Water, Water, Water
Diabetes Insipidus and Cushings Syndrome in the Dog and Cat

Ann Della Maggiore, DVM, DACVIM

Sunday, November 8, 2020

CVMA ACMV CHAPTER SBCV
CVMA-SBCV Chapter

VET STRATEGY

This session is generously sponsored by VetStrategy

VET STRATEGY

Goals for this Presentation

- Polyuria and polydipsia
- Diabetes insipidus
  - Central
  - Nephrogenic
- Hyperadrenocorticism
  - Diagnosis
  - Treatment options
  - Monitoring
- Species specific differences

Polyuria and Polydipsia

Polyuria: Increased urine production, large volumes of dilute urine

Polydipsia: Voluntary increase in water intake

Normal water intake: 50-80 ml/kg/day

Grey zone: 80-100 ml/kg/day

Polydipsia: >100 ml/kg/day

Normal urine production: 1-2 ml/kg/hour

~25-50ml/kg/day

History- Confirming PU/PD

Primary rule out would be LUT disease

- **Pollakiuria must be ruled out**
- **Other LUT signs: hematuria, dysuria, stranguria**
- **Urinary incontinence**

Urinary incontinence

- **PU/PD can lead to or worsen urinary incontinence**
- **Treatment of underlying PU/PD can lead to resolution of incontinence**

Additional clinical signs dependent on etiology of PU/PD

Lucy 4 yo FS St. Poodle

- Presented for urinary incontinence
- Unremarkable physical exam except for slight recessed vulva
- Unremarkable CBC, chem, UA: USG: 1.006
- Urine culture negative
- Anesthesia, urethral pressure profile, cystoscopy, trial of drugs for urinary incontinence
- No improvement…. What did I miss?

Documenting Polyuria

Polyuria can be more challenging than polydipsia to quantify and document

- **Rely on history:**
  - **Cat →Large clumps in litter box**
  - **Dog → Urinate for a long period of time/large volume**

Serial urine specific gravities:

- **First morning sample:**
  - **More concentrated**
  - **Represent a longer period of time**

- **A PU/PD patient should not concentrate urine**
  - **Usually USG <1.020**

Documenting Polydipsia

Measure 24 hour water consumption

Variables that alter water intake:

- **Ambient temperature**
- **Respiratory evaporation**
- **Fecal water content**
- **Physiologic state (pregnancy or lactation)**

Lucy… What did I miss?

Owners noted that she always drank a large amount of water

Drinking about 180 ml/kg/day….

Diagnosed with CDI and with treatment of her PU/PD and the urinary incontinence resolved!

Polyuria and Polydipsia
Differential Diagnoses

Common Causes

- Renal failure
  - Acute or Chronic
- Diabetes mellitus
- Hyperadrenocorticism
- Hyperthyroidism
- Hypercalcemia
- Pyelonephritis/pyometra
  - E. coli
- Iatrogenic/medications

Uncommon Causes

- Diabetes insipidus
  - Central vs Nephrogenic
- Psychogenic polydipsia
- Hepatic insufficiency
- Post-obstructive diuresis
- Hypoadrenocorticism
- Renal glycosuria
- Hypokalemia
- Polycythemia
- High salt diet

Primary Polyuria or Primary Polydipsia

- **PU/PD are often reported simultaneously**
- **Most conditions cause primary polyuria, and polydipsia is in response to polyuria**
- **Exception to this is psychogenic polydipsia**

Diagnostic Approach

- Pertinent History
- Physical examination
- CBC, serum biochemistry panel, urinalysis
- ± Urine Culture
- ± Diagnostic Imaging
  - Radiograph
  - Abdominal ultrasound
- Additional testing: Variable based on DDx
  - Examples:
    - If hyperadrenocorticism is a DDx consider: UCCR, LDDS
    - If hyperthyroidism is a DDx consider total T4

Diabetes Insipidus

No breed, age, gender predispositions

Primary clinical signs are PU/PD

Clinical signs and exam are dependent on etiology

Clinical Pathology Abnormalities:

- **CBC: Normal**
- **Chemistry: Normal, possibly low BUN**
- **Urinalysis: USG typically less than 1.006**

Severe dehydration may cause azotemia, hypernatremia, and hyposthenuria, which can occur with inappropriate water restriction

Diabetes Insipidus Diagnosis

Plasma osmolality: normal is 280-300 pOsm

- **If osmolality <280 pOsm suggest psychogenic polydipsia**

DDAVP (Desmopressin) trial:

- **Response consistent with CDI**
- **Some animals with hyperadrenocorticism may also respond**

Modified water deprivation test

- **Labor intensive, expensive, time-consuming**
- **Results can be confusing- partial DI**
- **Can be dangerous if not performed correctly**

Central Diabetes Insipidus

Partial to complete AVP deficiency

Congenital:

- **Developmental defects**

Acquired:

- **Neoplasia → 1^0 or met**
- **Inflammation**
- **Head trauma**
- **Idiopathic**
- **Surgery→ hypophysectomy**
- **Infection**
- **Cysts**
- **Hyperadrenocorticism**
- **Ischemic brain injury**

Central Diabetes Insipidus

- DDAVP (Desmopressin) trial
  - Tablets: 0.05-0.2mg/dog PO BID-TID
    - Sm dog 0.05 mg/dog
    - Med 0.1 mg/dog
    - Large 0.2 mg/dog
  - Drops: 1 drop in conj. of eye q 8-12 hours
- Expensive
- Life long therapy
- Once document response (serial USG's), taper down to lowest dose to maintain clinical effect
- Treatment is for owners!

Nephrogenic Diabetes Insipidus

Partial of complete inability of the renal tubule to respond to AVP

Secondary (acquired)

- **Pyometra/pyelonephritis**
  - **E. Coli endotoxins**
- **Hypercalcemia**
- **Hyperadrenocorticism**
- **Hyperthyroidism**
- **Polycythemia**
- **Hypokalemia**

Primary

- **Congenital- very rare**

Sadie 12 yo FS GSD

- Diagnosed with pituitary dependent hyperadrenocorticism over 2 years ago
- Medications: Trilostane 60 mg PO BID with food (2 mg/kg q 12hr)
- Determined to have good control of her HAC based on overall clinical appearance, ACTH stimulation test, UCCR.
- Owner noted profound PU/PD over the last 2 months. Increase urinary accidents in the house and urinary incontinence
- Serial USG: 1.004, 1.006, 1.006, 1.008 (first thing in the morning)

Sadie… continued

- Suspect CDI due to PDHAC
- Elected a trial of desmopressin 1 tablet (0.2 mg) PO BID
- Serial USG 1 week later:
  - USG: 1.035, 1.044, 1.045
- Owner noted complete resolution of incontinence and inappropriate urination
- Consistent with diagnosis of CDI

Polyuria and Polydipsia
Differential Diagnoses

Common Causes

- Renal failure
  - Acute or Chronic
- Diabetes mellitus
- Hyperadrenocorticism
- Hyperthyroidism
- Hypercalcemia
- Pyelonephritis/pyometra
  - E. coli
- Iatrogenic/medications

Uncommon Causes

- Diabetes insipidus
  - Central vs Nephrogenic
- Psychogenic polydipsia
- Hepatic insufficiency
- Post-obstructive diuresis
- Hypoadrenocorticism
- Renal glycosuria
- Hypokalemia
- Polycythemia
- High salt diet

Why Does Polyuria Occur?

Varies with etiology:

- **Diabetes Mellitus** → Glucose in urine causes osmotic diuresis
- **Renal disease** → Damage to kidneys prevents normal urine concentrating ability
- **Head trauma**→ Inability to produce AVP due to damage to the hypothalamic- pituitary tract → **Acquire Central DI**
- **Hyperadrenocorticism-** Inhibit AVP release by a direct effect within hypothalamus and/or pituitary and alters effect of AVP at the kidney

  → **Central and/or nephrogenic DI**

Secondary or Acquired Nephrogenic DI

- **Hypercalcemia→** High calcium prevents the kidney tubules from responding to AVP to concentrate urine
- **Pyometra/Pyelonephritis→** Bacterial endotoxins (E. coli) compete for AVP binding sites creates a insensitivity to AVP
- **Hypoadrenocorticism→** Impaired coupling of the AVP receptor to AC
- **Hypokalemia→** Down regulates AQP2

Diabetes Insipidus Variations in Dogs vs Cat

Minimal to no variation between species

- **Pathology**
- **Diagnostic evaluation**
- **Treatment**

Challenges to diagnostic evaluation in the cat, water deprivation test and serial urine specific gravity

Psychogenic Polydipsia

- Compulsive water drinking leading to compensatory polyuria
- Hyperactive dogs in exercise restricted environments
- Animal can concentrate urine with careful water deprivation
- Intact hypothalamic pituitary renal tubular axis but may have renal medullary solute washout from chronic diuresis which can inhibit normal concentrating ability

Psychogenic Polydipsia

Diagnosis

- Diagnosis of exclusion
- Serial USG's (ability to concentrate often up to 1.030)
- Evidence of behavioral abnormalities
- Modified water deprivation test (rarely performed)

Treatment:

- Behavior modification
- Careful water restriction
- Change in environment

Rye 9 mo FI DSH

Case report just accepted for publication!

Presented obtunded and non-responsive

- **Na>190, BUN 55, Cr: 3.4**

Owners had never seen her drink

Urinating large amounts compared to littermates

Rye 9mo FI DSH

Hospitalized for a week to slowly reduce sodium

Started on DDAVP and feeding tube placed to allow administration of water

Hyponatremia Hypodipsia

- **Rare**
- **Congenital**

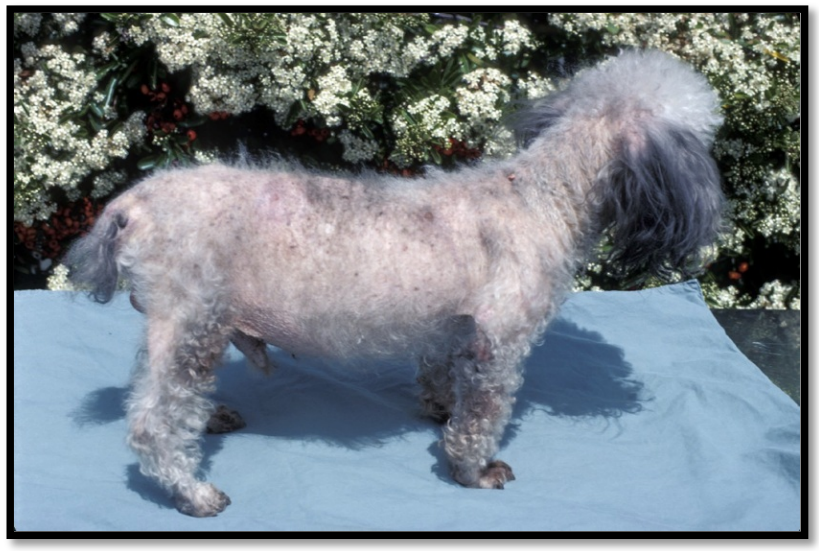
Questions?

Canine Hyperadrenocorticism

Middle age to geriatric patient

Breed predilection:

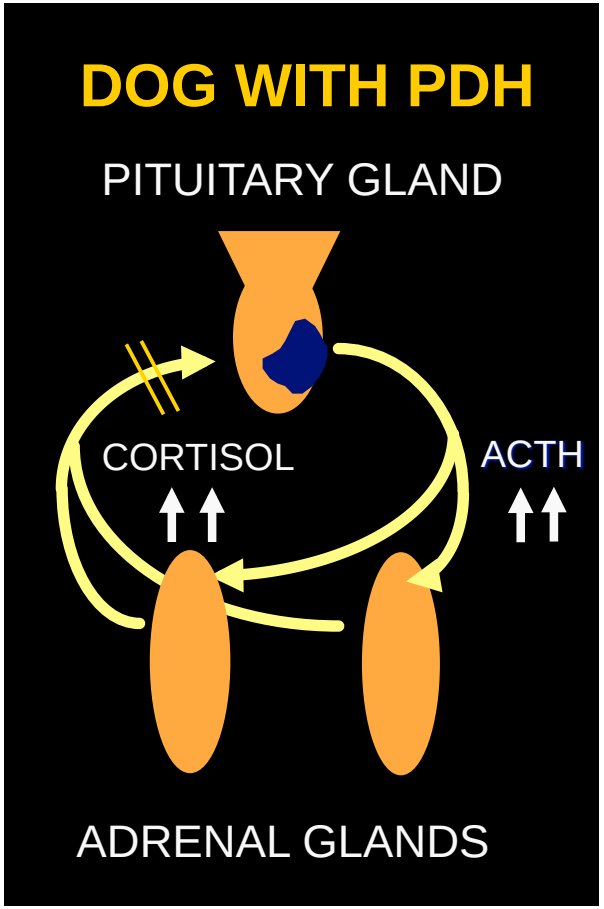
- **Poodles**
- **Dachshund**
- **Beagles**
- **Terrier Breeds**
- **Miniature Schnauzer**
- **Labrador retrievers**
- **Boxers**
- **Boston terriers**
- **Irish Setter**
- **Basset hound**



Hoffman et al. JSAP 2018

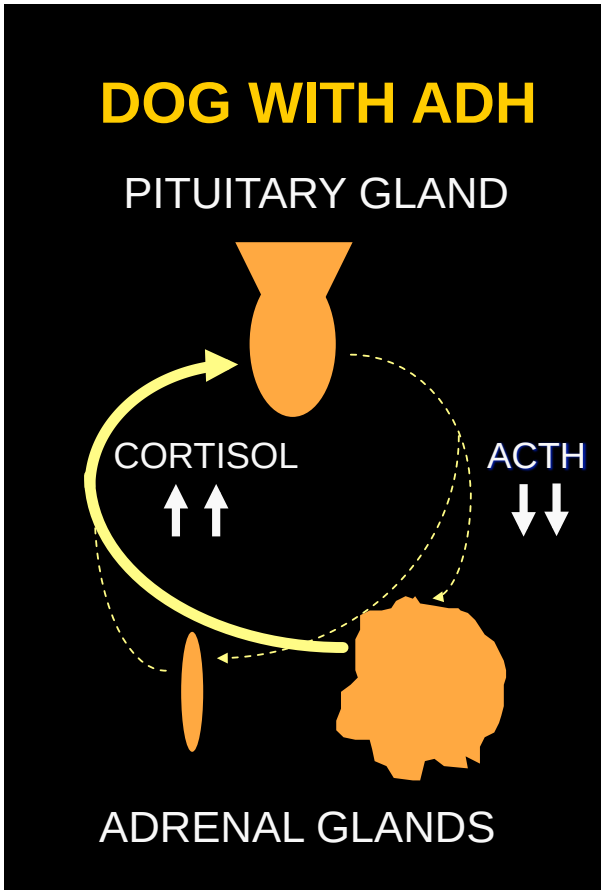
Hyperadrenocorticism

Etiology : Pituitary-dependent HAC (PDH)



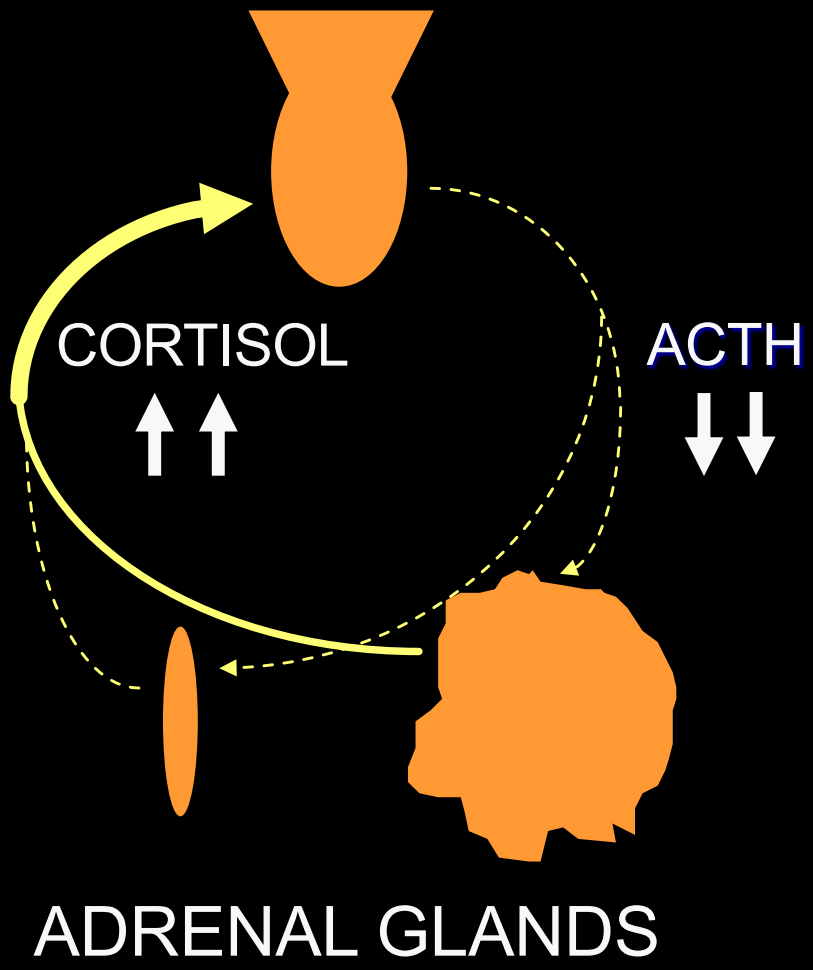
- 80-85% of cases
- Pituitary tumor (benign) secreting excess ACTH
- Macroscopic or microscopic
- Leads to bilateral adrenal hyperplasia
- Adrenal hyperplasia and continued ACTH stimulation leads to chronic excess of circulating cortisol

Hyperadrenocorticism
Etiology: Adrenal-dependent HAC (ADH)

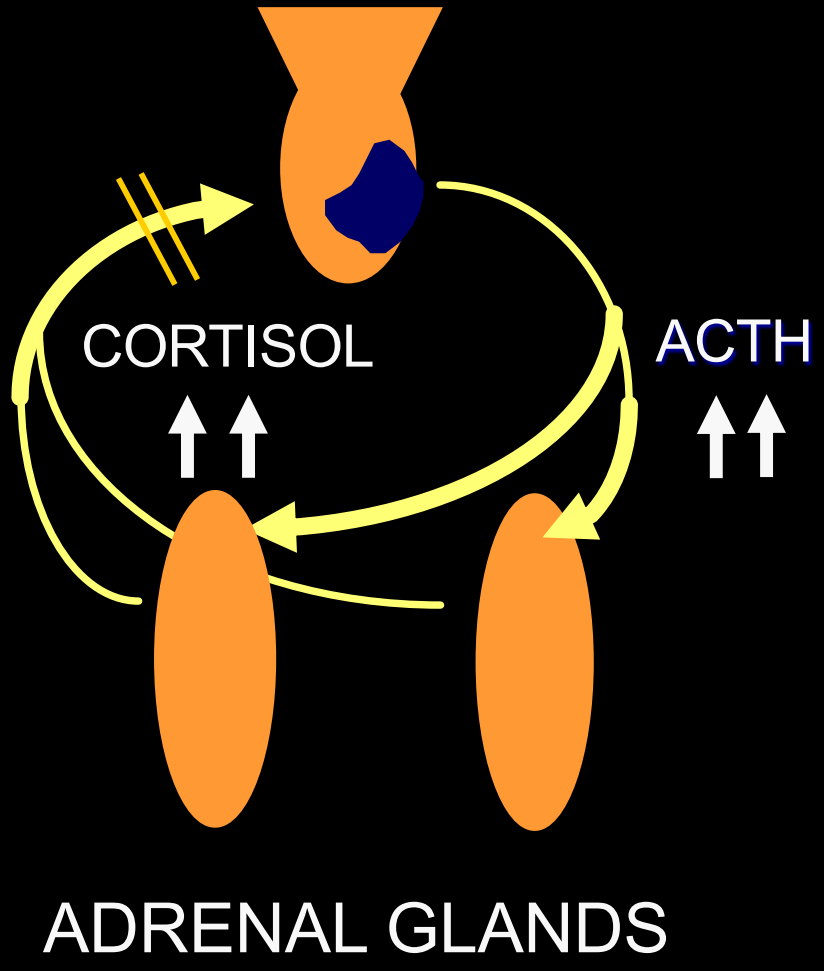


- 15-20% of cases
- Adenoma (50%) or carcinoma (50%)
- Tumor synthesizes and secreted excess cortisol chronically
- Non-tumor adrenocortical cells atrophy due to ACTH suppression

DOG WITH ADH



DOG WITH PDH



Clinical Presentation

Slow progression, mistaken for aging

- Occasionally rapid onset

Clinical signs:

- **PU/PD/PD**
- **Abdominal distension**
- **Dermatologic changes**
  - **Thin skin, alopecia, pyoderma, calcinosis cutis**

Improved awareness of HAC means the diagnosis is made earlier in course of disease. Clinical signs are less evident!

Basic Laboratory Findings

CBC:

- **Normal HCT or mild erythrocytosis**
- **Stress leukogram**
- **Thrombocytosis**

Chemistry:

- **Increased ALP →85% of cases**
- **Increased ALT → mild**
- **Hypercholesterolemia**
- **Hypertriglyceridemia**
- **Hyperglycemia**

Basic Laboratory Findings

Urinalysis

- **USG usually < 1.015**
- **Proteinuria- ~45% UPC>1.0 in the absence of UTI**
- **Glycosuria (+/-)**

Urine Culture

- **UTI difficult to identify, due to dilute urine and lack of inflammation**

Hyperadrenocorticism
Effects on Other Hormones

- Serum Total T4 can be decreased in ~70% of dogs
- Free T4 decreased in 60% of cases
- TSH usually decreased
- Serum PTH hormone can be increased
- Insulin concentration increased and exaggerated response to glucose

Indications for Additional Testing

HISTORY, CLINICAL SIGNS, PHYSICAL EXAM

Exam room diagnosis

Clinicopathologic alterations

Additional indications to test:

- **Incidental adrenal mass**
- **Hypertension**
- **Insulin resistance diabetes mellitus**

Differential Diagnosis

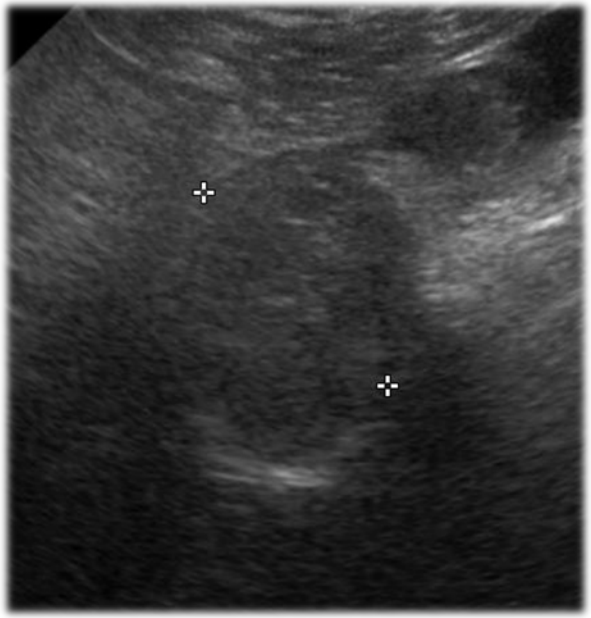
Depends on presentation, lots of overlap between conditions

- **Diabetes mellitus**
- **Diabetes insipidus**
- **Chronic kidney disease**
- **Hepatic disease**
- **Hypothyroidism**
- **Hyperthyroidism**
- **Ascites**
- **Iatrogenic- anticonvulsant therapy**

Imaging

Abdominal Radiographs

- Calcinosis cutis/soft tissue mineralization
- Cystic calculi
- Hepatomegaly



Abdominal Ultrasound

- Adrenal size
  - Normal = 0.4 to 0.75 cm
- Metastatic disease
- Other concurrent disease

Feline Hyperadrenocorticism

Rare; 80% PDH, 20% ADH

Presenting complaint

- **Resistant diabetes mellitus (>50%)**
- **PU/PD**
- **Fragile skin**
- **Weight loss, lethargy**

Biochemistry results

- **Hyperglycemia**
- **Stress leukogram**
- **ALP and ALT elevation in about 10%**
- **Cholesterol elevation in ~25%**

Testing for Hyperadrenocorticism

Screening Tests

→ **Does the dog have HAC?**

Differentiating Tests

→ **Does the dog have PDH or AT?**

Testing for Hyperadrenocorticism

Screening Tests->

Does the dog have HAC?

- **Urine Cortisol Creatinine Ratio**
- **ACTH stimulation test**
- **Low Dose Dexamethasone Suppression (LDDS) Test**

Diagnostic Evaluation
Endocrine Testing- Screening

Resting serum cortisol → Useless!

UCCR

- **Sensitive (95-99%); NOT Specific (<20%)**
- **Normal values suggests disease other than HAC**
- **Does not discriminate ADH and PDH**
- **Best run on a sample collected at home first thing in the morning to minimize stress artifact**

Diagnostic Evaluation
Endocrine Testing- Screening

ACTH stimulation test

- **Distinguishes natural occurring from iatrogenic**
- **Easy, quick, but expensive**
- **Does not differentiate**
- **Sensitivity (57-63% with ADH, PDH 83%)**
- **Specificity (85-93%)**
- **Inconclusive results common**
- **Baseline prior to starting medical management**

ACTH Stimulation Test Protocol

- **Collect a blood sample for a basal cortisol**
- **Inject 250µg of synthetic ACTH (Cortrosyn) IM**

  Or
- **Inject 5µg/kg of synthetic ACTH (Cortrosyn) IV**
- **Collect a second serum sample 60 minutes later for cortisol concentration**

What if it is given perivascular??

→ No problem… does not alter test results

Johnson CM JVIM 2017

What About The Expense?

Recent study looking at 2 different groups:

- **Newly diagnosed**
- **Monitoring during treatment**

Determined that in dogs suspected of having HAC best to use 5µg/kg of synthetic ACTH.

In dogs undergoing treatment 1µg/kg of synthetic ACTH can be used for monitoring.

Comparison of 2 Doses for ACTH Stimulation Testing in Dogs Suspected of or Treated for Hyperadrenocorticism

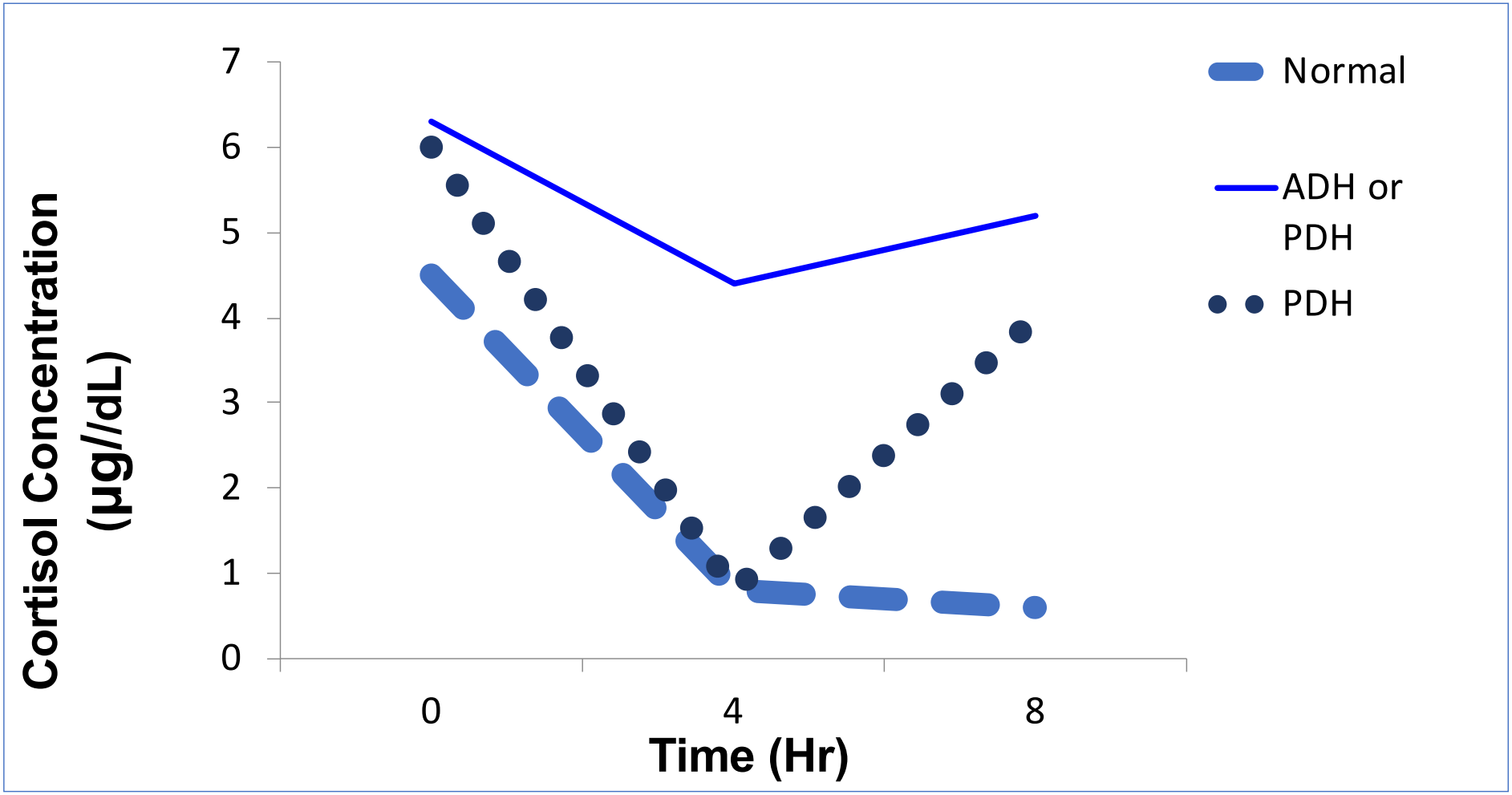
C. Aldridge, E.N. Behrend, R.J. Kemppainen, T.M. Lee-Fowler, L.G. Martin, C.R. Ward, D. Bruyette, J. Pannu, P. Gaillard, and H.P. Lee

Low Dose Dex Suppression Test

- Easy, inexpensive, 8 hr test
- Sensitivity 90%
- Specificity 80-90%

Protocol:

- Administer 0.01 mg of dexamethasone/kg IV and obtain blood at 0, 4, and 8 hours for cortisol conc.
- If 8-hour supports diagnosis→ 4-hour is used to differentiate between PDH and ADH



- **Dogs with PDH 66% suppress transiently**
- **33% do not suppress→ ADH or PDH**

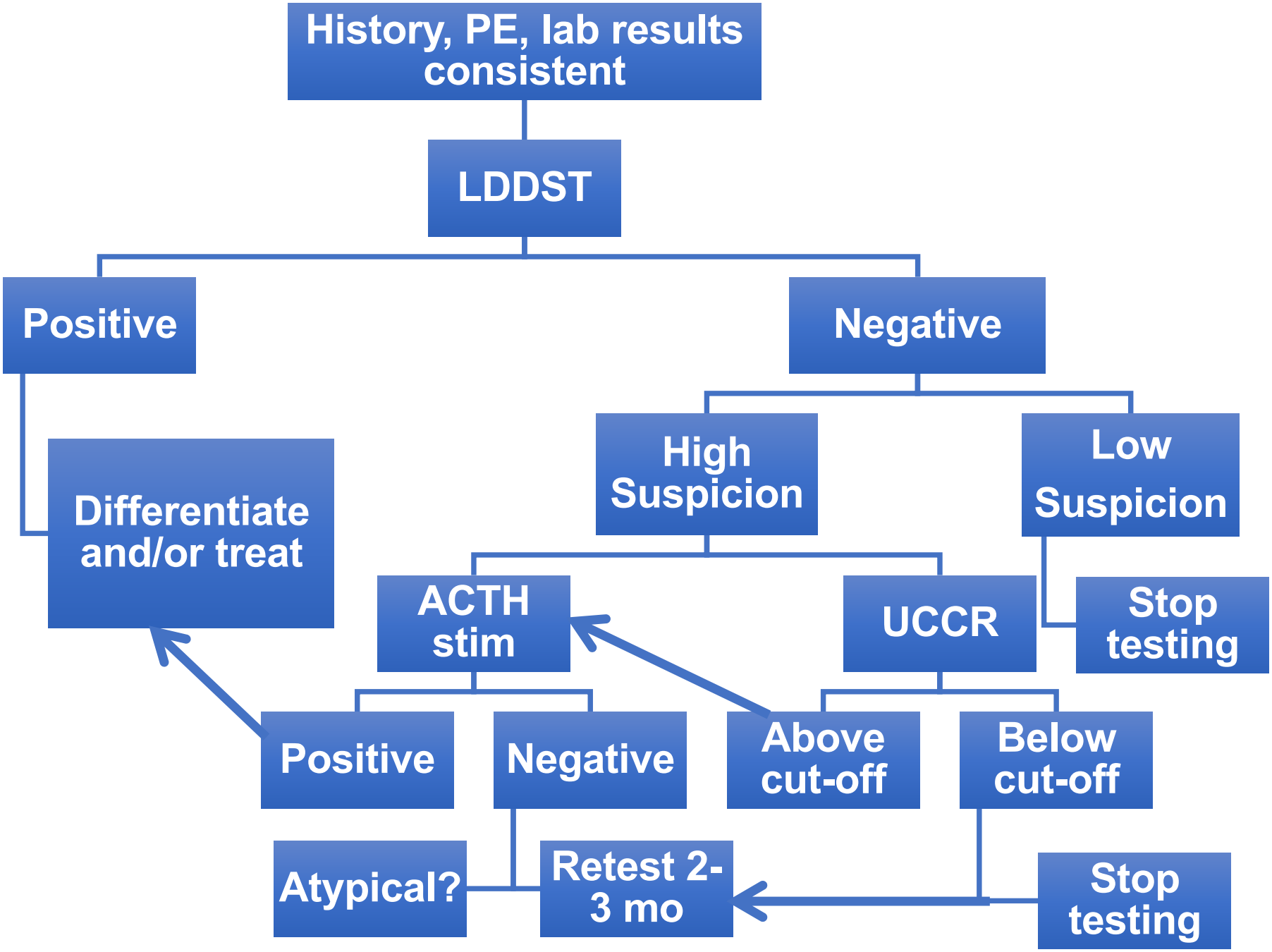
What defines suppression: <50% of baseline or <1.4 µg/dl

Should I use ACTH stim or LDDST for diagnosis?

- Its not just about sensitivity and specificity…
- PPV vs NPV

	ACTH Stim	LDDST
PPV	96%	94%
NPV	46%	71%

- What does this mean?


History, PE, lab results consistent
LDDST
Positive
Negative
Differentiate and/or treat
High Suspicion
Low Suspicion
ACTH stim
UCCR
Stop testing
Positive
Negative
Above cut-off
Below cut-off
Atypical?
Retest 2-3 mo
Stop testing

Testing for Hyperadrenocorticism (HAC)

Differentiating Tests→ Does the dog have PDH or AT?

- **Imaging- Abd. ultrasound, CT, MRI**
- **Low Dose Dexamethasone Suppression (LDDS) Test**
- **High Dose Dexamethasone Suppression (HDDS) Test**
- **Endogenous ACTH concentration**

Diagnostic Evaluation

Imaging

PDH- bilaterally symmetric normal-size or large adrenals (a maximum width greater than 0.75 cm)

ADH- anywhere from 1-8+ cm, ideally contralateral small or undetectable; normal does not rule out

- **>3cm concerning for carcinoma**

What about Bilateral Adrenal Masses?

Multiple nodules of varying size is suggestive of macronodular hyperplasia

Alternatively consider…

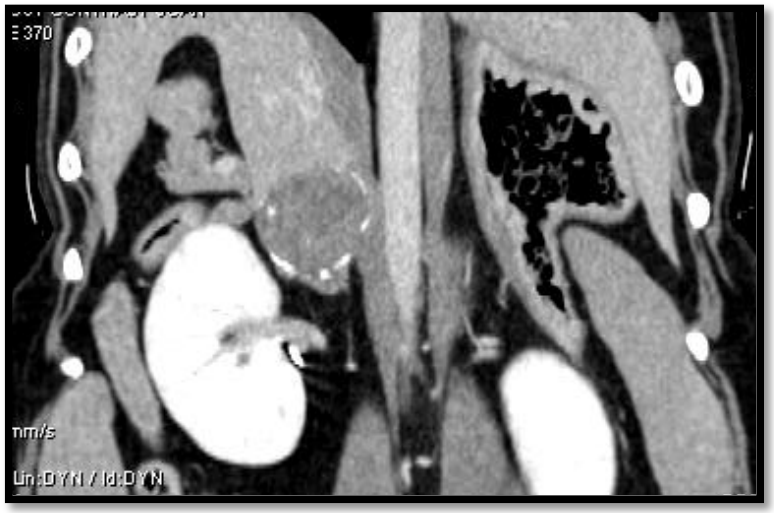
Bilateral adrenal masses/tumors

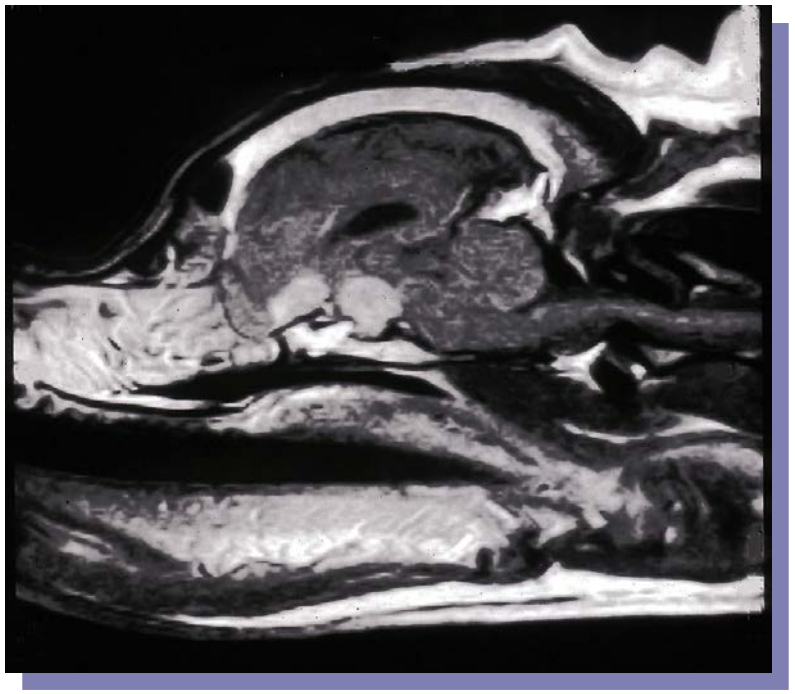
Diagnostic Evaluation

Imaging- Differentiating

CT/MRI

- Evaluate the pituitary gland for a macroadenoma
- Evaluate size, symmetry and vascular invasion of adrenal tumors





Diagnostic Evaluation
Endocrine Testing- Differentiating

LDDS Test

- **4 hr <50% of baseline or <1.4 µg/dl → PDH**

Endogenous ACTH level

- **Use results conflicting PDH vs. ADH**
- **ADH should result in ACTH below RR or undetectable. PDH should be in upper half of RR. Low normal RR non-diagnostic**
- **Labile test so sample handling critical!**

HDDS Test— Rarely used

Feline Hyperadrenocorticism Endocrine Testing

Rare condition, limited data

- **UCCR- higher reference range than dogs, sensitive but not specific**
- **LDDST- start at higher dose, 0.1mg/kg IV of dexamethasone**
- **ACTH stimulation Test- 125mg/cat IM, sample at 30minutes and 60 minutes.**
  - **Lacks sensitivity (~33%) and specificity**

CLINICAL SIGNS

Diagnostic Evaluation
Endocrine Testing

- **No test is perfect**
- **Any test can give false positive and false negative**

Diagnosis should never be made on the basis of endocrine tests alone!

Treatment of HAC

Adrenal Dependent:

- Ideally surgical removal
  - CT scan performed prior to surgery
  - Laparoscopic or ventral midline laparotomy
  - Medically managed for a minimum of 2-4 weeks prior to surgery
- Medical Management

Treatment of HAC

Pituitary Dependent:

- Medical management
- If a macroadenoma is present:
  - Radiation therapy
  - Hypophysectomy
- Bilateral adrenalectomy (less ideal)
- Surgery and radiation have the potential to cure

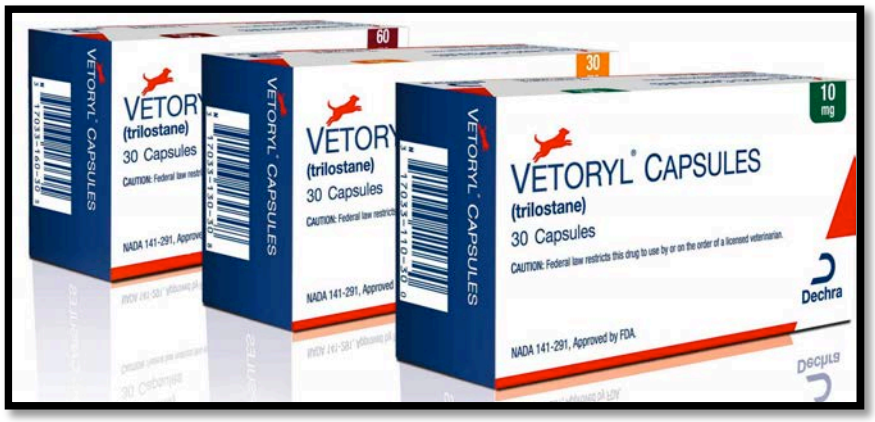
Treatment of HAC
Medical Management- Trilostane

- FDA approved
- Synthetic steroid, no inherent hormonal activity
- Blocks 3β-HSD; may also have activity against 11β-HSD and 11β-Hydroxylase
- Reversible (in theory…)
- Effective in both PDH and ADH
- 67-90% effective

Treatment of HAC
Trilostane

Requires monitoring to determine appropriate dosing avoid adverse effects

Side effects:



- **V/D**
- **Decreased appetite**
- **Hyperkalemia**
- **Signs of hypocortisolism, iatrogenic hypoadrenocorticism**
- **Failure to respond**

Adrenal cortical necrosis, adrenal enlargement

Reusch et al. 2007

Treatment of HAC
Trilostane

Starting dose 0.5-1mg/kg PO q 12 hrs

****Give with food to improve absorption through GI tract**

Additional points:

- **Larger dogs need lower dose/kg**
- **Can administer once daily or q 8hrs**
- **15% of dogs will develop transient hypoadrenocorticism in the first 2 years**

King and Morton VetJ 2017

Treatment of HAC
Trilostane- Monitoring

- **Consensus on ideal monitoring is limited**
- **ACTH stimulation test at 10-14 days or sooner if adverse effects noted performed 2-4 hours post pill, important that timing of test consistent in each patient.**

  Bonadio et al. JVIM 2014
- **Goals of therapy:**
  - **CONTROL CLINICAL SIGNS**
  - **Aim to get post-ACTH stim cortisol between 1-5µg/dL**
  - **Full effect of the drug may take 30 day**

Treatment of HAC
Trilostane- Monitoring

Once adequate dose achieved ACTH stimulation test performed q 3-4 months, serum biochemistry q 6-12 months.

Can see electrolyte changes and hepatotoxicity on high doses

Compounded drug:

- **Strongly advise against**
- **If compounded, have compounded from Vetoryl capsules if possible**

Changes in Monitoring Dogs on Trilostane

Three cortisol concentrations compared:

- **pre-trilostane**
- **3-hour post trilostane**
- **1-hour post-ACTH stimulation**

- **3hr post trilostane measurements better reflected clinical control compared to 1 hr post-ACTH stim.**
- **Do not assess adrenal reserve function.**
- **Alternatives for ACTH stim and monitoring being evaluated**

Macfarlane et al., 2016

Cushings Questionaire

Multiple scoring tools/calculators described to establish a clinical score

BOX 1 Questionnaire: Owner Perception of the Effectiveness of Trilostane Therapy[a,b]

Please rate your dog's behavior/appearance for the past 4 weeks in the following categories.

QUESTION	SCORE
1. Drinking. Do you think your dog has drunk:	
Less than normal	PI
Normal	1
More than is normal (e.g., 1 or 2 times normal)	3
Very much more than is normal	4
2. Urinating. Do you think that the volume or frequency of urination is:	
Less than normal	PI
Normal	1
More than is normal (e.g., 1 or 2 times normal)	3
Very much more than is normal	4
3. Appetite. Would you describe your dog's appetite as:	
Very poor (not eating at all or hardly eating)	PI
Poor (does eat some food but requires encouragement)	PI
Normal	1
Increased (eats all food quickly and will look for more)	3
Greatly increased (seems ravenously hungry all the time)	4
4. Vomiting and diarrhea. How often has your dog had sickness and diarrhea?	
Never had sickness or diarrhea	0
Has had sickness or diarrhea once	0
Has had sickness or diarrhea more than once but not every day	PI
Has had sickness or diarrhea every day	PI

Cushings Questionaire

5. Exercise. How active is your dog?	
Lies in one place nearly all of the time	4/PI
Goes for walks and will also play on occasions	3
Very active, happy to run off-leash but does get tired	2
I cannot tire my dog out!	1
6. Skin and coat. How would you describe your dog's coat and skin condition?	
Very poor (e.g., thin coat, bald patches, very dull)	4
Poor (e.g., slightly thin coat, hairs dull)	3
Reasonable (e.g., no bald patches, slightly dull)	2
Very good (e.g., thick coat, shiny, no dandruff)	1
7. Other problems. Does your dog have any of the following?	
Trembling/shaking/muscle twitches more than once a week	PI
Persistent panting even at rest	3
Pain (anywhere)	PI
Difficulty moving	PI
Mental depression	PI
8. General. How do you feel your pet enjoys life?	
Miserable most of the time	PI
Has more bad days than good days	0
Happy most of the time; occasional bad days	0
Happy all of the time	0
9. Overall. How good do you feel your dog's current treatment for Cushing's is?	
My dog has more clinical signs than before treatment	5/PI
There is no difference now than before treatment	4
There is some improvement since starting treatment	3
My dog is nearly back to his/her normal self	2
If I did not know, I would think there was nothing wrong with my dog now	1

PI=possible illness.

[a] *Dog is classified as unwell and is NOT scored if PI is selected 3 or more times.*

[b] *Adapted from: Macfarlane L, Parkin T, Ramsey I. Pre-trilostane and 3-hour post-trilostane cortisol to monitor trilostane therapy in dogs.* Vet Rec *2016;179(23):597.*

Treatment of HAC
Lysodren

- Drug of choice prior to trilostane
- Selective destruction of zona fasiculata and reticularis while preserving zona glomerulosa
- Multiple protocols available specific to PDH, ADH, medical adrenalectomy
- Primary consideration would be for ADH with a protocol aimed at adrenocortical destruction

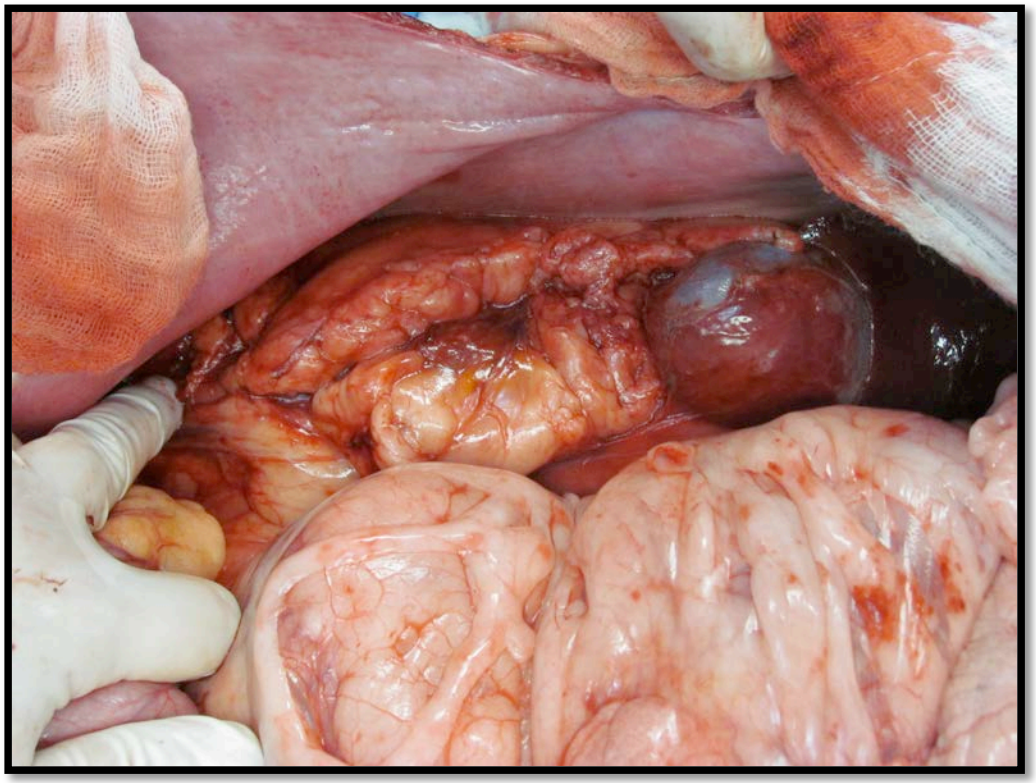
Treatment of HAC
Adrenalectomy

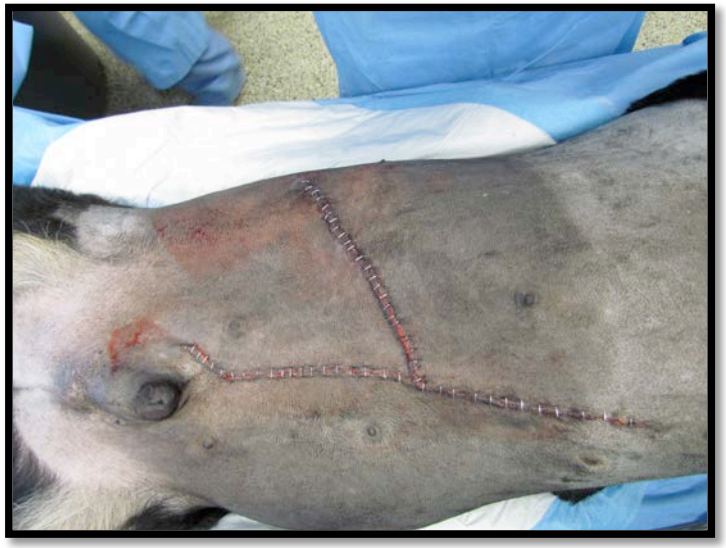
Pretreatment with Trilostane for 3-4 weeks

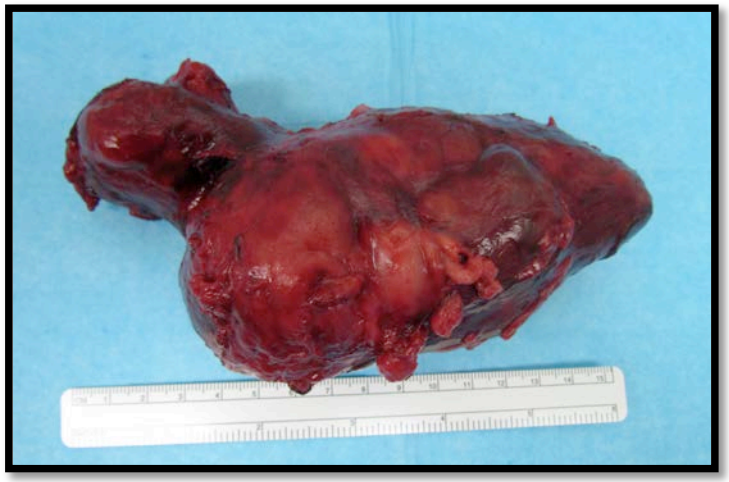
Complications:

- **Hypercoagulability- thromboembolism**
- **Poor wound healing**
- **Decreased respiratory function**
- **Pancreatitis**
- **Post-operative hypoadrenocorticism**

Treatment of HAC
Surgical Approach- Adrenalectomy







How does Caval Involvement Change Prognosis?

- **75% of cases with caval involvement survived to discharge**
- **Median survival of that population was 547 days.**
- **Bodyweight, tumor type, and size and extent of caval thrombus did not affect survival to discharge**
- **Post-diaphragmatic thrombus termination was associated with a greater risk of death.**

Mayhew et al. VetSurg 2019

Treatment of HAC
Laparoscopic Adrenalectomy

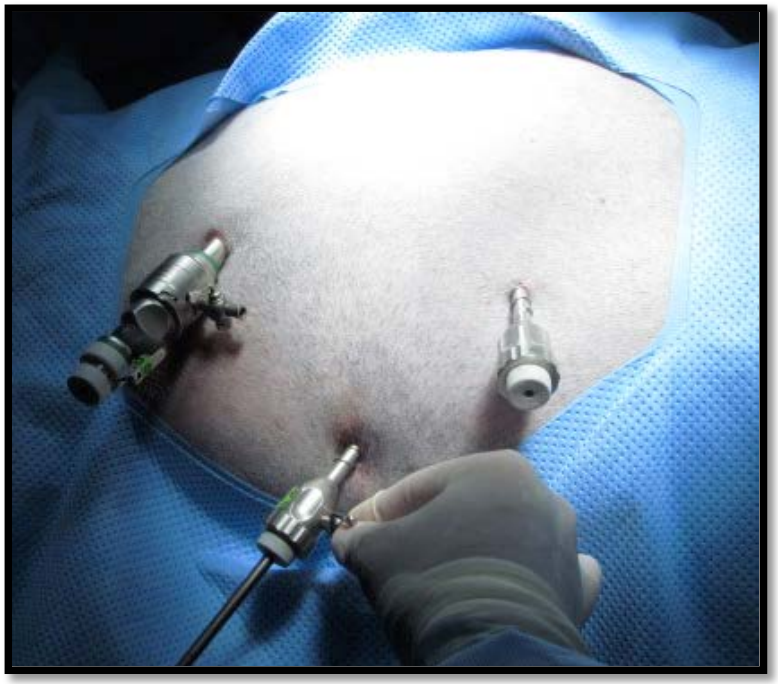
Preferred if possible, must be non-invasive adrenal mass

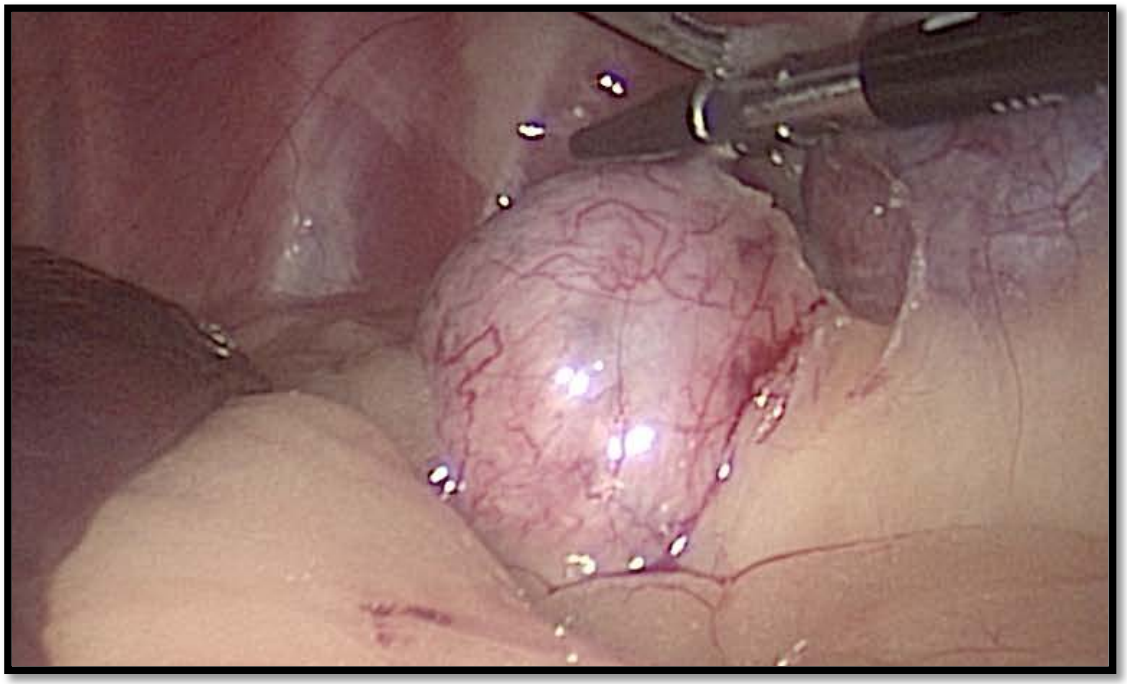
Benefits:

- **Limited manipulation of other abdominal organs**
- **Rapid surgical recovery, shorter hospitalization**
- **Less postoperative pain**
- **Decreased wound complications**
- **Reduced morbidity and mortality**

Mayhew et al. JAVMA 2014

Treatment of HAC
Laparoscopic Adrenalectomy





Treatment of HAC

Post-operative Management

- Monitor electrolytes
- Check ACTH stim 6-12 hrs post op
- Results should be <2 µg/dL post-ACTH immediately post operatively
- Dexamethasone SC or IV post operatively, and prednisone on tapering dose for 4-8 weeks post operatively.

Treatment of PDHAC
Hypophysectomy

Pituitary microsurgery using transphenoid approach

Offered very few places.

Post operative complications:

- **Hypernatremia**
- **Secondary hypothyroidism**
- **Diabetes insipidus**
- **KCS**

Treatment of HAC
Pituitary Radiation

Stereotactic radiosurgery

Offered more places throughout the US

Performed on patients with known macrotumor (>10mm) and neurologic signs

Goals:

- **Reduce tumor size**
- **Reduction of neurologic signs**
- **Reduction of clinical signs of HAC**
- **Prolonged survival with good quality of life**

Prognosis

Commonly diagnosed in older patients with concurrent disease

ADH Median survival time:

- **Medical management 353-427 days**
- **Surgery can be curative**

PDH Median Survival times:

- **Trilostane 662 to 852 days**
- **Mitotane 708 to 720 days**

Survival without treatment→ 359-506 days

Helm et al., 2011; Arenas et al., 2014

Feline Cushings Syndrome Treatment

Low numbers, minimal studies

Trilostane

- **High dose - 20-30mg/cat PO q 24hr or divided q12**
- **Minimally responsive**

Surgery

- **Laparotomy or laparoscopic adrenalectomy**
- **May secrete multiple hormones**
- **Hypophysectomy**

Radiation

Prognosis- guarded to poor

Key Points:

- DI is overall rare, important to rule out more common causes of PU/PD
- Hyperadrenocorticism is diagnosed on clinical signs and confirmed with endocrine testing
- Feline HAC is more challenging to diagnose and manage than canine HAC
- Recommendations for diagnostic testing and testing associated with monitoring is changing currently
- Surgical options are becoming safer and more readily available

CVMA ACMV CHAPTER SBCV

CVMA-SBCV Chapter

MarQueen

PET EMERGENCY & SPECIALTY

Thank You

VET STRATEGY