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The Low Down on Apoquel and Cytopoint: Making Sense of Allergy Control Medications

Trish Ashley DeVore, DVM, DACVD
Oregon Veterinary Medication Association Conference, Mar 2018

Key Point #1: Allergies are NOT the result of an underactive or suppressed immune system, but a trigger-happy one. Allergies are (probably a genetically-based) abnormal response to "normal" proteins / agents. E.g., chicken meat, cedar pollen, grass pollens are not pathogens (not expected to cause disease), but there are plenty of pets and people who react to these things. This is an "abnormal" response to a "normal" item present in our daily life.

More in-depth details: The exact pathogenesis of allergies, how and why they get started, is not 100% known and likely there are different pathways for different individuals. However, a key player in allergies is the production of *IgE*, an immunoglobulin or antibody, to an allergen. The involvement of IgE in the immune response triggers a cascade of events that leads to allergy symptoms (redness, itch, swelling, altered skin barrier, etc.). For example, the dog who has an allergic reaction to a vaccine has made IgE against the vaccine after a previous vaccinations. With subsequent vaccination, the IgE attaches to mast cells in the skin, the mast cells degranulate (explode) and release histamine and other cell proteins causing the allergic symptoms (hives, facial swelling, dilated blood vessels and red skin). We also believe part of the pathogenesis of allergies is an imbalance in the regulation / production of T-lymphocyte helper cells, an imbalance that favors forming lymphocytes that promote excessive and perpetual inflammation.

Key Point #1 Summary: In simplistic but relatable terms, chronic allergies are basically a trigger happy immune system that is stuck in a traffic circle firing at an armored car in front of it. And *to control allergies, we have either avoid the allergens (don't be exposed to other cars), calm down / suppress the immune system (take away the gun), or try to divert the immune system* (give it something else to do – get out of the traffic circle).

Key Point #2: Allergy "testing" is too inaccurate to use it to tell us what to avoid. The major allergies are food, flea and environmental triggers. Skin and blood tests for food allergies are very inaccurate, and skin / blood tests for environmental and flea allergy are, at best, moderately accurate. In an allergy work up, we start by "controlling what we can control" (food, flea) and see how much that helps. If at least one (preferably 2) different food trials have failed to help, we resign ourselves to the diagnosis of environmental allergies (also called **atopic dermatitis**; allergies to pollens, house dust mites, molds, etc.). Once we have this diagnosis, we have committed this pet to a life of management of allergies because avoidance is almost impossible. The best hope is to minimize long term medication use is to do *desensitization*, or *allergen immunotherapy (see notes below)*.

More in-depth details: Details of food trial are beyond the scope of this talk, but in short, there is no one perfect food; we have to consider previous diets, owner preference, need for treats, canned food. Owners need detailed instructions and encouragement during food trials. And many pets have both food and environmental allergies, such that we see only partial improvement during a food trial, complicating our assessment.

More in-depth details: *Allergen immunotherapy* ("AIT"; aka allergy shots, allergy vaccine, allergy serum) is giving to a pet, in a controlled and graduated fashion, the allergens to which they are allergic, trying to retrain their immune system, just as is done for people. We are not sure how this works, maybe by turning off IgE production, or altering the types of T-helper cells produced by the immune system, or maybe creates a diversion for the immune system.

Key Point #2 Summary: Diagnosis of allergies (food vs environmental vs flea) is a process of elimination. Avoidance of environmental allergens is almost impossible. Allergy testing is done to help us do allergen immunotherapy.

Key Point #3. Allergen immunotherapy (AIT) can be technically challenging, takes a dedicated pet parent, and often takes months (if not more than a year) to work. About 65% of pets started on AIT respond. Response rate is lower for pets with years and years of allergies. AIT is indicated for the relatively young (< 7-8 yrs of age) and cooperative pet. *All the negatives aside, AIT is the best chance of controlling allergy symptoms yet minimizing use of medications.*

More in-depth details: AIT involves either giving SQ injections 1-3 x /week or oral drops twice day. Not all pet owners have the skills or patience for this. Oral administration is usually easier for most owners, except for (most) cats.

So, for pets who do not improve with diet changes and flea control (which is >50% of our allergy patients), we have to find a way to control the pets' symptoms. Owners would like control without medications, which means AIT, but this is not right for everyone. For years, all we had were steroids and Atopica (cyclosporine), both with their pros and cons. In the last 5 years, we have 2 new exciting medications (Apoquel and Cytopoint) to help control canine allergic dermatitis. What do you as a technician need to know about these medications (how fast they work, how they are given, potential side effects, costs, etc.)?

Apoquel (oclacitinib):
This is a unique immune suppressive medication (a janus kinas inhibitor) used for control of allergy symptoms. It affects less of the immune system than prednisone, more than cyclosporine, more than Cytopoint. It works by affecting the production and function of some cytokines (chemical signals between immune cells).

Onset: Rapid onset, decreasing itch in 24 hrs or less. Effectiveness: 95% of dogs respond to BID Apoquel; 75% to once a day.

Route of administration: Oral pill; usually easy to give; give twice a day for up to 2 weeks, then DECREASE to once a day. Note: Apoquel should NOT be given BID long term.

Side effects: Uncommon unless given BID long term, which can be very immune suppressive. May increase predisposition to viral papillomas, some skin growths (histiocytomas). Question of association with cancer development is unproven / no association recognized at this time. Occasionally see behavior changes (increased aggression).

Okay for long term use? Yes, but not our "soundest medical advice" for a young dog

Cost: Variable – not cheap; mostly over $50 / mon. All 3 tab sizes cost the same but some dogs need a half of one size

No-Nos: Not labeled for dogs < 1yr of age, pregnant or lactating dogs; cautionary use if history of neoplasia or ongoing serious infection such as pneumonia. Do not use ID long term unless no other option.

What about cats? Not labeled for cats but there are several case reports using it in cats. They do not respond as well as dogs; often need a higher dose and more often. We have no long term safety data on its use in cats.

Cytopoint (lokivetmab):
This is a monoclonal antibody against IL (interleukin)-31. IL-31 is a cytokine whose only function, to our knowledge, is to generate itch and inflammation. The function of Cytopoint, an antibody to IL-31, is to bind or

lock up all the IL-31 in the dog's body. Cytopoint is considered an "immune modulator", *not* an immune suppressive therapy.

Onset effectiveness: Rapid, usually decreased itch in 24-48 hrs and works for, on average, 4 weeks (some longer some less). ~60-70% response rate

Route of administration: subcutaneous injection. Warm to at least room temp before giving! (stings when cold)

Side effects: No predictable side effects; possible to have an allergic reaction to it; it is a protein. We watch our patients for 10 min after administration until they've had 5 injections.

Okay for long term use? Yes. Labeled to give up to every 4 weeks. Okay for dogs less than 1 year of age. Has not been testing in pregnant or lactating dogs

Cost: Depends upon body size; one injection in a dog over 70 lb may be $100 or more. Less expensive than Apoquel for small dogs.

What about cats? NO!! Dogs only. This is a DOG antibody; cats will develop a reaction to it.

In review / in comparison:
Corticosteroids (prednisone, prednisolone, dexamethasone, Depomedrol, etc):

- Onset / effectiveness: rapid; helping with 24-48 hrs. 90+% response rate
- Route of administration: Various (pills, liquids, injections, topical, transdermal)
- Side effects: Numerous. Short term: pu/pd, polyphagia, panting, behavior changes. Long term: "breaks the body down faster than it builds itself up" Can cause diabetes in cats
- OK for long term use? Not without at least some side effects – less at lower doses and most important to give every other day or less.
- Cost: cheap financially, but not physiologically
- Comments: Steroids have their place, such as in conjunction with other meds. And many owners are wise to steroids and don't want to use them or use them very much

Atopica (cyclosporine):
An immune suppressive medication (dampens the function of lymphocytes, alters cytokine production and function). It helps control foreign body rejection; used to prevent transplant rejection. It is a little more suppressive than Apoquel, less than prednisone (but dose-dependent). It is used in dogs and cats.

- Onset / effectiveness: Takes 2 to 4 weeks to work; ~80% response rate
- Route of administration: oral caps or liquid (injectable available but costly, can sting, and can cause abscesses)
- Side effects: GI upset (in 20-25%); gingival thickening; increased hair growth; increased susceptibility to some unusual infections. Fewer side effects than steroids, in general.
- Okay for long term use? Yes, if tolerated but like other meds, not ideal for the young patient as a long term plan
- No-No's: pets should NOT be on a raw diet and taking Atopica; use with caution in pets with history of neoplasia
- Cost: "not cheap" – Elanco has a rebate for repeat purchases; generic on human side less expensive (still not cheap).

If I have to use a medication long term for environmental allergy control, what would I use?
As is so often the case in veterinary medicine, "it depends." But on the whole, my best medical advice:

For the young dog: I would start allergen immunotherapy (ideally based on allergy testing). While waiting for that to work, I would start with Cytopoint because it has the least effect on the immune system and fewest potential side effects. If Cytopoint does not help, I would try Apoquel. If that fails, I reach for cyclosporine. If that fails, they should see a dermatologist! (All this is in conjunction with regular bathing, giving fish oils, using regular flea control, etc.) Our best controlled patients are ones on a combination of therapies (the *multi-modal approach*).

For the middle-aged to older dog with chronic allergies: I tend *not* to encourage allergy testing and immunotherapy but skip to the medications in the order list above.

Even though neither medication highlighted in this talk is labeled for cats, I will offer an opinion here: For younger cats, like dogs, I encourage allergy testing and immunotherapy. While waiting for this to work, my first choice is cyclosporine, if tolerated and the cat can be medicated orally. If this fails, I think the potential side effects of long term steroid use and the unknowns of Apoquel use in cats are about equal risks. I would use my judgment and educate the owner on pros and cons of both. If we elect Apoquel use, I do recommend monitoring blood work in 8 weeks and every 4-6 months thereafter if normal.

Resources:
https://www.zoetisus.com/conditions/dogs/itchcycle/home.aspx
https://www.apoqueldogs.com
http://www.cytopoint4dogs.com

What Does a Dermatologist Do Anyway?
When a Pet Should See a Dermatologist

Trish Ashley DeVore, DVM, ACVD
Oregon Veterinary Conference, March 2018

Why am I talking about this? Because I've never heard a client say to me, "I'm so mad at my veterinarian for referring me to a dermatologist." But I've heard on numerous occasions a frustrated client express, "I'm so mad at my veterinarian for not sending us to a dermatologist sooner." And clients spend as much or more time talking to technicians and support staff than they do to the doctors. How can you help keep your clinic's clients happier and less frustrated?

Background: What Is It Like Dealing With Skin Problem?

Skin / Ear Problems Are Common
A Veterinary Pet Insurance 2012 database survey of almost 500,000 insured pets found the top diagnoses reported in the dog were skin allergies, ear infection and skin infection. Itching (pruritus) is among the most frequent complaints of pet owners, affecting roughly 1 in 6 dogs that seek veterinary help.[1]

Skin Problems: As Frustrating As They Are Common
Most dermatologic problems (such as environmental allergies, aka, atopic dermatitis or AD) are not curable, but need to be managed long term. This is frustrating for owners, vets and veterinary staff. A dermatology referral clinic in France asked owners of dogs diagnosed with AD questions about their pets' skin treatments and experiences prior to be referred to a dermatologist. Two of the main complaints were based in frustration: lack of a cure and the emotional impact of the disease.[2]

Owners, out of efforts to save money on vet visits, try changing diets without veterinary guidance, try unproven but well-advertised "therapies" such as supplements and herbal remedies or totally invalid commercial "allergy tests." Often by the time they bring a pet to their veterinarian, they've spent more on these things than the cost of an exam, and they arrive in the exam room already frustrated and financially tapped.

Adding to the Frustration: Many Skin Problems Look Alike
For example, the symptoms of food allergy and environmental allergies can look exactly alike. Sometimes fungal infections look like immune-mediated diseases. And we lack simple and accurate tests for the most common problem, allergies. This is not only frustrating for everyone who wants a quick and easy diagnosis, it leads to a higher likelihood of a wrong initial diagnosis. This is not (usually) the fault of the clinician but just a fact of dealing with dermatitis. When a pet fails to respond to a prescribed treatment, the owner gets frustrated, loses confidence in the veterinarian, and seeks another opinion. Often we fail to explain to the owner how often skin diseases look alike and special diagnostics are needed to determine the problem.

Chronic Disease Means Chronic Medications
Many pet owners are not satisfied to keep their pets on medications long term without being convinced of the need to do so. For many this means exploring other options, even the likelihood of success is relatively low, to assure themselves they have done the best they can for their pet. And medications are expensive, another stress for pet owners.

We Humans Need a Plan
It is important that we convey to pet owners that in dermatology cases we often have to take an approach of ruling out one thing at a time, make our best assessment, assess the pet's response, and that *we have a plan* if the pet does not respond to our treatment. The study from France cited 44% of pet owners complained of "absence of clear treatment decision and / or plan" by their primary care vets.[2] A study published in a human psychology

journal in 2011 suggested that having a plan toward meeting a goal may "free cognitive resources" (aka brain space!) "for other pursuits" as well as help reach the goal. In short, we feel and function better when we have a plan.[3] But, we (veterinary professionals) are not always good at expressing this to our clients. Often we lack the *time* in the appointment to explain all that we would like our clients to know.

Are General Practitioners Adept at Treating Skin Problems?
The primary care veterinarian is absolutely the first place a pet owner should turn if their pet has skin problems. Primary care vets can address many issues, assess for mites, infection, check for fleas, give guidance on diet management. As discussed, many pets have chronic problems that will bring them back repeatedly. And after 3 to 4 visits for the same problem without a long term plan or discussing the reality of chronic skin problems, pet parents become frustrated, even if their doctor is very competent. This is more problematic in multi-doctor clinics where clients see different doctors.

Educating pet owners about allergies takes time, usually more than the average 15-20 minute appointment can accommodate. Some primary care vets make skin appointments 30-45 minutes long.

Another concern is that dermatology has become an elective in some veterinary schools. Six of the 30 schools in the country have no full-time dermatologist, so these students do not have a dermatology rotation in their clinical year unless taken as an elective. I have personal knowledge of veterinarians who feel inadequately trained in dermatology after graduating from school; they have not had experience with the cases nor with the "human side" of dermatology cases and are frequently frustrated or lack confidence in recommendations. Even clinicians who had good dermatology exposure in school find it very frustrating. Heck, dermatologists are frustrated by dermatology cases!

What Can a Dermatologist Offer That a General Practitioner Cannot?
1) Greater knowledge base: A veterinary dermatologist is a veterinarian who has had specific training (a 2-3 year long residency) and experience in the diagnosis and treatment of animal skin, ear, hair, nail, hoof and mouth disorders. Specifically veterinary dermatologists have significant training and experience in the management of allergic skin disease.[4] Dermatologists may have the insight or experience to look at a pet and more confidently diagnose a problem or have more knowledge about the appropriate diagnostic tests (such as culture or biopsy).

2) We know *when* to do, *how* to do, and how to *interpret* allergy tests, both intradermal and blood tests. Blood allergy tests are accessible to primary care clinicians; skin testing is most often done by dermatologists, taking some special skill, access to testing allergens, and knowledge of the allergens. We believe the skin test is somewhat more accurate than the blood tests. Knowing *when* to do the test is also important (for example, doing a food trial first), as well as how to interpret the results (for example, how inaccurate food allergy test results are; what does it mean if there is a reaction to something not in the pet's environment.)

3) We have experience with managing allergen immunotherapy (AIT), the reason for doing allergy testing. Dermatologists are a little more familiar with the allergens, what is present when, which ones cross-react, etc., to help formulate the AIT. Immunotherapy is not a cookie cutter or "one size fits all" treatment. Not only do pet's have different reactions, each immune system responds differently to AIT and it must be adjusted based on the pet's response. This means regular communications with pet owners (not just visits but phone updates, emails) and having at least one support staff member (usually a technician) with knowledge and experience to answer questions for the doctor.

4) Expertise with biopsies: Some skin conditions require biopsy and histopathology for diagnosis. Like allergy testing, when to do this, what sample to take, how to take it, and to whom to send for histopathology are all important factors in getting a diagnosis and therefore appropriate treatment.

5) Otitis cases: Most cases of recurrent ear infections are from underlying allergies. Sometimes ear infections are the only symptom, and it is so tempting to just treat the ear problems as they occur. But after time, the infections worsen and the clients begin to wonder why are they seeing their vet to do the same thing over and over? A dermatologist will take time to discuss with the client the possible underlying causes of the infections and how to address them. Many dermatologists have video-otoscopes for more thorough evaluation of the ear canal.

6) Time: Because of the complexity and often long history of derm cases, dermatologist often allot 1 to 1.5 hrs for the new appointments to listen to the clients, take a thorough history and evaluate the pet. Clients are appreciative of the time given to them.

7) Initials behind the name. It's silly. Specialists are not necessarily smarter than any other veterinarian, but when clients hear advice from a board-certified specialist, they are more likely to trust our assessment and hopefully comply with recommendations.

What are Signs That a Pet with Skin Problems Should Be Referred?
1) *The pet has been seen for same problem 3 or more times ("gets better but it comes back") within 12-18 months without a diagnostic plan*. As technicians checking in pets for exams and discussing prescribed medications with clients, you are knowledgeable and in a position to look at the pet's chart for similar previous visits, and to listen to what the clients says about their pet's issues. Most clients are going to begin to feel some frustration by the 3rd visit if there is no plan to find a cause.

If you notice signs of rejection of treatment recommendations or frustration from the client such as eye rolling, heavy sighs, clenched fists, even light-hearted humor at their misfortune of having a pet with allergies, this is a clue they are ready for someone to take charge of getting long term comfort for their pet.

2) *The pet is not responding to appropriate treatment*. This goes back to the "many skin diseases look alike." We've guessed the incorrect diagnosis and the pet is not getting better or worsening. At a minimum these pets need to see the primary care vet again. The doctor may feel it's time for biopsy or other diagnostics; they may want to refer for this. Sometimes it is time for a fresh set of eyes on the case.

3) *A change in the skin condition / rapidly progressing dermatitis*. A significant change in a skin problem (for the worse) is alarming to both pet owners and veterinarians. It can be a sign of a very serious disorder and appropriate tests done in a timely manner can be critical.

4) *When the client is saying "I've tried everything I can think of…I've done everything the doctor has said to do… I've looked up allergies on the internet…. "* Clients (we humans in general) can drive ourselves absolutely crazy trying to fix our own problems. But when we lack professional oversite, there is always a nag in our heads "am I thinking of everything? What about this? What about that?" These clients benefit much from talking with a specialist so they have a more confident plan and education about their pet's skin problems.

How and to Whom Do You Suggest Referral Diplomatically?
When you suspect a pet with recurrent skin / ear problems would benefit from referral, it is helpful to briefly summarize the pet's visits for the doctor before they see the patient to point out the recurring nature of the problem, and to give the doctor a head's up to any client frustrations. This is best expressed as "our client is a little frustrated with *the problem* (not us) and might feel better if we or a dermatologist discusses the possible underlying problems in greater depth." And even if referral is not recommend or not accepted, you've done the pet, the client and the vet a great service by summarizing what has been done so far and encouraged taking time to investigate the issues.

When talking to clients on the phone / between their appointments, it may be acceptable to say, "Gosh, I realize you are concerned and frustrated (or whatever emotion the client is feeling); maybe we should talk to the doctor

about a more extended appointment time to investigate the underlying problem more thoroughly, or possibly if Rosco should see a dermatologist."

If there is a take-away message to pass along, it is that we've never heard a client say they were unhappy their vet recommended a referral, but many times we've heard clients complain they were not referred sooner, that they found specialty care by doing an internet search. Let the client know you have their and their pets' health as your top priority.

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4. ACVD (American College of Veterinary Dermatology) Website: http://www.acvd.org. This website has information about the types of problems dermatologist see, has some before and after photos of some common conditions, and contact information for dermatologists around the country.

Dermatology Procedures: The What, How, When, and Why

Trish Ashley DeVore, DVM, DACVD
Oregon Veterinary Conference March, 2018

Introduction

The nice part about dermatology is the body organ of interest is on the surface; no need for special imaging to examine it beyond good light and good magnification. The down side, as mentioned in other talks, is many skin conditions of differing causes look alike on the outside, making special diagnostic tests necessary. This is a discussion of common dermatologic diagnostic procedures, when, why and how they are done as well as the appearance of some findings.

Skin Scrapes – Deep: Deep skin scrapes are done (usually) to look for demodex mites, the follicular mite. Demodex mites cause some variation of hair loss, red skin, infection (bumps, papules, pustules) and maybe itch. The mites, when present and causing dermatitis, are usually easy to find with properly done scrapes. We are seeing fewer cases of demodex due to the use of isoxazoline parasiticides (Nexgard, Bravecto, Simparica), which often kill demodex mites. Note: these products are not labeled for the treatment of demodicosis but we have multiple case reports of their efficacy.

Supplies needed: mineral oil, microscope slide, microscope, cooperative patient.

How: Put a healthy drop of mineral oil on a microscope slide. In area in question on the dog or cat, the skin is gently but firmly squeezed for 2-3 seconds, 1 to 2 times. Then let the skin return to its normal position. Apply a small amount of mineral oil on the squeezed area. Use a dull, clean scalpel bladed to scrape in short strokes across the surface of the skin with slight pressure until pin-point bleeding is seen. Keep the edge of the blade *perpendicular* to the skin to avoid cutting it. The material accumulating on the blade is wiped onto the awaiting microscope slide with mineral oil. Apply a coverslip. Examine the collection on 10X magnification with the condenser at its lowest position.

Skin scrapes – Superficial: Superficial scrapes are done to look for itch-causing, surface burrowing mites such as Scabies and Cheyletiella. These mites are often very hard to find; just a few mites can cause intense itch. The burrowing mites are also killed by the isoxazoline parasite control products.

Supplies needed: same as for deep scraping.

How: Where deep skin scrapes focus on a small area deeply, superficial scrapes examine a broad area of skin just on the surface. Apply a healthy amount of mineral oil to the area to be examined (usually the belly, outer ear flap / pinna, lateral elbows) and using a dulled scalpel blade helped perpendicular to the skin, collect skin cells with broad sweeping strokes. Apply the collection to a microscope slide that has a dollop of oil on it already. Examine the material at 10X magnification with the condenser at its lowest position.

Trichogram: This is microscopic evaluation of hair shafts. It is done by gently (usually with fingers) plucking hairs from area of skin in question, place them in mineral or immersion oil, cover with a coverslip on a microscope slide, and examined at 4 and 10x magnification, sometimes 40X. We may be assessing what stages of growth the hairs are in, looking for parasites (demodex around the "follicle" end), or looking for fungal spores of ringworm. This is a nice alternative to trying to scrape the face of a very wiggly puppy and a better way to find demodex in thickened skin (such as feet).

Wood's Lamp Exam: This is a type of black light emitting ultraviolet light used to look for fungal infection (dermatophytosis or ringworm). This test for fungal infection is easy but not very sensitive. Should be performed in a darkened room; move patient to a darker room if necessary. Allow the lamp to warm up for 30-60 seconds

and allow your eyes to adjust to the darkness. Shine the light over areas of skin in question and look for apple-green fluorescence of the HAIR shafts (NOT scale). When this is seen, we are virtually 100% confident this is a fungal infection; if it is negative, we have not ruled out a fungal infection.

Fungal Culture: Fungal culture is the best test for fungal infection (aka ringworm or dermatophytosis). If hairs glow on Wood's lamp exam, these are the best to submit. *Gently wipe area in question with rubbing alcohol* to remove environmental molds. Use (ideally) autoclaved hemostats and pull the hairs in question, and either place on your DTM plate or place in sterile container such as a blood tube and submit to laboratory.

If no hairs fluoresce under the lamp but the pet has suspected lesions, the best way to sample is using a new *toothbrush*. Again wipe the affected areas with alcohol. Brush the affected areas for 15 secs / several brushings. If your clinic does fungal culturing in-house, remove the hairs from the toothbrush using a sterile hemostat and place on the media and also touch the brush gently to the surface of the media. Otherwise, send the whole toothbrush to the micro lab.

If the cat (or dog) has no lesions at all, use a toothbrush and comb the whole body, especially focusing on areas where ringworm likes to be, namely the face, ears, head and feet. Process hairs as described above.

In-house fungal cultures should be evaluated daily for color change and growth; a positive results is when color change happens first or at the same time as growth of hyphae. Environmental contaminants will eventually turn change the color of the media, but not until they've been growing for several days.

Ear Cytology: This should be done on all cases of otitis to identify any parasite (Otodectes), bacterial or yeast infection so we can tailor the treatment. Before collecting ear swabs, you need to know if the doctor is looking for mites, for yeast / bacteria, or both. How the swabs are processed differs depending what you are looking for. In either case when doing swabs from canals, I prefer to use "Q-tips," not the long wooden sticks with a minimalist's amount of cotton on then end. They hurt.

For Ear Mites: Best collection done by "wetting" Q-Tip with mineral oil first before swabbing the canal for debris. Insert Q-tip into vertical ear canal, gently rub the swab on the canal walls. Roll the collected material in a healthy drop of mineral oil on a microscope slide. Examine using low magnification (4x) and condenser at lowest point – mites are easy to find; large and active.

Ear Cytology for Yeast and Bacteria: Insert Q-tip into vertical ear canal to where the canal bends, gently rub the swab on the canal walls. Gently ROLL (not smearing) the collected material on to a dry microscope slide. I use one slide and make and "R" with the swab from the right ear, "L" with the left so I only have to stain one slide.

Fixing and staining: If the swab is very waxy, it is good to GENTLY heat fix. NO ROASTING NECESSARY! Just gently heat the underside of the slide with a flame from a lighter. If the debris is purulent, do NOT heat fix. If in doubt, don't heat fix. Stain with Diff Quik as you would normally (> 30 sec in each jar). Allow to dry and examine first under low magnification to find a representative area, then progress to oil immersion to identify yeast / bacteria / inflammatory cells.

What if the doc wants to look for mites and bacteria / yeast? Easy peasy: take swab using dry Q-Tips; make your cytology slide for staining first. Then roll the swabs in a healthy amount of mineral oil on another slide.

Skin Cytology: Cytology of the skin is a non-invasive way to give clinicians useful information about their patients. We can detect various bacterial infections, yeast infection / overgrowth, and sometimes immune mediated disorders. How we do a skin cytology depends on the type skin lesions present.

Surface Skin Cytology: Tape Cytology: This is the most common skin cytology done in derm practices, usually looking for yeast or bacterial infection on the surface of red, scaly, or thickened skin. It is a great way to get interdigital cytology. Most often used on allergy patients to determine their secondary infections. Tape cytology is not good for very moist areas such as wet lip folds or oozy, ulcerated skin.

Supplies needed: roll of clear acetate tape (not invisible but clear, such as Scotch Tape), ½ to ¾" wide, microscope slide, and the second 2 stains of the Diff Quik set.

How: Tear off a piece of tape that is not longer than your microscope slide (2" is plenty). Handle the tape by the end, trying to avoid a big thumb print and human skin cells in the middle of the tape. Gently press the adhesive side of the tape to the area(s) of skin in question and remove. After I've taken my sample, I attached on end of the tape to an end of a microscope slide, just to use the slide as a way of holding the tape for transport to the lab.

Staining: There are 2 ways to do this. One is the "quick and easy but not as pretty" when you are rushed; the other "a more thorough look at the borough." Regardless of technique: Do NOT put the tape in the fixative jar of Diff Quik (the first, clear). The specimen is already "fixed" to the adhesive of the tape.

1) Quick and easy: Put 1-2 drops of the blue stain (3rd Diff Quik jar) on the microscope slide, then place the slide adhesive-side down on the slide. Gently blot with a paper towel to remove excess stain. Ready to read.
2) More thorough: You are going to dip the tape in the orange and blue Diff Quik and rinse with tap water. Again, do NOT put in the fixative. To spare you from stained fingers, use a clothespin to grasp the tape as you dip it in the individual stains. You can also use the clothespin to hold the tape on the end of the microscope slide as you stain it. After the blue stain, rinse the tape gently with tap water, then put it adhesive-side down on the slide, blot with paper towel to remove excess liquid, and it is ready to read.

Reading tape cytologies: Not easy! These are "busy" slides with many skin cells and debris from the skin.

Sometimes you find pollens and mold spores. Yeast and bacteria are best seen under oil immersion objective. It takes much practice to be comfortable with reading tape cytologies.

Cytology of Crusting Dermatitis
Pustules and crusts are better assessed with a more gentle direct impression rather than the "extraction" of tape cytology. Direct impression means to touch a clean microscope slide right on to the area in question. Gentle pressure is key; excessive pressure damages inflammatory cells.

For crusts: Using a hypodermic needle sideways and parallel to the skin, you can gently lift / push the crust from the skin. Gently press a slide to the exposed, now ulcerated skin. Allow material to dry and stain routinely with Diff Quik. We may be looking for bacteria (intra or extracellular), neoplastic (cancer) cells, or cells indicating an auto-immune disease (such as acanthocytes of pemphigus).

Pustule Cytology: Pustules in dogs and cats are very short-lived and it is fortunate when any are present to help with diagnostics. Using a 25g needle or smaller, very gently "lift the roof" off the pustule: Hold the needle parallel to the skin and carefully disrupt the pustule, exposing a small amount of purulent material. VERY gently touch the slide to the purulent material. (If you press firmly, inflammatory cells will rupture, altering morphology.) Sometimes the material sticks to the needle. If so, gently touch the needle to the slide. Allow to dry and stain routinely.

Skin Bacterial Culture: Multi-drug resistant staphylococcus is becoming more common; culture to guide antibiotic choice is necessary. Appropriate sampling is important to capture the suspected pathogen and avoid as many contaminants as possible. Like cytology, how a culture is taken depends on the lesion.

Culturing *from epidermal collarettes*: An epidermal collarette is the skin lesion on a dog we want to call "ringworm" but is not, it's usually staph infection. The bacteria in these lesions is found under the scale at the periphery of the lesion. The best way to culture these lesions is use a 25 or 27g needle and gently lift some scale at the edge of the lesion, then touch the culture swab to the newly exposed skin under the scale. Sample from 2-5 areas.

Culturing *from a pustule*: Like we do for cytology, gently rupture the "roof" of the pustule, then touch a culture swab to the now exposed purulent matter; can also touch the tip of the needle used to the culture swab.

Culturing *from a raised draining lesion (a furuncle):* We usually look for an area of skin not yet ruptured and draining. An area of skin that has been open to the environment for a while will have non-pathogenic, contaminating bacteria in it, and you may miss identifying the pathogenic staph.

Find a raised but intact area of skin that looks like a little abscess about to rupture; often the skin has a red to purple appearance and a soft center. Wipe the surface gently with rubbing alcohol. Carefully poke the center (where the skin wants to rupture) with a 25 or 27 g needle. If material does not ready exit the skin, apply gentle pressure on the side of the swelling. Carefully touch a culture swab to the material that comes to the surface.

Culturing *thickened skin with no exudate* or an ulcerated skin (such as lick granulomas): in this situation we often have to take a biopsy and submit the tissue for culture. Ulcerated skin will be covered with contaminants and oral bacteria.

Skin Biopsy: When the cause of a pet's dermatitis is not apparent from exam, history and other diagnostic tests, skin biopsy for histopathology is often recommended. Most skin biopsies are done with a "punch biopsy," a round, 4 to 8 mm in diameter surgical blade that takes a small core specimen. Depending on the area to be biopsied, this can be done with just local anesthesia (lidocaine or carbocaine, often diluted with equal parts of saline to decrease the sting) +/- sedation. Biopsies from tender areas such as the nose, feet, or face require heavy sedation or anesthesia.

The skin to be biopsied should have *minimal preparation*. Scrubbing the area may wash off needed information! Pathologists can get valuable information from even crusts. Best to just carefully trim hair out of the way but do not dislodge any crusts. Outline the area to be biopsied with a marker, and the local anesthetic (usually ¾ to 1 ml) is injected under this marking. If the affected area has no hair, it helps the pathologist to draw a line on the top of skin with a marker in the direction of hair growth or surrounding hairs. The small biopsy specimen should be handled gently and put into formalin promptly.

The most important part of a biopsy is picking the correct area; the second most important is giving the pathologist a history of the patient's skin problems and list of symptoms. If your doctor does not fill out the submission form, put it in front of them with a pen, or gather as much information as you can and fill out the history section. Most important are what part of body sample came from, duration of the dermatitis, current medications, response to previous meds, and degree of itch.

Intradermal (Skin) Allergy Testing: This test is done (on dogs, cats, horses) to help identify a pet's allergens (proteins that trigger their allergies) so that we may formulate allergen immunotherapy. It is uncommon for primary care practitioners to do this test and you are unlikely to be called on to do this. However it is mentioned and photos shown so you have an idea what this is when talking to clients about testing.

Pain Medications and How They Work

Stephen Cital RVT, SRA, RLAT, VTS-LAM (Res. Anesthesia)
http://www.stephencital.com

Briefly, pain is a subjective experience with two complementary, but unpleasant aspects: one is a localized sensation in a particular body part; the other is a mechanical quality of varying severity commonly associated with behaviors directed at relieving or terminating the experience. Pain begins with specific receptors. Differing species will have varying numbers of receptors based on their evolutionary development and CNS physiology. Receptors are nerve endings, present in most body tissues, that respond in relation to pain by damaging or potentially damaging stimuli. Second, the messages initiated by these noxious stimuli are transmitted by specific, identified nerves to the spinal cord at the dorsal horn. The sensitive nerve ending in the tissue and the nerve attached to it together form a unit called the primary afferent nociceptor. The primary afferent nociceptor contacts second-order pain-transmission neurons in the spinal cord. The second-order cells relay the message through well-defined pathways to higher centers, including the brain stem reticular formation, thalamus, somatosensory cortex, and limbic system. It is thought that the processes underlying pain perception involve primarily the thalamus and cortex.

Opioids

Opioid drugs work by binding to varying opioid receptors in the brain, spinal cord, and more newly discovered peripheral areas of the body. They reduce the sending of pain messages to the brain and reduce (not eliminate) feelings of pain. Chronic use can lead to tolerance and even dependence like other addictive medications. New evidence in human and animals models is showing systemic inflammatory processes, making us have to re-evaluate the dose regiments to our patients of this still gold standard acute pain management drug classification. Opioids will have varying effects and potency on differing species and patient to patient. Opioids are generally safe for even the most critical patients at appropriate dosing.

Non-steroidal anti-inflammatories (NSAIDS)

This is a critical drug class that groups together medications that provide analgesia by processes of anti-inflammatory effects and antipyretic effects. This drug class is an ever evolving group with medications that have varying effects acting through inhibition of prostaglandin synthesis secondary to their inhibition of the enzyme cyclooxygenase (COX). This results in suppression of inflammation, and thus analgesia. Most NSAIDs not only inhibit prostaglandins at sites of inflammation, but systemically. Prostaglandins serve important functions in other parts of the body (kidneys and GI), a factor that accounts for some of the toxicity of these agents. The most frequent complications associated with NSAID usage are those involving the gastrointestinal tract and the creation of ulcers after administration. Studies on NSAIDS use the negative side effects on the gastrointestinal track as standard in safety testing. The evolution of NSAIDS has developed more specific (COX1, COX2) acting medications with improved safety and anti-inflammatory effects. NSAIDs that inhibit COX1, with increases up to 3 times with tissue injury were the first generation with more detrimental side effects on GI tissue. COX2 NSAIDS are more pain relieving by way of inhibiting the isoform synthesized by macrophages and inflammatory cells with tissue injury, which is the more pain stimulating concern able to produce severe inflammation and hyperalgesia.

NSAIDS do have positive synergy with other analgesics, such as opioids, and can actually help reduce the dose of opioids to achieve the same level of pain without the NSAID synergistic effect.

More and more evidence is supporting the use of certain NSAIDS pre-operatively, affectively alleviating the inflammation process BEFORE it starts for hydrated, elective and healthy patients over 6 weeks of age. It is critical renal infarct is avoided to reduce potential negative side effects.

Grapiprant is a member of a new piprant chemical class being developed by Arantana that works through a

specific targeted mechanism. Specificity at the EP4 prostaglandin receptor. Instead of inhibiting the cyclooxygenase enzymes, grapiprant has a specific target, at the EP4 prostaglandin receptor. What is particularly unique about this mechanism is grapiprant does not affect the function of the other prostaglandin pathways that are necessary to support normal kidney function, platelet function and other important physiological processes.

N-methyl-D-aspartate receptor (NMDA)
NMDA is a receptor for the excitatory neurotransmitter glutamate, which is released with noxious peripheral stimuli. The activation of these receptors is associated with hyperalgesia, neuropathic pain, and reduced functionality of opioid receptors. Increased spinal neuron sensitization, leading to hyperalgesia and neuropathic pain are a result of a heightened level of pain and can develop very quickly. When this occurs opioid receptors can become less sensitive leaving opioids less or totally ineffective and can lead to prolonged tolerance. Common NMDA medications include Amantidine, Methadone and Ketamine. All work to antagonize, or inhibit the action of the NMDA receptor.

Alpha-2 agonists
Dexmedetomidine (DxMd) is probably the most widely used alpha-2 in small animal practice today. Binding to alpha-2 adrenaline receptors in the central nervous system and the peripheral nervous system inhibits the release of norepinephrine and impedes transmission of further nerve impulses, which provides the dual effect of sedation and analgesia. Alpha- 2 agonists cause vasoconstriction by directly inhibiting K(ATP) channels. Constriction of these vessels increases the pressure, which in turn the heart senses and will decrease to the point of what might be considered severe bradycardia.

Dexdomitor is a purified derivative of the old formulation known at Domitor. Domitor contained the active molecule, medetomidine and the inactive levomedetomidine, which only added to the negative effects associated with Domitor, such as stress on the liver for clearance. DxMd is a highly selective alpha$_2$ agonist, ~1000 times more selective than Xylazine, which was formerly a common small animal sedative, used mostly for large animals and inducing feline emesis. The value of this medication is invaluable in many different ways other than sedation. Dexmedetomidine also has moderate analgesic properties, comparable to the analgesic effects of buprenorphine. It is important to note that while an animal is sedate and the analgesic properties are in effect, the analgesic effects will diminish before the sedation. Therefore it is recommended give pain medication well before the sedation wears off. The medication also has the added benefit of opioid potentiation, thereby reducing the total amount of opioids one will need. DxMd has a ceiling effect, meaning giving more will not depress the patient further, but only add to the time the patient is sedated. DxMd can be given IM, IV and PO (in cats). Dexmedetomidine can also be given at micro doses as a CRI and added to local anesthetics to prolong their effects.

Local Anesthetics
Many of us are familiar with the common sodium channel blockers lidocaine and bupivacaine, among others. When the fast voltage gated sodium channel is blocked by this drug group the membrane of the postsynaptic neuron will not depolarize and so fail to transmit an action potential leading to its anesthetic effect. Sodium channel blockers are the only pain medication that offer complete pain relief and are now recommended to be given for any elective tissue trauma, such as surgery. Adding adjuncts such as steroids, DxMd and opioids have all shown to extend the efficacy of local blocks to vary degrees and are being more and more encouraged.

Neurotoxins
While most people think of neurotoxins as a bad thing the saying “everything in moderation” comes to mind. Practitioners and researchers in the human world have been using various types of neurotoxins derived from shellfish, snakes, fish and bacteria in various forms of pain relief systemically and locally. Saxitoxin, a shellfish derivative had success in multiple human studies for significantly increasing local anesthesia (~72hrs). In canine bone cancer studies substance-p saporin was used to target pain-transmitting neurons after injection into the spinal

cord and provided excellent pain relief compared to more traditional methods.

Magnesium
Magnesium (Mg) is a common cation in the body playing a fundamental role in many cellular functions. Mg possesses calcium antagonistic properties and is involved in transmembrane ion fluxes and regulates neuronal activity. Mg is also an antagonist of the NMDA receptors as the magnesium ion blocks the central canal of the ionic receptor inhibiting calcium influx and preventing neuronal depolarization. With these physiological principles Mg has gained interest again as a non-narcotic analgesic systemically and epidurally. Human studies have reported conflicting reports of analgesic effects of systemic administration intraoperatively. Multiple studies including veterinary studies do show reduction of intra- and post- operative opioid requirements during soft tissue surgery, orthopedic surgery and thoracotomy. Additionally a study in dogs showed analgesia after an epidural of Mg with no motor deficits, but no potentiation of morphine antinociception like seen in systemic combination. Further studies are needed to determine onset and half-life of the technique.

References available upon request.

Local-Regional Anesthesia: Practical Implementation

Stephen Cital RVT, SRA, RLAT, VTS-LAM (Res. Anesthesia)
http://www.stephencital.com
San Jose, CA

Local and regional (L&RA) anesthesia is the technique of applying or infiltrating tissues with a sodium channel blocking agent (most common in veterinary medicine: lidocaine, bupivacaine, ropivicaine) to completely numb a specific area. We can literally take an animal in severe pain, such as broken ribs, and make them comfortable again within minutes. There are several adverse effects of continued pain, many of which delay healing and impact the patient psychologically. Local and regional blocking techniques are one of the few techniques we have to completely stop pain signaling to the spinal cord, further reducing sensitization, which in the worst-case scenario could lead to neuropathic pain. Local and regional blocking used for the anesthetized patient also shows a decrease in mobility and mortality and decrease complication in the post-operative period. There are multiple terms used for differing techniques of L&RA.

Topical or Surface Anesthesia is using sodium channel blocking agents in creams or solutions on the skin or mucous membrane providing some relief. Unfortunately, many of the agents we use are not readily absorbed through the skin surface unless left on for quite some time prior. The use of lidocaine patches over wounds or incisions has also been described but has not been found to alleviate the need for other analgesic medications.

Local Infiltration is a less precise means of infiltrating tissue with a blocking agent to achieve pain sensation loss. Basically, where the surgeon plans to incise or tissues will be manipulated in a way that causes discomfort the local blocking agent should be used. There are many studies on the efficacy of using this technique for any stable surgical patient. The efficacy of this technique has even found its way into the Pain Management Guidelines published by AAHA.

Regional or Nerve Blocking techniques are a bit more precise using anatomical land marks, palpation or devices to infiltrate the blocking solution within millimeters of a nerve. A good knowledge of the nervous system anatomy is desirable before implementing such techniques. It is important we are not piercing the actual nerve or infiltrating the nerve, like commonly done during leg amputations. More recent research has shown the infiltration of the nerve, stretching the fibers can sensitive the remaining nerve component adversely.

Neuraxial Anesthesia is the technique of infiltrating blocking agents in the epidural space. This is a very effective technique for essentially anything on the caudal half of the animal and can be useful for such conditions as pancreatitis or thoracotomies when using opioids instead of a blocking agent.

There are a few basic tools one will need for local blocks. Basic tools include: A variety of hypodermic and spinal needle gauges and lengths, preferably luer lock syringes. Red rubber catheters with male end adapters for infiltration OR pre-made wound infusion catheters. For more advanced techniques: A nerve stimulator and ultrasound.

There are a few techniques in veterinary medicine used in practice to reduce the sting of blocking agents, as they are usually a weak base. Adding sodium bicarbonate to the blocking agent does alkalinize the agent for a less dramatic sting in awake patients. When adding sodium bicarbonate, the mixologist should keep in mind some proportional ratios as adding too much can cause precipitation and decrease efficacy. A 1 part sodium bicarbonate to 3 parts blocking agent solution is usually safe and still effective. If the patient is anesthetized you may forgo this technique altogether. If you need a greater volume of the blocking agent and have not added sodium bicarbonate you can add regular saline to the blocking agent at no greater ration than 1:1 or efficacy will be

compromised. There is some evidence that adding saline to the mixture will better facilitate tissue distribution of the blocking agents.

The technique of mixing two different local blocking agents, having one agent with a quicker onset (Lidocaine) and one with a longer onset and lasting effect (bupivacaine), has largely been via summation. There is a larger body of evidence that shows when mixing the two agents the bupivacaine may be washed out of the system before any real beneficial effects. One more practical strategy is to provide the initial 60-90 minutes of anesthesia using a less irritating agent (lidocaine) and then reinject the anesthetized tissue with bupivacaine to provide analgesia well into the postoperative period.

Several adjunctive agents added to local blocking agents have been described and are a favorite technique of the author. Micro doses of opioids (0.005mg/kg buprenorphine or 0.01mg/kg morphine), steroids, and dexmedetomidine (0.25mcg/kg) can dramatically prolonged the effects of local blocks from a couple of hours to 24-48 hours. Another more recent option is NOCITA by Aratana. This is a liposomal bupivacaine solution that last for 72 hours. Although labeled of canine CCL repair and feline fracture repair at this time the author has used it in many other procedures such as hemilaminectomies, general incision closures, dental work and much more. A poster describing the use of the human form of the medication showed analgesic effects last up to 96 hours in mice.

With the insertion of a needle into any tissue we can expect some risk and possible complications. From the needle insertion aspect, we can cause mechanical trauma to several different types of tissue depending on where you are inserting. Most commonly with peripheral blocking techniques we may see nerve injury. This occurs as the needle pierces through the nerve instead of adjacent to the nerve.
We can also cause trauma to the nerve by what is termed *injection pressure*. This is the rate at which the operator of delivering the blocking agent either intra or perineural. We can also see nerve injury what using advanced tools such as a nerve stimulator for electrolocation. In general, if the operator is near the nerve and feels resistance during injection of insertion, like going into a different type of tissue, they should stop and re-adjust the needle placement. Typical compilations from these types of injuries usually manifest 48 hours after the injury as motor loss, the patient biting or scratching at the site from a tingling sensation or injury to the tissue over time form chronic numbness and lack of self-awareness to injury.

Other complications we may see include neurotoxicity. Although evidence has shown that the efficacy of blocking agents (and other medications) used in epidurals with preservatives is the same the preservatives like EDTA has been associated with severe back pain in canine and human studies.

- References available upon request. Please email the author for a copy of the slides for needle insertion sites and techniques at http://www.stephencital.com

Bupivacaine Body Cavity Analgesia Protocol

Usage: This treatment is to provide local analgesia for the thoracic and abdominal cavities.

Supplies needed:
- Bupivacaine 5mg/ml
- 0.9% NaCl
- Sodium Bicarbonate 8.4%
- Sterile syringe
- Sterile needle

Procedure:

1. Draw up 1.5mg/kg Bupivacaine, dilute with three parts 0.9%NaCl.
2. Then add 1 part of Sodium Bicarbonate 8.4% to 9 parts Bupivacaine/0.9% NaCL solution. (Take your total volume in mls of Bupivacaine/0.9% NaCl solution and divide by 9.)
3. Aspirate the tube per protocol prior to instilling the analgesia solution.
4. This is a per animal dose. If the patient has bilateral chest tubes, divide the dose among them.
5. Infuse the solution slowly over 1-2 minutes.
6. This solution should remain in the pleural space for at least 30 minutes.
7. Aspirate the chest tube per protocol.
8. This treatment should be repeated every 4 hours, as needed for analgesia. The total dose of Bupivaciane should not exceed 9mg/kg/day.
9. Please document on the treatment sheet the volume instilled and aspirated.

Please note:

- Higher doses of Bupivacaine may cause signs referable to:
    - Neurologic signs
        - Altered mentation, tremors, seizures
    - Cardiovascular signs
        - Myocardial depression, arrhythmias, hypotension
- Some patients may experience stinging when the Bupivacaine solution is administered, so use caution.
- ***Do not use in cats***
- ***Do not use for pericardectomy patients***

Nursing on the Front Line: Recognizing Hemodynamic Changes

Stephen Cital RVT, SRA, RLAT, VTS-LAM (Res. Anesthesia)
http://www.stephencital.com

A holistic approach to the patient and watching trends rather than focusing on specific numbers is still standard. Goal directed monitoring also has its place in intensive care settings, but can lead to missed clinical signs or features. It is crucial to have trained staff monitor critical patients. It may even be necessary to have one dedicated technician for a particular animal in the ICU setting with little distraction, especially of the patient was a ASA IV or above. Advanced techniques to monitor hemodynamtic parameters can involve placing a central line for central venous pressure measurement or even placing and maintaining an arterial catheter. This sort of monitoring is still a gold standard. As we all know this may be difficult and not practical for the duration of a case or feasible in a certain species. Not to mention financially burdensome to the client or out of the scope of your practice's needs and abilities. This, in turn, can lead to less effective treatment and response in our patients. There is now non-invasive technology which includes Perfusion Index (PI) and Pleth-Variability Index (PVI). These are two newer monitoring parameters that can be very telling when obtained correctly. The perfusion index is the ratio of the pulsatile blood flow to the non-pulsatile or static blood in peripheral tissues. What this means is now we can monitor peripheral tissue perfusion in our patients non-invasively giving better insight into our fluid therapy management, cardiac/renal output, and efficacy of medications. A defined reference variable is not yet established in the canine or feline patient or any other species for that matter, other than humans, which tends to be quite broad. However, the PI parameter, as well as all of the other non-conventional parameters, is great for trending and monitoring. The PI is also a great tool for assessing the efficacy of opioids and epidurals. When full onset of the opioid or epidural occurs, we see a spike in the PI showing via vasodilation.

The Pleth Variability Index (PVI) is a new technology even in human medicine. It is a measurement of the change of perfusion index with a complete respiratory cycle. With this in mind, PVI is most reliable with patients undergoing mechanical ventilation. In a scientific abstract presented at the American College of Veterinary Anesthesiologists conference, one research group found that the PVI had a good correlation in detecting hypovolemia and return to normovolemia in dogs, but could not be used in definitively stating hypervolemia. Several more recent veterinary papers on PVI have come out with positive conclusions as to the reliability of predicting fluid responsiveness using this non-invasive tool.

History- A clear history should be gathered about the patient, from its primary veterinarian, surgeon, anesthetist and any other sources.

- Patient background (age, sex, code status)
- Type of operation and outcome if applicable
- Indications for operation and pre-operative diagnosis
- Current inotropes, vasopressors, or anti-hypertensives (if any)
- Need for cardiac pacing when applicable
- Bleeding risks and clotting times
- Other significant co morbidity, with emphasis on those conditions that may alter the post-operative management or course (asthma, diabetes, renal failure, hepatic failure, etc.)
- Medications
- Allergies

Physical exam and assessment

- Verify that the patient's oxygen saturation is adequate. Check the ABG results as soon as they are available. If this is not available an SpO2 and monitoring of the pH, bicarbonate and electrolytes must be evaluated.
- Verify correct ventilator settings if applicable.

- Check the initial hemodynamic readings (HR, BP, cardiac output and index, CVP) and determine what vasoactive infusions the patient is on and at what rates.
- Check the patient's heart rhythm. Verify pacemaker settings if the patient is connected to one.
- Examine heart sounds. Listen for murmurs.
- Check all peripheral pulses. Do repeated assessments if there is concern for acute limb ischemia. A Doppler can be placed for on a peripheral limb for continuous evaluation.
- Do a more complete neurologic exam

Labs and tests Electrocardiogram
- Note any changes of ECG
- Rhythm - post-operative bradycardias, blocks, or atrial fibrillation
- ST-T changes - diffuse non-specific changes are not uncommon and may reflect pericardial inflammation or ischemic events
- Chest X-Ray
- Rarely used in the non-research sector, verify correct position of the Swan-Ganz catheter.
- FAST scan U/S

Laboratory Results
- Hemoglobin
- Coagulation parameters (PLT, PT, PTT, ACT)
- Renal and liver chemistry
- Potassium, magnesium, calcium - a vigorous diuresis is common in the first few hours after the OR. This can lead to significant hypokalemia and hypomagnesaemia which increases the likelihood of post-operative dysrhythmias. Standing orders are in place to replace these electrolytes.
- Glucose - tight glycemic control post-operatively reduces morbidity in humans.
- Cardiac markers - elevations of CPK, CPK-MB, and troponins are non-specific. They should be assessed as part of the overall clinical picture including the hemodynamic status of the patient and the EKG.

Warming
Effects of hypothermia
- Predisposes to ventricular dysrhythmias and lowers VF threshold
- Increases SVR; increases afterload and myocardial workload
- Patient shivering causes increased peripheral O2 consumption
- Decreases CO2 production; a patient who has a respiratory alkalosis (low PCO2) on initial ABG usually will increase their PCO2 with rewarming
- Coagulopathy; impairs platelet function and the coagulation cascade. Rewarming is an important part of the treatment of a bleeding patient.

Hemodynamic management
Hypotension and low cardiac output
1. *BP = CO x SVR*
2. *CO = HR x SV (stroke volume)*
3. *Stroke volume is determined by preload, contractility, and afterload*
4. *Bradycardias or tachydysrhythmias that decrease ventricular filling can decrease CO.*

There are numerous causes for hypotension post-operatively. Proper management of the hypotensive patient in the ICU requires that the precise etiology for the hypotension is determined and therapy is directed towards reversal of this specific problem. Equation 1 demonstrates that hypotension can be caused by a "pump problem" (low cardiac output) or a low SVR (arterial "circuit" problem). The following is an approach to managing the hypotensive patient:

1. Look at the recent hemodynamic parameters.
2. Assess the cardiac output/index. Is this a "pump" problem? Or is it due to low SVR?
3. Look at the cardiac rhythm.
4. Look at the CVP to assess preload.
5. Is the afterload high?
6. Is contractility decreased?

- Is this tamponade? Look at the recent hemodynamic parameters obtained from the Swan-Ganz catheter or evaluate via echo.
- Assess the cardiac output/index.
- If the cardiac index is in the normal range or high, then the patient does not have a significant "pump" problem and the cause of the hypotension is secondary to diminished peripheral arterial tone (low SVR). A vasopressor agent should be considered. The differential diagnosis of low SVR includes;
  - SIRS - a proportion of patients post CPB will have significant cytokine increases
  - Sepsis
  - Anaphylactic or anaphylactoid reactions
  - Drug-induced, toxicological - nitrates, antihypertensives, narcotics and sedatives, etc
  - Adrenal insufficiency (Was the patient steroid dependent pre-operatively?)
  - Hyperthyroidism, hypothyroidism
  - Neurogenic (spinal) shock
- If the cardiac index (CI) is low then the cause of the hypotension is inadequate flow or a "pump" problem.
- Look at the cardiac rhythm. Absolute or relative bradycardias or tachycardias can lead to decreased CO and should be corrected.
- Look at the CVP to assess preload. A patient with a low CI and a CVP that is "relatively" low should be given a fluid challenge. Remember, what you really are interested in is a volume measurement (preload= right or left end-diastolic volume), but what you are measuring are pressures (CVP = Right or left ventricular end-diastolic pressures).
- High afterload. Secondary to vasoconstriction and hypertension.
- Decreased contractility. This should be managed with inotropic agents while simultaneously looking for the cause.
- Tamponade
- Acute valvular regurgitation. Check for a new regurgitant murmur.

Appropriate systemic arterial blood pressure is vital for survival in any species. In practice, we are faced with many reasons and conditions to obtain and interpret a patient's blood pressure, such as anesthesia, cardiovascular disease and kidney disease. Both high and low blood pressure can be detrimental to our patients, so careful and accurate monitoring techniques are necessary. The two most common methods of non-invasive blood pressure (NIBP) measurement are Doppler ultrasound with a sphygmomanometer and oscillometry (Cardell or other machine). High definition (HD) oscillometry is a newer indirect technique and is becoming more common. Whenever taking a NIBP, it is important to note the trends of the data collected. NIBP is not an exact reflection of the patient's true blood pressure, as there are many variables that can affect the results.

Doppler ultrasound measurement
Doppler ultrasound measurement is the most common blood pressure measurement technique used in small animal practice. In dogs, the reading attained most closely correlates to the systolic blood pressure, whereas in felines recent research is showing that it is more closely correlated to the mean arterial pressure (MAP). Some specialists will add 15 points to a Doppler derived pressure to better estimate the systolic pressure. Limitations when using the Doppler method include the user's acute hearing, patient movement and cuff placement. A minimum of four readings should be taken to show a trend with the readings, discarding the first reading.

Supplies
Sphygmomanometer, blood pressure cuffs, ultrasound transmission gel, tape, clippers OR a NIBP machine

General tips
Blood pressure readings should be measured in a quiet, comfortable environment after the patient has become acclimated (i.e., without disturbance) for at least a few minutes, but acclimation is rarely possible for acutely or critically ill patients. Cats have good control of peripheral vasoconstriction and can become peripherally constricted under stress.

The patient should remain still in lateral recumbency during measurements to optimize accuracy. If necessary the patient can be restrained gently or have a towel placed over the head as a relaxation method. If the patient cannot be restrained in lateral recumbency because of agitation, respiratory distress, or other reasons, a standing BP can be obtained and a corrective equation used. Chemical restraint should be avoided.

The size of the cuff is important and should measure 40% of the appendage circumference in dogs and 30% in cats. Using a cuff that is too large will typically give falsely low blood pressure readings, while the opposite is true for a cuff that is too small.

Placement of the cuff is important. The most important thing is that the limb you use is at heart level whenever possible. Options for cuff placement when using the Doppler technique include mid-radius on the forelimb and proximal to the hock on the hindlimb. The base of the tail is also an effective site in small dogs and cats.
For oscillometric techniques, place the cuff mid-radius on the forelimb, the mid-tarsus on the hindlimb, or the base of the tail. The cuff tubing is placed over the artery on all locations and methods. The transducer tubing will ideally be directed away from the patient and towards the monitoring device. The cuff should be placed at the most even and cylindrical portion of the appendages.

To acquire the most accurate blood pressure values, cuff height off the floor or table should be as close to the level of the right atrium as possible.

Do not secure the cuff with tape tightly, as this will restrict airflow to the cuff's bladder and cause inaccurate readings. A very loose piece can be used if absolutely necessary.

Do not place the cuff on a compromised limb or a limb with an arterial catheter, an IV catheter or a pulse Ox probe.

Technique
1. When using the Doppler unit, shave a small area on the posterior aspect of the paw, just above the largest pad on the front or hindlimb. If using the tail, shave a small area on the ventral aspect of the tail below where the cuff will be. When using the hindlimb, you can also shave and place the crystal over the more medial dorsal pedal artery. Apply a small amount of ultrasound conduction gel and use the Doppler crystal to find the clearest audible pulse. Secure the crystal head lightly with tape or hold still in place.
2. Using the sphygmomanometer, inflate the cuff until the pulse is no longer audible. Slowly release the pressure while listening for the first audible trace of the pulse to return and record this number. The first blood pressure measurement should be discarded.
3. Additional blood pressure measurements should be obtained for a minimum of 3 to 7 consecutive measurements. Consistent recordings with less than 20% variability are acceptable. Once these values have been obtained, they should be averaged to yield the blood pressure measurement.

If the patient is sitting or standing and the height difference between the cuff and the right atrium exceeds 10cm, subtract 0.8mmHg for every 1cm the cuff sits below the right atrium.

Example:
Initial systolic reading via Doppler or oscillometric reading was 160mmHg.
The top of the cuff is 23cm below the right atrium.
23cm X 0.8mmHg = 18.4mmHg

Corrected value:
160mmHg- 18.4mmHg = 141.6mmHg
If oscillometric machines are used repeat steps 1-3 and record readings. Use corrected value equation if patient is standing or sitting.
4. Once a pressure is taken, record the cuff size, patient positioning, appendage used and placement of crystal in the medical record for future readings. Keeping the method consistent is key.

Rabbit Anesthesia: What is Everyone So Scared Of?

Stephen Cital RVT, SRA, RLAT, VTS-LAM (Res. Anesthesia)
http://www.stephencital.com
San Jose, CA

Rabbits have quite the reputation for increased anesthesia risk, but why? A study comparing anesthetic death rates of various pet species reported a rate of 0.17% for dogs, 0.24% for cats, 1.39% for rabbits. Explanations for increased anesthetic death rate include greater intolerance to pain, stress, anesthetist comfort level, elective procedures in this species, and the ability to hide underlying illness.

Elective anesthesia for procedures such as those requested by project protocols should be thought out carefully and completely. Indications for emergency surgery also occur such as gastrointestinal obstruction. Whenever possible base line pre-anesthetic blood work is favorable.

Abnormal blood parameters and hypovolemia should be corrected prior to an anesthetic even whenever possible. Traditional parameters are used to estimate dehydration in small animals, including decreased skin turgor, dry mucus membranes and lethargy can be used as with the glossy appearance of the large rabbit eye. The rate of balanced replacement fluid administration depends on the estimated rate of fluid losses and clinical status of the patient. Based on human and now more and more veterinary research it has become clearer that we have grossly overestimated insensible fluid losses. Demonstrations show that fluid lost through skin and airway added up to approximately 0.5 mL/kg/hr. Fluid loss increased to 2 mL/kg/hr with an open abdominal cavity. This increases to 8-10 mL/kg/hr when the abdominal cavity was open with organs and intestines exteriorized. Fasting for a few hours prior to anesthetic events, although not critical in rabbits due to their inability to vomit, likely has limited impact on intravascular volume in the average patient as long as extra losses from loose stool, weeping wounds and blood loss are not in occurrence.

There is a paradigm shift on intra-operative fluid rates. Historically rate of 10-20 ml/kg/hr were once recommended for small animals. Current literature is suggesting dramatically lower rates of only 2-5ml/kg/hr, which can be extrapolated for rabbits as well.

Intravenous access is highly recommended for any anesthetic candidate. Intravenous catheterization in appropriately restrained awake rabbits or with mild sedation is attained by using the marginal ear, cephalic or saphenous vein, but may be difficult in smaller, or hypovolemic species. Intraosseous catheterization using the proximal tibia at the tibial crest, or the proximal femur at the greater trochanter are just as efficacious as IV routes and can be more easily accomplished in emergent situations. IV flow rates via the intraosseous route are equal to those by traditional IV access. Micro neonatal IO needles, spinal needles may be beneficial can be used but the author prefers 22-20g hypodermic needles for IO catheterization. When placing an IO catheter a local anesthetic and sterile technique over the insertion site should be use along with systemic form of pain management. Confirmation of correct placement can be done via test administration of boluses, but definitive confirmation is through radiography. Always perform two views to confirm the catheter is in place.

The tradition of mask or boxing down rabbits for anesthesia induction is more and more becoming a concern for animal welfare and a significant variable with research project looking at physiological function or cardiac injury models with an immense release of catecholamine's. The technique is also responsible for increased mortality rates post operatively. If an IV or IO catheter is unattainable there are IM or SQ agents that can be used instead that have a wide safety margin or can be reversed, such as Alfaxalone or DexMedetomidine.
The benefits and safety of "multi-modal" anesthesia have been demonstrated in many species including rabbits. Pre-anesthetic agents can provide a smoother, anxiety relieving induction while reducing the amount of general anesthesia required. They other added benefit is analgesia many of the agents some of the choices we have can provide.

Opioids are the authors preferred drug choice for premedication and gas inhalation reduction. Even the most critical patient can tolerate appropriate dosing of opioids. One study using an intra operative fentanyl CRI reduced the amount of anesthetic gas needed while keeping the blood pressure at more favorable ranges.

Anxiolytics such as midazolam are also a preferred medication of choice by reducing the amount of opioids, anesthetic gas and systemic release of catecholamine's rabbits produce as high stress prey species.

Dexmedetomidine has great clinical relevance in rabbits with its sedative, anxiolytic and analgesic properties. This medication also has the added benefit of allowing opioids to better attach to receptor sites making the opioid essentially more effective. Dexmedetomidine also has cerebral protective properties, which is particularly useful with rabbits during apnea and intubation. Xylazine has been associated with cardiac injury for models undergoing repeated anesthesia using this agent.

Local anesthetics can be effective in rabbits and performed the same way as in small animals. The general "safe" dose of lidocaine and bupivacaine is 2mg/kg. If more volume is needed of either a 1:1 ration of the local anesthetic and saline can be used without major loss to the effectiveness of the medication. Lidocaine and bupivacaine should not be mixed as this reduces the half-life of both medications.

Airway management:
Rabbits can pose a challenge to the anesthetist. Since ET intubation in rabbits is commonly done blindly, the skill requires practice. ET intubation performed blindly in rabbits and some other rodent species requires the animal to be moderately sedate or anesthetized.

- While having passive supplemental oxygen administered, the anesthetist will hold the head up, straightening the tracheal as much as possible. If attempting to intubate a rabbit with only one person, insertion of an oxygen nasal cannula provides a pre-oxygenation source
- A few drops or a mist from an atomizer of a local anesthetic can be applied to the supraglottic region.
- After allowing the local anesthetic to take effect to reduce laryngeal spasm an appropriate sized ET tube can be inserted.
- The anesthetist will listen at the end of the ET tube where it connects to the anesthetic circuit for breath sounds.
- When listening, the anesthetist should listen for the loudest and most clear breath sounds and attempt to feed the tube into the presumed trachea. Hair plucked from the patient can also be used to detect whether or not the ET tube is in the trachea. Some anesthetists prefer to use a modified esophageal stethoscope connected to the end of the ET tube to listen, rather than the ear to tube methods. Others choose to visualize the condensation created by a breath on the walls of a clear ETT.
- Multiple attempts only using minimal pressure may be indicated.
- Attention to not cause soft tissue trauma and swelling is always advisable with this method. This technique can also be used, but via the nasal passages. Generally, a slightly smaller tube will need to be used with lubricated.

Rigid or flexible laryngoscopes or careful use of a 0-0 Miller blade, or other modified laryngoscope blades are methods that some practitioners use, but time for setup and limited oral space can make this method less practical.

Docsinnovent introduced the v-gel™ for rabbits and felines, which is a modified tube that only covers the supraglottic region. Although ET intubation is the author's preferred method, v-gel™ offers a quick and easy approach when ET intubation proves too difficult. They are not ideal for animals requiring oral surgery as they take up a decent amount of space in the mouth and the potential for fluid leakage around the tube if undergoing a dental procedure. The other limiting factor is they require capnography to ensure proper placement. The tubes themselves have a build in port for sidestream capnography, but also support mainstream capnography with an adaptor that will add to the total deadspace. Older sidestream machines may not be ideal for small patients as some machines require taking 50-200 ml/min of the ventilated gasses for sampling.

In the event an animal cannot be intubated, the forced mask ventilation technique may need to be utilized

- This technique consists of fitting a patient with a mask that covers the nose and mouth with as few leaks as possible.
- The head should be placed so that the trachea is fully extended and as straight as possible to allow easier movement of air.
- There are various pre-manufactured masks, but at times it is necessary to create a homemade mask out of syringe cases, small bottles or tubing. Taking advantage of the patient's bottom incisors case be useful for masks made form syringe cases. The technique involves using a larger suture or small sized string tied/looped around the top incisors and pulled through the mask. Letting the string hang out the end and pulling taught as the non-rebreathing circuit is connected will ensure a sealed mask. Careful attention should be paid to not create ocular trauma with careful positioning and plenty of eye lubricant.
- If the forced mask ventilation technique is used, it is important to remember to protect the animal's eyes. It is too often a mask is left putting pressure or digging into the inferior eye socket.
- This technique can also lead to gas in the stomach that may need to be treated postoperatively by tubing or carefully expressing the air out. Rabbits in particular have a thin stomach wall, over inflation can cause rupture and hinder normal tidal volume intake

In an emergency situation a tracheotomy can be performed or the animal may be intubated by using the over the needle technique. The over the needle technique or "retrograde intubation" technique uses a hypodermic needle inserted percutaneously from the ventral aspect into the trachea. A wire or heavy gauge suture is used as a guide wire and passed through the needle into the trachea, aiming towards the mouth and pushed through the trachea/oral cavity until it can be used as a stylet. However, these methods are not recommended unless dire circumstances exist, and the practitioner is willing to assume and deal with the potential risks and complications.

Monitoring

Basic anesthesia monitoring greatly mirrors cat and dog anesthesia. Titrating anesthetic agents up or down to alleviate patient discomfort and keeping an appropriate anesthetic plane is identical to domestic species anesthesia. Special monitoring equipment used in the research setting can better accommodate higher heart rates seen with smaller mammals and give more reliable ECG tracings. Pediatric settings on all monitors should be utilized if conventional monitoring equipment is used. Laboratory animal vendors also offer miniaturized anesthetic machines and tubing, eliminating dead space seen in regular sized anesthesia tubing even compared to the use of non-rebreathing circuits. Micro ventilators, specially designed for patients in the 150-400 gram weight range, are also commercially available.

Spending more time setting up a patient with every bell and whistles may prolong anesthetic time. It may be more advantageous to simply finish the procedure without every monitoring gadget attached and go back to basics. Being able to use ones stethoscope, eyes, ears, touch and intuition are just as vital as any piece of monitoring machine.

It is important to maintain all patients as normothermic as possible. Heating pads, warm IV fluids, warmed/humidified anesthetic gases, and radiant heat are common and effective methods. Plastic bubble wrap (for warmth, insulation, and soft bedding) is also used. Using a continuous thermometer placed in either the rectum or esophagus will help the anesthetist better gauge the patient's temperature intra-operatively and allow for quicker responses to ever fluctuating temperature changes.

Because the surface area to body mass is high in rabbits, the cooling effects from surgical scrubbing used during aseptic technique can make it challenging in maintaining a normal body temperature, yet is critical. A rabbit's ears comprise around 12% of the animals' surface area and a bat's wings comprise about 85% total body surface area and can be used to cool quickly or warm a patient.

Normothermia will help keep a steady metabolic rate and gut flow. A normal temp will also aid in keeping a

normal blood pressure to perfuse our patients' vital organs.

Wrapping the patient in the bubble wrap or merely using it like a blanket is most effective, due to the lightweight property of the wrap. This will allow for better inspired tidal volumes by not adding additional weight on top of the patient. Using warming devices such as warm water bags and circulating warm water blankets work well. Caution should be taken to not allow direct contact to the patient in the event a thermal burn. Warm water bags eventually cool and have recently been shown to have the opposite effect and can steal heat from an anesthetized patient. Warm air blowers are ideal, but can be cumbersome with such tiny patients. A personal favorite is using bubble wrap. It not only is cheap and disposable, it offers a lightweight and insulated option in thermoregulation. Tiny knitted socks work well in covering limbs. Heat and moisture exchange devices are also a good option, but can add to dead space and respiratory resistance. These devices work by inserting the device between the ET tube and the breathing hose. The paper filter keeps warm moist air in the chest cavity. They also help protect the anesthesia machine from aerosolized bacteria the patient may be harboring with expiration. As a last resort, warm water enemas can be used in extremely cold patients, but a cooling evaporation effect can occur if the patient becomes wet during the process.

Appropriate and inappropriate gas exchange in rabbits has the same positive and detrimental effects as it does in dogs and cats, but resiliency to hypercarbia can be less appreciable. The general normal range for end tidal carbon dioxide is the same as cat and dog values, between 35-45mmHg. The gold standard in monitoring the respiratory system and function in any species is by arterial blood gas sampling. Many blood gas machines only require a small sample size, which is ideal for exotic species, but collection complicates this method is an arterial line is not places beforehand. Capnography is also a useful tool in any small exotic species, but considerations based on the size of the patient and breath quality should be made as adding the sensor may increase dead space further. Reducing dead space from sidestream monitors can be done by sticking an 18g needle directly into the hub of the ETT connector and then attaching the sidestream line. With this technique you go from almost 8mL of dead space with traditional elbow or straight sidestream adapters to only 0.07mL. Mainstream capnography is preferred over sidestream machines. In humans sidestream capnographs can be less accurate in neonates and pediatric patients. This is because the sidestream machines sample a relatively large amount of the total ventilation, ~20% of ventilator requirements. The EMMA by Masimo is an excellent choice when working with small exotic animals for mainstream capnography. The small monitor only has 1 mL of dead space when using the pediatric sensor.

Blood pressure management can be one of the most difficult parameters to keep within normal ranges for the anesthetized rabbit. Rabbits have a poor response to dopamine and less pronounces effects to dobutamine and phenylephrine compared to cat and dogs.

Stressors, Pressers and Heartache Disasters

Stephen Cital RVT, SRA, RLAT, VTS-LAM (Res. Anesthesia)
http://www.stephencital.com

Appropriate blood pressure is vital for sustaining a healthy cardiovascular, cerebral and internal organ system. Without healthy blood pressure these systems can suffer, leading to chronic disease processes and death. In veterinary medicine monitoring blood pressure is still relatively new when compared to the human medical field and refining the best techniques in accurate measurement and treatment is under constant research. Nearly every species we deal with has physiologic differences and tolerances when it comes to their blood pressure and how effective various modalities work in each species. The following is a very broad overview of common medications used to treat abnormal blood pressure under anesthesia, with a focus on hypotensive correction.

Treating hypertension or hypotension (MAP <60mmHg for many species) while under anesthesia should never first start with pharmacological agents unless the patient has a pre-diagnosed cardiovascular condition. Treatment for either hyper or hypotension should first start with a carful patient assessment. These are some questions that should run through your head before moving to pharmaceutical intervention. Is the patient too light or painful? Is the gas inhalant or total IV anesthesia rate too high? Is the fluid therapy adequate? Are my monitoring devices (i.e. Doppler or mechanical blood pressure monitors) appropriately placed and do the numbers appear to match with how the animal looks clinically? Does my patient have an underlying cardiac issue? Does my patient have an arrhythmia?

Atropine- Although not typically considered a blood pressure medication, atropine plays an important role in treating certain arrhythmias under anesthesia. Neonates and immature animals are more reliant on heart rate for appropriate cardiac output and thus blood pressure. While atropine has fallen out of favor in the pre-medication plan it is a drug that warrants shelf space in all operating rooms.

Ephedrine- Ephedrine is a safe, yet somewhat expensive option in the first line of pharmaceutical intervention of hypotension. Ephedrine increases cardiac output, heart rate, blood pressure, coronary blood flow, and myocardial oxygen consumption. It reduces the need and time for CRI's of other vasopressors and inotropes with its longer duration of action. Ephedrine is used in human medicine second to phenylephrine for hypotension during pregnancy and fetal surgery. One study found in dogs a bolus only lasted 5 minutes and the increases of cardiac output and increased BP were merely transient. Ephedrine also causes stimulation of the respiratory centers and bronchodilation.

Dopamine- This is a highly dose dependent drug. For effective increases in MAP in dogs and cats research suggests rates starting at 7μg/kg/min to increase the A1-adrenergic agonist effects take the lead. The effect will increase systemic and pulmonary vascular resistance, venous return, and possibly PVC's due to splenic contraction. Tachycardia can occur at higher dose rates. When using dopamine it is recommended to decrease the CRI in a stepwise manner. The receptor effects of dopamine are dose dependent. Dopamine stimulates the release of endogenous norepinephrine from presynaptic storage sites at adrenergic receptors causing an endogenous sympathomimetic effect. There is some debate on the use of dopamine in felines as they lack the typical distribution of dopamine receptors found in canines.

Dobutamine- Dobutamine is another commonplace inotrope used to treat hypotension related to poor cardiac output. Current thought regarding the medication for treatment of hypotension during inhalation anesthesia is that it lacks good predictable effects, especially in cats. Higher doses ~10mcg/kg/min were needed to produce vascular resistance in dogs and an increase in cardiac output, but caused vasodilation in cats. In both studies minimal to no increase in blood pressure was noted.

Phenylephrine- Phenylephrine is a direct-acting sympathomimetic amine with strong Alpha1-adrenergic receptor

agonist effects. It is used intravenously during anesthesia to increase systemic vascular resistance therefore increasing blood pressure. Phenylephrine is the first line medication for hypotension during fetal surgery.

Norepinephrine- This medication has largely B-adrenergic receptor mediated effects. At sufficient doses we see an increase in cardiac output, increased SYS, DIA and MAP, along with systemic and pulmonary vascular resistance. Coronary arterial flow is also increased via vasodilation. Tachycardia is less pronounced compared to epinephrine.

Epinephrine 0.01–1 μg/kg/min
Norepinephrine 0.01–0.2 μg/kg/min
Dobutamine 1–20 μg/kg/min
Phenylephrine 0.2–2 μg/kg/min
Dopamine 1–10 μg/kg/min, >10 μg/kg/min Primarily α effects
Ephedrine 0.05–0.5 mg/kg

Airway management:

Traditional endotracheal intubation uses a sterile or thoroughly disinfected endotracheal tube for each patient to prevent the spread of infectious disease. The endotracheal tube should be lubricated with a very this layer of sterile xylocaine or K-Y jelly. The author also uses an extremely thin layer of sterile non-water based eye lubricant to avoid the drying out of the lubricant and subsequent traumatizing removal of soft tissue during longer procedures. The is also a commercially available spray for ETT's called SILKOSPRAY by Rusch. Operators should avoid using a lubricant containing benzocaine, as this can lead to a dose-dependent methemoglobinemia (MetHb), which does not bind oxygen and most hospitals are unable to test for MetHb in clinic.
Intubation may stimulate the vagus nerve, increasing parasympathetic tone especially in dogs. This may result in bradycardia, hypotension and cardiac dysrhythmias. If the animal has an underlying cardiovascular disease, cardiac arrest may occur. Atropine or glycopyrrolate can be given as part of the premedication. This is also a consideration when eye surgery is going to be performed or repeated movement of the head/throat region in preventing the parasympathetic stimulation.

Operators should not be forceful in the intubation technique as this can damage the larynx, pharynx, or soft palate and lead to tissue edema. Ideally, the tip of the endotracheal tube should be past the larynx and not beyond the thoracic inlet. If the tube is advanced too far, it may enter one bronchus, resulting in ventilation to only one lung. Premeasure the length of the endotracheal tube and the distance between the nose and the thoracic inlet prior to anesthesia. A general rule of thumb is once the cuff of the ETT has pas the arytenoids advance about a centimeter more, then stop. The end of the tube should be at the level of the animal's incisors to eliminate dead space and respiratory resistance.

Laryngeal Mask Airways (LMA's) for the veterinary patient were introduced by Docsinnovent with the v-gel™ for rabbits and felines (soon to have a canine and equine model), which is a modified tube that only covers the supraglottic region. Although ET intubation is the author's preferred method, v-gel™ offers a quick and easy approach when ET intubation proves too difficult. They are not ideal for animals requiring oral surgery as they take up a decent amount of space in the mouth and the potential for fluid leakage around the tube if undergoing a dental procedure. The other limiting factor is they require capnography to ensure proper placement. The tubes themselves have a build in port for sidestream capnography, but also support mainstream capnography with an adaptor that will add to the total deadspace. Older sidestream machines may not be ideal for small patients as some machines require taking 50-200 ml/min of the ventilated gasses for sampling.

In the event an animal cannot be intubated, the **forced mask ventilation technique** may need to be utilized. This technique consists of fitting a patient with a mask that covers the nose and mouth with as few leaks as possible. The head should be placed so that the trachea is fully extended and as straight as possible to allow easier

movement of air. There are various pre-manufactured masks, but at times it is necessary to create a homemade mask out of syringe cases, small bottles or tubing. Taking advantage of the patient's bottom incisors case be useful for masks made form syringe cases. The technique involves using a larger suture or small sized string tied/looped around the top incisors or canines and pulled through the mask. Letting the string hang out the end and pulling taught as the non-rebreathing circuit is connected will ensure a sealed mask. Careful attention should be paid to not create ocular trauma with careful positioning and plenty of eye lubricant. If the forced mask ventilation technique is used, it is important to remember to protect the animal's eyes. It is too often a mask is left putting pressure or digging into the inferior eye socket. This technique can also lead to gas in the stomach that may need to be treated postoperatively by tubing or carefully expressing the air out.

If direct visualizationof the glottis or a portion of it is not possible, *Jane* E. *Ouandt,* DVM., M.S., DACVA descrives the following techniques; in the case of a pharyngeal or an oral mass, one method to use is **retrograde intubation**. A hypodermic needle is passed through the ventral aspect into the skin of the neck and into the trachea at the junction of the second and third tracheal rings. A guide wire, or canine urinary catheter that will pass easily through the needle, is maneuvered through the needle cranially into the larynx, pharynx and oral cavity. It is then used as a guide for the passage of the endotracheal tube. After the tip of the tube is within the larynx, the needle and guide wire can be removed. The endotracheal tube is then advanced into the final position. Subcutaneous emphysema and pneumothorax are possible complications with this technique.
In an emergency situation a **tracheostomy** can be performed. Indications for a tracheostomy include relieving an upper respiratory tract obstruction, facilitate removal of respiratory secretions, decrease dead space, provide a route for inhalant anesthesia when oral or facial surgery is complex, reduce resistance to respiration, when you are unable to orally intubate, reduce the risk of closed glottis pressure, or cough, following pulmonary or cranial surgery.

To perform a tracheostomy make a midline skin incision on the ventral neck equidistant from the larynx and the manubrium. Part the two sternohyoid muscles on the midline and continue blunt dissection down to the tracheal rings. Make an incision transverse between the rings; keep the incision small, only big enough for the tracheostomy tube. Alternatively, make a longitudinal incision to include two or three tracheal rings. Don't place the incision too close to the first tracheal ring, or it could potentially damage the cricoid cartilage and lead to subglottic laryngeal stenosis. Place stay sutures around the tracheal ring adjacent to the incision on either side of the surgical opening. The sutures will aid in placement of the tube and are left in, labeled cranial and caudal, to help when the tube is routinely replaced or cleaned, or if it gets dislodged.
The tube ideally is two-thirds to three-fourths of the tracheal diameter. If a specifically designed tracheostomy tube is not available, an endotracheal tube can be used but may need to be cut so it is short enough that it does not go into one bronchus. Fasten the tube in place by tying it around the neck with umbilical tape or gauze. The soft tissue is loosely closed with sutures and the skin is closed with non-absorbable sutures. It is important to allow any air escaping around the tube to vent to the outside and not accumulate under the skin.

External pharyngotomy is a type of intubation that can be performed for oropharyngeal surgery or orthopedic procedures of the mandible or maxilla. This type of intubation aids in the visualization of the area and allows for normal dental occlusion so that proper reduction of jaw fractures can be achieved. Initially place the endotracheal tube orally. Make a skin incision near the angle of the mandible. Pass hemostats bluntly through the incision into the caudal part of the pharynx. Remove the endotracheal tube adapter. grasp the tube. and pull it through from the pharynx through the subcutaneous tissue and skin incision. Replace the adapter and connect the tube to the breathing circuit. Secure with tape and suture. Extubation is done with the cuff deflated and the tube pulled through the skin incision.

Jet insufflation is a technique similar to the retrograde intubation except a small oxygen tube is connected to the need or catheter tip and the air is forcefully pushed into the lungs. It is imperative that the air can escape, otherwise causing lung injury.

Drug	Drug Class	Dose	Time to Effects	Effects on BP	Effects on HR	Efficacy in Species	Fetal Safety	Mechanism of action	Notes
Dopamine ($0.70 per ml)	Adrenergic /Dopaminergic inotrope	10-12 mcg/kg/min (Alpha effects)	2-5 minutes, last 5-10 minutes post cessation	++	+++	C, F, NHP, O, S, B	Most likely safe, but use with caution	Precursor to norepi and indirectly releases norepi causing vasoconstriction	2-10 mcg/kg/min, organ and GFR are increase sans vascular resistance
Dobutamine ($0.40 per ml)	Beta adrenergic inotrope	1-20 mcg/kg/min	2-5 minutes, Peaks at 10 minutes lasts 5-10 minutes post cessation	++ Dose dependent	++ Dose dependent	C, F, NHP, O, S, B, R(High dose)	Most likely safe	Increase myocardial contractility (can increase cardiac O2 demands)	Good for alveolar fluid buildup, patient must be hydrated, Liver is primary metabolizer. Can produce ectopic HB's, chest pain, palpitations, nausea and headaches.
Phenylephrine ($7.72 per ml)	Alpha adrenergic agonist Vasopressor	0.5-3 mcg/kg/min in NaCl or D5W	Immediate, lasts 5-10 minutes post cessation	+++	0-+ (report of reflex bradycardia, correct with atropine)	C, F, NHP, O, S, B, R, L	Safe and preferred in humans. Can cause uterine contraction.	Vasoconstriction, slight decrease in cardiac output with increase in coronary flow.	Use in hydrated patients
Ephedrine ($26.50 per ml)	Sym. Bronchodilator/ Vasopressor	0.03-0.25 mg/kg bolus, q 5 min	immediate	+++	+++	C, F, NHP, O, S, B, R, L	Safe for healthy fetus (excreted in milk)	releases norepi causing vasoconstriction (Can deplete norepi)	Do not use in cardiac patients
Norepinephrine ($39.00 per ml)	Alpha & Beta Adrenergic Vasopressor Cardiac inotrope	0.01-0.2 mcg/kg/min, max dose of 2 mcg/kg/ min. Add 4mg to 1L 5% Dextrose sol. run at 0.75-1.5 **mls**/kg/hr	immediate	+++	0-+ (report of reflex bradycardia, correct with atropine)	C, F, NHP, O, S, B, R, L	Do no use for viable pregnancy	Vasoconstriction, coronary artery dilator, slight increase in cardiac contractility	Use in hydrated patients. Good for Septic patients, high doses can lead to poor perfusion
Epinephrine ($6.00 per ml)	Alpha & Beta Adrenergic agonist	0.01-1 mcg/kg/min, START LOW	immediate, lasts 5-10 minutes post cessation	+++	+++	C, F, NHP, O, S, B, R, L	Do no use for viable pregnancy	Vasoconstriction, Increased cardiac contractility, increase in coronary and pulmonary flow	Better for increasing systolic BP, can Decrease Diastolic. Use in hydrated patients only. Can cause poor tissue perfusion at high doses.
Vasopressin ($69.50 per ml)	Hormone	20 pressor U/ml, 1-4mU/kg/min	Immediate	+++	0-+	C, F, NHP, O, S, B, R, L	Do no use for viable pregnancy	vasoconstriction	Hepatic flow increase

Capnography and Pulse Oximetry: Becoming BFFs

Stephen Cital RVT, SRA, RLAT, VTS- LAM (Res. Anesthesia)
http://www.stephencital.com

Technology as we all know is ever changing and advancing. Currently there are actually iPhone® cases that read ECG's (AliveCor®) as well as phone applications designed to help diagnose arrhythmias. The launch of a SpO2 sensor attachment (iSpO2®, Masimo Corp.) that can be used as a personal SpO2 monitor for fitness, endurance and aviation training increases the number of portable point of care devices.

*Not all devices are marketed, nor have FDA approval, for diagnostic use in the medical/veterinary setting.

I hope to enlighten readers on advances now available in veterinary medicine. Only one device is featured in this article, the Radical-7® Pulse CO-Oximeter by Masimo Corp., because it is the only FDA approved non-invasive CO-Oximeter now being marketed for veterinary use. The technology does not necessarily have heaps of data for animal efficacy yet, but it is an important advancement we as veterinary medical professionals should know is available. The utilization of the machine is gaining popularity in human medicine and now is the time for its utilization in veterinary medicine. Multiple human studies have found the individual components of the Masimo Radical-7® to be accurate within a reasonable margin of error for a point of care device.[1,2] Our facility, the California National Primate Research Center at UC Davis, recently received the Radical-7® non-invasive Co-Oximeter to test and decide if this piece of monitoring equipment can provide accurate enough information to be used regularly in our anesthetized and critical patients.

The use of standard pulse oximetry in veterinary medicine is widespread and a highly accepted form of non-invasive monitoring of oxygen saturation and pulse rate in critical, non-critical and anesthetized patients; the gold standard still being arterial blood gas sampling and EKG's. Superior oxygen saturation and perfusion is essential in the ICU and OR for the best patient outcome and reduced healing times as well as organ function.[1, 3] Standard pulse oximeters use two wavelengths of light to calculate oxyhemoglobin, which is a combination of total hemoglobin, methemoglobin, carboxyhemoglobin, as well as other forms of dyshemoglobins. Then a combined total as a percent is calculated and displayed as the SpO2%. The accepted reference interval for most species is 98-100%. With using the standard 2-wavelength pulse oximetry it is easy to misinterpret SpO2% readings as "within normal limits" when a dyshemoglobin may be present. [4]

Can you remember the last time you ordered a dyshemoglobin profile or have ever had to treat one? Not only are dyshemoglobins under diagnosed in human medicine, but more so in veterinary medicine. Dyshemoglobins are caused by a metabolic disorder, toxin ingestion, toxin exposure or acquired from medications.[1, 2, 4]

Now you might be asking how dyshemoglobins are assessed? Invasive Co-oximeters have been around for a while in human medicine, but with little use in veterinary medicine. Invasive Co-Oximetry entails an actual blood sample being run through a Co-Oximetry machine.

The Masimo Radical-7 non-invasive Co-Oximetry only entails clipping or taping a regular looking SpO2 sensor to the patient. One might be asking how a SpO2 sensor will differentiate the different types of hemoglobin's? The answer being, the new, advanced non-invasive Co-Oximeter uses >7 wavelengths of infrared light to calculate a combined oxyhemoglobin reading as a total SpO2% and due to the increased sensitivity from the extra wavelengths of infrared light it also calculates and shows readings for two

dyshemoglobins, methemoglobin and carboxyhemoglobin. The percentages of the two dyshemoglobins are then displayed as percentages on the screen. In our use of the machine we discovered detectable levels of methemoglobin on the Radical-7, confirming with invasive Co-Oximitery. This was startling and an important discovery as it allowed us to change our anesthesia protocols to minimize acquired MetHb buildup for better patient care.
The standard hemoglobin reading the machine also provides can be very useful for animals undergoing blood transfusions or hemodilution. The machine in addition gives a total arterial oxygen content reading for further non-invasive monitoring. This parameter is excellent for ventilator cases or respiratory compromised patients.

Cardiovascular perfusion and balanced fluid therapy is yet another fundamental part of traditional patient monitoring. However, advanced techniques to monitor such parameters can involve placing a central line for central venous pressure measurement or even placing and maintaining an arterial catheter. As we all know this may be difficult, not practical for the duration of a case or feasible in a certain species. Not to mention financially burdensome to the client or out of the scope of your practices needs and abilities. This in turn can lead to less effective treatment and response in our patients. The Masimo Radical-7 includes the features of Perfusion Index (PI) and Pleth-Variability Index (PVI). These are two new monitoring parameters and can be very telling when obtained correctly. The perfusion index is the ratio of the pulsatile blood flow to the non-pulsatile or static blood in peripheral tissues. What this means is now we can monitor peripheral tissue perfusion in our patients non-invasively giving better insight into our fluid therapy management, cardiac/renal output and efficacy of medications. A defined reference variable is not yet established in the canine or feline patient or any other specie for that matter, other than humans, which tends to be quite broad. However, the PI parameter as well as all of the other non-conventional parameters, is great for trending and monitoring. When monitoring the PI for anesthetic or pain management purposes the perfusion status is usually the converse of supporting a good PI in non-anesthetized critical patients by the drugs causing vasodilation. When proper onset of anesthetic or analgesics has taken effect we see a spike in the PI showing efficacy. This technology is particularly exciting for patients undergoing kidney transplant, open-heart surgery, thrombin disorders and trauma patients.

The Pleth Variablity Index (PVI) is a new technology even in human medicine. It is a measurement in the change of perfusion index with a complete respiratory cycle. With this in mind, PVI is most reliable with patients undergoing mechanical ventilation. In a scientific abstract presented at the American College of Veterinary Anesthesiologists conference, one research group found that the PVI had good correlation in detecting hypovolemia and a return to normovolemia in dogs, but could not be used in definitively stating hypervolemia.

The last parameter is the acoustic respiratory monitor. What this acoustic respiratory monitor (aRR) has to offer is continuous respiration monitoring without endotracheal tube placement. It is perfect for the ICU setting, especially with quiet or recumbent patients. It is also idea for field knockdowns or large or exotic animals. The readings of the aRR on the Radical-7 have been nearly identical to our intubated patients with use of a traditional respirometer.

In all the Masimo Radical-7 offers an all-inclusive and advanced monitoring device. It has brought attention to parameters never monitored before as well as reminded us of the importance of drug selection. Formal validating studies are underway with the Masimo Radical-7 for the veterinary community and we anticipate positive results.

Capnography: Although capnographs are becoming more and more standard on multiparameter monitors correct or accurately interpreting of the waveforms is still fairly low in veterinary medicine. Typically, the waveforms are looked upon as just breaths. However, each wave is indicative of a breath or lack thereof.

There is so much more that can be gathered from the wave stature and anatomy. Understanding the waveforms better will allow anesthetists to gauge the quality of the breath, possible occlusions or leaks and perfusion quality of the animal.

References upon request.

Becoming an Anesthetic Mixologist

Stephen Cital RVT, SRA, RLAT, VTS-LAM (Res. Anesthesia)
http://www.stephencital.com

There are literally thousands of combinations of sedatives/tranquilizers, muscle relaxers, neurosteriods, opioids, dissociative agents, paralytics and so on to choose from. Selecting what is safe, practical and even cost efficient is necessary for our patients, keeping in mind the specific signlamnet and health status of our individual patient.

Benzodiazepines are reported to enhance the positive subjective effects of opioids (euphoria) but it is unclear whether the reinforcing effects are additive or synergistic. Either way we see a great MAC sparing effect with the combination of the two medications.

When creating a multimodal anesthetic plan utilizing volatile anesthetic or TIVA we should always consider this formula.

Analgesia + Muscle relaxation + Sedation

Often one of these medication on the equation will have anxiolytic effects as well.

OPIOID CONTINUOUS RATE INFUSIONS
Premedication with a mu opioid agonist will provide an effective loading dose for any mu opioid CRI

Fentanyl (50mcg/mL or 0.05mg/mL)

- Commonly used in a CRI as the sole agent or can be combined with ketamine +/- lidocaine.
- A single IV bolus will only last approximately 20-30 minutes.
- Fentanyl has a context sensitive half life. When used as a CRI for greater than 2 hours the drug will start to accumulate in the tissues. Once accumulation has occurred the plasma concentration does not decrease rapidly once the CRI is discontinued. To prevent a prolonged recovery, it may be beneficial to decrease the fentanyl CRI rate and/or make adjustments to the vaporizer about 30-40 minutes prior to the end of surgery. The effects tend to last much longer in cats compared to dogs.
- Extremely high dosages may depress ventilation and cause bradycardia.
- Fentanyl does not require dilution when used in a syringe pump
- An IV bolus (loading dosage) of 1-5mcg/kg should be given prior to the start of the CRI if no other mu agonist opioid has been administered.
- CRI rate (intra-op): 0.1-0.7mcg/kg/min (6-42mcg/kg/hr) **It is recommended to start with 0.1mcg/kg/min and adjust the dosage up as needed depending on patient response to surgical stimulus. If the patient responds to surgical stimulation then it is recommended that a bolus (1-3mcg/kg) be administered and the CRI rate increased in 0.1 increments until no further surgical stimulation occurs.
- CRI rate (post-op): 0.03-0.05mcg/kg/min (2-3mcg/kg/hr)

Remifentanil (1mg powder)

- Commonly used alone in a CRI or can be combined with ketamine +/- lidocaine.
- Metabolized by nonspecific plasma esterases to inactive metabolites. This makes remifentanil superior to fentanyl for patients with renal or hepatic dysfunction.
- Rapid onset of action and short duration of action. It must be administered as a CRI because the short duration of action limits the use as a bolus injection.
- It has non-cumulative effects within the body so recovery is rapid after CRI is discontinued.
- Extremely high dosages may cause profound sedation, respiratory depression and bradycardia.
- Supplied as a 1mg powder that must be reconstituted with sterile saline prior to use.

Dilution: mix 1mg powder in 20mL NaCl → 50mcg/mL or mix 1mg powder in 10mL NaCl → 100mcg/mL
- Loading dosage: 1-5mcg/kg IV should be given prior to the start of the CRI if no other mu agonist opioid has been administered.
- CRI rate: 0.1-0.7 mcg/kg/min

Hydromorphone (2mg/mL)
- Can be used alone or in combination with ketamine +/- lidocaine.
- Does not cause histamine release.
- Dilution: add 2mg (1mL) to 9mL NaCl → 0.2mg/mL
- Loading dosage: 0.03-0.05mg/kg IV prior to starting the CRI if no other mu agonist opioid has been administered.
- CRI rate: 0.3-0.8mcg/kg/min (0.02-0.05mg/kg/hr)

Morphine (15mg/mL)
- Commonly used alone or in combination with ketamine +/- lidocaine.
- Caution with use in cats. Morphine CRIs are not commonly administered alone to cats when awake due to the likelihood of causing excitation.
- Morphine is light sensitive. The syringe or fluid bag should be covered when using a morphine CRI long term.
- Dilution: add 15mg (1mL) to 9mL NaCl → 1.5mg/mL or add 30mg (2mL) to 8mL NaCl → 3mg/mL
- Loading dosage: 0.1-0.2mg/kg IV (very slowly) should be given prior to the start of the CRI if no other mu agonist opioid has been administered.
- CRI rate: 2-6mcg/kg/min (0.1-0.3mg/kg/hr)

Methadone (10mg/mL)
- Can be used alone or in combination with ketamine +/- lidocaine.
- Also acts as an NMDA receptor antagonist to help treat and prevent central sensitization.
- Dilution: add 10mg (1mL) to 9mL NaCl → 1mg/mL
- Loading dosage: 0.1-0.5mg/kg IV prior to starting the CRI if no other mu agonist opioid has been administered.
- CRI rate: 0.05-2mg/kg/hr

ADJUNCT CRIs FOR ADDITIONAL PAIN MANAGEMENT

Ketamine (100mg/mL)
- Classified as an NMDA receptor antagonist that effectively blocks central sensitization from occurring in the dorsal horn of the spinal cord and helps prevent hyperalgesia and allodynia.
- Ketamine does not have any direct analgesic effects but it is used as an adjunct to other analgesic drugs such as opioids. It may help improve opioid receptor sensitivity. DO NOT use ketamine as the sole analgesic agent.
- Dosages used for the CRI are given at sub-anesthetic levels so none of the dissociative effects are seen during CRI administration.
- Starting a ketamine CRI prior to a painful stimulus will provide the best means of preventing CNS sensitization but it is still effective in patient's that present with established pain.
- Loading dosage: 0.5mg/kg IV of ketamine should be given prior to starting the CRI in order to achieve initial therapeutic blood levels. Induction with ketamine/diazepam or Telazol® will provide an effective loading dose.
- CRI rate (intra-op): 10-20mcg/kg/min

- CRI rate (post-op): 2-10mcg/kg/min for at least 24 hours

Lidocaine (20mg/mL)

- MAC sparing and analgesic effects when administered as a CRI intra-op.
- Classified as a sodium channel blocker and a class IB antiarrhythmic.
- Displays free radial scavenging effects which may be helpful at preventing reperfusion injury.
- Acts as an inflammatory modulator by decreasing neutrophil chemotaxis and platelet aggregation.
- Acts as a prokinetic that enhances gut motility and helps prevent ileus.
- NOT recommended for use in cats due to its potential for toxicity. If used, do not exceed a dosage of 10mcg/kg/min and monitor closely for seizure activity and bradycardia.
- Commonly used as a first line treatment for ventricular premature complexes (VPC) or ventricular tachycardia.
- Some brands of lidocaine are sensitive to light. If lidocaine comes in a brown bottle the syringe or fluid bag containing the lidocaine should be covered when used as a CRI long term.
- Loading dosage: 1-2mg/kg IV of lidocaine should be given prior to starting the CRI in order to achieve an appropriate therapeutic level.
- CRI rate: 25-75mcg/kg/min

Dexmedetomidine (500mcg/mL or 100mcg/mL)

- Generally combined with an opioid CRI to enhance analgesia and sedation when an opioid CRI alone is not enough.
- Will greatly reduce MAC of inhalants when used intra-operatively.
- Commonly used during the post-operative period as a treatment for emergence delirium or when the patient would benefit from long term sedation during the post-operative period.
- Can be given in combination with ketamine, lidocaine and opioids
- Cardiovascular effects (significant bradycardia, biphasic effects on blood pressure) will likely be seen during CRI administration. Vital signs should be monitored closely. It is best to avoid a dexmedetomidine CRI if the patient has cardiovascular disease.
- Inhibits antidiuretic hormone (ADH) so an increase in urine production may be seen. The bladder should be expressed prior to recovery if used as an intra-operative CRI.
- Inhibits insulin release so a transitory hyperglycemia may be seen. Avoid a dexmedetomidine CRI if serial glucose values need to be obtained.
- Loading dosage: 0.5-1mcg/kg IV should be given prior to starting the CRI in order to achieve an appropriate therapeutic level.
- CRI rate: 0.5-3mcg/kg/hr

Medetomidine

- Used in the same manner as dexmedetomidine.
- Loading dosage: 1-2mcg/kg IV prior to starting the CRI.
- CRI rate: 1-2mcg/kg/hr

* Used with permission from Palmer, D. (2013). Information originally published in the VSPN Notebook®, 4th ed. Veterinary Support Personnel Network/Veterinary Information Network (http://ww.vin.com). Davis, CA.

TABLE 1: Dosages for constant rate infusions (CRIs) used in CATS.

Drug	Loading Dose	CRI dose	Quick Calculation	Comments
Morphine (M)*	0.10 mg/kg IM	0.03 mg/kg/hr (0.5 mic/kg/min)	Add 15 mg to 500 ml fluid & run at 1 ml/kg/hr	Cat may need light sedation; can be combined with K &/or L
Hydromorphone (H)	0.025 mg/kg IV	0.01 mg/kg/hr	Add 5 mg to 500 ml fluid & run at 1 ml/kg/hr	May cause hyperthermia; can be combined with K &/or L
Fentanyl (F)	0.001-0.003 mg/kg IM or IV (1-3 mic/kg IV)	2-5 mic/kg/h (0.03-0.08 mic/ kg/m)post-op 5-20 mic/kg/h (0.08-0.3 mic/ kg/m intra-op	For 5 mic/kg/h, add 2.5 mg to 500 ml fluid & run at 1 ml/kg/hr	2.5 mg=50 ml F, remove 50 ml LRS before adding F; can be combined with K &/or L.
Methadone	0.1-0.2 mg/kg IV	0.12 mg/kg/hr	Add 60 mg to 500 ml fluid & run at 1 ml/kg/hr	MAY cause sedation; can be combined with K &/or L.
Butorphanol	0.1 mg/kg IV	0.1-0.2 mg/kg/hr	Add 50 mg to 500 ml fluid & run at 1 ml/kg/hr for 0.1 mg/kg/hr	Only moderately potent & has ceiling effect - use as part of multimodal protocol
Ketamine (K)*	0.25 mg/kg IV	0.12-0.6 mg/kg/hr (2 -10 mic/kg/ min)	Add 60 mg to 500 ml fluid & run at 1 ml/kg/hr for 0.12 mg/kg/hr	Generally combined with opioids; may cause dysphoria
Lidocaine (L)	0.25 mg/kg IV	1.5 mg/kg/hr (25 mic/kg/min) Some sources recommend no more than 10 mic/kg/min in cats	Add 750 mg to 500 ml fluid & run at 1 ml/kg/hr 10 mic/kg/min would be 300 mg lidocaine in 500 ml fluid with a rate of 1 ml/kg/hr	750 mg=37.5 ml, remove 37.5 ml LRS before adding L; can be combined with opioid &/or K; **Lidocaine MAY be contraindicated in the cat due to cardiovascular effects.**
Medetomidine (Med) or Dexmedetomidine (D)	1-5 mic/kg Med 1-2 mic/kg D Can be IV or IM May not be necessary	0.001-0.004 mg/kg/hr Med (1-4 mic/kg/hr) 0.0005-0.002 mg/kg/hr D	Add 500 mic Med or 250 mic D (0.5 ml of either) to 500 ml fluid and run 1-4 ml/kg/ hr	Provides analgesia and light sedation. Excellent addition to opioid CRI, or can be administered as solo drug CRI.
Morphine* / Ketamine*	M: 0.10 mg/kg IM K: 0.25 mg/kg IV	0.03 mg/kg/hr M & 0.12 mg/kg/hr K	Add 15 mg M & 60mg K to 500 ml fluid & run at 1 ml/kg/hr	Can be administered up to 3 ml/kg/hr but dysphoria MAY occur. Can substitute, F, or methadone for M.
Morphine / Ketamine / Lidocaine (MLK)	M: 0.10 mg/kg IM K: 0.25 mg/kg IV L: 0.25 mg/kg IV	0.03 mg/kg/hr M, 0.12 mg/kg/hr K; 1.5 mg/kg/hr L	Add 15 mg of M, 60 mg K and 750 mg (or 300 mg) L to 500 ml fluid & run at 1 ml/kg/hr	Can substitute H, F or methadone for M.

* Any of the drug amounts in the bag of fluids can be decreased and the fluids administered at a higher rate if necessary. For example, for morphine, ketamine and morphine/ketamine infusions, 7.5 mg of morphine & 30 mg of ketamine can be used and the CRI administered at 2 ml/kg/hr if more fluids are needed.

TABLE 2: Dosages for constant rate infusions (CRIs) used in DOGS.

Drug	Loading Dose	CRI dose	Quick Calculation	Comments
Morphine (M)*	0.5 mg/kg IM (or 0.25 mg/kg SLOWLY IV)	0.12-0.3 mg/kg/hr (2.0 mic/kg/min-3.3mic/kg/min	Add 60 mg to 500 ml fluid & run at 1 ml/kg/hr for 0.12 mg/kg/hr	MAY cause sedation; can be combined with K &/or L.
Hydromorphone (H)	0.05-0.1 mg/kg IV	0.01-0.05 mg/kg/hr	Add 5-24 mg to 500 ml fluid & run at 1 ml/kg/hr	MAY cause sedation; can be combined with K &/or L.
Fentanyl (F)	0.001-0.003 mg/kg IM or IV (1-3 mic/kg IV)	2-10 mic/kg/h (0.03-0.2 mic/kg/m)post-op 3-40 mic/kg/h (0.05-0.7 mic/kg/m intra-op	For 5 mic/kg/h, add 2.5 mg to 500 ml fluid & run at 1 ml/kg/hr	2.5 mg=50 ml F, remove 50 ml LRS before adding F; can be combined with K &/or L; Intra-op dose can be up to 20-40 mic/kg/h
Methadone	0.1-0.2 mg/kg IV	0.12 mg/kg/hr	Add 60 mg to 500 ml fluid & run at 1 ml/kg/hr	MAY cause sedation; can be combined with K &/or L.
Butorphanol	0.1 mg/kg IV	0.1-0.2 mg/kg/hr	Add 50 mg to 500 ml fluid & run at 1 ml/kg/hr for 0.1 mg/kg/hr	Only moderately potent & has ceiling effect - use as part of multimodal protocol
Ketamine (K)*	0.25 mg/kg IV	0.12-0.6 mg/kg/hr (2 -10 mic/kg/min)	Add 60 mg to 500 ml fluid & run at 1 ml/kg/hr for 0.12 mg/kg/hr	Generally combined with opioids; may cause dysphoria; post-op dose may be higher
Lidocaine (L)	0.5 – 1.0 mg/kg IV	1.5-3.0 mg/kg/hr (25-50 mic/kg/min)	Add 750 mg to 500 ml fluid & run at 1 ml/kg/hr for 25 mic/ kg/min	750 mg=37.5 ml, remove 37.5 ml LRS before adding L; can be combined with opioid &/or K.
Medetomidine (Med) or Dexmedetomidine (D)	1-5 mic/kg Med 1-2 mic/kg D Can be IV or IM May not be necessary	0.001-0.004 mg/kg/hr Med (1-4 mic/kg/hr) 0.0005-0.002 mg/kg/hr D	Add 500 mic Med or 250 mic D (0.5 ml of either) to 500 ml fluid and run 1-4 mls/kg/hr	Provides analgesia and light sedation. Excellent addition to opioid CRI, or can be administered as solo drug CRI.
Morphine* / Ketamine*	M: 0.5 mg/kg IM K: 0.25 mg/kg IV	0.12 mg/kg/hr M & 0.12 mg/kg/hr K	Add 60mg M & 60mg K to 500 ml fluid & run at 1 ml/kg/hr	Can be administered up to 3 ml/kg/hr but sedation or dysphoria MAY occur. Can substitute H, F or methadone for M
Morphine / Ketamine / Lidocaine (MLK)	M: 0.5 mg/kg IM K: 0.25 mg/kg IV L: 0.5 mg/kg IV	0.12 mg/kg/hr M, 0.12 mg/kg/hr K; 1.5 mg/kg/hr L	Add 60 mg of M, 60 mg K and 750 mg L to 500 ml fluid & run at 1 ml/kg/hr	Can substitute H, F or methadone for M. Dr. Muir's dose is 3.3 mic/kg/min M, 50 mic/kg/min L; 10 mic/kg/min K.

*Any of the drug amounts in the bag of fluids can be decreased and the fluids administered at a higher rate if necessary. For example, for morphine, ketamine and morphine/ketamine infusions, 30 mg of morphine & 30 mg of ketamine can be used and the CRI administered at 2 ml/kg/hr if more fluids are needed.

Nursing for the Post-Operative Cardiac Patient

Stephen Cital RVT, SRA, RLAT, VTS-LAM (Res. Anesthesia)
http://www.stephencital.com

While the title of these proceedings can suggest surgical intervention or techniques performed on the actual heart of the patient the intent is the post-operative care for animals with pre-existing cardiac disease. Nonetheless, for those of you that do perform cardiac surgery the following techniques still apply, sourced from the McGill University protocols of critical care management. A holistic approach to the patient and watching trends rather than focusing on specific numbers is still standard. Goal directed monitoring also has its place in intensive care settings, but can lead to missed clinical signs or features.

It is crucial to have trained staff monitor critical patients. It may even be necessary to have one dedicated technician for a particular animal in the ICU setting with little distraction, especially of the patient was a ASA IV or above. Advanced techniques to monitor hemodynamtic parameters can involve placing a central line for central venous pressure measurement or even placing and maintaining an arterial catheter. This sort of monitoring is still a gold standard. As we all know this may be difficult and not practical for the duration of a case or feasible in a certain species. Not to mention financially burdensome to the client or out of the scope of your practice's needs and abilities. This, in turn, can lead to less effective treatment and response in our patients. There is now non-invasive technology which includes Perfusion Index (PI) and Pleth-Variability Index (PVI). These are two newer monitoring parameters that can be very telling when obtained correctly. The perfusion index is the ratio of the pulsatile blood flow to the non-pulsatile or static blood in peripheral tissues. What this means is now we can monitor peripheral tissue perfusion in our patients non-invasively giving better insight into our fluid therapy management, cardiac/renal output, and efficacy of medications. A defined reference variable is not yet established in the canine or feline patient or any other species for that matter, other than humans, which tends to be quite broad. However, the PI parameter, as well as all of the other non-conventional parameters, is great for trending and monitoring. The PI is also a great tool for assessing the efficacy of opioids and epidurals. When full onset of the opioid or epidural occurs, we see a spike in the PI showing via vasodilation.

The Pleth Variability Index (PVI) is a new technology even in human medicine. It is a measurement of the change of perfusion index with a complete respiratory cycle. With this in mind, PVI is most reliable with patients undergoing mechanical ventilation. In a scientific abstract presented at the American College of Veterinary Anesthesiologists conference, one research group found that the PVI had a good correlation in detecting hypovolemia and return to normovolemia in dogs, but could not be used in definitively stating hypervolemia. Several more recent veterinary papers on PVI have come out with positive conclusions as to the reliability of predicting fluid responsiveness using this non-invasive tool.

History- A clear history should be gathered about the patient, from its primary veterinarian, surgeon, anesthetist and any other sources.

- Patient background (age, sex, code status)
- Type of operation and outcome
- Indications for operation and pre-operative diagnosis
- Current inotropes, vasopressors, or anti-hypertensives (if any)
- Need for cardiac pacing when applicable
- Bleeding risks and clotting times
- Other significant co morbidity, with emphasis on those conditions that may alter the post-operative management or course (asthma, diabetes, renal failure, hepatic failure, etc.)
- Pre-operative medications
- Allergies

Physical exam and assessment

- If indeed the animal did endure a cardiac surgery (typical in research or university settings) assure that the endotracheal tube is in proper position and the patient has equal air entry bilaterally. Remember that tube displacement or pneumothoraxes' can occur or become apparent at any moment.
- Verify that the patient's oxygen saturation is adequate. Check the ABG results as soon as they are available. If this is not available an SpO2 and monitoring of the pH, bicarbonate and electrolytes must be evaluated.
- Verify correct ventilator settings.
- Check the initial hemodynamic readings (HR, BP, cardiac output and index, CVP) and determine what vasoactive infusions the patient is on and at what rates.
- Check the patient's heart rhythm. Verify pacemaker settings if the patient is connected to one.
- Examine heart sounds. Listen for murmurs.
- Check all peripheral pulses. Do repeated assessments if there is concern for acute limb ischemia. A Doppler can be placed for on a peripheral limb for continuous evaluation.
- Do a more complete neurologic exam when the patient begins to awaken from GA.

Labs and tests Electrocardiogram

- Note any changes from pre-op ECG
- Rhythm - post-operative bradycardias, blocks, or atrial fibrillation
- ST-T changes - diffuse non-specific changes are not uncommon and may reflect pericardial inflammation or ischemic events
- Chest X-Ray
- Rarely used in the non-research sector, verify correct position of the Swan-Ganz catheter.

Laboratory Results

- Hemoglobin
- Coagulation parameters (PLT, PT, PTT, ACT)
- Potassium, magnesium, calcium - a vigorous diuresis is common in the first few hours after the OR. This can lead to significant hypokalemia and hypomagnesaemia which increases the likelihood of post-operative dysrhythmias. Standing orders are in place to replace these electrolytes.
- Glucose - tight glycemic control post-operatively reduces morbidity in humans.
- Cardiac markers - elevations of CPK, CPK-MB, and troponins are non-specific. They should be assessed as part of the overall clinical picture including the hemodynamic status of the patient and the EKG.

Warming

Effects of hypothermia

- Predisposes to ventricular dysrhythmias and lowers VF threshold
- Increases SVR; increases afterload and myocardial workload
- Patient shivering causes increased peripheral O2 consumption
- Decreases CO2 production; a patient who has a respiratory alkalosis (low PCO2) on initial ABG usually will increase their PCO2 with rewarming
- Coagulopathy; impairs platelet function and the coagulation cascade. Rewarming is an important part of the treatment of a bleeding patient.

Transfusion

The principle objective when giving PRBC's is the improvement of inadequate oxygen delivery and the minimization of adverse outcomes as a result of this. In a patient who is actively bleeding and thus who's hemoglobin mass is not in a steady state, one must be more liberal in transfusing PRBC's to avoid severe impairments in peripheral oxygen delivery. However, with a patient who is not bleeding rapidly, one can take a more deliberate approach to transfusion. Transfusions are often over prescribed. If the patient appears to

have a normal neurologic status and respiratory pattern/rate, a transfusion may not be warranted. There are several potential risks associated with the transfusion of red blood cells.

Hemodynamic management

Hypotension and low cardiac output

1. *BP = CO x SVR*
2. *CO = HR x SV (stroke volume)*
3. *Stroke volume is determined by preload, contractility, and afterload*
4. *Bradycardias or tachydysrhythmias that decrease ventricular filling can decrease CO.*

There are numerous causes for hypotension post-operatively. Proper management of the hypotensive patient in the ICU requires that the precise etiology for the hypotension is determined and therapy is directed towards reversal of this specific problem. Equation 1 demonstrates that hypotension can be caused by a "pump problem" (low cardiac output) or a low SVR (arterial "circuit" problem). The following is an approach to managing the hypotensive patient:

1. Look at the recent hemodynamic parameters.
2. Assess the cardiac output/index. Is this a "pump" problem? Or is it due to low SVR?
3. Look at the cardiac rhythm.
4. Look at the CVP to assess preload.
5. Is the afterload high?
6. Is contractility decreased?

- Is this tamponade? Look at the recent hemodynamic parameters obtained from the Swan-Ganz catheter or evaluate via echo.
- Assess the cardiac output/index.
- If the cardiac index is in the normal range or high, then the patient does not have a significant "pump" problem and the cause of the hypotension is secondary to diminished peripheral arterial tone (low SVR). A vasopressor agent should be considered. The differential diagnosis of low SVR includes:
    - SIRS - a proportion of patients post CPB will have significant cytokine increases
    - Sepsis
    - Anaphylactic or anaphylactoid reactions
    - Drug-induced, toxicological - nitrates, antihypertensives, narcotics and sedatives, etc
    - Adrenal insufficiency (Was the patient steroid dependent pre-operatively?)
    - Hyperthyroidism, hypothyroidism
    - Neurogenic (spinal) shock
- If the cardiac index (CI) is low then the cause of the hypotension is inadequate flow or a "pump" problem.
- Look at the cardiac rhythm. Absolute or relative bradycardias or tachycardias can lead to decreased CO and should be corrected.
- Look at the CVP to assess preload. A patient with a low CI and a CVP that is "relatively" low should be given a fluid challenge. Remember, what you really are interested in is a volume measurement (preload= right or left end-diastolic volume), but what you are measuring are pressures (CVP = Right or left ventricular end-diastolic pressures).
- High afterload. Secondary to vasoconstriction and hypertension.
- Decreased contractility. This should be managed with inotropic agents while simultaneously looking for the cause.
- Tamponade
- Acute valvular regurgitation. Check for a new regurgitant murmur.

Inotropes and vasopressors

Inotropes

1. Adrenergic (catecholamine)

- Dobutamine - beta-agonist (ß1 >ß2). Increases contractility and HR. ß2 effect can sometimes decrease SVR and BP. ß1 effect can cause dysrhythmias. Start at 2.5 mcg/kg/min. Titrate upward by 2.5 mcg/kg/min until adequate cardiac index.
- Epinephrine -alpha and beta agonist (ß > alpha). Increases HR, CO, and SVR. Generally a second-line inotrope. A subset of patients who do not respond to dobutamine will respond to epinephrine. Potential detrimental effects include significant increases in myocardial oxygen consumption, increased lactic acidosis, arrhythmias. Start at 0.5 to 1.0 mcg/min and increase by these amounts until adequate cardiac index.
- Dopamine - stimulates dopaminergic, beta, and alpha receptors in dose-dependent fashion. May be less effective in cats. Inotropic effect (beta-effect) predominates in the 5 to 10 mcg/kg/min range. In humans, low doses (2 - 4 mcg/kg/min) it has been purported to have beneficial renal protective effects ("renal-dose dopamine"). While it can increase urine output by several mechanisms, there is little evidence that it improves creatinine clearance or decreases the incidence of acute renal failure.

Vasopressors

1. *Adrenergic (catecholamine)*
   - Norepinephrine -Strong alpha agonist with beta activity as well. Causes vasoconstriction and thus increases SVR and BP. Theoretically, since it has inotropic activity as well, it is less likely to cause a decrease in cardiac output due to increased afterload compared to a pure alpha agonist such as phenylephrine. Negative effects include myocardial and mesenteric ischemia, dysrhythmias, and decreased cardiac output due to afterload increases.
   - Phenylephrine (Neosynephrine) - Pure alpha agonist. Can be used as a continuous infusion but more commonly used as bolus for sudden severe hypotension not responding to volume infusion.
2. *Peptides*
   - Vasopressin - used for hypotension with a normal or high cardiac output and low SVR state that is refractory to norepinephrine. Has a significant side effect profile including myocardial and mesenteric ischemia.

ECG's: The Basics

David Liss, BA, RVT, VTS (ECC, SAIM)

Basic Conduction:
ECG: Electrocardiogram- Studies the electrical activity generated by the heart. The heart has two components, a mechanical pump, for blood flow, and an electrical generator to push the pump.

Depolarization/Repolarization
Depolarization- Electrical current transfer across cardiac cell membranes causing contraction. Ca++/K+ ions move into cell.

Repolarization- Movement of ions back out of cells, relaxing of muscles. K+/Na+ move out-K+(quickly to establish negative gradient)

This process allows the muscle cells to contract in sequence, allowing a synchronized contraction of many, many muscle cells, allowing the heart to pump blood to the lungs and body.

Normal Conduction Pathway:
ALL POSSESS AUTOMATICITY (or the ability to discharge without outside stimulus)

SA Node
Initiates electrical signal-Under normal physiologic conditions
Is in right atrium
Firing of SA node sends contraction signals through the right atrium

AV Node
Directs electrical signal towards the ventricles
Is just above the right ventricle
Failsafe- Can fire on its own (automaticity)-If the SA node stops working

Bundle of His
Large bundle of electrical fibers directing the signal towards the Bundle Branches

Right/Left Bundle Branches
Separate groups of electrical fibers powering the right and left ventricles

Purkinjie Fibers
Terminal fibers at the end of the conduction sequence
Can fire on their own (automaticity)

Cellular Electrical Potentials:

Resting
Cardiac cell is polarized (relative negative inside as opposed to outside cell)
Na+/Cl- Highly Extracellular
K+-Mainly Intracellular

Depolarization
Stimulus applied
Na+/Cl- rush into cell causing a net positive effect intracellularly
Ca2+- also shifts inside

Magnitude of electrical activity is proportional to the length and diameter of the muscle
Dilitation- Lengthening and Hypertrophy-Thickening can see an increase in electrical activity on ECG

Repolarization

K+ begins to leave cell as permeability increases
The Sodium-Potassium Pump removes intracellular sodium

Normal ECG:

The Waves

P-wave

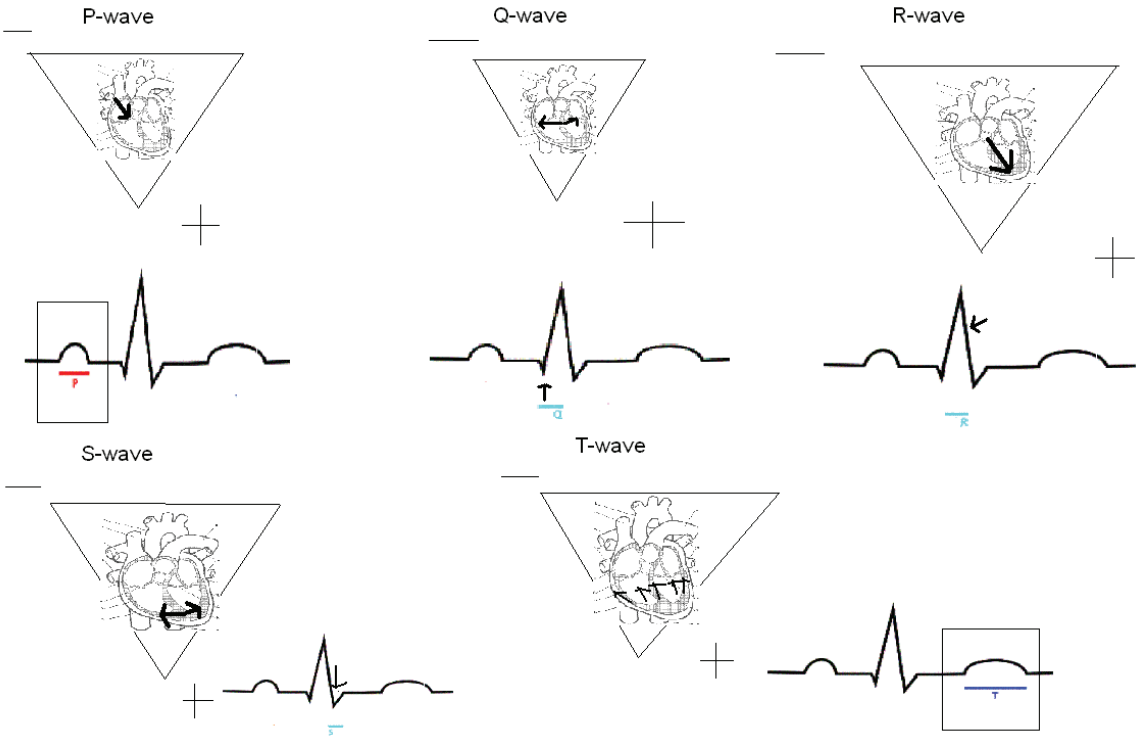
First component of normal ECG and is produced by depolarization of the atria.
Width indicates time for contraction
Height indicates amount of current generated
Positive in Lead II

QRS Complex

Second Component comprised of 3 separate waves in the ECG
Represents ventricular depolarization
Q-wave- First negative wave following the p-wave
R-wave- First positive wave after the p-wave
S-wave- Second negative wave after the p-wave

T-wave

Final positive wave after the QRS Complex
Represents Ventricular Repolarization (atrial repolarization hidden in QRS Complex)



Basics of Arrythmias:

Questions to ask when interpreting ECG's:

-Is the heart rate normal or abnormal?

-Is the rhythm regular or irregular?
-Can I identify P waves and QRS-T complexes?
-Is there a P-wave for every QRS and a QRS for every P-wave?
-Do all the complexes look roughly the same length/shape/size?

Slow rhythms:

Sinus Bradycardia

Abnormal rate
Regular rhythm
Yes PQRST
Yes P for QRS and QRS for P
Complexes look the same-Complexes look "sinus" Or Normal QRS

First Degree AV Block

Heart rate abnormal
Irregular
There are p-wave and QRS Complexes
Yes P for every QRS or QRS for every P
Complexes do not look the same, elongated P-R interval

Third Degree AV Block

Heart rate abnormal (slow)
Irregular rhythm
There are p-waves and QRS Complexes
Not a p for every QRS or QRS for every P
Complexes do not look the same (VPC's interspersed)
There is no relation between P-waves and QRS complexes

Fast rhythms

Sinus Tachycardia

Abnormal rate
Regular rhythm
Yes PQRST
Yes P for QRS and QRS for P
Complexes look the same-QRS is Sinus

Ventricular Tachycardia

Abnormal rate
Appears Regular
No P waves QRS Complexes wide and abnormal
No P for QRS or QRS for P
Complexes may look the same or different depending on where in the ventricle the electrical signal has originated from

Ventricular Fibrillation

Unable to determine rate
Not regular
No P waves/QRS Complexes
No P for QRS or QRS for P
There are no identifiable complexes
THIS IS A RHYTHM OF CARDIAC ARREST!

Atrial Fibrillation

Atrial rate-Very fast
Ventricular rate-Slow to fast
Irregular rhythm
No stable P-waves present, appear as fibrillation
Not a regular PQRST configuration
QRS Complexes sometimes normal morphology

Supraventricular Tachycardia

Atrial/Ventricular rate very fast, sometimes 300+ BPM
Irregular (may speed up and/or slow down-Paroxysmal)
P-waves may or may not be present
Appears to be a QRS for every P wave
QRS Complexes are regular to thin

Rhythm's with Premature Beats

APC's

Premature firing from an atrial focus
Heart rate usually normal to slow
Irregular rhythm (premature beat present)
P-wave may or may not be present
QRS for every P of non premature beats
QRS of normal morphology because atrium initiated contraction

VPC's

Heart rate normal to slow or fast
Irregular rhythm (Premature beats present)
P-wave not present with premature beats
QRS for every P with normal Sinus beats
QRS of wide and bizarre morphology of premature beats
Premature firing of the ventricles-Can occur at multiple foci-Multi-focal

The A, B, and Cs of Transfusion Medicine

David Liss, BA, RVT, VTS (ECC, SAIM)
Los Angeles, CA, USA

Introduction

Transfusion medicine, in veterinary medicine, has come a long way from the days of bleeding a donor dog and immediately infusing it into another without any regard for blood type or reactions. With the advent of commercial blood banks and clinical research, the veterinary team can support a critically ill patient with blood products/component therapy in a safe controlled manner. Understanding the need for transfusions, products available, the physiology of transfusions, and how to administer/monitor a transfusion is essential in any veterinary setting. Technicians are paramount to the successful completion of a transfusion, as we are often charged with monitoring the patients receiving the various products available. This presentation will serve as a foundation for the veterinary technician to be able to perform high-quality transfusion medicine.

The need for a transfusion

The need for a blood/blood product transfusion can arise in various situations. With acute hemorrhage, oxygen-carrying red blood cells are lost, as well as essential plasma proteins. Water, proteins contributing to oncotic pressure, and red blood cells which carry oxygen to organs are all not able to provide their normal physiologic functions. Replacing the vascular volume with crystalloid or colloid fluids enhances vascular volume and perfusion to an extent, but does not replace the oxygen-carrying capacity the red blood cells provide. Whole blood transfusions might be used here to replace what was lost. In the case of non-hemorrhagic anemia, a transfusion of packed red blood cells may be required. These patients do not need the plasma component, and having access to blood components might be advantageous to minimize the potential for a reaction. Patients with coagulopathies or other conditions may require a plasma transfusion to replace those and slow hemorrhage caused by the loss of clotting factors. There are also other rare conditions (such as von Willebrand's disease (vWD) that may receive a transfusion as a medical therapy. Finally, transfusions of albumin (human or canine), intravenous immunglobulin (IVIG), or antivenin should be considered "transfusions" and monitored according to the same guidelines as blood products.

Transfusion products

Blood products used in transfusion medicine are numerous. They include: fresh whole blood, stored whole blood, packed red blood cells, fresh frozen plasma, frozen plasma, cryoprecipitate, cryo-poor plasma, platelet-rich plasma, albumin products (human or canine). A product rarely used in veterinary medicine but somewhat in human medicine is packed platelets. Non-blood product transfusions include antivenin, and IVIG. All of the above products can cause some sort of an antigenic reaction in the patient they are administered to.

The whole blood products include fresh whole blood and stored whole blood. Fresh whole blood is harvested from a donor and contains red blood cells, white blood cells, platelets, and plasma. It contains labile (fragile) clotting factors (which only last for a short time in refrigerated products) and platelets (which are also fragile and do not last long). Stored whole blood is typically kept for upwards of 28 days (depending on the anticoagulant used). Stored whole blood contains red blood cells, white blood cells, and plasma proteins (excluding labile clotting factors). It should be noted that cells in stored whole blood (notably RBC's) are not "normal" and do not have the same oxygen-carrying abilities as normal red blood cells. There can also be an accumulation of chemicals in stored whole blood from cell breakdown. The whole blood products are colloid solutions, meaning they will increase blood pressure greatly by the effects of the large plasma proteins. Fluid overload can be a concern with use of these products. Antigenic reactions of all the products will be discussed later.

Packed red blood cells are prepared by taking a whole blood donation, centrifuging the blood and removing the red blood cells after the plasma has been decanted off. The red blood cells are washed and suspended in 0.9% normal saline to rid them of any remaining white blood cells or plasma proteins. The PCV of a packed red blood

cell transfusion is approximately 70-80%. Packed red blood cells are typically not considered colloids; however, they are suspended in a crystalloid solution and can till be implicated in volume overload.

Plasma products include: fresh frozen plasma, frozen plasma, cryoprecipitate, cryo-poor plasma, and platelet rich plasma. Fresh frozen plasma (FFP) is prepared by the removal of the plasma portion of the whole blood donation, and freezing it immediately. Fresh frozen plasma contains labile clotting factors (V, VIII, and vWF) as well as the remainder of the non-labile clotting factors, relatively small amounts of albumin and immunoglobulins, fibrinogen, alpha-macroglobulin, antithrombin III, and other physiologically important proteins. FFP has a freezer life of 4 years. After 4 years it becomes Frozen plasma (FP) and contains all of the above proteins with the exception of the labile clotting factors. Cryoprecipitate is the supernatant from a special centrifugation of plasma (after it has been removed from the red cells). This contains high levels of vWF and is often removed and stored for infusion during a vWF crisis. The remaining plasma, after the cryo has been separated off, is cryo-poor plasma and contains all parts of the fresh frozen plasma, except for vWF. Platelet rich plasma

Miscellaneous transfusion products include: human albumin, canine albumin, IVIG and antivenin. Human/canine albumin is typically used to treat severe hypoalbuminemia. IVIG is an anti-inflammatory infusion used in severe hematologic/dermatologic autoimmune disease states (IMHA, ITP, etc). Finally, antivenin is an immunoglobulin infusion that has been sensitized to snake venom and is used to treat snake envenomations.

Blood types

Although not all transfusions involve blood, giving blood products requires knowledge of the physiology of blood types in the canine and feline patient. Canine patients have a receptor and allele system, where their red blood cells express receptors for various different receptors corresponding to a corollary blood type. Canines may have upwards of 18 different blood types; the most notable being DEA (dog erythrocyte antigen) 1, (with 1.1, 1.2 and 1.3 subgroups), DEA 2, DEA 3, DEA 4, DEA 5, DEA 6, etc. There is another antigen that may be of importance called the dal antigen present in dalmation dogs. Canines are either positive or negative for the various receptors and can have multiple receptors present on their red blood cells. The most antigenic receptors are the DEA 1.X antigens which is why commercial kits test for the DEA 1.1 antigen. Dogs do NOT have naturally occuring alloantibodies, meaning they are not sensitized to different blood types initially. However, after they receive a transfusion they now create circulating antibodies against the blood type infused. Most dogs (99%) are positive for the DEA 4 allele, so a DEA 4+ dog can be considered a universal donor if they are negative at all other allele sites. Ideally, dogs should be typed and cross-matched prior to infusion of DEA 4+ (only) blood products.
Feline patients have a slightly different system: the AB system. Cats either have an A protein, B protein, or A AND B protein present on their red blood cell. Cats DO possess naturally occuring alloantibodies and can react to any blood type but their own on the first transfusion. A type cats have weak anti-B antibodies and will typically have a delayed hemolytic reaction where some, but not all, of the infused red blood cells (if Type B blood was infused to a Type A cat) will become destroyed and unusable. In contrast, type B cats have very strong anti-A antibodies and can die if transfused with type A blood. As of 2006, 90% or greater of all domestic shorthair cats in the US and in most european countries are type A. Type B occurs in a decent percentage of Abyssinian, Birman, British Shorthair, Cornish Rex, Devon Rex, Exotic shorthair, Himalayan, Persian, Scottish fold, Somali, Sphinx, and Turkish Angora/Van cats. All cats should be typed if a transfusion might be in their future, given type-specific blood products, and cross-matched prior to infusion as well. There is a small percentage of cats that are type AB. They contain both the A and B-type sugar on their red blood cells and have no natural alloantibodies.

Recommendations for transfusing AB cats is to use Type A blood.

There are various kits available to type patients in the hospital. The canine versions typically screen for DEA 1.1 to see whether the patient is 1.1 positive or negative. Many canine patients are DEA 1.1 negative and should only be transfused with DEA 1.1 negative blood. DEA 1.1 positive patients can receive DEA 1.1 positive or negative blood. The biggest concern is transfusing DEA 1.1 positive blood to a DEA 1.1 negative patient. Feline blood typing kits identify Type A and Type B blood types, and potentially Type AB cats.

Blood typing is very important to prevent serious life-threatening mis-matched blood types. Crossmatching can identify blood type incompatiibilities and other potential antigen-antibody reactions by causing agglutination. Crossmatching can be done with a manual method or a gel tube method (using a commercial kit).

Administering/Monitoring Transfusions

Regardless of the product being administered, transfusions are not entirely safe. There are several categories of transfusion reactions, each of which with their own pathogenesis and clinical signs. There are several different classes of reactions. The acute immunologic reactions include: acute hemolytic reactions (typically mis-matched blood types), allergic reactions (antigen-antibody reactions to foreign proteins), febrile non-hemolytic transfusion reactions, and transfusion-related acute lung injury (TRALI). The acute reactions occur immediately and tend to be life-threatening. Delayed immunologic reactions include: delayed hemolytic transfusion reactions (possibly from mis-matched blood types), and post-transfusion purpura. Acute non-immunologic reactions include: volume overload (TACO), citrate toxicity, hypothermia, and bacterial contamination (sepsis). And finally, the delayed non-immunologic reactions include infectious disease transmission.

Reaction category	Type of reaction	Cause	Clinical signs	Treatment
Acute immunologic	Acute hemolytic	Blood type mismatch	Collapse, vomiting, hemolysis	Stop transfusion, administer fluids, steroids
	Allergic	Antigen binding in donor blood.	Typically dermatologic: urticaria, pruritis, edema	Slow/stop transfusion, diphenhydramine
	Febrile non-hemolytic	Leukocyte antigens on donor leukocytes and platelets. WBC's release inflammatory mediators during storage	Elevation of body temperature by 1 C above baseline after starting transfusion	Slow or stop transfusion, diphenhydramine or NSAID.
	TRALI	Donor leukocytes in the product react with recipient leukocytes inciting an immune response	Pulmonary edema with normal cardiac function	Fluids, Oxygen, potentially mechanical ventilation
Delayed Immunologic	Delayed hemolytic	Antibody response to donor antigens. Cells are prematurely removed from circulation	Drop in PCV, Rise in bilirubin	No treatment typically
	Post transfusion purpura	Platelet fragments get targeted and initiate immune response destroying host platelets	Spontaneous bleeding (gums, teeth, nose)	Immunosuppressive therapy
Acute non-	Transfusion-	Volume overload	Coughing, edema,	Administer the

immunologic	Associated Circulatory Overload (TACO)	from administration of high molecular weight fluids or over-zealous administration	pulmonary edema, chemosis, nasal discharge	transfusion at a slower rate, administer diuretics
	Citrate Toxicity	Anticoagulant in blood products, can bind calcium if given in large quantities	Tetany, cardiac arrhythmias	Slow/stop transfusion, administration of parenteral calcium. ADMINISTER IN ANOTHER LINE
	Hypothermia	Giving cold blood products	Hypotension, cardiac arrhythmias	Provide warming measures (towels, forced air warming, IV fluids warming, ets). Warm blood product
	Bacterial contamination	Administering contaminated or expired blood products	Hypotension, collapse, SIRS, Septic syndromes	Fluids, antibiotics, stop/slow transfusion, supportive care
Delayed non-immunologic	Infectious disease transmission	Non-screened blood products transmit a blood-borne disease	Clinical signs of disease	Treatment of the respective disease.

Monitoring the infusion of a transfusion is incredibly important and veterinary technicians should be diligent about doing so. The transfusion is typically administered slowly (1/4 of the normal rate) and the rate is slowly increased if the patient can tolerate the transfusion. Most transfusions of blood products NEED to be given over 4 hours to reduce the chance of bacterial contamination. All blood product transfusions (plasma and red blood cell products) must be given through filters to prevent foreign material (notably clots) from entering the patient. Special filters are made to screen for bacteria and WBC's. Filters contained within pre-made intravenous sets can typically handle an entire transfusion load. In-line filers (often used for smaller doses of transfusion that are administered on a syringe pump) must be used according to the manufacturer directions. There is a brand of filter that can only handle 25-30 cc of packed red blood cells; after which it cannot be considered effective. Blood transfusions are often administered on an IV pump, but red blood cell viability is determined, in part, by the mechanism of administration. If IV pumps that are NOT rated for blood are used RBC's can be lysed before administration, or may have their cell walls weakened and they will not survive in circulation long. Thus, recommendations include to free-drip the transfusion (or hand push it) if you are not using a blood-rated pump. Contrary to popular belief, the HESKA IV pumps are not actually rated for blood products, although their technology is typically the style used for blood transfusions.

Although administration of blood products is somewhat commonplace, diligent monitoring and knowledge of the biology of blood types is essential to perform high-quality transfusion medicine. The veterinary technician needs to stay updated on new and emerging trends in transfusion medicine and should be proactive about monitoring patients who are receiving a transfusion. Transfusions can be life-saving, but can also do more harm than good if not administered properly.

Pain Management in the ER/ICU

David Liss, RVT, VTS (ECC, SAIM)
Los Angeles, CA

Introduction

The recognition and treatment of pain is an incredibly important part of the hospitalized veterinary patient's regimen. Patients that do not have their pain addressed might suffer longer hospitalization times or face increases in morbidity and mortality from pain. Pain is known to elicit a sympathetic response worsening or inciting shock states which promote decreased wound healing and decreased organ perfusion. The veterinary technician is invaluable in assessing and reporting findings to the veterinarian. The veterinary technician, in conjunction with the veterinarian, can discuss and implement a multi-modal approach to managing pain in critically ill patients.

Pain Physiology and Pathophysiology

Pain is typically thought of as an adaptive response to prevent injury. If you stick your arm over a fire, it hurts so you can pull away and minimize tissue damage. However, severe injury, or failure to treat pain can cause detrimental physiologic effects, beyond the positive effects of self-preservation. In the periphery, specialized nerves, called nociceptors, exist and transmit pain signals to the spinal cord and to the brain. The free nerve endings, which terminate in soft tissue, have various receptors that can be activated in response to thermal, chemical, or mechanical noxious stimuli. For example, an acid burn will stimulate different fibers than a laceration. The first step in the pain process is transduction, where nerve endings convert stimuli into electrical signals. The two main nerve fibers (each with separate ability to transmit various stimuli) are the A-delta and C fibers. The A-delta fibers tend to fire faster, sending quicker signals to the spinal cord. The C-fibers tend to have a slower ability to reach their threshold. Thus, pain that is felt immediately upon exposure to noxious stimuli (crushing, pinching, tearing) is transmitted through A-delta fibers. Pain that is felt with a bit of a pause, cold temperature, etc is transmitted across C-fibers. Another important point is that there are various A-delta and C nerve fibers contain endings that typically do not transmit pain signals (initially) but can be "woken up" and recruited in severe circumstances. The next step in the pain process is transmission; after the nociceptors have converted the stimulus to energy it is sent to the spinal cord for initial processing. The signal travels through the nerve fibers to the dorsal part of the spinal cord. As discussed earlier, C-fibers are 10x slower than A-delta fibers in their transmitting speed. After pain signals reach the spinal cord, modulation occurs. Here, the spinal cord either dampens or increases the pain signal according to various neurotransmitters or chemicals that are activated/deactivated in the spinal cord. The majority of pain signals that make it to the spinal cord and are sent on are mediated by glutamate, a neurotransmitter. Glutamate acts on the AMPA, KAI, and neurokinin (NK) receptors and stimulates a response by sending the signal up the spinal cord to the brain. The NMDA receptor (upon which ketamine exerts it's effects) is responsible for amplifying pain signals, whether they are incredibly strong or not. The NDMA receptor is thought to be important in prolonged/amplified pain states. A neurotransmitter called Substance P activates the NMDA receptor. Finally, GABA receptors, when activated, tend to inhibit signals from crossing into the spinal cord to be processed. There are other important neurotransmitters involved in modulation of signals in the dorsal horn of the spinal cord. These include: serotonin, norepinephrine, and opioid receptors. Serotonin, norepinephrine and opioid receptors, when activated, inhibit excitation of neurons, thus agonists of these drugs have analgesic properties. The last, and final, step of the pain pathway is perception. Perception occurs in multiple parts of the brain and is then perceived as an unpleasant sensation associated with real or perceived tissue damage. Pain can be categorized in various different ways: disease or anatomy related (pancreatic, etc), location (superficial, visceral, deep), duration (acute, chronic) or intensity (mild, moderate, or severe). These often require some objective input) from the patient, so categorizing these in veterinary patients can be challenging.

A few other important concepts in pain management in the acute patient include: Allodynia, sensititzation, windup, and referred pain. Allodynia refers to an exaggerated reaction to a stimulus that is normally not painful. This can occur due to an exaggerated pain response where the pain threshold of nociceptors is lowered.

Sensitization and windup are the result of peripheral and central physiochemical changes that occur during tissue damage and the inflammatory response. Peripherally, inflammatory mediators and cells can reduce the threshold of normally high-threshold nociceptors, and awaken "sleeping" nociceptors causing an exaggerated pain response. Central sensitization (windup) occurs as another mechanism for an exaggerated pain response, and because this occurs in the spinal cord, can result in severe pain that lasts much longer than the initial tissue insult. Repeated signaling to the spinal cord activates excitatory neurotransmitters which activate various receptors (NMDA, notably) and secure open-channels for pain stimuli to pass through. It appears that central sensitization can be responsible for allodynia. Referred pain is pain in a body part that is not affected by tissue damage. This might occur in a limb that was not amputated (phantom limb pain), or pain in limbs where the source of the pain is in the abdomen, for example.

Pain Pharmacology

Drugs used in the treatment of pain are best described by their effects on the pain pathway. Major classes of drugs used for pain in the acute setting include: opioids, NSAIDS, alpha-agonists, NMDA-antagonists, and local anesthetics.

Opioid medications act peripherally (transduction) and centrally (modulation) on opioid receptors. There appear to be three sub-types of receptors: mu, kappa, and delta. There are various types of opioid drugs including agonists, antagonists, and partial agonists/agonist-antagonist drugs. The below table summarizes these drugs.

Drug	Primary receptor	Secondary receptor	Level of pain appropriate for	Duration of action	Species	Routes to be administered
Morphine	Mu	NA	Moderate-Severe	Up to 4 hours	Cat, Dog	IV, IM, SQ-IV Can cause histamine release
Hydromorphone	Mu	NA	Moderate-Severe	Up to 4 hours	Cat, Dog	IV, IM, SQ
Oxymorphone	Mu	NA	Moderate-Severe	Up to 4 hours	Cat, Dog	SQ, IM, IV
Fentanyl	Mu	NA	Moderate-Severe	Single injection up to 30 minutes	Cat, Dog	IV- CRI
Buprenorphine	Mu (partial agonist)	NA	Mild-moderate	Up to 6 hours	Cat, Dog	SQ, IM, IV
Methadone	Mu	NMDA antagonist	Moderate-severe	2-6 hours	Cat, Dog	SQ, IM, IV
Butorphanol	Kappa	Mu	Mild-Moderate	1-6 hours (Dogs typically 1 hour or less)	Cat, Dog	SQ, IM, IV
Tramadol	Mu agonist	Serotonin/ Norepinephrine reuptake inhibitor	Mild-moderate	Twice-four times daily dosing	Cat, Dog	PO
Naloxone	Mu antagonist		Reversal agent	NA	Cat, Dog	IV

The second class of important analgesic drugs are the non-steroidal anti-inflammatory drugs (NSAIDS). These drugs have a potent ability to slow/stop inflammatory processes which are responsible for pain signaling. Although tissue damage may exist, of the inflammatory cascade can be prevented, pain signals will not be transduced. NSAIDS work on transduction of pain, working locally to prevent cytokine release, cell recruitment, and other inflammatory signs. They do have some significant side-effects and their use in critical patients are limited. Examples include: carprofen, meloxicam, aspirin, etodolac, piroxicam, deracoxib, fibrocoxib, tepoxalin, and ketoprofen.

Next, alpha-agonists, such as dexmedetomidine, can act in the spinal cord to prevent modulation of pain signals through agonizing norepinephrine at the alpha-receptors in the dorsal horn. Alpha-agonists tend to have severe cardiopulomonary effects, even at low doses, and so their use in critical patients is also limited. However, they remain an important part of the pain arsenal in dealing with anesthetic delirium, or as a continuous rate infusion for sedation with desired analgesic effects.

Local anesthetics are the next major class of analgesic drug to discuss. These drugs, ending in -caine, are Na-channel blockers. Influx of Na+ ions into the neuron is responsible for the creation of an action potential in the nerve. The action potential propagates and the signal travels along the neuron to the spinal cord. Blocking Na+ influx would stop the action potential and prevent transmission of the painful stimulus. Examples include: lidocaine, bupivicaine, proparacaine, and tetracaine. A summary of these drugs is found below.

Drug	Duration of action	Routes administered	Notes
Lidocaine	60-120 minutes	Local, SQ/Intradermal, IV	Can provide effective adjunctive analgesia as a CRI Reduce dosages in cats****
Bupivicaine	180-480 minutes	Intrathecal, Intrapleural, NOT IV	Only to be used
Proparacaine	Variable	Topically (ocular)	

Finally, the adjunct drug that might be used in analgesia in the critically ill is ketamine. Ketamine functions as an NMDA-antagonist, preventing or stopping exaggerated pain signals from passing through these channels to the brain (windup). Ketamine does not have analgesic properties on it's own. Rather, it seems to potentiate the effects of other drugs (opioids notably) by blocking NMDA-receptors and lowering the needs for the other analgesic drug (opioid) by itself.

Assessment of Pain

Assessing pain in small animals in the ICU can be somewhat difficult. There has been alot of research into physiologic and behavioral responses to pain. This research has allowed the veterinary professional to better assess and categorize pain states in animal patients. While it might seem somewhat intuitive that a patient who was hit by a car and growls is painful, the veterinary community didn't always see things that way. The best recommendation is to implement a comprehensive pain scale in the hospital and use that when assessing pain in your patients. A commonly used chart is the Colorado State University pain scales found here:

- Canine: ivapm.evetsites.net/refid.20468/refDownload.pml
- Feline: ivapm.evetsites.net/refid.20467/refDownload.pml

Behaviors associated with pain can be found in the following charts:

Canine pain behaviors		
Anxiety	Decreased desire for interaction	Submissiveness
Reluctance to move	Whimpering/Howling/Growling	Guarding
Aggression	Anorexia	Self-mutiliation

Feline pain behaviors		
Hiding	Decreased desire for interaction	Hissing/spitting
Reluctance to move	Excessive licking/grooming	Attempting to escape
Lack of grooming/unkempt coat	Tail flicking	Crouching

Vitals alone (blood pressure, heart rate) have been found to be poor predictors of pain. Many animals with normal vital signs are in pain. Approaches to a patient for a pain assessment might include:

- Observation of the animal in the cage
- Observing the patient interacting with another staff member
- Taking vital signs: HR, RR, Temp, Mentation, BP
- Attempting to elicit a painful response: palpating incision or limb/organ affected
- Observing quality of life: eating/drinking, coat, ambulation

Once the assessment is complete, the decision is made to institute analgesic therapy or modify current therapy, if it is inadequate.

Treatment of Pain

Treating a patient with acute pain involves a multi-modal approach. The first step is to assess the pain and make judgments as to the level of pain, location, and analgesic therapy that is appropriate. This involves thinking of where the pain occurs, what stimuli is causing it, and if there is a windup component.

Options for treating pain in the ICU include: injections of analgesic medications, continuous rate infusions of analgesic medications, use of local anesthetic blocks near site of pain, epidural injection and catheter placement, transdermal patches, continuous infusion of analgesics into pain site ("soaker catheters), and non-allopathic interventions such as acupuncture and/or physical therapy.

An example of a multi-modal approach to analgesia in a thoracotomy patient:

Pre-medication:
Hydromorphone (pure u opioid) + Midazolam
Induction:
Fentanyl (pure u opioid) + Lidocaine (Na-channel blocker) + Ketamine (NMDA antagonist) + Midazolam
Intra-operatively:
Fentanyl + Lidocaine + Ketamine CRI
Intercostal block (local anesthesia)
Post-operatively:
Bupivicaine infusion into thoracostomy tube
FLK CRI
+/- NSAID

References available upon request.

Cheap, Easy Blood Tests for the Emergency Patient

David Liss, BA, RVT, VTS (ECC, SAIM), CVPM

Introduction

There are many cheap and easy blood tests to run in the veterinary ER that can yield a large amount of emergency information. A patient will not usually die of an extremely high ALT value, but could certainly die of anemia or hyperkalemia. These tests identify life-threatening abnormalities quickly and easily and can greatly enhance care of the veterinary emergency critical care patient. Tests covered in this lecture include: PCV, TS, Buffy coat, Blood Glucose, BUN/USG, lactate, Electrolytes, and a blood smear.

PCV/TS/Buffy Coat

The Packed Cell Volume represents the volume of red blood cells in a column of serum from 0-100%. This measurement can indicate many things about the hemodynamic/fluid balance status of the patient. A low PCV indicates anemia, or absolute/relative lack of red blood cells, and a high PCV can indicate hemoconcentration, dehydration, polycythemia, and hyperviscous blood. A PCV value should be approximately 35%-40% depending on reference range or reference text. A PCV >45% typically indicates hemoconcentration/dehydration/ hypovolemia and/or polycythemia. A PCV <35% indicates anemia. The PCV tends to be interpreted as a relative number, with no absolute reference. A PCV of 47% may be perfectly appropriate in a patient or it might indicate anemia and hemoconcentration and after IV fluid therapy will drop to <30%. Typically the PCV and TS are measured together to fully assess the protein influence on the presence or absence of hemoconcentration or hypo/hyper proteinemia. There are typically 6 categories: Low PCV/Normal TS, High PCV/Normal TS, Normal PCV/Low TS, Normal PCV/High TS, High PCV/High TS, Low PCV/Low TS.

The total solids represents a measurement of all large molecules in the serum contributing to oncotic pressure. These are typically proteins and so the measurement is often called the total protein. Proteins in serum include globulins, albumin, fibrinogen and others. Albumin and globulin have the largest contribution to the TP and so hypoalbuminemia, hyperalbuminemia, hypoglobulinemia and hyperglobulinemia are typically expressed as a low or high TS respectively.

Low PCV/Normal TS (PCV <30%, TS = 7) g/dL or 70 g/L): IMHA, non-regen anemias (aplastic, PRCA, chronic disease)
High PCV/Normal TS (PCV >60%, TS = 7 g/dL or 70 g/L): Polycythemia, Endocrine diseases, HGE. HGE is an important rule/out as IV fluids will unmask the hypoproteinemia through hemodilution.
Normal PCV/Low TS (PCV = 40%, TS <5 or 50): Protein-losing disease, liver failure, hemorrhage
Normal PCV/High TS (PCV = 40%, TS >9 or 90): FIP, Multiple myeloma, severe dehydration with anemia
High PCV/High TS (PCV > 60%, TS > 8-9 or 80-90): Hemoconcentration
Low PCV/Low TS (PCV <30% and TS <5 or 50): Hemorrhage, Chronic blood loss (GI bleed)

Additionally, the PCV tube can yield important other information including: serum color and buffy coat. Serum colors are summarized below:

Red: Hemolysis (presence of free hemoglobin in serum)
Yellow: Icterus (presence of bilirubin in serum)
Milky/White: Lipemia (increased triglycerides in blood)

Buffy coat is a fairly inaccurate estimate of the WBC count. A normal buffy coat should be <1-2% and higher values can indicate a leukocytosis.

Blood Glucose

Blood glucose is maintained in normal healthy animals through the production, storage and release of this simple carbohydrate in physiologic processes. Glucose is produced through the digestion of carbohydrates, or through the breakdown of stored glycogen (glycogenolysis), or produced through other substrates (gluconeogenesis). Through these processes blood glucose levels fluctuate to only minimal levels throughout the day. This also allows for short periods of starvation to occur. During hypoglycemia, hormones including cortisol, glucagon and epinephrine are released. These hormones raise the BG through inhibiting insulin secretion, enhancing gluconeogenesis and glycogenolysis, and by causing peripheral resistance to insulin, thereby raising the plasma BG level. During hyperglycemia, insulin is released from B-cells in the pancreas and causes glucose transport proteins in cells to facilitate the transport of glucose intracellularly. Insulin also inhibits gluconeogenesis and glycogenolysis.

Normal BG levels in dogs and cats are not the same. Dogs can have a resting BG level of 53-117 mg/dL (2.9-6.5 mmol/L) and cats can fluctuate from 57-131 mg/dL (3.1 to 7.2 mmol/L). Hypoglycemia occurs with a BG of typically <50 mg/dL (<2.7 mmol/L) although a more rapid drop in glucose can occur and therefore a higher BG than 50/2.7 could show signs of hypoglycemia. This may occur due to decreased production or increased utilization of glucose. Typical causes include puppy hypoglycemia, insulinoma, iatrogenic insulin OD, xylitol toxicity, sepsis and fulminant hepatic failure. Hyperglycemia typically occurs with a BG of greater than 117/6.5 in the dog and 130/7.2 in the cat although cats can have a significant stress hyperglycemia. Hyperglycemia typically occurs in the absence of insulin or the presence of insulin resistance. The most common causes are diabetes mellitus or stress, although it can occur in pancreatitis, Cushing's disease, steroid administration and iatrogenic causes like administration of dextrose bolus/IV fluids.

Measurement of blood glucose is typically easy in the veterinary hospital. BG can be measured with arterial, venous or capillary blood and can also be measured from plasma/serum OR whole blood. Plasma/serum measurements are higher than whole blood as the water content is higher. As glucose can freely diffuse across RBC membranes the glucose will typically be hidden in the RBC in a whole blood measurement. Venous samples will most likely be slightly lower than capillary samples but in patients who have fasted all sample types (venous, arterial, capillary [ear in cats]) are basically the same.

Blood glucose can be measured most commonly via in-house analyzers and point-of-care analyzers/portable glucose monitors. Since portable/point of care instruments are readily available these are typically used in the emergency setting. These analyzers do not provide numbers that are identical to in-house analyzers but they are considered close enough to be clinically useful.

Blood Urea Nitrogen (BUN)/USG

Urea (or urea nitrogen) is a waste product of hepatic metabolism. Because it is a waste product and is kept in relatively low blood concentrations and is filtered by the glomerulus elevated BUN indicates an issue with glomerular filtration rate (GFR) and is termed azotemia. There are three kinds of azotemia- pre-renal, renal, and post-renal. Pre-renal azotemia refers to decreased renal blood flow which lowers the GFR typically caused by hypovolemia/dehydration, renal azotemia is typically caused by renal disease/failure, and post-renal azotemia is caused by ureter/urinary obstruction. A urine specific gravity (if obtainable) aids in the support of categorizing the azotemia.

BUN	USG	Azotemia
High	Concentrated	Pre-renal
High	Isosthenuric	Renal
High	Concentrated	Post-renal

A BUN can be easily estimated from an AZOstick which gives a range of BUN either 35-50 or 50-80 mg/dL. An elevation of BUN can indicate hypovolemia/dehydration or possible acute kidney injury and is a valuable test in the emergency setting.

Lactate

Lactate production is the result of anaerobic cellular metabolism. It is a bypdroduct of metabolizing pyruvate to produce ATP for cellular energy. Glycolysis produces pyruvate in the cytoplasm of cells. Under aerobic conditions pyruvate traverses into the mitochondria and produces lots of ATP via the Kreb's cycle. Red blood cells do not have mitochondria, so lactate dehydrogenase converts pyruvate to lactate to produce ATP. The excess lactate diffuses out of the cell and back to the liver to assist in making glucose via gluconeogenesis. This is called the Cori cycle.

In states of cellular hypoxia, only glycolysis occurs, producing pyruvate. Because pyruvate cannot travel into the mitochondria, LDH converts pyruvate to lactate and produces a small amount of ATP. As more and more lactate accumulates, it cross into the intravascular space and travels around the body. If global hypoxia is present, other tissues will be unable to utilize lactate to make glucose and it accumulates in the intravascular space. Lactate is dissociated with a Hydrogen ion at physiologic pH. Thus the excess hydrogen lower blood pH. Lactate is metabolized by the liver, and excreted by the kidneys.

There are two types of Lactic Acidosis. Type A, where tissue hypoxia is present with normal mitochondrial function and Type B, where oxygen delivery is adequate, but carbohydrate metabolism/mitochondrial dysfunction are present. Type A lactic acidosis indicates decreased DO_2 (oxygen delivery to tissues) through either, a decreased cardiac output, hypovolemia, or decreased oxygen content (as in anemia) or a decreased ability to extract O_2 (edematous states). Type B lactic acidosis has 3 subtypes. Type B-1 comes from decreased clearance of lactate. This may occur with liver failure, diabetes (from abnormal carbohydrate metabolism), renal failure, or in neoplasia. Type B-2 lactic acidosis occurs with drugs/toxins that affect a portion of glycolysis called oxidative phosphorylation. These include ethylene glycol, carbon monoxide, salicylates and acetaminophen. Type B-3 lactic acidosis occurs with mitochondrial diseases.

Lactate is typically measured to indicate cellular hypoxia as a result of decreased oxygen delivery (shock). Typically a lactate of greater than 2.5-3 mmol/L indicates this condition. Lactate can be easily measured on point-of-care devices or portable lactate meters.

Electrolytes

Sodium, Potassium, and Chloride can be easily run on in-house lab analyzers and can provide a wealth of information. While chloride is an important electrolyte to consider, abnormalities of sodium and potassium will be discussed in this manuscript.

Sodium is the most abundant cation and represents a large portion of positively charged ions in the extracellular fluid. Sodium is typically thought to indicate fluid and water balance in the body, with elevations in sodium (hypernatremia) representing a deficit in free water, and decreases in sodium (hyponatremia) indicating elevations in body water. Although this is not always true, a simplified discussion of this electrolyte is covered in this lecture. Hypernatremia is a "water" problem, not necessarily an excess sodium condition. Typical causes include: dehydration/hypovolemia, lack of intake of water, sodium toxicity (rarely), diabetes insipidus. Rapidly correcting hypernatremia that has a chronic nature can lead to cerebral edema and brain injury. Hyponatremia is not common and has several causes including: pseudohyponatremia (hyperglycemia), SIADH, liver failure. Hypo and hypernatremia can be life-threatening if severe enough.

Potassium, in contrast to sodium, is the most abundant intracellular cation and has very low serum concentrations. However, since it is important in muscle contraction (cardiac and smooth) hypo or hyperkalemia can be life-threatening and produce cardiac arrhythmias or hypoventilation due to intercostal/diaphragm muscle paralysis. Hyperkalemia causes include: acute kidney injury, ureteral or urethral obstruction, Addison's disease, or massive hemolysis/rhabdomyolysis. Hypokalemia causes include: urinary loss (DKA, renal failure), B-agonist overdose, diuresis and use of medications (furosemide).

Blood Smear
Preparation and reading of a blood smear is an extremely inexpensive and highly valuable test in the veterinary ER. A smear can quickly evaluate red blood cells, white blood cells, platelets, amongst other abnormalities and aid in, or even actually diagnose a patient's condition. Technicians should be proficient at reading blood smears.

A blood smear can be rapidly prepared using EDTA blood and placing one drop of blood on the end of a clean glass slide. The spreader slide should be applied and a nice even smear made with a visible "fingerprint" appearing feathered edge. The slide should then be stained as per hospital policy.

Evaluation of the smear starts on the 10x objective (lower power) and should evaluate the feathered edge for general cell distribution, presence of platelet clumps, microfilaria etc. RBCs can be evaluated for general density and rouleaux/agglutination patterns identified. WBC numbers can also be estimated. Once this initial scan is complete the objective should be enhanced to higher power while focusing in on the monolayer, which is just slightly away from the feathered edge.

A differential can be performed and various cells can be evaluated. Neutrophils should be evaluated for cytoplasmic appearance, toxic change, evidence of increased immature band neutrophils, and the other leukocytes evaluated as well.

Red blood cells can be evaluated for their color strength (poly or hypochromasia), shape (discoid or spherical), evidence of fragmentation like schistocytes, evidence of Heinz bodies, echinocytosis (indicating envenomation), and spherocytes which can rapidly diagnosis an immune mediated process. Also RBC inclusions like parasites can be identified.

Platelet counts can be estimated as well and platelet clumping identified. Macroplatelets can indicate regeneration in thrombocytopenic animals and a low platelet count can rapidly identify thrombocytopenia.

Conclusion
There are many cheap and easy tests to perform in the emergency patient which can yield a ton of information. All of these tests can be performed by just about any veterinary facility and can be rapidly performed by and interpreted by a veterinary technician. Employing these tests in your practice will enhance the quality of emergency care provided to your patients, as well as amplify the amount of data the veterinarian gets to diagnose the condition.

Oxygenation and Ventilation: Don't Sing the Blues!

David Liss, RVT, VTS(ECC, SAIM)
Los Angeles, CA

Introduction

Veterinary technicians effect, interpret and assess the respiratory system every day on almost every patient. To fully comprehend this amazing system one must review its basic and "advanced" physiology. This presentation will serve to outline the basic anatomy of the respiratory system and delve into more complicated aspects, such as pressures, blood flow and resistance, and finally briefly discuss the role of the respiratory system in metabolism and disease.

Basic Anatomy

There are two major parts to the respiratory system: the conducting airways and the blood-gas interface. The conducting airways allow air to travel from the environment to the blood-gas interface to facilitate oxygenation and removal of carbon dioxide. The trachea is the initial tube connecting the mouth and larynx to the right and left mainstem bronchi. Following are the lobar and segmental bronchi. Finally, the last are the terminal bronchioles and respiratory bronchioles that contain alveoli. Within the alveoli is the blood-gas interface. Here, simple diffusion allows oxygen and carbon dioxide to flow across concentration gradients. The reason this works so well is Fick's law of diffusion. Fick's law states that gas movement is influenced by the area of the sheet and proportional to it, but inversely proportional to the thickness. Therefore, a large and thin sheet will transmit more gas than a short and thick sheet. Being that the blood-gas interface is around 50-100 meters long and about 1/3 mm thick, it is primed to move gas from capillary to alveoli and vice-versa, quickly.

The amount of air (volume) from the trachea to the terminal bronchioles is not involved in gas exchange and thus contributes to anatomic dead space. This is approximately 150mL in a human . Physiologic dead space is made up of areas of the lung that do not eliminate CO2. In normal patients anatomic and physiologic dead space are essentially equal, with adequate areas of alveoli available for gas exchange. However, in disease, physiologic dead space can increase from collapsed or alveoli filled with pus, blood or other fluid.

Blood flow to and from the lung is achieved from the right side of the heart, to the pulmonary artery, and into the lung via the pulmonary arteries. Blood then passes through a rich and dense network of capillaries, where it travels one cell at a time to millions of alveoli. Oxygenated blood then travels into the pulmonary veins and the pulmonary vein to the left atrium. The only thing separating a red blood cell from the alveoli lumen is the thin capillary and alveoli wall. The bronchial circulation is often forgetten. These vessels perfuse the lung parenchyma at the level of the bronchi and bronchioles.

Two important processes to note include the role of surfactant and the removal of foreign material from the lung. Due to the high pressures alveoli face and their liquid lining, one would think they would collapse much like popping packing material. However, they must stay open for gas exchange to occur. To help offset the surface tension across the alveoli wall they produce a thick (relatively) substance called surfactant. This help lower the surface tension by strengthening the wall of the alveoli. Additionally, the respiratory tract (essentially open to the environment) is constantly collecting dust and other foreign particles. The mucociliatory apparatus functions to move particles from the lower airways to the upper airways and out of the system. Dust that travels down to the bronchioles will be trapped in mucous and cilia will slowly raise the mucous up through the bronchi and into the trachea where it can be expelled. Foreign material in the alveoli gets trapped and cannot be effectively removed.

Lung Pressures and Volumes

Understanding respiratory physiology cannot occur without discussing properties of volume and pressure in the lung.

Tidal volume: The amount of air taken in during a normal inhalation (and released during exhalation) (10-15 ml/kg)

Vital capacity: The amount of air taken in and expelled during a maximal inhalation/exhalation.

Residual volume: Amount of air left in the lungs after a maximal breath

Functional residual capacity (FRC): Content of air left in lungs after a normal inhalation

Total lung capacity: Total volume of lung- Vital capacity + residual volume

Total ventilation: Respiratory rate x tidal volume

Alveolar ventilation: (Tidal volume – anatomic dead space) x respiratory rate per minute

Pulmonary Blood Flow

In contrast to the systemic circulation, pulmonary blood "pressures" are quite low. If the average systemic mean arterial pressure is around 100 mmHg (Systolic of 120, Diastolic of 80mmHg) then the MAP of the pulmonary circuit is 15 mmHg with a systolic of 25 mmHg and a diastolic of 8 mmHg. This represents the ultimate function of the lung in regards to blood flow. Blood flow to the lung is never regulated like other organs and the blood must receive the entire cardiac output from the right side during every heart beat. To facilitate ultimate diffusion by passing blood cells one by one through the capillaries, increases in pressure from the right heart could damage capillaries, and any constriction of the smooth muscle of the pulmonary vasculature would certainly cause the right side of the heart to work harder.

Transmural pressure: The pressure difference between the inside and outside of a capillary. If the pressure outside a capillary rises above the pressure inside the capillary will collapse. The capillary pressure is typically equal to atmospheric pressure. The alveolar pressure also is roughly equal to the atmospheric pressure. However if pressures inside the alveoli or surrounding the capillary increase the transmural pressure increases (greater pressure gradient) and the capillary collapses.

Intrapleural pressure: Pressure within the pleura surrounding and lining the thoracic cavity. Typically this pressure is negative, and becomes more negative on inspiration. However, pleural disease, such as a pneumothorax can increase it above atmospheric level.

Pulmonary circulation can deal with great changes in increased flow and resistance through some amazing attributes. Typically, systemic blood pressure will increase with changes in various cardiovascular parameters. If you run on a treadmill your blood pressure may increase from vasoconstriction or increased heart rate/stroke volume. However, if we increase the flow of blood to the respiratory system, the resistance often stays the same or lowers. This occurs from two different processes. The first process is termed recruitment. Recruitment refers to capillaries that previously did not conduct blood (not needed) to start doing so. If the pressure inside one capillary increases because of increased flow, and an additional capillary opens up to assist the overall resistance is lowered and kept the same. The second process is distension. And unlike vasodilation that is a widening of the arteries from the relaxation of smooth muscle, distension is a passive swelling of the capillaries from increased blood flow. These two processes function to maintain the resistance or pulmonary "blood pressure" under a wide variety of normal physiologic conditions.

Gas Exchange

Does oxygen passively enter the lungs and diffuse into the pulmonary circulation? The answer is somewhat complex. The partial pressure of oxygen in room air and in our alveoli can be calculated. The partial pressure is equal to the inspired concentration (21%) multiplied by the difference between atmospheric pressure and the gas' water vapor pressure. In this case the PO2 is (0.21 x [760-47]) = about 150 mmHg. By the time that oxygen reaches our lungs the PO2 is around 100 mmHg. Oxygen is pulled from the alveoli at varying rates during demand. The rate of alveolar ventilation correlates to the delivery of oxygen to the capillaries. Thus, hypoventilation can cause hypoxia/hypoxemia.

Additional causes of hypoxemia include: diffusion impairment, shunt, and V-Q mismatch. Diffusion impairment is an uncommon cause of hypoxemia but can result from thickening of the blood-gas barrier. Shunt refers to deoxygenated blood that avoids oxygenation and travels from the right side of the heart to the left side. Shunt can be caused by several different mechanisms including: thromboembolism, recumbency (certain areas don't receive ventilation) or cardiac abnormalities like septal defects. Shunt results in hypoxemia as deoxygenated blood enters the arterial circulation. Shunt typically does not respond to 100% oxygen administration. The PO2 will rise but not to the projected value because the blood volume that is bypassing the lungs never becomes oxygenated.

Additional dissolved oxygen may be detected when a blood gas is performed, but the hemoglobin may not respond to the additional oxygen. The last cause, V-Q mismatch, refers to ventilation and perfusion "mis-matching" across the lung. For ventilation and oxygenation to occur the alveoli must be open , air must flow into it, and blood must run across the alveolus. However, if one of these elements does not occur removal of CO2 and delivery of O2 may not occur. V-Q mismatching refers to examining the entire lung field and deciding whether there is a mismatch occurring- blood may not be flowing to certain areas, and certain alveoli may have blood flow, but no ventilation. If a ventilation obstruction occurs (airway blockage) the oxygen levels will fall but if there is blood flow, CO2 may elevate slightly but may not immediately increase. This is evident in the severe hypoxemia and cyanosis see in upper airway obstructions. Now on the other hand, if a blocked capillary does not deliver blood flow to the alveolus, the oxygen in the alveolus will increase as more is delivered to it, and the CO2 will eventually fall to 0 because inspired air contains no CO2. This represents a V-Q relationship of 0 (airway obstruction, no ventilation means V = 0) and one of infinity (perfusion = 0, Q = 0). This relationship between alveolar and arterial oxygen can be measured in the A-a or Alveolar-arterial gradient calculation.
A-a gradient:

$$\text{A-a gradient} = P_AO_2 - P_aO_2$$

$$P_AO_2 = P_IO_2 - P_ACO_2/R$$

SO: A-a gradient $= [P_IO_2 - P_ACO_2/R] - PaO2$

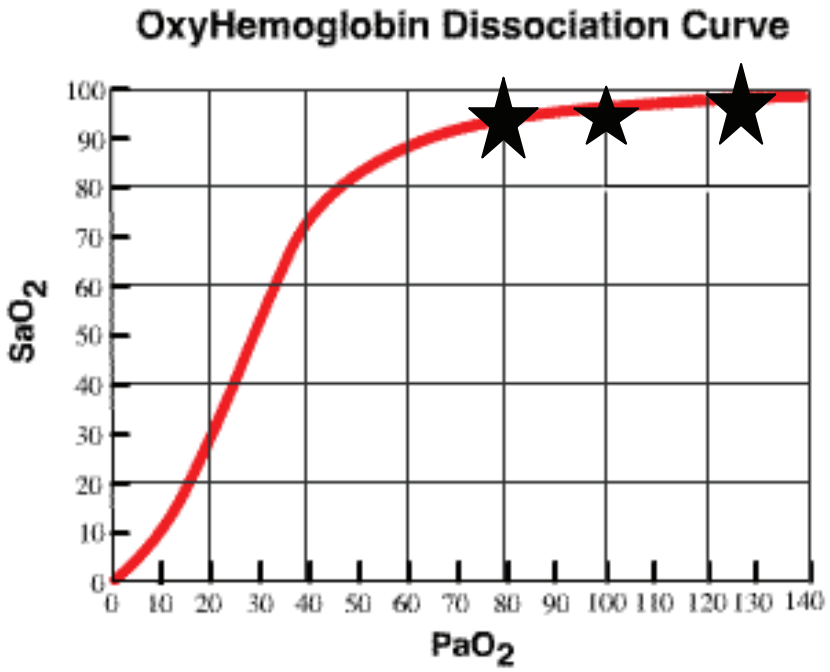
[R = respiratory quotient. Normally 0.8]

Note: this is at sea-level and cannot be used in >21% oxygen
Assume that PIO2 = 150 mmHg (oxygen partial pressure at 21%
Assume that alveolar (A) CO_2 = arterial (a) CO_2

A-a gradient $= [150 - P_ACO_2 / R] - P_aO_2$

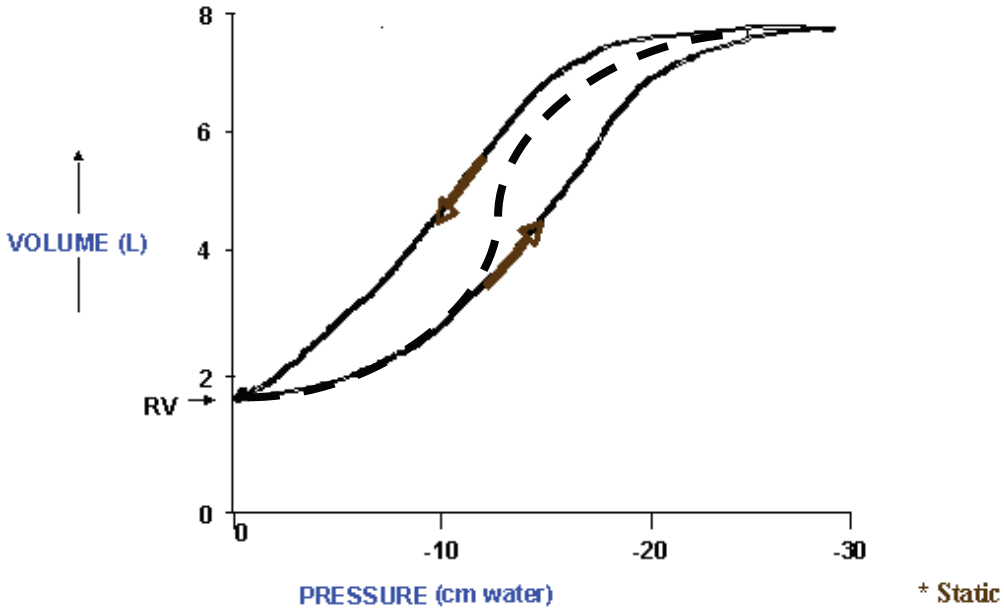
Example: An arterial sample is drawn that reveals a P_aO_2 of 72 mmHg on room air. The blood gas also reveals a CO_2 of 40 mmHg. We know that a PaO2 of 72 mmHg on room air is too low.
The A-a gradient is: (150 – 40/0.8) – 72 = 28. This is a high number. Meaning a gradient exists between the oxygen in the alveoli and the arterial oxygenation indicating a V-Q mismatch situation. Normal is less than 10. The gradient, under ideal conditions should be equal.

Relationship of Dissolved Oxygen to Hemoglobin Saturation
The PaO2 represents the dissolved oxygen gas in the bloodstream. However, metabolic delivery of oxygen depends on its rate of saturation with hemoglobin which is pumped around the body by means of the cardiovascular system. The key points here are that this is not a linear curve. If one looks at the ideal P_aO_2 level at room air (approximately 100mmHg) this corresponds to an S_aO_2 of about 94%. The P_aO_2 can continue up to 500 mmHg (ideal for breathing 100% O2) but the S_aO_2 only continues from 94-100%. A patient with a pulse ox (S_pO_2 = S_aO_2) of 98% or less under anesthesia breathing 100% O_2 has serious hypoxemia.



Airway Mechanics: Pressure Volume Curves, Compliance and Resistance

Pressure and its relationship to volume are very important in lung physiology. Below is a pressure-volume curve (loop) of a normal lung. The slope of the line (represented by the solid line) is the change in unit volume per change in unit pressure and is termed "compliance." Note that even at zero pressure there is still some residual volume in the lung (FRC).



The dashed line represents the compliance of the lung. A low compliance means the lung does not expand well at increasing pressures. It is a "stiff" lung. Reduced compliance can be seen in diseases causing alveolar flooding (harder to expand lung), atelectasis (hard to inflate alveoli), and fibrotic states. Loss of alveolar surfactant can also contribute to decreased compliance resulting in atelectasis, and alveolar flooding.

The last property to discuss in the lung is resistance. Resistance is the pressure difference between the alveoli and the mouth. Bronchoconstriction will narrow the airways and cause increased resistance to airway flow. Collapsed airways (small and large) will contribute to resistance as well.

Metabolic Functions of the Lung

The lung is involved in several different aspects of metabolism. First it regulates CO2 in the bloodstream which directly corresponds to acid contribution to the acid-base status of the body. Hypercarbia leads to retained carbonic acid and a respiratory acidosis. In contrast, it can serve as an acid-release conduit in metabolic acidosis situations. If a patient is in a diabetic ketoacidotic crisis, tidal volume or minute volume can increase, leading to CO_2 loss and an increase in blood p_H. The lung is also responsible for the conversion of angiotensin I to angiotension II via angiotensin-converting enzyme (ACE). The reaction takes place in the lungs and the release of angiotensin II leads to systemic vascoconstriction and heart remodeling in cardiac disease. In addition, the inflammatory mediators of the arachidonic acid cascade (leading to leukotriene and prostaglandin formation) are metabolized in the lung and then distributed into the systemic circulation.

Conclusion

The respiratory system is much more complicated than we once thought. As technicians we need to have a thorough understanding of respiratory physiology to better serve our patients, whether they are in respiratory distress, or simply receiving sedation.

References available upon request.

What's for Dinner? Nutrition in the ICU

David Liss, RVT, VTS (ECC, SAIM)
Platt College & VCA Veterinary Specialists of the Valley, Los Angeles, CA

Introduction

Critical patients in the ICU are expending calories at an exponential rate. While a normal patient who is "recumbent" might only need a limited amount of calories to sustain life, patients with critical illness need supra-physiologic calorie administration to maintain vital functions. Nutritional treatment of a patient with severe illness should not be an after-thought but an integrated part of the treatment plan. Veterinary technicians are typically administering nutritional therapy, and as such are responsible for assessing nutritional status, and providing nutritional treatments for these patients.

Starvation in critical illness

Starvation experienced by a patient who is critically ill is different than that of a patient who is simply starving (lack of access to food, etc). The latter patient experiences simple starvation in which fat stores are broken down into energy to maintain vital functions. The patient conserves their lean body mass (skeletal muscle) in place of fat. In contrast, the critically ill and starved patient undergoes "stressed" starvation and typically recruits protein stores (in the form of skeletal muscle) for energy. This protein catabolism affects immune function, wound healing, and strength. Mammals do not create their own nutrients for energy, we are dependent on food intake to meet energy and metabolic needs.

Nutritional plan

The elements of the nutritional plan include:

1- Who to feed
2- When to feed
3- Where to feed
4- What to feed

According to nutritional specialists, every patient who can have feedings (not contraindicated) should be fed. There are very few actual contraindications to feeding, so this means most of our sick patients should be fed. Some issues, like vomiting or pancreatitis, are more relative contraindications to feeding and studies have shown how to feed patients through vomiting or even early feeding in pancreatitis. Patients in severe shock should not be fed as these nutrients will not be absorbed.

All patients should be assessed for nutritional status when entering the hospital. Withholding nutrition should not be performed for more than 2-3 days at most. This gives the clinician time to stabilize life-threatening issues such as fluid volume/dehydration, arrhythmias, and electrolyte abnormalities. Additionally, this 2-3 days really represents the time of onset of anorexia, so if they have not been eating well for 3 days, and we wait an additional three days in hospital, that is 6 days without food and there are sure to be some physiologic consequences to that. Ideally, placing a bowl of food into the cage, and having the patient eat readily is the ultimate scenario, but this is not always the case. In addition to interventions like warming the food, trying different foods, patients may need a more invasive nutritional plan to provide nutrients.

Routes to feed

There are typically two routes to provide nutrition: enteral and parenteral. Enteral nutrition is preferred because enterocytes receive nutrition as well. Intravenous feeding (such as in parenteral nutrition) provides nutrients for other organ systems, but not for the GI tract. Enteral nutrition options involve using the gut in some capacity. Voluntary feeding, force feeding, and placement of a feeding tube represent the options in this area. While voluntary feeding is not always achieved force feeding shouldn't be thought of as a viable alternative. Several problems exist with force-feeding. Risk of aspiration is great, and typically the amount of food administered isn't

actually what the patient receives. Food ends up on the walls, in towels, or on the staff member performing the treatment, Additionally, force feeding may result in food aversion and a dis-interest in eating on the part of the patient, which is certainly a setback.

Feeding tubes

Feeding tubes are a great option for providing nutrition to critically ill patients. Options include: nasoesophageal and nasogastric tubes, esophagstomy tubes, gastric tubes (PEG or otherwise), and jejunostomy tubes. The least invasive tubes to place are the nasal tubes with gastric tubes requiring an endoscope or surgical placement. Jejunostomy tubes are usually always placed with a surgical technique. Various aspects of feeding tubes are summarized in the below table:

Tube	Location of feeding	Long or short term	Types of diets infused	Complications
Nasoesophageal	Esophagus	Short	Liquid, not bulky	Very few
Nasogastric	Stomach	Short	Same	Aspiration, mechanical damage, discomfort
Percutaneous Endoscopic Gastrostomy (PEG) tubes, or Gastrostomy tubes	Stomach	Long	Larger diets- some bulk, blended diets	Stoma infection, accidental disconnection/removal, leakage of stomach contents
Jejunostomy	Jejunum	Short	Liquid/elemental diets	Accidental removal, leakage of intestinal contents
Esophagostomy	Esophagus	Long	Liquid to blended diets	Very few, stoma infection.

Typically critical care patients need a diet that is very high in calories per unit of food- called a calorically dense diet. This allows us to feed a large number of calories in a reasonable amount of food. Various diets are available for critical care use including Hill's A/D, Iams Maximum Calorie, Royal Canin Recovery, and Clinicare (Abbott Animal Health). Clinicare is the only true liquid diet and can be infused through most tubes. Other diets must be blended as they are chunky and can result in tube clogs.

Feeding through feeding tubes is done either with bolus feedings or a constant rate infusion. Typically a patient needs a caloric amount equal to the resting energy requirement (RER) which is a daily amount of calories in a sedentary patient. This is 30 x BW (kg) + 70. For example, and 10 kg patient would need (30 x 10 = 300, +70 =) 370 kcal per day. If doing bolus feedings, this amount is typically divided into 4-6 feedings. A constant rate infusion would infuse the total amount of calories divided by 24 hours. So our 10 kg patient would get (370 / 24) = about 15 calories an hour. Amounts of calories per mL / ounce varies, but on average Clinicare is 1 kcal per mL. Easily enough this patient could receive 15ml/hr of Clinicare. Typically this is divided into 30-50% to be started on Day 1. So 30-50% of the RER is started on Day 1, if the patient tolerates it well, that can be doubled (to 60%) and then finalized on day 3. The nutritional conversion (kcal per ounce or ml) can usually be found on the back of most cans of food.

Complications of enteral feeding

Complications of tube feeding can present some challenges to the ongoing management of these patients. Clogged tubes can occur with any kind of diet and can be so serious as to necessitate tube removal. However, troubleshooting can often resolve the problem. Typically pressurizing the tube with some water, or infusing some soda can break up a clog. Potentially a stylet or wire can be used to break up the clog. Flushing with warm water

after feedings can help prevent clogs. Other rare complications include aspiration, esophageal erosion, inadvertent removal, and potentially pressure necrosis.

Parenteral nutrition

If the enteral route is not available, parenteral nutrition is an option in small animal patients. Partial parenteral nutrition (PPN) also called peripheral PN can be administered through a peripheral catheter but only meets a portion of the patient's metabolic needs. Total parenteral nutrition or TPN (also called Central PN) can be formulated to meet most of a small animal patients metabolic and caloric needs. However, TPN is hyperosmolar and needs to be administered through a central line. These solutions can be technically challenging as they can be sources of sepsis (infection because of their high glucose concentration), and significant metabolic derangements. The clinic needs to be ready for intensive monitoring with a patient receiving TPN.

Conclusion

Critically ill patients are faced with having to "fight off" their critical illness through the use of calories to fuel their metabolic processes. Without an external source of nutrition, these patients may break down fat and protein leading to severe instability and impaired healing and recovery from illness. Various options for feeding critical patients are available including enteral and parenteral options. Veterinary technicians are essential in assessing nutritional status of patients and implementing nutritional therapies.

References available upon request.

Sepsis, SIRS, DIC, Oh My! Critical Care Syndromes

David Liss, BA RVT VTS (ECC, SAIM), CVPM
davidlissrvt@gmail.com

As in every specialty there are specific syndromes which are within the realm of the highly trained specialists. Inflammatory bowel disease, polyradiculoneuritis, and eosinophilic granuloma complex represent syndromes that internist, neurologist, and dermatologists treat and consider unique to their specialty. In critical care medicine we have the same types, although they tend to have high mortality rates. Systemic Inflammatory Response Syndrome (SIRS), Sepsis, Multiple Organ Dysfunction Syndrome (MODS) and Multi-organ failure (MOF), and Disseminated Intravascular Coagulation (DIC), are critical care syndromes which typically cause the high rates of morbidity/mortality in these highly debilitated patients. The frustrating element of these syndromes is that various disease processes (pancreatitis, GI disease, etc) can cause them and thus they must be anticipated and watched for in any of the critically ill we treat in our ICU's.

SIRS

SIRS represents a syndrome of widespread systemic inflammation which can be incited by an infectious or sterile source. SIRS in the face of bacteremia is typically termed sepsis. Patients can be mildly affected or severely affected by SIRS. If an infectious cause is the culprit gram-positive and negative bacteria can both produce endotoxins (lipopolysaccharide, or LPS for example) that activate monocytes and macrophages. This is a normal response from the immune system in the face of an infection. These white blood cells (WBC's) release inflammatory mediators that are responsible for the characteristic localized signs of infection: redness, swelling, heat, and pain. The inflammatory mediators, termed cytokines, include tumor necrosis factor- α(TNF-α), IL-1 and IL-6. There is also an anti-inflammatory reaction that serves to balance this influx of inflammatory cytokines; IL-10, IL-13 are prototypical anti-inflammatory cytokines.

As the inflammatory process continues, inflammatory responses move from the local level to the systemic level. As inflammation has many positive characteristics and serves to protect and alert the body to infection/trauma, free-flowing cytokines can wreak havoc with bodily systems if left to their own devices. It appears that these cytokines can cause three major negative effects to the body's natural processes:

1- Vasodilation
2- Endothelial barrier damage
3- Activation of coagulation pathways

Vasodilation, resulting from a loss in vascular tone, reduces cardiac output, perfusion to tissues, and ability to maintain vascular volume. Nitric oxide, a potent vasodilator, is often produced in inflammatory states (leading to redness- increased blood flow to areas to deliver macrophages). Systemic release of nitric oxide leads to systemic vasodilation, as opposed to beneficial localized vasodilation of a small capillary bed. An additional theory of vasodilation in SIRS is vasopressin deficiency. Lack of vasopressin reduces systemic vascular resistance as vasopressin binds to V1 receptors in vascular smooth muscle to induce vasoconstriction during low volume states.

Endothelial barrier damage

Activation of the coagulation pathways can lead to coagulation factor exhaustion and a hypercoagulable state. Cytokines induce tissue factor (TF) production and thus activate the tissue factor pathway in vivo. As TF is produced, micro-clots are formed and may deposit in the microvasculature. These micro-clots cause cell death, and tissue hypoxia leading to organ failure. As widespread coagulation ensues, systemic anti-coagulant systems are consumed and exhausted, which shifts the patient into coagulation over-drive.

SIRS Criteria

Published criteria for SIRS in small animals tend to be somewhat general and non-specific. This means that many patients may or may not ACTUALLY have SIRS but fit these criteria. Several bio-markers have been researched

in humans but authoritative studies are lacking in veterinary medicine. Hence the following criteria:

	DOGS	CATS
Temperature	<100.6 F or >102.6 F	<100 F or > 104 F
HR	>120 beats/minute	<140 or >225 beats/minute
Respiratory rate	> 20 breaths/minute	>40 breaths/minute
WBC count or percentage of bands	<6,000, >16,000, >3% bands	<5,000 or >19,000

Hopper, Silverstein. Small Animal Critical Care Medicine. pg. 48

Patients with SIRS may also manifest the following clinical signs:

- Tacky, injected mucous membranes
- Bounding peripheral pulses
- Vomiting/diarrhea
- Neutrophilic leukocytosis with toxic changes. Left shift may be present
- Hypoglycemia
- Hyperbilirubinemia
- Cholestasis

Treatment of SIRS

There is no specific treatment of a patient experiencing systemic over-inflammation. Treatment is aimed at supportive care and should follow a systematic approach. Important patient concerns include:

- Supporting appropriate fluid balance
- Suspecting GI bacterial translocation if gut integrity compromised
- Treating and reversing hypoglycemia
- If septic: providing source control, appropriate Anti-microbial selection, culture/sensitivity
- Oxygenation support
- Nutritional support
- Stress ulcer prophylaxis
- Preparing for and treating refractory hypotension

Sepsis

Sepsis is essentially SIRS + infection. Sepsis represents the hyper-inflammatory state that arises when a bacteria, virus, protozoa, or fungus infection incites the inflammatory cascade to begin. Sepsis may cause or be caused by bacteremia, or the presence of a bacterial infection in the bloodstream. Severe sepsis is defined as sepsis and organ dysfunction. Septic shock is defined as sepsis with refractory hypotension, despite volume resuscitation. These categories are important as a patient can be septic and have systemic inflammation, and not necessarily be hypotensive, or have organ dysfunction or failure.

As with SIRS there are criteria to diagnose/suspect sepsis in a patient. Many are similar to SIRS with the addendum that infection is proven or highly suspected. In addition to those listed above the following should increase suspicion for sepsis:

- Hyperglycemia
- Altered mental status
- Edema
- Hypotension
- Organ dysfunction (lungs, kidney, liver)
- Oliguria/anuria
- Hyper/hypocoagulability

- Hyperlactatemia

Staging of sepsis
An international sepsis staging schema exists to categorize the severity of a patient's septic response. Some patients may be pre-disposed to septic inflammation, but do not yield severe systemic manifestations, while other patients may have multiple concomitant co-morbidities all rooted in the septic syndrome.

PIRO:

P- Predisposition
Signalment, underlying disease process
I- Insult/infection
What is the underlying infection? Organism?
R- Response
SIRS? Hypotension? Shock?
O- Organ dysfunction
Signs of organ dysfunction: Oliguria, azotemia, pulmonary edema, increased liver enzymes/cholestasis, etc.

Identifying the septic focus
Source control, referring to finding the septic source and eliminating it, can be achieved by searching for and identifying the septic focus. We often go on a "hunt" for sepsis in patients we suspect it in but cannot initially prove it (no outward abscess as culprit). Septic foci can be present in essentially every organ system, but septic peritonitis is a common cause of sepsis. Other foci for sepsis, other than the abdomen, can be the respiratory tract, thorax (pyothorax), GI system, cardiac (endocarditis), urinary tract (urosepsis), pancreas, and integument (abscess, bite wounds). These systems should be ruled in or out when doing a sepsis "hunt."

Bacterial sepsis
Bacterial sepsis is most commonly identified and responsible for systemic dysfunction. Gram-negative bacteria produce lipopolysaccharide, an endotoxin, which stimulates the systemic response. Gram-positive bacteria produce various toxins including: lipoteichoic acid, and peptidoglycan. Common species of bacteria implicated in sepsis include: E. Coli, Enterococcus sp, α/β-hemolytic Streptococcus, Pseudomonas, and Clostridium.

Sepsis "hunt"
Some recommendations to investigate sepsis in a patient who has developed a clinicopathologic suspicion for sepsis:

- Evaluate invasive devices: unwrap and inspect catheters and tubes
- Perform abdominal/thoracic ultrasonography
- Blood culture collection
- Urine culture submission
- Consider joint taps, CNS tap, abdominal tap

Treatment of sepsis
In patients that have a highly suspicious septic focus early anti-microbial therapy is warranted. Antibiotics administered within 1 hour of presentation reduced morbidity and mortality. If possible cultures should be obtained to ensure adequate and targeted anti-microbial therapy. Patients should have all critical care parameters monitored and special attention paid to fluid balance, vascular volume/blood pressure, and blood glucose levels, as these tend to fluctuate most often. Specific treatments include:

- Supplementation with 50% dextrose as needed
- Insulin therapy to control hyperglycemia
- Vasopressor therapy to assist with vascular tone as needed

- Consider ACTH stim and/or low-dose steroids if hypotension is refractory to vasopressors
- Consider Vasopressin CRI for vasopressor therapy

Multi-organ dysfunction syndrome/multi-organ failure
MODS or MOF can occur secondary to septic insults or hyper-inflammatory states. Many factors influence organ dysfunction including:

- Hypoperfusion from aberrant fluid balance/cardiac output/vascular tone
- Vasodilation amongst organ specific tissue beds
- Decreased oxygen delivery to organs
- Micro-thrombi causing ischemia
- Direct damage caused by infectious agents

Systems approach to treating MOF
- Acute renal failure/oliguria
- Acute liver failure
- Acute mentation change
- ARDS

Nursing Care of the Ventilator Patient

David Liss, RVT, VTS (ECC)
Platt College, Los Angeles, CA

Effective and authoritative nursing management of a sedated and ventilated patient requires pre-requisite knowledge of four different areas:

1. Blood gas analysis
2. Basic knowledge of physiology and mechanics of mechanical ventilation
3. Understanding of physiology and ability to troubleshoot various monitoring devices such as: ECG, Blood pressure (often invasive), Capnography and Pulse Oximetry
4. Understanding of complications of ventilation including: ventilator-associated pneumonia (VAP), ventilator-induced lung injury (VILI), Barotrauma, Oxygen toxicity and cardiovascular effects of mechanical ventilation

After the critical care technician has a foundation in these four content areas, the nursing management (described in this presentation) will define the fifth crucial area of knowledge for veterinary technicians dealing with ventilator patients. Reasoning behind suggestions that these are requisite areas of knowledge for technician working with ventilated patients points to the fact that unless an ICU is extremely lucky to have a boarded critical care specialist and/or emergency veterinarian available at all times to tend to a ventilator patient, often VT's are left with the patient in the ICU and the DVM must go see incoming patients, attend to surgical anesthetic emergencies, deal with other critical patients, or float to a different department. Many times an ICU may not have more than one criticalist, meaning a resident, intern, student or other non-boarded DVM may be involved making the role of the technician all the greater.

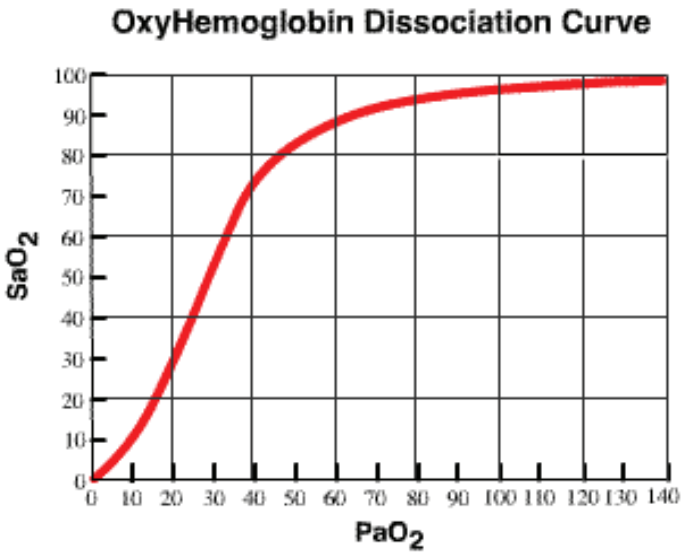
Major nursing areas and topics to be described in this talk include: Basic overview of blood gases, basic overview of monitoring in a ventilated patient, major nursing domains such as: oral and airway care, recumbency care, catheter and tube care, etc and an overview of ventilator alarms and basic troubleshooting.

Blood Gases
More important than metabolic acid-base information in the mechanically ventilated patient would be understanding the oxygenation/ventilation information provided by an arterial blood gas. Traditional blood-gas machines report a partial pressure of oxygen and carbon dioxide delineated with an "a" in the subscript if arterial. Thus, the P_aO_2 and P_aCO_2 are the partial pressures of dissolved oxygen and carbon dioxide in arterial blood. The carbon dioxide level is closely related to alveolar ventilation and directly corresponds to ventilatory rate or tidal volume. Hypercarbia indicates hypoventilation and hypocarbia indicates hyperventilation. CO2 can also be elevated in cases of severe lung disease (alveolar flooding). Normal Co2 is typically 35-45 mmHg.

Interpreting oxygenation indices is slightly tricker. If normal PaO2 is 80-100mmHg on room air, what should the PaO2 be when a patient is receiving 100% oxygen? 60% oxygen? The PaO2 should always be roughly 5 times the inspired oxygen content (FiO2). So a patient breathing 100% oxygen should have a PaO2 of 500mmHg or so. A patient on 60% oxygen should have a PaO2 of around 300mmHg. Now if one receives a blood gas report indicating sub-normal PaO2, how do we know if it is hypoxemia and clinically significant? The PaO2/FiO2 ratio gives a quick estimate of lung function and can be used on patients breathing FiO2's >21%. The traditional measure of lung function is the Alveolar-Arterial (A-a) oxygen gradient, but can only be reliably used on patients breathing 21% oxygen. If the patient's PaO2/FiO2 is <300 (no units) than the patient can be considered at risk or demonstrating clinical signs of acute lung injury. If the patient's PaO2 is <200 the patient is at risk or showing signs of Acute Respiratory Distress Syndrome (ARDS).

The last piece of information to discuss is the relationship between saturation of oxygen at the level of hemoglobin (SO2 or SaO2 if arterial) and the dissolved oxygen tension (PaO2). The oxyhemoglobin dissociation curve gives us this information:

Figure 1 Oxyhemoglobin Dissociation Curve



Points to consider:

1- An SaO2 of 98% or greater corresponds to a PaO2 of 100 mmHg OR greater (flat curve)
2- An SaO2 of 94-95% corresponds to a PaO2 of 80 mmHg indicating appropriate oxygenation at room air
3- Patient's breathing 100% oxygen should not truly have an SaO2 of less than 98%.

Monitoring the Ventilated patient

Monitoring devices employed in the mechanically ventilated patient include: EKG, Blood pressure (non-invasive or invasive), end-tidal Co2 monitoring (capnography), temperature, and pulse oximetry. Understanding each of these is essential to properly administering effective nursing. A in-depth discussion of each of these monitoring devices is beyond the scope of this presentation. Only an overview will be presented.

- **ECG**: Electrocardiography measures electrical conduction in the heart. The heart rate reported by the ECG should always be double checked with auscultation or a manual pulse rate. Cardiac arrhythmias can certainly occur in critically ill patients and can contribute to worsening organ perfusion or cardiac arrest.
- **Capnography:** Capnography (more specifically end-tidal CO2 measurement) reports the CO2 in the patient's endotracheal tube at the very last part of their breath (end-tidal volume). This most closely approximates alveolar CO2 and is fairly accurate. It tends to underestimate alveolar CO2 so normals are about 3-5mmHg lower than arterial measurement of CO2. Mainstream and side-stream options are available. The small T-connector that is placed at the end of the patient's ET tube contributes to additional mechanical deadspace. Constant CO2 measurement can be of great importance in a ventilated patient, not only indicating appropriateness of ventilator settings but also can indicate impending disaster such as barotrauma or cardiac arrest.
- **Blood pressure**: Blood pressure is the measurement of pressure within the arterial system and is affected by heart rate, stroke volume, and systemic vascular resistance. Bradycardic or tachycardic patients may be hypotensive, hypovolemic patients or patients with heart failure may be hypotensive, or patients with vasodilation or vasoconstriction may also have blood pressure abnormalities. It is a very important parameter to measure in ventilated patients as PEEP increases intrathoracic pressure and may decrease venous return and if the patient does not have pre-existing heart disease, septic and SIRS inflammatory mediators can induce cardiomyopathy, causing hypotension and decreased organ perfusion. Non-invasive

blood pressure is typically measured using Doppler technology or an oscillometric model. It is important to note that studies have validated the Doppler and invasive (arterial catheter) methods of blood pressure monitoring in the critically ill, but not oscillometric models. Arterial catheter placement has advantages in the mechanically ventilated patient in that you can measure direct arterial blood pressure and sample for arterial blood gases. The advantages of direct arterial blood pressure include: second-to-second readings, direct measurement of MAP (not calculated), and an arterial waveform.

- **Pulse Oximetry:** Discussed mainly above in the blood gas section. Pulse oximetry is a non-invasive measurement of hemoglobin and if kept normal, assumes oxygen is traversing the blood-gas barrier in the lungs. However, because only slight changes in pulse oximetry can relate to larger changes in PaO2 it must be used with caution.

Physical Exam

Ventilated patients should have regular full physical exams by a veterinarian or technician. This includes a nose-tail assessment of every major body system. Eyes and ears should be examined for any abnormalities such as ocular discharge, conjunctivitis, development of corneal ulcer, or otitis. Cotton balls in the ears should be removed, counted, and changed as needed. The oral cavity should be examined for ulcer development or glossal/hypoglossal swelling. Lymph nodes should be palpated and the front limbs massaged and rotated through full range of motion. The heart and lungs should be ausculted regularly for any crackles, wheezes, changes in heart sounds, or abnormalities in rate. The abdomen should be palpated and the genital area examined. Rear legs should be palpated for muscle atrophy and moved along their full range of motion. Finally, the tail and anogenital areas should be examined for any scald or other developments. Temperature, pulse/heart rate, pulse quality, auscultation, and mucous membrane color/CRT, blood pressure, sedation scores, end-tidal CO2, pulse oximetry, ECG, ventilator settings and laboratory results should be performed and charted at regular intervals.

Oral/Airway Care

Development of oral ulcers, presence of regurgitation, or glossal swelling may occur in a ventilated patient. In addition, an artificial airway bypasses the normal mucociliatory apparatus involved in cleaning the upper airways of debris. If an endotracheal tube cuff is not properly inflated, sedated recumbent patients may regurgitate and are at risk of aspiration pneumonia. Oral/airway care in the ventilator patient involves the following protocols: oral examination, care of the mouth/tongue, and management of endotracheal/tracheostomy artificial airways. Humidification and circuit changes will also be discussed.

Protocol #1: Oral examination/mouth/tongue care

- The oral cavity should be examined on a regular basis for the presence of regurgitation, development of ulcers, or swelling of the tongue
- The mouth should be rinsed or swabbed with a dilute chlorhexidine solution q4-6 hours
- Hard to reach areas, such as under the tongue or the pharynx, can be swabbed with Q-tips dipped in the chlorhexidine solution
- Tongue swelling may occur; applying a small amount of a glycerin solution will often relieve this
- The nursing staff at UC Davis developed an effective nursing intervention protocol for oral lesions which was performed every 6 hours:
    - Gently suction secretions from mouth/oropharynx
    - Lavage oropharynx with dilute chlorhexidine solution
    - Examine oral cavity; describe any lesions and their location
    - Moisten oral mucosa with glycerin
    - Wrap/pad soft tissue structures with glycerin-soaked gauze
    - Reposition ET tube, pulse oximeter, mouth gag, and tongue

Fudge, et al. Oral Lesions Associated with Orotracheal Administered Mech. Vent. In Crit. Ill Dogs. JVECC 7 (2): 1993

Protocol #2: Care of an endotracheal tube

- Endotracheal tubes are considered artificial airways, and as such need to be cared for
- They by-pass the normal defenses of the mouth, larynx and trachea
- If initial intubation was not performed with a new, sterile ET tube, the tube should be changed to a sterile one when possible
- To prevent pressure necrosis the endotracheal tube cuff should be relieved of pressure and the ET tube moved gently in or out about 1" every 4-6 hours
- Even with a humidification system instilling a small amount of normal saline through the endotracheal tube assists with moisturizing the airways and breaks down mucous as saline is mucolytic
- Mucous secretions may develop at the end or inside the endotracheal tube so suctioning is needed. After the instillation of saline a sterile suction catheter, red rubber catheter, or in-line suction catheter should be used to suction the airway
- Make sure to increase the patient's FiO2 to 100% for 1 minute to increase the FRC. Then suction the airway quickly and immediately re-apply the ventilator circuit.

Protocol #3: Care of a tracheostomy tube

- Many patients on long term ventilation may have a tracheostomy tube placed so they can have increased function of their mouth
- The typical tracheostomy setup includes either a permanent tube settled in the trachea and an inner lumen that can be changed, or a permanent tube that must be changed ever so often
- As the patient has normal mouth function secretions build up quite often with a tracheostomy tube
- The patient should be hyper-oxygenated prior to suctioning or nebulizing their airway
- The breathing circuit can be quickly disconnected, the suctioning performed, and the circuit re-attached
- The incision site around the tracheostomy tube can be gently cleaned with sterile Q-tips and sterile saline and dilute chlorhexidine

Protocol #4: Humidification and circuit changes

- Circuit changes may be necessary if they become clogged or what was assumed to be best practice (in the past) to change them.
- However, frequent circuit changes is considered a risk factor for VAP and thus the CDC recommends only changing the ventilator circuit NO MORE than every 48 hours

Recumbency/Passive Range of Motion

Ventilator patients are recumbent and require thick bedding to prevent the development of decubital ulcers. Any pressure points (elbows, hips) can develop ulcers quickly and prevention involves thick padding and rotating the patient to disperse the pressure placed on these areas. Keeping the patient sternal is preferred and then the patient's hips may be rotated from side to side every 4hours. Careful monitoring for any excrement is necessary to prevent scald and tissue trauma leading to secondary bacterial colonization. Passive range of motion and massage therapy can help maintain appropriate blood flow to the periphery and is important to do during "down-time" between treatments.

Catheter/Tube Care

Ventilator patients OFTEN have the following "tubes" that need care: Peripheral IV catheters, jugular/saphenous central lines, arterial catheters, urinary catheters, feeding tubes (NG, E-tube, PEG), and potentially chest tubes, abdominal drains. All of these should be monitored for any signs of inflammation or infection. Catheters (peripheral or central) should have catheter care performed at least every 24 hours: strip the bandage material and tape off of the catheter and inspect the site, while wearing non-sterile gloves. If there are any signs of phlebitis the

catheter may need to be removed. Urinary catheters should be cleaned with dilute chlorhexidine from the prepuce to the urinary catheter bag by wiping distally to move any bacteria/contamination away from the urinary tract. The vaginal vault or prepucial sheath should be flushed with chlorhexidine solution as well. Urinary lines (if not completely closed systems) should be changed every 24 hours.

Charting/Record Keeping

Charting and record keeping is very important with ventilator patients. Flowsheets should include hourly treatments both ventilator-specific and those accompanying the primary disease process. If this is too much information, a second ventilator-specific flowsheet should be created to facilitate administering all of the appropriate medical care. Ventilator settings (tidal volume, RR, I:E ratio, PEEP levels, airway pressures, mode, FiO2) should be recorded every hour. Regular treatments: suctioning, cuff deflation, body positioning, etc can be listed on this sheet and then highlighted or marked to be performed at the appropriate intervals. Clear and legible handwriting is very important.

Ventilator Alarms/Troubleshooting

Common problems in ventilator patients include: Ventilator-induced lung injury (VILI) and ventilator-associated pneumonia (VAP). Recommendations from human medicine been quite successful in implementing care bundles to identify patients at risk of VAP and institute care immediately if VAP is suspected. The care bundle is presented here:

1- Wash hands and do not wear rings when dealing with ventilator patients.
2- Elevate patient's head 30-45° when possible.
3- Avoid gastric overdistension
4- Avoid intubating and re-intubating if possible
5- Use of a cuffed ET tube plus in-line subglottic suctioning

A common problem with ventilator patients is ventilator-patient dysynchrony or "bucking." This is usually caused by one of two things: 1- Sedation/analgesia is not adequate and patient is "light" or 2- Ventilator settings are not adequate for patient causing distress. Re-assess the ventilator settings and the patient's sedation levels if you find the patient is reacting to the ventilator.

Common ventilator alarms include:

*****Make sure all alarms are set to appropriate patient settings. No sense in the low FiO2 alarm going off IF the patient is supposed to be on 21% oxygen!*****

Low tidal volume alarm: This occurs when the machine senses a tidal volume breath that did not make it to the pre-set tidal volume. This can occur if there is a leak in the ventilator circuit, ET tube or cuff, or the cuff is not sealed appropriately.

Low pressure alarm: This occurs for similar reasons the low tidal volume alarm would go off. However, it is important to note that an early pneumothorax can trigger a low pressure alarm as there ventilator driven pressure is now "leaking" into the pleural space.

High pressure alarm: This may occur because there is a blockage in the ET tube or circuit, or the patient has developed a pneumothorax from barotrauma. This is a very serious alarm .

Low FiO2 alarm: Another serious alarm sensing that the delivered oxygen concentration is not what the pre-set levels are supposed to be. This may indicate an oxygen failure, machine failure, or inadequate supply of oxygen in the hospital.

References available upon request

Critical Care Nursing Tales from the Trenches

David Liss, BS, RVT, VTS (ECC, SAIM), CVPM
Los Angeles, CA

Introduction

Dealing with emergencies often involves life-saving procedures and team efforts to bring patients back from the brink of death. This lecture will present two cases from admit to discharge and show how the veterinary critical care team can jump into action and deal with the most complicated and critical cases.

Case 1

Signalment: 2y MI Yorkshire Terrier
Presenting complaint: Hit by car
Physical exam: HR: 40 BPM, RR: Apneic, MM- Gray, Large laceration across thorax
Treatment summary:

- IV Catheter and fluid bolus
- Intubation/CPR
- Open chest CPR
- Chest tube placement/suture of thorax
- Analgesic medication
- Jugular catheter placement
- Urinary catheter placement
- Sedation/Intermittent Positive pressure ventilation
- Weaned from IPPV- oxygen cage
- 2 days of intensive care
- Pelvic fracture repair
- Discharge 8 days later

Case 2

Signalment: 2y MN DSH
Presenting complaint: ADR, Diarrhea
Physical exam: HR 60 BPM, Cyanotic, Apneic
Treatment summary:

- IV Catheter- fluid bolus
- ECG
- Blood gas
- Administration of insulin, calcium, dextrose
- Intubation/CPR
- Recovery from CPR
- Urinary catheter placement
- 1 day later- nasogastric tube placement
- Urinary output monitoring
- Peritoneal dialysis
- Jugular catheter placement
- Preparation for hemodialysis transfer
- 2 days ICU treatment
- Surgery for bilateral ureteroliths
- Discharge 6 days post-op

Parasitology, Microbiology, Cytology and Histopathology (and Chemistries)

Saundra E. Willis DVM DACVIM

Parasitology

Fecal testing in health:

- If routine deworming done: double or single centrifugation, or passive floatation
- If routine deworming not done: double centrifugation for optimal accuracy

Fecal testing in disease: consider one or more of the following

- Direct smear in clinic
- Double Centrifugation Fecal
- Giardia/Crytosporidium IFA
- Giardia elisa
- Prophylactic deworming
- Other testing
- (note: for salmon poisoning, trematode eggs are best found on a complete fecal exam including direct smear and double centrifugation)

Direct Mount:

- Wet mount made by mixing drop of fecal sample in drop of saline or water
- Mix gently
- Coverslip and examine for motile parasites, worms and other larvae, 10x and 40x
- Best done immediately, ie. in clinic

Tritrichomonas InPouchTF for identification of *Tritichomonas foetus*

- Fecal sample size should be no larger than the size of a peppercorn
- Incubate pouch in vertical position at room temperature as refrigerator temperature will kill the trichomonads
- Avoid cat litter
- (please read: https://cvm.ncsu.edu/wp-content/uploads/2016/05/ownersguide-to-feline-t-foetus.pdf)

Fecal Cytology:

- Fresh sample (less than 15 minutes old). Technique: Spread thin on slide, air dry, heat fix, stain
- Normal fecal cytology: >90% mixed population of rods, Few epithelial cells, Few/no WBC's, no RBC's
- Scan 10x
- Epithelial cells, abnormal cells
- Intestinal irritation, neoplasia
- Scan 40x
- Increased WBC's: colonic inflammation
- Increased RBC's: intestinal bleeding
- Bacterial numbers and types

Technique	Whipworm (false negatives)	Roundworm (false negatives)	Hookworm (false negatives)
Direct Smear	92.61%	85.38%	72.82%
Ovassay	32.02%	25.88%	4.85%
Centrifugation	4.93%	10.53%	0.97%

Internal Parasites can be difficult to Diagnose

Fecal Solutions:

Sodium Nitrate	1.18-1.20	Good all purpose
Zinc Sulfate	1.18-1.20	Best general all- purpose
Sheathers Sucrose	1.27	Excellent all-purpose
Water	1.00	(example only)

Fecal (O/P) Double Centrifugation:
Fecal Sedimentation: Concentrates feces and therefore ova and larvae, Used to gather heavy ova such as Trematode eggs

Fecal Centrifugation: Centrifugation increases flotation of ova, oocysts, etc to the top of the solution
Creates highest yield of ova, decreases amount of debris to sort through

Double Centrifugation:
Prepare a fecal emulsion using one to six grams feces and 10-12 mls. of water in a paper cup, strain the emulsion through a gauze square into a second paper cup, examine for larvae and worms.

Label one 15ml conical centrifuge tube with the specimen number, Pour the fecal emulsion into the labeled tube to the 15ml mark, Add water to the tubes to balance them (if necessary), Centrifuge for 10 minutes at 1,500 rpm

When the centrifugation is complete, decant supernatent, add sugar solution to the halfway point on the tubes, Mix the fecal sample with the sugar solution with an applicator stick. Complete mixing is important to avoid chunks of material on the slide. Avoid sample contamination by always using new mixing sticks between samples, and not allowing pipettes to touch samples. Add more sugar solution to each tube to reach the 15ml mark. Centrifuge the tubes again for 10 minutes at 1,500 rpm.

Carefully set up tubes in the test tube rack. Use a plastic pipette to gently run additional sugar solution down the side of the tube, create a slight positive meniscus. Disturb the contents as little as possible
Set a 22mm square coverslip on top of each tube. Let stand for 5-10 minutes. Remove the coverslip by lifting straight up and place on a slide. Label the slide
Examine for parasites
10X magnification
40X magnification

Recipe for Sheather's Solution

- Heat 36 ml (1.5 c) water to just boiling
- Add 454g (2.25 c) granulated white sugar
- Mix until completely dissolved
- Pour into plastic container and cover immediately
- Store at 4C for long periods of time. Store at room temp. when container is being used daily

Fecal Quantification: Large Animal, Modified McMaster Test
1 gram feces/15 mls water. Double centrifugation using Sheather's Sugar. Flotation for one hour. Every egg counted. Number given per species per one gram.

Microbiology
Sample Submission:
Culturette
Culturing tissue: Piece of tissue in saline, CTT
Culturing urine: Send urine, cystocentesis preferred
Mycoplasma cultures: Standard c/s best
Provide history, and note anything you are looking for.

Cytology
History:
Size of mass (measure! be clear on units)
Location (site: cutaneous, subcutaneous)
Nature of mass (firm, hard, soft, fluid-filled)
Known duration and any changes in that time
Relevant medical history
Blood work results – hypercalcemia, neutrophilia, etc.
Avoid abbreviations unless common
If multiple sites, please be clear in history and on slides
Large masses – it is helpful to note different aspirate sites so know where to biopsy

Equipment:

- Gather and prepare ALL supplies prior to aspiration

- Needle size: 21-22 gauge, Larger gauge (16-18) – bone
- Syringe: 6 mL (or 3 mL)
- Glass slides – frosted edge
- Tubes – EDTA, serum/clear top

How to Approach the Lesion

- Is it ulcerated: If so, go deep
- Impressions – often of limited diagnostic utility. Exceptions: fungal infections, some well-exfoliating tumors
- Is it large: aspirate multiple sites
- Is it fast growing (and large): avoid the center

Collection Techniques:
Non-aspiration/fenestration: Helps minimize blood contamination and often better preserves cells. Have air-filled syringe ready prior to collection. Have syringe attached when aspirating internal structures. Finger over hub of needle in case of a fluid-filled structure

Aspiration: Good for more sensitive areas or very firm/hard masses. Pull back on plunger and quickly release to minimize blood contamination. Ensure vacuum is released prior to withdrawing needle
Imprint/scrapes for biopsy specimens: Dab on gauze/paper towel until surface is dry/tacky, press slide onto surface – do NOT drag/smear along the slide surface

Can also use backside of scalpel blade for firm tissues (similar to skin scrape) and then smear those cells onto a slide

A note when using ultrasound…Use as little gel as possible!

Application to the slides:
Use the air-filled syringe to push the aspirated material onto the slide. Aim for the "butt" of the slide (near the frosted edge0 such that there are more cells to look at (less "wasted" cells). This will make sure slides are correctly stained, given the variation in type of automated stainer between labs and that high power can be used to view the slide.

In-House Evaluation:
Stain 1 slide prior to submission. Assess cellularity, cell intactness: have I sampled the tissue I thought I did? SUBMIT THIS SLIDE WITH THE OTHERS

Dif-Quick Stain:
Do not heat fix. Have chemical fixative. Have 2 sets – "dirty" and "clean". Change and/or filter regularly. Dye will precipitate out with time and/or will become exhausted. Abundant stain precipitate on slide: Quick dip in the solvent (blue stain/1st step). It is easy to understain – appropriate times are crucial. Thick smears require longer time.

Packaging
Ensure slides are COMPLETELY air-dried. Exception: lipomas/fat-rich samples will NEVER appear dried. Do NOT refrigerate any slides. Ensure slides are all labeled: Name, Site. Use pencil or markers specific for slide labeling (sharpies and other "permanent" markers can be dissolved).

Specific Tissues and Samples:

Cystic Fluid-Filled Masses
Fluid often of limited diagnostic usefulness on its own. Always try to aspirate the wall of the mass. Both the wall and the fluid count as one site, so still submit both! Put sample into BOTH an EDTA and serum/plastic-topped tube . EDTA: cytology, Serum: chemistry testing, culture. If may want both cytology and culture, recommend two separate tubes

Always prepare at least 1-2 slides at time of collection and submit with fluid.

Urine:
Split sample into 2 tubes. Centrifuge 1 and prepare 1-2 smears of the pellet as soon as possible following collection. Submit unspun urine in tube, spun pellet in tube, and air-dry slides on a cytology form

Mammary Gland:
Often CANNOT differentiate benign from malignant mammary tumors. REQUIRE HISTOPATHOLOGY.
Good to diagnose mast cell tumors, lipomas, and other non-mammary origin tumors

Skin (non-mass lesions):
Cytology very limited utility in many chronic skin diseases, Require histopathology for assessment of architecture
Mites, pyoderma should be able to be identified in-clinic.

Histopathology
Sample Submission: Right proportion of formalin to sample, 1 to 10. Formalin only penetrates 1cm into tissue. Make cuts into larger masses to facilitate penetration of formulin. Larger tissues can be wrapped in bags and placed on ice. Call for help in sending in tissues. From our technicians: Tightly close jar, and tape shut.

Chemistry Submission
1. 1-2 mils of serum required (always send a bit more for additional testing)
2. Allow blood to clot for 30 minutes to begin clot retraction, decreases hemolysis
3. Centrifuge in SST or RTT
4. Avoid hemolysis: undertake a clean venipuncture, allow vacuum in tube to draw sample into tube
5. Avoid lipemia: 8-12 hour fast is ideal. Ultracentrifugation can be done at the laboratory to remove lipemia by creating a "creme layer" on top of the cleared serum. Serum is then aspirated from below the "creme" layer with a pipette.

Blood-Chemistry Panel
A blood-chemistry panel measures electrolytes, enzymes and chemical elements of the blood to assess several different organ systems in the body. This will help detect endocrine disease, renal and kidney disease, hepatic or liver disease, gastrointestinal disease (including the pancreas); disorders of acid-base, chemicals such as calcium, electrolytes such as potassium, protein disorders and blood lipids such as cholesterol.

Blood Chemistry Analytes include:
- Albumin- one of the major proteins in the blood. With globulins, comprise total protein. Low levels can be seen with hepatic (liver) disease, certain types of kidney disease, and gastrointestinal disease. Blood loss can also lower albumin and globulin levels.
- ALP (alkaline phosphatase) – enzyme that increases in liver disease and with elevations in cortisol. The highest ALP increases are seen in hyperadrenocorticism, termed Cushing's disease, or when the dog is on corticosteroids due to increased production of this enzyme. Increased ALP in cats always reflects hepatic disease; steroids do not cause an elevation of this enzyme in the cat.

- ALT (alanine aminotransferase) - enzyme found primarily in the liver cells, termed hepatocytes. ALT increases reflect hepatocyte damage from may causes.
- Amylase-enzyme produced in the pancreas that digests carbohydrates in the gastrointestinal tract. Elevated levels can but don't always indicate pancreatic inflammation.
  AST(aspartate aminotransferase) - enzyme found in skeletal and heart muscle and liver. When elevated with ALT, most likely reflects liver disease. AST elevations alone support muscle disease.

Bilirubin – bilirubin is produced by the liver from old red blood cells and is excreted in the urine and stool. Bilirubin is increased in some types of liver disease, particularly gallbladder disease and in autoimmune hemolytic anemia due to destruction of red blood cells in the circulation. When bilirubin levels in the blood reach a certain level, we will see a yellow coloring to non haired areas like the gums and ears. This is called jaundice or icterus. An increase in bilirubin will also color the urine a deeper yellow (see section on urinalysis)
BUN (blood urea nitrogen) - BUN is a waste product of protein metabolism It is produced by the liver and excreted by the kidneys. Decreased levels can indicate hepatic dysfunction but can also be seen with consumption of a low protein diet. Elevated values are seen with dehydration, kidney disease and with high protein levels in the diet or in the gut such as seen with gastrointestinal bleeding. A urinalysis (specifically the specific gravity) is necessary to determine if the elevation in BUN (and creatinine) is due to kidney disease or dehydration.

Calcium-calcium in the blood stream comes from bones. A hormone called parathyroid hormone (PTH) from the parathyroid gland (small glands next to the thyroid in the neck) regulate blood levels of calcium. High blood calcium, termed hypercalcemia, can reflect disorders of PTH including a hyperactive parathyroid gland, termed hyperparathyroidism, malignancy, or idiopathic hypercalcemia (cause unknown) seen in the cat. In these conditions, the phosphorus level may be low. Low blood calcium, termed hypocalcemia, can occur due to a malfunctioning parathyroid gland resulting in hypoparathyroidism but other conditions such as eclampsia (nursing pets) and antifreeze toxicity can lower calcium levels.

Chloride – chloride, sodium and potassium are electrolytes. They serve many different functions within the body. Low values of chloride, termed hypochloremia, are most often seen with vomiting. High values suggest dehydration or loss of water from the body.

Cholesterol - sterol in the blood. Cholesterol is produced in the liver, and low values, termed ypocholesterolemia, can be seen with hepatic dysfunction, gastrointestinal disease, and hypoadrenocorticism or Addison's Disease. High cholesterol values can be seen with diabetes, hypothyroidism and Cushing's Disease. In the advanced stages of glomerular disease, diagnosed by finding high proteins in a urine that shows no evidence of inflammation (high urine protein:creatinine ratio), cholesterol can be elevated, and albumin decreased. With ascities, this triad of signs is termed nephrotic syndrome.

CO2-reflects bicarbonate (HCO3) levels in the blood. Low levels support acidosis, high levels alkalosis. CO2 can be helpful in some instances but measurement of blood gases using an arterial or venous sample is more accurate. Blood gas analyzers are usually available at emergency and/or specialty veterinary hospitals.

Creatinine - creatinine is a waste product from muscles. It is eliminated by the kidneys. As with BUN, creatinine increases with both dehydration and kidney disease thus the urine concentrating ability, determined by urine specific gravity, must be analyzed to determine cause of an increased creatinine. In thin cats, creatinine may be falsely decreased due to their reduced muscle mass.

Creatinine Kinase – creatinine kinase or CK is released from damaged muscles. CK elevations support some type of acute muscle damage including heart muscle. Transient and sometimes significant elevations can be seen with injections and blood draws.

Globulin - blood protein that most often increases due to inflammation but also can increase with rickettsial disease and with neoplasia. Protein electrophoresis can help us determine if the globulins are elevated due to inflammation or neoplasia.

Glucose- blood sugar. Hyperglycemia, support a diagnosis of diabetes. However, stress in the cat can result in transient hyperglycemia. Pancreatitis can cause hyperglycemia. Hypoglycemia, low blood sugar level, can be seen with starvation, an insulin producing tumor and other neoplastic conditions, liver dysfunction, and systemic infection, sepsis.

Lipase – produced by the pancreas. Elevations may indicate pancreatic inflammation but can be seen with kidney and gastrointestinal disease. Lipase and amylase elevations are not helpful in the diagnosis of pancreatitis in the cat.

Osmolality – osmolality is a measure of the concentration of substances such as sodium, chloride, potassium, urea, glucose, and other ions in blood. An increase in osmolality can help determine if a toxin such as ethylene glycol (antifreeze toxicity) has been ingested.

Phosphorus – phosphorus and calcium levels in the blood are controlled by PTH. Phosphorous increases in the blood in kidney disease, generally in chronic renal disease, and also in dehydration. Cats with hyperthyroidism can have higher phosphorus levels. A low calcium level and high phosphorus level is consistent with hypoparathyroidism, diagnosed by a low PTH level. A high calcium and low phosphorus level in the face of a high PTH level diagnoses hyperparathyroidism.

Potassium – an electrolyte. High levels of potassium, termed hyperkalemia, can be seen with acute renal failure (such as seen with antifreeze toxicity), lower urinary tract obstruction, and with Addison's Disease. Hyperkalemia can be life threatening as it can cause a dangerously low heart rate, termed bradycardia. Low blood potassium, termed hypokalemia, is seen most often in cats due to decreased appetite or but can present in dogs and cats with potassium wasting through renal and gastrointestinal disease. Hypokalemia can cause severe muscle weakness.

Sodium - one of the major salts in the body fluid, sodium is important in the body's water balance and the electrical activity of nerves and muscles. High sodium values can be seen with dehydration and water loss. Low sodium values, termed hyponatremia, can be seen with vomiting and loss from the gastrointestinal tract, kidney disease, and with Addison's disease.

Total Protein – total protein is made up of albumin and globulin. Total protein can be elevated when either one is elevated but if both are elevated, dehydration/water loss is generally the cause. A decrease in both albumin and globulin may reflect gastrointestinal disease or hemorrhage. Clinical findings will help determine which cause is more likely.

Total T4 – tetraiodothyronine or T4. Basic test for hypothyroidism (dog) and hyperthyroidism (cat).Various illnesses can cause a low T4 (nonthyroidal disease) which can make a diagnosis of hypothyroidism in an ill or stressed dog difficult. In the older cat, concurrent disease with hyperthyroidism such as kidney disease can push the Total T4 down into the mid to high normal range. Thus confirmatory testing may be needed to accurately diagnose hyperthyroidism in a cat for which the total T4 level is not elevated.

Groups of Tests by Organ System

Although a basic screen provides the most information, we often group tests by the organ systems they are testing, for example:

- Kidney Disease - BUN, creatinine, phosphorus, potassium, urinalysis
- Liver Disease - ALT, AST, ALP, GGT, bilirubin, albumin, cholesterol, BUN

- Gastrointestinal Disease - albumin, globulin, cholesterol
- Pancreatic Disease – amylase, lipase

Additional Testing
After reviewing the results of screening tests, we will narrow our differential list. Some potential diseases we were considering now are ruled-out or seem less likely, others more likely. Perhaps our screening tests have given us the diagnosis or enough information to make a tentative diagnosis. But often we need additional testing to get that diagnosis or further refine our differential list.

Hematology and Urinalysis: Optimize Your In-Clinic and Reference Laboratory Testing

Saundra E. Willis DVM DACVIM

Introduction

Complicated medicine disease and diagnostic testing can be challenging for pet owners. It can be frustrating for owners when they don't understand the VALUE of testing. They will often decline an option that is not understood. This is particularly true if the veterinarian has not clearly and concisely outlined what they think the problem or problems might be, the potential disease(s) causing the problem(s) and the diagnostic tests needed to rule-in or out those potential diseases.

"Enough tests already just treat" owners say. Or one may hear from an owner: "They are just going to do a bunch of tests" when a friend suggests a visit to the veterinarian for an ill pet. The veterinarian may or may not do a "bunch of tests" but how does the veterinarian know how to treat the patient if they don't know what is wrong? The most direct way to achieve lasting relief and return to health is by uncovering what the problem really is, by diagnosing the disease, so that treatment can be definitive and not simply empirical. Diagnosing disease also makes financial sense. Money is not wasted on treatments for diseases the pet does not have.

As we move through these sections on diagnostic laboratory testing, continue to think of how you would describe testing to an owner to optimize their understanding of what can often be a complicated process. Understanding will help them make the best choices for their pet.

The tests: A Practical Guide

Generally, we start diagnostic testing with screening blood work which most often includes a Complete Blood Count (CBC), blood chemistry profile and (ideally) a urinalysis. The blood chemistry profiles differ only slightly between diagnostic laboratories. The screening panel provides the most information cost effectively. Depending on the initial results and the pets response to our supportive therapy, we may order more specific blood tests.

Below are the most common tests included in a general panel, a brief description of the test, and why we use it. I have also included a few more specific blood tests that we may order depending on the results of the screening tests.

Complete Blood Count (CBC)

A CBC is performed to assess for disorders of the red cells anemia and polycythemia (too many red cells), disorders of the WBC's including infection, non-infectious inflammation, cancers like leukemia, allergies and parasitic infections, and disorders of platelets the main one being thrombocytopenia, or low platelet count.

Red Blood Cell Count (RBC) – number of red cells per volume of blood (/UL)

Hematocrit (HCT) or Packed Cell Volume (PCV) - calculated percentage of red blood cells in the circulation. It is either determined by the hematology analyzer as hematocrit or HCT, by multiplying the RBC count by the MCV or by centrifugation of a small blood sample to pack the RBC's and determine a Packed Cell Volume or PCV. The HCT and PCV should agree.

Hemoglobin (Hgb) - essential oxygen carrying molecule within RBC's
Note: Although RBC, HCT/PCV and Hgb all equally measure RBC mass, we tend to use HCT or PCV to assess for anemia or polycythemia.

Mean Corpuscular Volume (MCV) - average size of the red blood cells. A high MCV may indicate certain vitamin deficiencies. A low MCV consistent with microcytosis on a slide review often supports iron deficiency

most often attributed to chronic blood loss or hepatic disease. Poodles and poodle mixes can have a hereditary macrocytosis.

Mean Corpuscular hemoglobin Concentration (MCHC) – average concentration of hemoglobin in each red blood cell. We most often see a low MCHC in iron deficiency which appears as hypochromasia on a slide review

Reticulocytes - immature red blood cells. A regenerative anemia has an increased number of reticulocytes meaning that the bone marrow is responding. Regenerative anemias are seen with acute hemorrhage or coagulation disorders and immune-destruction of RBCs. Nonregenerative anemias reflect anemias due to chronic disease such as kidney disease, cancer, and bone marrow disorders and a more chronic, protracted hemorrhage resulting in iron deficiency. After an acute hemorrhage, it can take up to 3-5 days to result in an adequate regenerative response.
Note: MCV and MCHC help us to classify the type of anemia and determine its cause.

White Blood Cell Count (WBC) – number of total white blood cells per volume of blood (/UL)

Differential
The differential provides an analysis of the different types of white blood cells that make up the total WBC count. The differential can be done manually by counting the number of each cell type in a total count of 100 cells; this provides a percentage of each cell type. The absolute number of each white blood cell type is calculated by multiplying the percentage of each type by the total white blood cell count. It is the absolute value and not the percentage that we use to identify how or low cell counts. Automated hematology cell counters provide a differential. The accuracy of the machine differential depends on the type of analyzer and essentially the normality of the CBC. Automated analyzers will hint at the presence of abnormal white cells such as bands, toxic change, neoplastic lymphocytes, mast cells, etc. Only a manual slide review can confirm the accuracy of an automated differential and detect the presence of these significant morphological changes.

Neutrophils - primary white blood cells responsible for fighting infections. Neutrophils increase in inflammation due to causes such as infection and neoplasia. Low neutrophil counts can indicate a severe overwhelming infection, viral disease or a bone marrow disorder.

Lymphocytes – component of the immune-system that produces antibodies and other substances involved in immunity. Low lymphocyte numbers, termed lymphopenia, can occur in viral infections and with metabolic stress. High numbers, lymphocytosis, can occur with certain endocrine diseases, rickettsial diseases and with tumors/leukemias of lymphoid tissues..

Monocytes – circulating form of tissue macrophages, that are phagocytic. They ingest large particles including bacteria and work to clean up inflammation. Their numbers increase with inflammation, tissue and tumor necrosis. Low numbers have no clinical significance.

Eosinophils - primarily involved in allergic reactions and parasitic infections. Eosinophilia can occur in allergic and hypersensitivity conditions, parasitism, and as a paraneoplastic syndrome. Eosinophilia has no clinical significance.

Basophils - uncommon WBC but can be seen in certain parasitic infections including heartworm and with allergic conditions. Similar to eosinopenia and monocytopenia, low numbers have no clinical significance.

Platelets - play an important role in blood clotting. Platelets in a blood sample may clump falsely decreasing the analyzer count. When the analyzer cound is low a blood smear review is required to assess and estimate platelet numbers. Increased platelet numbers, termed thrombocytosis, can occur from a variety of problems most

commonly inflammation. Low platelet numbers, termed thrombocytopenia, can indicate immune-destruction of platelets, rickettsial disease or a coagulation disorder.

Urinalysis
The urinalysis includes the physical, chemical and microscopic evaluation of the urine. Clean free catch urine samples are fine for the analysis of most components. Culture of a free catch sample often yields bacterial growth due to contaminants thus urine collected by cystocentesis is preferred for urine culture. A urinalysis is essential for determining whether azotemia (increased BUN and creatinine) is due to dehydration or kidney disease. A urinalysis is also crucial to the diagnosis of endocrine disease causing polyuria/polydipsia such as diabetes mellitus and Cushing's Disease causes of hematuria and urinary tract inflammation including infection and urolithiasis and prostatic disease and urinary tract neoplasia.

Parts of the Urinalysis:
Color and Turbidity - urine is yellow in color, the depth of color varying with the urine concentration. Hematuria, hemoglobinuria, and bilirubinuria are the most common causes of discolored urine; the former two making the urine red, the latter, more orange. The most common causes of increased turbidity are pyuria (increased WBC's), crystalluria, and lipiduria (increased fat).

Specific Gravity - determined by refractometer, not by dipstick. A measure of urine concentration, and therefore of kidney function. A specific gravity of 1.000 is equivalent to water whereas 1.090 is highly concentrated urine. A specific gravity greater than 1.030 in the dog and 1.035 in the cat indicates that the kidney is functioning to concentrate the urine. Urine specific gravity below 1.008 is termed hyposthenuria, the kidneys are actively diluting the urine, and between 1.008 to 1.012, isosthenuria, in which the kidneys are neither diluting nor concentrating the urine.

Chemical Analysis:
pH - a pH of 7.0 is neutral; most dog and cat urine is acidic with pH ranging from 5.8 to 6.5 but any pH can be normal. Alkaline urine, pH > 8.0, may be due to infection.

Protein - normal urine should not contain protein. Protein in the urine can be due to RBC's, WBC's, bacteria, epithelial cells or in the presence of an inactive sediment (normal sediment), a type of kidney disease called glomerulonephritis.

Glucose - normal urine should not contain glucose. Glucosuria can be seen with diabetes mellitus and a high blood sugar, or with kidney disease when the blood sugar is normal. Cats can become transiently hyperglycemic and glucosuric with stress.

Ketones - normal urine should not contain ketones. Ketones and glucose in the urine support a complication of diabetes called diabetic ketoacidosis. Treatment may involve IV fluids and insulin given IV or IM and a search for an underlying complication to the diabetes.

Bilirubin - bilirubin in the urine can be normal in dogs particularly in concentrated urine. Bilirubin is never normal in cat urine. Bilirubinuria in both the dog and cat can indicate liver disease or a hemolytic anemia.

Blood - positive heme reactive on the dipstick can be due to RBC's in the blood or hematuria, dissolved blood or dissolved myoglobin due to muscle damage. Hematuria is seen with many conditions: bladder inflammation or infection, uroliths or stones, bladder neoplasia, prostatic disease. Bleeding disorders can also cause hematuria.

Hemolysis and RBC lysis such as seen with an immune mediated anemia will also cause a positive heme reactive. (Dipsticks are not accurate for Specific Gravity, Urobilinogen, Nitrate, and WBC's/leukocytes)

Urine Sediment Exam:
RBC's - RBC's occur from conditions similar to those listed under hematuria.

WBC's - WBC's support inflammation which can be due to infection, noninfectious inflammation such as bladder stones, and neoplasia.

Epithelial Cells - Epithelial cells come from the lining of the urinary tract and may originate from the kidneys, ureters, bladder, urethra, prostate (in a male) or external orifice of the urethra/vulva. One cannot determine the origin of the epithelial cells based on their appearance.

Bacteria - Urine is sterile but easily contaminated. Even a sample taken by cytocentesis may contain low number of contaminating bacteria. Bacteria are rods singly or in chains/groups or cocci in pairs or chains. Debris and amorphous crystals can appear to be bacteria. Sediment exam is best done unstained to avoid stain artifact. Air dried slides of urine sediment can be prepared and stained with Dif Quik to better assess for presence of bacteria, epithelial cells, etc.

Crystals - Crystals in the urine (crystalluria) means that the urine is oversaturated with the substances making up crystals. Urine pH and urine concentration can influence crystalluria. Crystalluria may not be clinically significant and must be interpreted with the history and clinical signs. Some crystals, such as calcium oxalate and struvite can form as the urine cools and may not be present in the patient at body temperature. Crystals such as urate and cystine support a metabolic problem in the patient.

Casts - Casts originate in the kidney and are actually microscopic "casts" of the kidney tubules. Their presence supports kidney disease but they are very fragile and not always seen in the urine. Cats occur more often in diseases that cause proteinuria and in cases of renal toxicity such as antifreeze toxicity.