

Alabama State Board of Veterinary Medical Examiners
P. O. Box 1968
Decatur AL 35602

Gentlemen:

Ref: Your ASBVME Letter, 5 Jun 2008, Complaint Response (Case # ?)

For a week after Asproolee's death NO ATTENDING VETERINARIAN voluntarily contacted me to express either his/her sympathy or provide an explanation for his cause of death. They were either "out of town" or "in surgery". However, I was desperate and in shock due to the outcome of his health ordeal and I wanted answers. After all, I was and am a veterinary services consumer and tax payer and had a right to expect more than silence. Since all five veterinarians would not/could not give me answers, I decided to file a complaint with the state board in order to find the answers from a group of professionals such as yourselves. Your decision: "The board, upon recommendation. . . .has dismissed the matter. . . ." really puzzled me. You could not really give me a cause of Asproolee's death, even with all his medical records available to you, OR you covered wrong/lack of diagnosis and omission in an effort to protect your colleagues' reputation. I understand. . you could be in their shoes some time. . .but you could never be in my shoes, trying to cope not only with grief , but also having unanswered questions.

Your practice code reads:

PRACTICE OF VETERINARY MEDICINE.

- a. To diagnose, treat, correct, change, relieve, or prevent animal disease, deformity, defect, injury or other physical or mental condition; including the prescription or administration of any drug, medicine, biologic, apparatus, application, anesthesia, or other therapeutic or diagnostic substance or technique on any animal including but not limited to acupuncture, dentistry, animal psychology, animal chiropractic, theriogenology, surgery, including cosmetic surgery, any manual, mechanical, biological, or chemical procedure for testing for pregnancy or for correcting sterility or infertility, or to render service or recommendations with regard to any of the above.
- b. To represent directly or indirectly, publicly or privately, an ability and willingness to do any act described in paragraph a.

In Asproolee's case there **was a failure to diagnose, misdiagnosis, and wrong treatment (surgery) which caused his death.** If this outcome is not in violation of your code, then what is it??

Your practice also reads:

The board directs its activities to protecting the interests of the public which it serves and in aiding the practicing veterinarian in the state by its efforts directed toward maintaining a high standard of integrity and skill in the practice of veterinary medicine and insuring that facilities, where veterinary medicine is practiced, meet all standards set forth in the Veterinary Practice Act and its Administrative Code.

You not only did not protect my interests as the public but you insulted my intelligence! Dr. Pitman, the investigative DVM, in his phone call on May 16, answered as follows per our **Memo for Record: (A = Asproolee)**

Called by Dr Pitman from AL Vet Bd at 1615 16 May 08

Said vets did nothing wrong.

Said A died from natural causes. (after the surgery?? Coincidental??)

Said he knew 2 of them personally. When I asked him how he could be objective evaluating his friends he said he saw no problem with it.

*When I asked him why Emergency clinic did no diagnostics or blood work as per their charter he said they treated the animal for a seizure. We asked him why A had a seizure and he said **he didn't know.***

I asked why the surgeon didn't provide us a detailed discussion of the possible outcomes of surgery for A. He didn't know.

*I asked him why the surgeon didn't keep A until he was eating/drinking normally before he released him back to us. **He didn't know.***

*I asked him why the only post operative instructions the surgeon gave us was "bring him back in 2 weeks for a check up" instead of details on what action to take if things weren't going properly, etc. **He didn't know.***

WHY didn't the investigator have these answers if he just completed a "thorough" investigation??

Please give us credit for some intelligence! Dr. Pitman, your parallelism of your mother's (94) hospitalization and bill payments is to say the least very unfortunate. Was your mother misdiagnosed, given the wrong treatment and died as a result?

Did Dr. Ballagas's grandfather die? He mentioned to me that his grandfather, 97, fell and broke his hip. He was not sure if his grandfather would make it even though the doctors did the right thing. . . Another unfortunate parallelism. I was only wondering if there is an epidemic among veterinarians's closed relatives' health problems and bill payments.

Dr. Ballagas asked me if Asproolee was on Prednisone and I said that the name of the medication definitely rang a bell. Yes, Asproolee was on Prednisolone eye ointment, and it was very well documented in Asproolee's U.S. Army vet medical records. In fact, the label of the medication was affixed to the particular medical record page. The Federal veterinarians adhere to the Federal and Military regulations to the letter. If any vet had bothered to check Asproolee's records, he/she wouldn't have missed it. He (Ballagas) said "I thought that his liver was not normal because of age!" He thought. . . ! Did he X-ray Asproolee prior to the surgery? What kind of anesthesia did he use?? Wasn't he aware that Asproolee had mitral valve insufficiency?

During our telephonic conversation and several days after Asproolee's death, Dr. Ballagas asked me what I needed the surgery and anesthesia reports for. He said that everything was fine, and that I should be talking to Dr. Lothrop since it was the internist who recommended the surgery. He said quote "If it wasn't for him, you would never have met me. The guy isn't working for me, and I don't work for him. . ."

I also mentioned to Dr. Ballagas that Asproolee's voice had changed a couple of days prior to the surgery. He believed that it was a thyroid problem, but he never offered any diagnostics for this condition. Was it laryngeal paralysis? Did it also contribute to Asproolee's fatal outcome? Yes, he was 94 years old, but that didn't seem to be of concern to any of the vets prior to the surgery. Did everyone realize that he was old for the surgery after he DIED?? What would they have said if Asproolee was diagnosed correctly and the operation was successful? Any mention of the age then?

I requested several local veterinarians and academic professionals, including Dr. Horne, for a second opinion as to the cause of Asproolee's death. I also requested ACVS to provide a list of diplomate surgeons in Alabama for a second opinion. They responded to my request, and Dr. Horne's name was on that list. At that time, I did not know that he was a member of the board, and I asked him for a second opinion. **Dr. Horne, you have seen now all the medical records, including X-rays and ultrasound. What is your opinion as an academic professional?? NOT ONE**, in the state of Alabama, was willing to help me in finding closure to my dearest pet's death. Only one very honest academic professional declined my request and wrote:

I have considered your request and I have decided to decline. I have worked directly with one of the surgeons in Birmingham and know the other quite well, therefore I do not feel comfortable becoming involved. Although I would do my best to be fair and unbiased, I would not want that to become even a minor factor. There are other board certified surgeons in the southeast that might do this for you.

Yet, Dr. Pitman had no problem with investigating fellow vets whom he knows personally. Camaraderie is highly encouraged and sought after in our armed forces, but camaraderie among professionals like you and law is bad for the public and harms your reputation.

I wrote and sent copies of Asproolee's medical records (except X-rays, ultrasound and surgery report) to several veterinarians outside the state of Alabama. I received second opinions from three veterinarians from three different states. Here are their second opinions:

- 1. Opinion: I can say that the liver biopsy supports an underlying Cushing's disease but beyond that I'm not sure. The hypoglycemia could explain the seizure but and could fit w/Addison's but Addison's doesn't fit w/the steroid hepatopathy in the liver. The rest really does sound like Addison's though: ultrasound that looked like intestinal obstruction, hypoglycemia, shock and death. What did the adrenals look like on the ultrasound? Big or small?**

Not sure what you mean by the internist failed to perform ultrasound on the adrenal glands. They were too small to be seen. That pretty much rules Cushing's disease out and puts more evidence under the Addison's disease category. Normal adrenal glands are easy to see and Cushing's adrenals are larger than normal. It is highly significant that they were not seen.

- 2. Opinion: My top suspicion is hypoadrenal or Addison's disease (the "great imitator").** The electrolyte results did not support, but atypical cases can have normal electrolytes. One big question not answered with Addison's is the histopath report from the liver tissue submission postop. It talked about changes caused by exposure to corticosteroids (from oral meds or from Cushing's). So, the test result I would like to have seen comes from an ACTH stimulation test. Abnormally high results indicate possible Cushing's, while low results indicate possible Addison's. If this was Addison's, it would have been an atypical presentation. Interestingly, the ACTH stimulation test would have addressed both possibilities. I've learned to at least ask myself about Addisonian disease when planning an exploratory.
- 3. Opinion: Lethargy, anorexia, panting, hind legs weakness, gastrointestinal problems, vomiting, and hair thinning were the symptoms that forced you to take your dog to the vet initially.** These symptoms can be signs of endocrine disorders, specifically Hypoadrenocorticism (Addison's Disease), Hyperadrenocorticism (Cushing's Syndrome), and hypothyroid.

I think that your dog may have had undiagnosed Addison's disease

(Hypoadrenocorticism). The cause of Hypoadrenocorticism is unknown, although immune-mediated destruction of the adrenal gland is suspected in most cases. Addison's disease is a deficiency of hormones normally produced by the adrenal glands. **However, your dog's blood work was atypical for Addison's, so Addison's disease would have to have been diagnosed** with additional tests such as: ACTH stim test or Low dose dex. test., and unfortunately it is too late for such tests so we must just speculate.

The enlarged heart and poor eye sight may have been congenital or developmental and may not have been related to the endocrine disorder. Your dog had already been on heart medications for 3 years prior to this episode. However, electrolyte abnormalities secondary to Addison's disease can disrupt heart function and cause arrhythmias, which could worsen an existing heart condition.

Addison's disease can also cause seizure and coma due to hypoglycemia. Your dog's last listed glucose level was 29. A glucose level of 29 is extremely low and is considered to be

hypoglycemic. Hypoglycemia by itself can cause seizures and coma. Addison's disease is not the only cause of sudden hypoglycemia, though. Other causes that could have occurred in your dog's case include Insulinoma (a tumor of the pancreas that produces excess insulin) and overwhelming infection (possibly secondary to the surgery, in your dog's case). There was a sign of possible infection in your dog: "SQ emphysema" in the integument at the suture site, this can be indicative of infection.

No surgeon will perform surgery unless he/she takes a look at all diagnostics, such as the X-Rays and the ultrasound,. The surgeon will not rely only on another doctor's recommendation, but will make his/her own decision. Your dog's surgeon must have been convinced that there was an obstruction of some sort, or a very good reason for performing surgery, or the surgeon would not have performed the surgery, therefore, the blame cannot be given only to the internist on this one.

Though there is no way to be definitive about the cause of death for your dog the following rule outs are possible: Surgical complications secondary to Addison's disease, Surgical complications secondary to heart disease, Infection following surgery, Adrenal carcinoma causing adrenal failure, or Pancreatic cancer (Insulinoma), among other possibilities.

4. Asproolee's case is under study by a Federal veterinarian and I will forward the results to you.
5. A prominent professor of a western major university Veterinary Medicine College will also provide his second opinion as soon as he receives radiographic and ultrasound examinations. Asproolee's attending veterinarians' office called and said that they have 22 (!!!) X-rays for copying at \$35.00 a piece! I had no idea that they X-rayed Asproolee 22 times, and they certainly never discussed any detailed results with me. Didn't Asproolee and I pay already very dearly for their wrong diagnosis and death? I guess they still want to profit from his death. However, it's hard to believe that after all these examinations the veterinarians still could not diagnose Asproolee!

As you can see **Asproolee died of Addison's disease** which the attending, the internist (who should have been immediately alerted when he saw NO adrenals!), and the surgeon failed to diagnose although all the symptoms were present. Does it require an Addison's veterinary specialist to diagnose it? Why did none of the vets think to run an ACTH blood test on Asproolee?? Addison's is a disease to which poodles are predisposed. With all the symptoms presented it should have been their FIRST thought!

You were only the first step in my long and difficult path to do justice for Asproolee's death. I am certain that you are aware of the many powerful tools the veterinary consumers and tax payers have now to have their interests protected. Burying 13 years of my life with Asproolee is not as easy as you might think. It has caused me post-traumatic stress, and I am in grief counseling trying to cope with the horrific and

agonizing way he died which Dr. Pitman described as “natural causes!” And then again, I was very well aware of the boards’ reputation in dismissing 95 percent of the cases.

I have two more canine pets. When they pass away (hopefully of natural causes), I will no longer have any more pets **because I do not trust veterinarians** anymore. I used to blindly, and that was my TRAGIC ERROR! Just the idea that my pets need some veterinary care now scares me to death! If something happens to my pets there is no one to turn to for help, and the veterinarians can get away with any wrong-doing!

Asproolee died because of Addisonian crisis. Seizure/Shock, collapse, coma, death. The emergency veterinarian did not perform any diagnostic tests in an effort to diagnose the seizure although we wrote on the admittance form that he had an exploratory surgery. We told him that Asproolee had never experienced any seizures prior to the surgery, and we asked him to administer IV fluids because he had continued with anorexia even though we tried to force feed him. Why did he ignore our request? Even the surgeon asked us if the emergency veterinarian had performed any diagnostic tests.

I offer some very interesting reading from Dr. Deborah S. Grero, DVM, PhD, DACVIM: Hypoadrenocorticism in dogs and cats, Symposium, June 2000, Veterinary Medicine. Also, more interesting literature-- “Addison’s disease—Uncommon or Underdiagnosed? A Round Table Discussion.”

I have also enclosed an excerpt from Cornell College of Veterinary Medicine. .all the signs they list were symptoms Asproolee exhibited. If we can find all this information, why couldn’t the vets have taken the time to do a little research—it **could have saved his life!**

Sincerely,

Raymond Hale

Tina Hale

Enclosures

SYMPOSIUM

PEER-REVIEWED

Hypoadrenocorticism in dogs and cats

Can you recognize the signs of hypoadrenocorticism? You'll have no doubts after reading this review on how to diagnose and treat this disease, which has a favorable prognosis once it's brought under control.

DEBORAH S. GRECO, DVM, PhD,
DACVIM
Department of Clinical Sciences
College of Veterinary Medicine and Biomedical Sciences
Colorado State University
Fort Collins, CO 80523

A 3-YEAR-OLD SPAYED standard poodle weighing 44 lb (20 kg) presented to a local veterinary hospital for evaluation of intermittent anorexia, vomiting, and diarrhea of seven months' duration. The dog's history included recurrent episodes of lethargy and anorexia that responded to fluid therapy. Physical examination revealed a thin dog. Results of a complete blood count, a serum chemistry profile, and urinalysis showed a mild, normocytic, normochromic anemia and eosinophilia. Fecal examination was negative for parasites. The tentative diagnosis was inflammatory bowel disease, dietary allergy, or occult parasitism. The dog was dismissed, and treatment consisted of limited antigen dietary therapy and fenbendazole.

Six months later, the dog presented to Colorado State University's Veterinary Teaching Hospital in hypovolemic shock after an acute onset of vomiting and diarrhea. The dog had been at a boarding facility for five days. The dog's heart rate on admission was 50 beats/min. Abnormal results of an emergency serum chemistry profile included azotemia (blood urea nitrogen concentration = 187 mg/dl; normal = 17 to 32 mg/dl; creatinine concentration = 4.3 mg/dl; normal = 0.6 to 2 mg/dl), hyponatremia (sodium concentration = 125 mEq/L; normal = 145 to 160 mEq/L), hyperkalemia (potassium concentration = 7.8 mEq/L; normal = 3.7 to 5.4

mEq/L), hypochloremia (chloride concentration = 98 mEq/L; normal = 112 to 129 mEq/L), and hypercalcemia (calcium concentration = 12.3 mg/dl; normal = 9 to 11.5 mg/dl). Urine specific gravity was 1.012.

The dog was treated with intravenous 0.9% sodium chloride solution (at a shock dose of 90 ml/kg/hr) and glucocorticoids (1 mg/kg dexamethasone sodium phosphate). An ACTH response test performed at admission revealed a low baseline serum cortisol concentration (< 0.1 µg/dl; normal = 1 to 4 µg/dl) with no response to ACTH administration (0.25 mg cosyntropin intravenously). The post-ACTH cortisol concentration was less than 0.1 µg/dl (normal > 6 µg/dl and < 20 µg/dl) one hour later.

The dog recovered from acute adrenocortical insufficiency. It will receive mineralocorticoid and glucocorticoid replacement therapy consisting of desoxycorticosterone pivalate (DOCP) (Percorten-V—Novartis; 2 mg/kg intramuscularly every 30 days) and prednisolone (0.2 mg/kg orally once a day) for the rest of its life. A complete blood count, a serum chemistry profile, and urinalysis are monitored every six months. The dog's weight has stabilized at 55 lb (25 kg).

Diagnosis

Given this dog's signalment, history, and clinical signs, hypoadrenocorticism (Addison's disease) should have been a differential diagnosis during the

Addisonian crisis

Acute adrenocortical insufficiency is a life-threatening emergency, so initiate therapy immediately. Treating an Addisonian crisis consists of 1) administering fluid therapy and stabilizing electrolyte concentrations, 2) replacing glucocorticoids, 3) managing gastrointestinal hemorrhage, and 4) replacing mineralocorticoids.^{1,2,15,18}

Of primary importance is rapidly administering large volumes of intravenous fluids; 0.9% sodium chloride solution is the fluid of choice. Fluid delivery is best accomplished by using a jugular catheter. You can

HYPOADRENOCORTICISM, ADDISON'S DISEASE, IN DOGS

Description: Primary hypoadrenocorticism results from adrenal cortical failure, commonly atrophy of unknown etiology, leading to deficiency of mineralocorticoids and/or glucocorticoids. An autoimmune pathogenesis is suspected and there is a genetic basis in dogs although the mode of inheritance is undetermined. Typically affects young to middle aged dogs, females more than males. Other causes include: destruction of the adrenal cortex by neoplasia; infarcts; infiltrative disease such as tuberculosis, histoplasmosis, blastomycosis, amyloidosis; or administration of treatments for hyperadrenocorticism. Secondary hypoadrenocorticism due to insufficient ACTH stimulation results from hypothalamic or pituitary lesions or prolonged exogenous glucocorticoid administration. Clinical signs can wax and wane. Acute severe disease (adrenal crisis) might be due to adrenal infarction or termination of the chronic phase. Concurrent renal failure is common. Megaeosophagus might occur.

Species: Canine

Signs: **A**lopecia, **A**norexia, **A**taxia, **B**loody stools, feces, hematochezia, **B**radycardia, **C**old skin, **C**olic, **C**oma, **D**eceased amount of stools, absent feces, constipation, **D**ehydration, **D**iarrhea, **D**isoriented, **D**ullness, **D**ysmetria, **D**yspnea, **E**xercise intolerance, **F**orelimb spasms, **G**eneralized weakness, **H**ematemesis, **H**indlimb lameness, **H**indlimb spasms, **H**ypothermia, **I**nability to stand, **I**ncreased respiratory rate, **M**elena or occult blood in feces, stools, **P**ain on external abdominal pressure, **P**ale, **P**araparesis, **P**olydipsia, **P**olyuria, **P**rolonged capillary refill time, **R**eluctant to move, **R**ough hair coat, **S**eizures or syncope, **S**udden death, **T**achycardia, **T**etraparesis, **T**rembling, **T**remor, **U**nderweight, poor condition, thin, emaciated, unthriftiness, ill thrift, **U**rinary incontinence, **V**omiting or regurgitation, **W**eak pulse, **W**eight loss

References: **Boysen SR.** Fluid and Electrolyte Therapy in Endocrine Disorders: Diabetes Mellitus and Hypoadrenocorticism. Veterinary Clinics of North America: Small Animal Practice 2008;38:699-717 [Web Reference]

Greco DS ed. Adrenal disease. Clinical Techniques in Small Animal Practice 2007;22:1-40 [Web Reference]

Adler JA et al. Abnormalities of Serum Electrolyte Concentrations in Dogs with Hypoadrenocorticism. Journal of Veterinary Internal Medicine 2007;21:1168-1173 [Web Reference]

Hughes AM et al. Clinical features and heritability of hypoadrenocorticism in Nova Scotia Duck Tolling Retrievers: 25 cases (1994-2006). Journal of the American Veterinary Medical Association 2007;231:407-412 [Web Reference]

Lennon EM et al. Use of basal serum or plasma cortisol concentrations to rule out a

diagnosis of hypoadrenocorticism in dogs: 123 cases (2000–2005). Journal of the American Veterinary Medical Association 2007;231:413-416 [Web Reference]

Thompson AL et al. Comparison of classic hypoadrenocorticism with glucocorticoid-deficient hypoadrenocorticism in dogs: 46 cases (1985–2005). Journal of the American Veterinary Medical Association 2007;230:1190-1194 [Web Reference]

Burkitt JM et al. Relative Adrenal Insufficiency in Dogs with Sepsis. Vet Intern Med 2007;21:226–231 [Web Reference]