

OVTAA JOURNAL

Oregon Veterinary Technician and Assistant Association

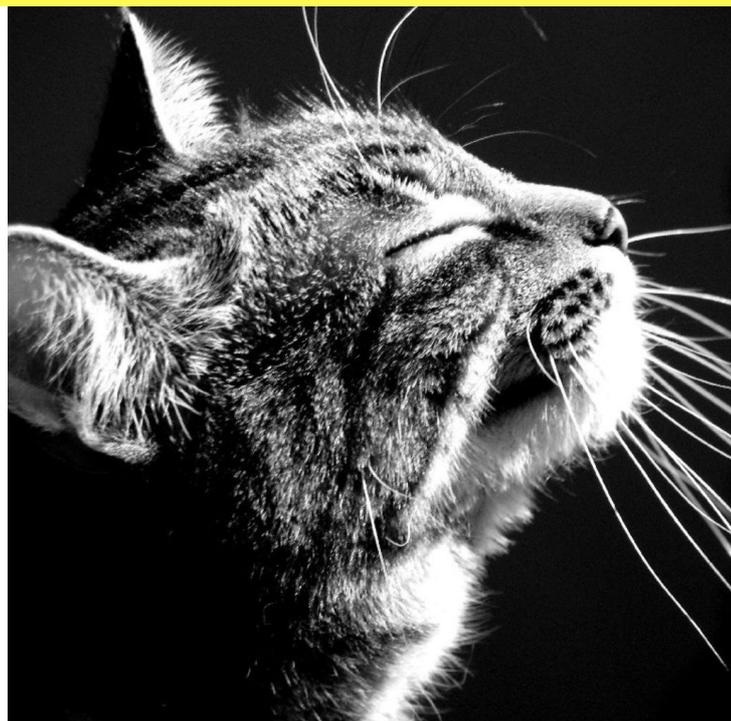
A MESSAGE FROM THE PRESIDENT

Dolores Galindo, CVT

OVTAA is gaining momentum in working for you, the membership. We are working for both Veterinary Technicians and Veterinary Assistants to promote our profession and to get you all recognized for the important work you do. Some of the things we have done in the last year include working with PVMA offering a wellness summit, organized a town hall meeting for techs and assistants to share their concerns about issues they face in practice and offered an event focusing on the utilization of veterinary technicians, to name a few.

We have offered some continuing education events and have one of our board members on the Oregon Veterinary Medical Examining board participating in legislative issues that affect veterinary technicians and assistants. We have a new webmaster for our website and while we are still under construction we are making some headway! We want to focus right now and into the immediate future on informing the public about who veterinary technicians and assistants are and what they do.

The OVTAA board members are a small group of dedicated individuals with full time jobs who volunteer their time to work to keep our organization active and growing. We need dedicated people like you to stand up and commit to the improvement of our profession overall, to offer yourself as a speaker for CE, to help out with our website and so much more. We have created an online option to conduct some of our meetings so that it is easier for our members to attend and we continually strive to determine where we can help our members. Our dream is to have a strong association that maintains its strengths and continues to grow so that we can create a profession where a sustainable living can be made and where we are recognized as medical professionals where we can work within our veterinary team to to continue to help the public and the animals they care for.



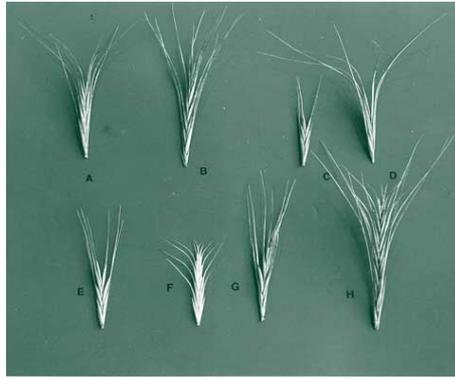
OVTAA WEBSITE

The OVTAA website is currently under construction. Soon you will be able to access membership information, CE opportunities, job postings and much more. Stay tuned!
<https://ovtaa.org>



THE BAD SEED

By Danielle Nelson, CVT
Bethany Family Pet Clinic



Shown: Common wheat grass awns / Image
credit: Smith Veterinary Hospital

The client calls and says: 1. “My dog has been scratching at his ears since we went hiking yesterday.” 2. “My dog has been licking at his paws since we came home from camping.” 3. “I took my dog to do scent work training and she’s been sneezing since we got back.” 4. “Something’s not right with my Search and Rescue dog” 5. “I took my dog bird hunting and she doesn’t seem to be breathing right.”

The scenarios are varied, but the common rule-out should be grass awns, foreign body migration and/or infection.

Grass awns are pieces of grass seeds, sometimes barbed and/or plumed, that can penetrate skin, be inhaled, ingested or get tangled in a dog’s coat as they run through an area with these grasses. Towards the end of summer these grasses can go to seed and be problematic, even fatal, to dogs and cats that have a grass awn foreign body infection or migration.

As a CVT with only five years of experience at a general practice, I’ve only seen two of these cases. One recent case, the grass awn was lodged in the dog’s ear and the other dog had many grass awns that had not been brushed out of the dog’s long coat, eventually penetrating the skin and causing severe irritation with a mild infection.

Both dogs needed heavy sedation for foreign body removal due to the pain, neither had pyrexia. However, as a Sporting Dog Handler, grass awns are a constant topic of conversation, story swapping and end of day routine examination of the dogs. As more canine sports become popular, such as lure coursing, barn hunt, scent work, etc., awareness and client education of grass awns in our late summer months is critical in veterinary medicine.

Clinical Signs: Clinical signs can often be subtle or obvious, depending on the affected system, or secondary to other common rule-outs: ADR, pyrexia, licking or scratching affected area, seemingly crying out for no reason, decreased exercise intolerance, decreased eating, difficulty breathing, ataxia, interdigital swelling, excessive lacrimation, etc. The list is endless...

Treatment: Treatment varies depending on diagnosis, however, the most critical element is time. Untreated bacterial infections can be fatal. Complications can lead to pyothorax, a deep corneal ulcer, tympanic membrane perforation, abscesses, etc., depending on the case. Identification: In Oregon, foxtails and cheatgrass are big violators. Cheatgrass is in rural areas, everywhere in central and eastern Oregon, but you’ll commonly find foxtails in urban areas as well.

Education: Client education is a huge part of veterinary medicine and grass awn complications can be dramatically reduced with our help. Educate your clients about physically examining their dogs after a day in the field. A five minute palpation exam, brushing out a long coat, examining pinnas, nares and paws, can potentially save their dog’s health, as well as their pocketbooks.

Anesthesia

Capnography Cheat Sheet

Differentials for increasing and decreasing waveforms

What is a normal ETCO₂ value?

- Close to arterial carbon dioxide pressure
- ETCO₂ approximately = PaCO₂ = 35-45 mmHg ❓❓

In the perfect normal patient

- **However...in real patients** ❓❓

ETCO₂ is usually 6-10 mmHg lower than PaCO₂ ❓❓

And in severely compromised patients, ETCO₂ can be very low compared to PaCO₂

Differentials for decreasing waveform signal, or ETCO₂ values

Hyperventilation
Hypovolemia
Hypothermia
Vasoconstriction

Rapidly progressive drop

Thromboembolism
Cardiac arrest
Massive hemorrhage

Rapid non progressive drop

Airway obstruction
Disconnect in ET tube or circuit
Bronchospasm
Incorrect (esophageal) intubation

Differentials for increasing waveform, signal or ETCO₂ values

Hypoventilation
Hyperthermia, even malignant hyperthermia (very high values) *True malignant hyperthermia is a rare occurrence*
Catecholamine release (adrenal, thyroid, mast cell)
Reperfusion
Tiny patient in need of non rebreathing circuit

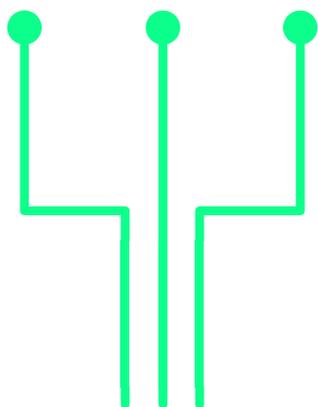
With baseline increase

Rebreathing due to faulty/stuck one way valves
Depleted carbon dioxide absorbent
Low sampling rate of capnography

HOW YOU FEEL
WHEN.....

YOU GET AN IV PUMP THAT
WON'T STOP BEEPING, BUT
YOU CAN'T FIGURE OUT WHY

Chantal Faraudo, CVT, CVPP
Certificate in Small Animal
Anesthesia for Preoperative
Analgesia and Critical Care



Case Study: What would you do?

ANSWERS ON PAGE 5

Signalment: 2 year old neutered male DSH "Sinbad" presents with stranguria, pollakiuria, painful when touched, vocalizing for 24 hours. Owner reports kitty hiding under bed, in and out of the litter box multiple times an hour and producing only drops of blood tinged urine.

Vital signs: Mentation: Dull, HR 120, Temperature: 96.6 degrees F rectally
Pulses: weak RR: 14 MM: pale pink CRT: 3 sec., BP 90 systolic Doppler
PE: Bladder large and firm, hematuria (drips only) on table, lung sounds normal, BCS 7/9, resented abdominal palpation, thoracic auscultation reveals bradycardia. No murmur ausculted. Penis is dark purple with crystallized plug present. EKG: Tented T waves, absent P waves, HR 110

Lab Results:

pH 7.0	Hct 48
PO2 38.8	Cl 116.2
Na 154	Glu 89
K 9.9	Lac 2.1
Ca 0.93	Creat 16.5
	Mg 0.61



What do you do first?

What do you think is happening?

What are your concerns?

What is your first priority?

Why is your kitty so bradycardic? Explain.

Why does your EKG show tented T waves, and no P's?

Consider doing this case with your staff at a Technician meeting or doing it with a colleague before you read the answer! And...Go!



Answer to case study

"Sinbad" has a urethral obstruction. Pain medication: pure mu opioid, place IV catheter. A minimum database (PCV/TS/GLU/AZO) should be obtained at catheter placement in addition to a venous blood gas to evaluate blood electrolytes, especially potassium, and acid/base status. It is not uncommon for sick blocked cats to have a severe metabolic acidosis, hyperkalemia, and low ionized calcium (likely secondary to hyperphosphatemia).

Priorities and Goals

Severity of signs can depend on the length of time the cat has been obstructed. They can present stable all the way to life threatening hyperkalemia (elevated Potassium levels) and azotemia.

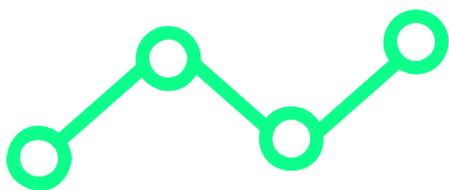
First priority is to stabilize the cardiovascular system. **DO NOT UNBLOCK UNTIL THIS IS DONE!**

2. Relieve the obstruction
3. Post-obstruction therapy

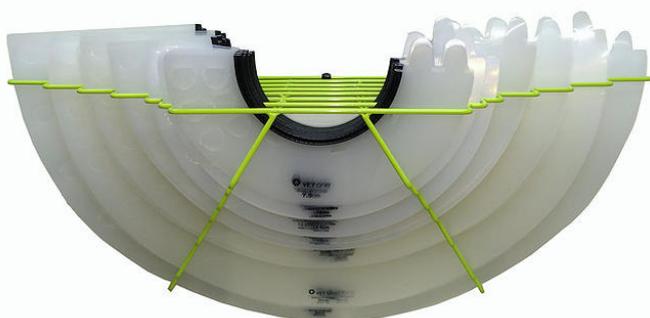
This kitty is severely hyperkalemic and needs the following in this order:

1. Intravenous fluid therapy with isotonic crystalloids: intravenous fluids will dilute the potassium lowering its serum concentration. Any isotonic crystalloid is appropriate (even crystalloids containing small amounts of potassium such as Normosol R or Plasmalyte). There is some evidence to suggest that resolution of a concurrent metabolic acidosis may occur faster in cats given Normosol-R or LRS compared with 0.9% Saline. In severely compromised patients, it may be necessary to administer a fraction of a shock bolus (10-20 ml/kg over 20-30 minutes).
2. IV 10% Calcium Gluconate: This medication works by increasing the cell's threshold membrane potential re-establishing the normal difference between resting membrane potential and threshold potential. This will allow the cardiac cells to depolarize. Dose: 3 ml/cat (1-1.5 ml/kg) given over about 3-5 minutes while evaluating the ECG. This dose may be repeated if needed. The effects of this drug are immediately apparent on the ECG and once the cardiac arrhythmias are resolved, no more drug should be given.
3. IV Dextrose OR IV Regular Insulin + Dextrose: When insulin binds to its receptors on cells this stimulates the $\text{Na}^{++}/\text{K}^{+}$ ATPase causing intracellular movement of potassium. Dextrose is given to prevent hypoglycemia. When dextrose is given alone, this will stimulate endogenous insulin production from the pancreatic beta cells. Dose: 1 unit of regular insulin/cat, 0.5 g/kg of 50% Dextrose diluted 1:3. It takes about 20-30 minutes to see an effect from this medication and the insulin will be active for approximately 1 hour. The cat should be supplemented with 2.5% dextrose in its intravenous fluid bag for several hours to prevent hypoglycemia
4. Intravenous Sodium Bicarbonate (**rarely given**): Administration of this medication will increase the pH in the extracellular space. This stimulates the exchange of intracellular H^{+} ions for extracellular potassium pushing potassium into the cell. Dose: 1 mEq/Kg once or 0.3(BWkg) – BE (usually 1/3 – 1/2 of this dose will be given). It will take about 20-30 minutes to see an effect.

Once the kitty is stable then proceed with unblocking. There are many protocols for sedation to general anesthesia for unblocking. The addition of a sacrococcygeal block will prevent urethral spasm, decrease medications and/or inhalants needed and make placing the catheter easier. The reduction of inhalants will reduce MAC and mitigate all of the side effects that come with it. To reduce the chances of urethral tearing you want the kitty very relaxed. Don't forget to collect a urine sample for urinalysis and culture. The kitty will need continued IV fluids post unblocking, close monitoring of urine output (ml/kg/hr) every 4 hours. Urine output at minimum should be 1-2 ml/kg/hr. Close monitoring for signs of fluid overload: murmur, fluids dripping from the nose, crackles in lungs. Pain medication on a regular schedule with pain score assessed every 4 hours. Prazosin, to help prevent urethral spasms. Nutritious, yummy can food to help increase water intake and treat crystals if present. Owner education about home care, medications, diet, warning signs of recurrence are critical.



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