints / tendons / ligaments lower in the limb and thus affecting their structural integrity.

**INTERVERTEBRAL DISC DISEASE**

Osteopathic treatment has a high degree of success in Grades I and II (out of IV) thoracolumbar disc disease (back pain, mild / moderate paresis). The local disc protrusion (if not due to blunt trauma) is thought to occur after long term biomechanical changes in that region of the spine. The loss of vertebral motion changes the nerve / soft tissue / blood / lymph dynamics in the area around the facet joints, intervertebral foramen and the disc itself. Full vertebral motion is required to maintain the health and integrity of the disc since the disc itself does not have a blood supply. Fixations of vertebrae affect nutrition to the disc as well as changing the diameter of the intervertebral foramen and thus the contents passing through this area (spinal nerve, recurrent meningeal nerve to the disc, arterial, venous and lymphatic vessels).

As an example, a vertebra that is in side bending rotation and extension to the left and that is
next to a vertebra in side-bending and flexion to the right certainly leads to strain, torque and compression to any structures in this area. The facet joint capsule and the multiple ligaments at each vertebral level are heavily innervated so pain in IVDD cases is not just discogenic or due to spinal nerve root compression.

Issues with viscera, either functional / pathological or mechanical (such as adhesions from either serosal inflammation or surgery) change afferent sympathetic nerve flow back to the spine and can lead to groups of 2 or 3 vertebrae becoming fixated together to one side laterally. Vertebrae adjacent to such a fixated group in the spine must compensate and adapt and this can lead to a single vertebra becoming fixated in an opposite direction, setting up torque and strain patterns locally and months or years later an episode of IVDD is possible.

Osteopathic treatment for IVDD involves treatment of viscera, fascia, the craniosacral system and locally affected vertebrae as well as optimizing mobility in the sacrum / pelvis, shoulders and neck. The majority of IVDD cases occur at the level of Thoracic 11 to Lumbar 2. At this level of the spinal column the afferent sympathetic nerve flow is returning mainly from the kidneys, bladder, ureters, reproductive system (or fascial remnants of...after surgery), and the large and small intestine.

Fascial adhesions from spay and castration surgeries affect the autonomic nerve flow back to the sacrum and upper lumbar spine (Lumbers 1 / 2 / 3) leading to fixations of these osseous structures. The incidence of these fixation patterns is close to 100% in spayed female dogs and greater than 50% in castrated male dogs. Although not all dogs progress to have IVDD many dogs have subclinical back issues and mobility challenges as they age. It is my belief that there is a connection between these extremely commons surgeries and the very high incidence of IVDD / back issues and hind limb issues (like ACL problems) in dogs.

DEGENERATIVE JOINT DISEASE / OSTEOARTHRITIS

Osteopathic treatment can help patients with DJD / osteoarthritis cope better with their condition by improving mobility elsewhere in the body. For example, dogs with elbow arthritis can be greatly helped by improving biomechanics of the hind end and back. A totally normal healthy dog bears 60% of body weight towards the front end and a dog with loss of mobility in the sacrum / pelvis / back will shift even more weight to the front end affecting the joints of the front limbs negatively. This approach along with local treatment of the affected joint(s) can also slow down the natural progression of the joint changes.

HIP DYSPLASIA / ELBOW DYSPLASIA
Osteopathy can help dogs with these conditions by optimizing mechanics elsewhere in the body. Osteopathic veterinarians will often use other complementary therapies such as acupuncture, Chinese herbal medicine, homeopathy and nutrition for these more complex and genetically oriented conditions.

Unilateral hip dysplasia that is mild may actually be less genetic and more of a mechanically induced change in the coxofemoral joint. If caught early and is in a milder state, normalization
of mechanics of the sacrum / pelvic area can slow down its progression and in very young dogs (less than 2 years of age) can possibly reverse radiographic changes since growth and remodelling are altered by the positive changes in mechanics and movement in that area.

BACK PAIN / NECK PAIN
Vague back and neck pain can respond so well to osteopathy since the entire body is assessed and treated for any restrictions thus allowing tissues to heal. Neck pain is not always due to a disc issue. The spinal facet joints are richly innervated and can be a source pain due to irritation from mechanical restrictions.

LUMBOSACRAL STENOSIS
Osteopathic treatment involves normalizing biomechanics in the sacrum / pelvic bones and lumbar spine through visceral manipulation (affecting SI movement and upper lumbar spine), joint manipulation and lastly craniosacral therapy to address excessive dural tension in the spinal column and sacrum where the dura is attached to bone. The dura mater is attached only at the cranium, upper cervical area and sacral area so addressing dural torque and strain throughout this system does alleviate mechanical stress due to the stenosis in the L-S area. Results are mixed depending on how advanced the weakness and neurologic deficits are.

VERTEBRAL SPONDYLOSIS
This is a radiographic lesion that is almost considered an ‘incidental’ finding for many veterinarians however to the chiropractor or osteopath it is highly significant. The bone spurs and calcified bridging of ventral spinal ligaments is the attempt of the body to stabilize an area of the spine that is experiencing hyper-mobility or excessive motion. The easiest comparison is the medial buttressing of medial collateral ligaments of the knee with ACL tears and ruptures. The hyper-mobility in one area is a response to hypo-mobility in another adjacent area of the spine and this is where the osteopath can intervene and resolve the hypomobility or restrictions whether by direct treatment of vertebrae or underlying visceral causes.

LICK GRANULOMAS
This is a condition that frustrates most conventional veterinarians but is highly rewarding to treat by veterinary chiropractors and osteopaths. Most front limb lick granulomas are caused by spinal nerve compression in the mid to lower cervical spine with radiculopathic or referred sensations / tingling / pain in the extremity. In just a few treatments (sometimes just one!) the irritation is gone and the dog stops licking the area, allowing the tissue to heal. In some cases treatment needs to be repeated over time so that there is no recurrence. Hind limb granulomas are due to nerve compression in the mid-lower lumbar spine.

WOBBLER SYNDROME
The instability and hyper-mobility in the cervical spine in this condition cannot be alleviated directly however a general osteopathic treatment and addressing any excessive dural tension in the craniosacral system does improve the dog’s mobility and slows down progression of the disease. There is better success in milder cases where the ataxia is not severe.
URINARY INCONTINENCE

In some cases urinary incontinence is caused by irritation of efferent parietal and autonomic nerves coming off the sacral plexus and going to the bladder. The sacrum can be fixated due to past mechanical trauma (slips, falls, other injuries) and is often found in middle aged or older dogs. Dogs that do not respond to hormone treatment for incontinence may be candidates for this mechanical approach.

POST-SURGICAL ADHESIONS

Adhesions are inevitable after any surgery. What veterinarians don’t know is that any kind of surgically-induced excessive fascial tension in the thorax or abdomen is an opportunity for ANS-related fixations in the spine and pelvis. The very long term repercussions of these fixations are possible lameness, pain or mobility issues years later.

CANINE ATHLETES

Osteopathic care is fantastic for addressing any ‘biomechanical unsoundness’ or asymmetries in movement that can affect performance in agility, obedience, conformation and other disciplines. Even more important is that dogs under osteopathic care can be much less prone to injury due to efficient, symmetrical and fluid movement at all times.

GERIATRIC DOGS

The body of an older dog sure has a story to tell! Their bodies have a lot of mileage on them! There are more ‘layers’ of joint / fascial / visceral restrictions to address but it is always rewarding to improve the mobility, flexibility and stamina in older dogs. They do not have to have overt lameness or pain to warrant a treatment; it is a given that they are candidates for osteopathic care.

PREVENTATIVE

Since fascial / joint / visceral restrictions are a fact of life at some level every dog is eventually a candidate for osteopathic care. By treating dogs at a younger age restrictions are caught earlier and therefore some musculoskeletal issues can actually be prevented or delayed until much later.

FREQUENCY OF TREATMENT

This depends on what condition / how chronic / level of current vitality / and the age of patient. Some I see once or twice a year for ‘tune-ups’ and with really geriatric dogs or cats we can keep them going well with monthly treatments. Typically I might see an animal 2 - 3 times a year to maintain improvements. Some issues are completely resolved in 1 - 3 treatments. With canine athletes I may assess and treat them frequently (every few months) to keep them at optimal levels of performance and these sessions are brief and findings can be quite subtle. More and more I am encouraging clients to bring me their young asymptomatic dogs because I know that they probably already have SI joint fixations after spay / neuter surgery. With early care we are likely preventing various musculoskeletal issues much later.

CLINICAL INDICATIONS FOR OSTEOPATHY IN CATS
Since cats are small, light and agile they compensate for orthopedic problems better than dogs. Cats are also notorious for hiding pain and discomfort. As with dogs, osteopathic care in cats is excellent for any musculoskeletal condition such as back or neck pain, lameness, osteoarthritis, lumbosacral joint disease and ACL tears / ruptures. Cats will demonstrate musculoskeletal problems by reduced activity, difficulty with jumping, irritability, licking local joints or areas of the spine, and decreased grooming.

**CLINICAL INDICATIONS FOR HORSES**

Lameness is a common presentation in a generalized equine practice and naturally in a specialized sport horse practice. Based on what is taught in veterinary school the main focus in lameness cases are the joints and soft tissues such as the ligaments and tendons of the lower legs. The focus is also on where the presenting complaint is such as swelling and inflammation. While this kind of ‘local lesion’ is very important to treat if present, more important really is the underlying areas of immobility that lead to wear and tear and eventual tissue changes in that local area. These lower leg issues are often the result of a long cumulative process of biomechanical compensations and adaptations from loss of mobility elsewhere in major structures such as the back, pelvis and shoulder blades. These areas of immobility or ‘osteopathic lesions’ are often remote from the area of the presenting complaint. In fact they can be at the other end of the body. I have seen numerous front end issues resolve when a gelding scar / adhesion or fascial restrictions of the ovaries, uterus and / or bladder were addressed and freed up the pelvis and low back.

Performance issues can completely stump the conventional veterinarians because they do not have the skill set to either find or treat their root causes. The horse is not lame and has no back pain but is ‘not right’ either, this is biomechanical unsoundness. The horse has a problem but diagnostic tests come up normal and the veterinarian cannot give the problem a name. Common performance issues are things such as resistance to bending in one direction, trouble fully engaging the hind end / lack of impulsion, trouble with one canter lead and many other forms of resistance, including many that are labeled as behaviour and ‘attitude’ problems. These are often the beginning of what eventually turns into pathology in a local area such as suspensory ligament problems or arthritic hocks. Restoring mobility to any restricted tissue in the body (joint, connective tissue, spine and organ) frees up the body to self-heal whatever inflammatory or degenerative process is going on, especially if the joint and soft tissue changes are not too severe.

Back pain can be incredibly elusive to treat successfully. Too many times the saddle is blamed when it really is just icing on the cake. Using a combination of chiropractic and acupuncture for chronic back pain I got modest results for many years or was just palliating and needing to treat on a monthly basis, or even more frequently. A long term cure was elusive. Visceral osteopathy changed all that. Restoring mobility to vertebrae relieves joint issues and alleviates back muscle tightness and spasm. But greater than 50% of fixations / subluxations in the thoracolumbar spine come from visceral and autonomic nervous system influences. Once visceral issues are addressed the affected vertebrae then free up all by themselves. This is the ‘magic’ part of osteopathy. It is uncommon now for me to use acupuncture for back pain......after a few
osteopathic sessions the pain is gone (given other influences are addressed via things such as good dentistry and a good fitting saddle). The other joint fixations in the body (50% or less) that do not have visceral root causes are treated locally via manipulations.

FRONT LEG LAMENESS / PERFORMANCE ISSUES

An osteopathic approach helps horses with overt pathologies in soft tissues or joints in terms of helping them cope better through optimum biomechanics of the whole body. The biggest strength of osteopathy is that this system of medicine can detect and solve lameness issues where no obvious pathology was determined. The majority of horses are biomechanically unsound meaning that in several areas of their bodies there is loss of normal range of motion of joints.

In a normal weight bearing situation 60% of the body weight is distributed towards the front end. The forelimb relies on multiple suspension systems that will absorb mechanical forces and shock during daily activities of life and sport. The structures that absorb these forces include the scapula and shoulder joint, hoof wall and bones of foot, carpal bones, fetlock and the connective tissues between the knee and fetlock. There is no clavicle in quadrupeds so the shoulder blades are responsible for all kinds of large coupled motions, combinations of dorsal, ventral, extension, flexion, abduction and adduction.

The gliding motion of the scapular bone on the muscles and fascia of the body wall is considered an ‘osteopathic joint’. When this ‘joint’ loses its full range of motion (coupled motions of extension / flexion, abduction / adduction, dorsal / ventral or upsip / downsrip) there are many repercussions in the rest of the front limb and other structures must take on more shock absorbing duties. Probably the most common area is the area of tendons and ligaments along the cannon bone. Add in the factor that shoes dramatically reduce the shock absorbing capacities in the foot and you have a recipe for heightened stress in the areas of greatest elasticity such as tendons and ligaments. This is where much focus of time and money is spent on lame horses but this is often futile with limited or short term results.

FOUR COMMON CAUSES OF LOSS OF MOTION OF THE SCAPULA

It is beyond the scope of this paper to explain in detail but these causes once addressed result in a much more mobile scapula and this has been born out in thousands of cases.

1: Sacrum-ilium restriction - via the dural membranes of the craniosacral system and the direct reciprocal tension system between the ilium and temporal bone on the same side, and then via the brachiocephalicus muscle from the temporal bone to the medial shoulder area

2: Dental imbalance - issue with lower 11 molars, lack of precise incisor reduction and influences of the hyoid apparatus on the TMJ and thus the temporal bone / brachiocephalicus muscle / shoulder connection

3: Viscera in cranial abdomen

Stomach / duodenum influence mainly the LEFT scapula (via the branch of afferent vagus nerve)

Liver (mechanical and / or functional issues) influences mainly the RIGHT scapula (via branch of afferent vagus nerve)
4: Local biomechanical compensation from the hind end, often on the diagonal
   It is rare to find fully mobile shoulder blades on the first osteopathic visit. And the majority of
   horses have multiple or stacked causes of scapular immobility. Some can be addressed
   immediately….restoring pelvic bone motion through osteopathic treatment (visceral or local
   mechanical issue) immediately restores motion in the scapula on the same side. However other
   causes take more time and other approaches…proper dentistry, treatment of the duodenum /
   stomach with diet changes, neutraceuticals, herbs etc. Once these organs reach a higher state
   of function then via normalization in the autonomic afferents the motion often returns
   automatically to the areas affected by such structures… (scapula, affected thoracic vertebrae).
   If the shoulder area is restricted by all 4 causes together then treating only a few will result in
   only partial freedom in the shoulder area.

HIND LEG LAMENESS / PERFORMANCE ISSUES
   After common pathologies such as arthritis / DJD, soft tissue inflammation and OCD are
   ruled out, the most common causes of lameness or performance issues involving the hind end
   (diagnosed via osteopathic exam) are:
Sacro-iliac joint immobility / pain – the 2 broad categories of root causes are:
- MECHANICAL  (incidence = approx. 50%)
  - local sacral rotation due to micro-trauma (slips, etc.) or a depressed sacrum due to major
    trauma
- VISCERAL  (incidence = approx. 50%)
  - castration scars, ovaries / uterus / bladder / ureter and hindgut acidosis, via the ANS
Lower lumbar fixations (spinal nerve root irritation)
Stifle (entrapment of lateral and / or medial meniscus is the most common)
Hock (fixation of 4th metatarsal or cuboid bone is the most common)

VISCERAL ‘DIAGNOSIS’ VIA PALPATION OF THE THORACOLUMBAR SPINE
   I call the thoracolumbar spinal area ‘organ land’ as it provides a treasure trove of
   information about what may be going on internally for horses and what may be at the root of
   some of their most basic musculoskeletal problems and offers a real advantage in solving
   problems in horses.

   A ‘3 in a row spinal pattern of fixation’ is a visceral pattern. The only caveat here is that all
   three vertebrae are fixated in a side-bending pattern laterally to the SAME side and all 3 must
   be in extension or all in flexion. Otherwise there are local fixations patterns and are not caused
   by autonomic nerve changes from organs. This is why competence in motion palpation of the
   spine is critical as well as being accurate in knowing which vertebrae one is palpating.

This system is not 100% perfect due to a complex autonomic nervous system and visceral
osteopathy continues to evolve, however to date what has been discovered has been validated
in the treatment of thousands of horses.

Examples of common visceral spinal patterns:

<table>
<thead>
<tr>
<th>Th 7 – 10</th>
<th>liver</th>
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<tbody>
<tr>
<td>Th 12 – 14</td>
<td>stomach</td>
</tr>
<tr>
<td>Th 13 – 15</td>
<td>duodenum</td>
</tr>
<tr>
<td>Th 15 – 17</td>
<td>adrenal glands</td>
</tr>
<tr>
<td>Th 17, 18, L 1</td>
<td>kidney(s), ureter</td>
</tr>
</tbody>
</table>
If I find any kind of sacro-ilial restriction (which is most of the time) then the next question I always ask is....is this a local mechanical problem or is there a root visceral cause? Spinal patterns in the thoracolumbar area tell me the answer to that question.

If experienced veterinary chiropractors rechecked some of their manipulations right at the end of their treatment they would find that a percentage of their adjustments actually did not hold for very long. These are osseous ‘blocks’ due to a root visceral cause and will never hold adjustments no matter how often they are done. This is knowledge that is missing in veterinary chiropractic training. What they do not know is that by addressing the root visceral cause (whether right away if it is a mechanical adhesion or over time addressing organ function) that the fixations in the musculoskeletal system go away on their own without the need for direct manipulation.

**TOP VISCERAL ISSUES THAT CONTRIBUTE TO LAMENESS AND / OR PERFORMANCE ISSUES**

**Pelvic viscera** (incidence = 50+ %) (Lumbars 1-3 (also TH 17 / 18 if ureter is involved), and fixation of one or both sacro-iliac joints

- **Geldings - gelding scar / spermatic cord adhesion**, or ureter or bladder issues
- **Mares – mechanical restrictions in ovaries and/or uterus and/or ureter and/or bladder** (fascial restrictions of mesovarium / mesosalpinx / mesometrium and ligaments of the bladder)

**Stomach / Duodenum** (incidence = 60+ %)

- **Stomach** (T 12 – 14 fixations, and often left scapular downslip / fixation)
  - most commonly would be an ulcer (osteopathic exam does not ensure there is an ulcer, only that the autonomic nervous system is saying there is something wrong or ‘off’ with that organ).

- **Duodenum** (T 13 – 15 fixations and often left scapular downslip / fixation)
  - this could be an ulcer but can also be an impairment of digestive enzymes arriving in the duodenum from the pancreas and an overall disturbance of duodenal function and nutrient absorption.

**Worm aneurysm / Verminous Arteritis**

- larvae of the large strongyle worm spend part of their life cycle in the cranial mesenteric artery inciting inflammation and fibrosis and thus changes to the ANS (sympathetic chain and ganglia)

- causes fixations in side-bending and extension to the right from Lumbar 6 to Thoracic 12).....these are the horses with obvious **right-sided stiffness** in the entire back and not wanting to bend to the right compared to the left.....**this is a unique ‘13-in-a-row’ spinal pattern**

- there is a huge spectrum to this very common but insidious problem...everything from the high level sport horse with mild right-sided back stiffness to the very obvious parasitized horse with a pot belly and a back that feels like ‘cement’ on the right side.....routine deworming is not often enough to adequately deal with this problem, many cases are ‘subclinical’ and only detected via osteopathic exam (or trans-rectal ultrasound of the artery wall), fecal tests can be
negative despite presence of larvae in the arterial system

**Hindgut acidosis** (clinical / sub-clinical) (incidence = 50+ %)
- Lumbars 4 – 6 fixated, right sacro-iliac joint fixation.....right side possibly more common due to location of cecum on the right side? I have not seen lameness cases with this issue but for sure it can affect their comfort in athletic endeavours and cause poor performance... with these cases you will also see a distended abdomen (not related to parasites or obesity), the diaphragm and 18th rib further caudal than normal, repetitive rings or grooves on the hooves and increased warmth in the coronet area on all four feet.
- Large quantities of grain or fructan-rich grasses pass through as undigested starch to the large intestine, the starch is rapidly fermented causing elevated levels of lactic acid (so pH drops below 6.5)....this increased hindgut acidity kills beneficial fibre-digesting bacteria allowing pathogenic bacteria to proliferate
- in the wild, horses eat up to 16 hours a day and walk 10 miles a day, a domestic horse has a vastly different lifestyle and constant negative challenges to the gastrointestinal system
- hindgut acidosis can also be a precursor to colic or colonic ulcers the latter which goes vastly undiagnosed in conventional medicine...many horses with colonic ulcers also have gastric ulcers

**Clinical signs or performance issues indicating presence of musculoskeletal problems:**
- back or neck pain, ‘cold-backed’ horses
- short-striding
- stumbling, tripping
- head-shaking
- problems on one lead
- resistance to bending laterally
- difficulty with collection
- difficulty maintaining impulsion
- need for training aids
- bucking, rearing
- trouble fully engaging hind end
- lots of warm up needed
- refusing jumps
- not standing squarely on all four limbs
- tail is held off to one side
- generalized stiffness
- lameness unresponsive to conventional approach, or vague lameness
- temporary results with chiropractic, massage or acupuncture treatments

**Reproductive issues:** infertility, inability to maintain a pregnancy, excessive or absent heat cycles

**Digestive issues:** diarrhea, recurrent colic, weight loss

**FREQUENCY OF TREATMENT**
- Frequency depends on what condition / how chronic / level of current vitality / and age of
the patient. Typically I might see a horse 2 - 4 times a year to maintain improvements. Some issues are completely resolved in 1 - 3 treatments. With sport horses I may assess and treat them frequently (every few months) to keep them at optimal levels of performance and these sessions are often more subtle. With osteopathy a lot of bigger visceral issues are resolved early on (they do not recur since the root issue was often addressed) therefore with repetition what is found and treated are more and more refined levels of problems. The goal is to always have the sport horse reach its full potential and then stay at that higher level. Also examining sport horses frequently (every few months) provides the opportunity to find common subclinical organ issues that may be starting up such as gastric ulcers, duodenal issues or subclinical hindgut acidosis.

Treatment of gastric ulcers can be done conventionally with products like omeprazole however there are numerous holistically-oriented approaches that can work also such as green clay, papaya extract (Stomach Soother), SUCCEED supplement and combinations with aloe, among many others.

Treatment of the duodenum and that area of the upper small intestine (remember that we have the spinal findings to confirm an issue there) has presented more of a challenge since treatments for stomach ulcers often do not resolve duodenal issues. The current thought is that it is more of a digestive enzyme imbalance (rather than an ulcer) and there have been some good results when using very high quality supplemental digestive enzymes (e.g. RiteZyme).

Treatment of subclinical hindgut acidosis is done using a buffer such as EquiShure, restoring hindgut flora with prebiotics / yeast / probiotics and lowering grain consumption. The other logical approach is to correct upper small intestine imbalances (often present along with hindgut issues) with digestive enzymes and thus ensuring that starches and sugars get properly assimilated in the upper gut and do not arrive at the hindgut undigested and thus lowering the pH of the large intestine. This approach has been very successful with cases suspected of having colonic ulcers (girthiness, abdominal pain and distension without colic signs).

REFERENCES
Please see References for Lecture #1 ‘PRINCIPLES OF VETERINARY OSTEOPATHY’

ADDENDUM:

DEWORMING PROTOCOL FOR VERMINOUS ARTERITIS

1: 5 days of PANACUR (10% fenbendazole) at DOUBLE the regular dose (once daily)
   - Pancur Liquid / Suspension - 50 ml (cc) once daily for 5 days (orally or on feed)
   - or if using 20% generic fenbendazole then the dose is 25 ml per day for 5 days
   (above are the already doubled doses for a 900 to 1100 pound horse)
2: Wait 7 days.
3: Repeat step #1.
4: Wait 7 days.
5: Give single dose of QUEST
6: Wait 10 days.
7: Give single dose of QUEST (or QUEST PLUS if not dewormed for tapeworms recently)
8: A full osteopathic treatment can be given, (at the earliest), a week after the 2nd Quest dose
Note: I have done this protocol on over 1000 horses in my practice over 6 years with no real negative issues. All horses have had a myriad of improvements in health and performance due to the unique strategic use this protocol. Contraindications for this protocol include very young horses (less than 1 year) or very geriatric horses (late 20’s or older), an underweight horse or a horse that has had obvious negative reactions to chemical dewormers. Horses that are suspected of having severe colonic ulcers are initially treated for that (hindgut buffer, digestive enzymes and prebiotics / yeast) and then the deworming is done once they have clinically improved.

If veterinarians want more information about this protocol, including testimonials, the author can be reached at holisticvet@shaw.ca