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OPHTHALMOLOGY

Lens Instability (Luxation/Subluxation) in the Dog & Cat

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Part One: Anatomy & Diagnosis

ANATOMY OF LENS STABILITY

The lens of the eye sits in the patellar fossa. Although you might have thought this structure to be found in the knee, it is actually the depression in the anterior face of the vitreous that itself is formed by the posterior convexity of the lens. Anteriorly, the lens abuts the iris, which provides it a measure of support. Around the equator of the lens are the lens zonules (or Zonules of Zinn, which form the suspensory ligament of the lens). These zonules anchor the lens capsule to the ciliary body, which is responsible for accommodation or focusing of the lens.

DIAGNOSIS OF LENS LUXATION

Lens luxation is the full dislocation of the lens out of the patellar fossa, either anteriorly (into the anterior chamber in front of the iris) or posteriorly (into the vitreous).[1] Both types of luxation can be surprisingly hard to diagnose, either due to very subtle changes or dramatic changes (e.g., corneal edema from glaucoma) that preclude full visualization of the lens.

Anterior lens luxation with cataract is often very obvious (Figure 1), but when the lens is clear or when corneal edema from glaucoma is present, it can be hard to visualize. One feature to look for in this scenario is the positioning of the iris. Generally, the curvature of the iris (which is

(CONTINUED ON NEXT PAGE)

SURGERY

Laparoscopic Ovariectomy and Gastropexy

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Laparoscopic ovariectomy (OVE) is a method for neutering that has been widely accepted for many years now. Among numerous sought after benefits of this laparoscopic technique are advantages such as a faster recovery period, improved visualization and a significant decrease in postoperative pain.[1,2,3] This procedure also has been shown to cause less surgical stress than a conventional ovariohysterectomy (OVH) and to be potentially more appropriate for an outpatient setting.[4] There also is less risk of dehiscence and hemorrhage, and less risk of postoperative wound complications.[3]

In humans, laparoscopic surgery has been proven to have many advantages over traditional celiotomy techniques, including decreased postoperative stress and pain, faster recovery periods, decreased hospitalization, improved cosmesis and improved visualization of abdominal organs.[5] Pet owners have become increasingly aware of these benefits for themselves and therefore have increased interest in minimally invasive surgical techniques for their own pets.

The common laparoscopic technique performed for female sterilization is OVE, during which only the ovaries are removed. It has been documented in veterinary studies that there is not an increased risk of complications such as pyometra, urinary sphincter mechanism incontinence and weight gain when compared with OVH.[6] The benefit of just performing OVE instead of OVH is a faster surgery time, a smaller incision and less manipulation of the female genital tract.[6]

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OPHTHALMOLOGY

FIGURE 1

↘ Anterior cataractous lens luxation in a cat.

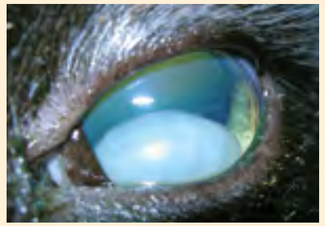
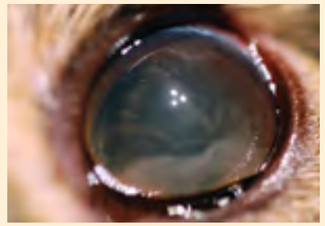


FIGURE 2

↘ Anterior lens luxation. Note the refractile edge of the lens from the 9 to 12 o'clock positions.



normally convex only because it lies over the convex lens surface) should be almost parallel with the curvature of the cornea. A slit beam from a transilluminator can illuminate both structures well. If you see a concave surface to the iris (bending away from you centrally), this can imply the lens is in front of the iris, and at this point you should look peripherally around the limbus to attempt to visualize the edge of the lens, which should reflect light as a bright crescent (Figure 2).

If the iris is lying flat ("D-shaped" anterior chamber), the lens may be posteriorly luxated (Figure 3). The best way to visualize this is sometimes via retinal fundic examination, or in the event of visual axis impairment (e.g. from hyphema), ocular ultrasonography (Figure 4).

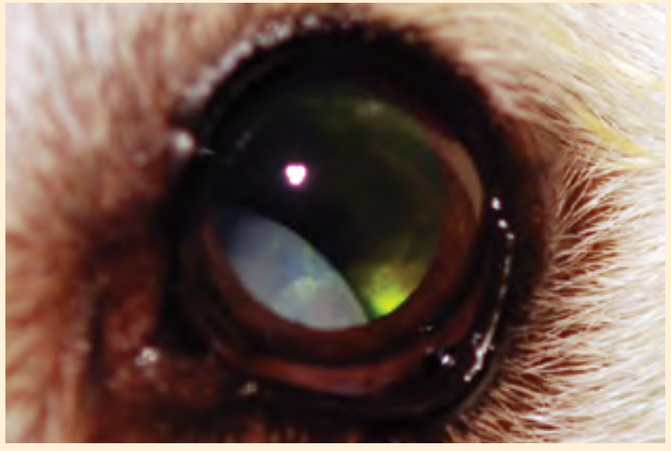
DIAGNOSIS OF LENS SUBLUXATION

Lens subluxation is the partial detachment of the lens from the ciliary body due to breakdown or weakness of the zonules. Although the presence of an "aphakic crescent" is the classic sign of lens subluxation (Figure 5), evidence of lens subluxation can be very subtle. Signs to look for can include an asymmetrically shallow anterior chamber (from one side of the lens tilting and pushing the iris forward), fluttering movement of the lens (phacodonesis) or iris (iridodonesis) as the eye moves, and very commonly, liquefied vitreous in the anterior chamber. Subluxation can also be accompanied by an increase in intraocular pressure (IOP), i.e., secondary glaucoma. To confirm a diagnosis of lens subluxation when some of these symptoms are present, examination in a darkened room with a dim slit beam from an ophthalmoscope can allow enough natural iris dilation to observe the subluxated edge of the lens when looking across the eye at an angle.

Even though it would help diagnosis, it bears emphasis that an eye with a suspected lens luxation or subluxation should never be iatrogenically dilated with tropicamide, atropine or any other mydriatic drug. Dilating an eye with a subluxated lens removes the support of the iris from the lens' anterior face and can precipitate full luxation anteriorly. Dilating an eye with posterior luxation increases the risk that the lens will move forward in the eye. Additionally, pupil dilation can close the filtration angle, increasing IOP and potentially precipitating a glaucoma crisis.

FIGURE 3

↘ Posterior cataractous lens luxation. The retina is also degenerate resulting in marked tapetal hyperreflectivity.



That said, mydriatic agents are sometimes used by ophthalmologists in the management of lens luxation. Examples include when pupillary block glaucoma is present (when the lens is anteriorly luxated and the pupil is constricted around the vitreous, or when the pupil is tight against the lens like a closed ball valve); attempts to replace an anteriorly luxated lens with a procedure called "couching";[6] and in post-surgical (lensectomy) management (to reduce synechia formation, help with comfort and stabilize the blood-ocular barrier).

CAUSES OF LENS INSTABILITY

Lens luxation is known to be of genetic origin in many canine breeds (e.g., Terriers, Chinese Crested, American Eskimo, Australian Cattle Dog), likely autosomal recessive.[3,4,8] DNA testing (for the ADAMTS17 substitution) is now available for these breeds from multiple sources: the Orthopedic Foundation for Animals, Optigen and, in the UK, the Animal Health Trust. Otherwise, secondary lens luxation can occur from many conditions, including trauma, chronic glaucoma (buphthalmos, as stretching of the globe can tear the lens attachments), chronic uveitis and hypermature cataracts.

OPHTHALMOLOGY

FIGURE 4

↘ Annotated ultrasound of an eye with posterior lens luxation. Direct visualization was impossible due to hyphema.

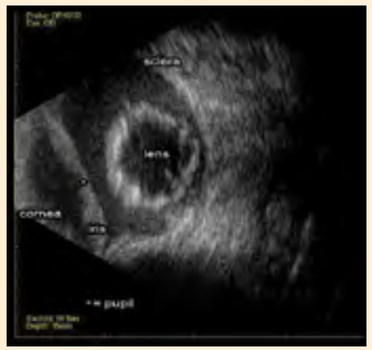
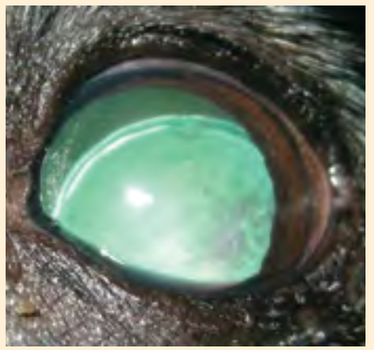


FIGURE 5

↘ Lens subluxation. Note the aphakic crescent superiorly. The lens appears iridescent due to retro-illumination from the tapetal reflection. There is also a cataract, an opacity to retro-illuminated light at the righthand (lateral) aspect of the lens.



In the cat, uveitis is the most common cause of lens luxation, which presumably occurs due to inflammatory breakdown of the lens' suspensory ligament. Therefore, in cats, an infectious disease workup (FeLV, FIV, FIP, toxoplasmosis, etc.) is important when presented with a lens luxation.[7] Congenital anatomic defects have also been reported.[5]

SEQUELAE TO LENS INSTABILITY

Lens instability can lead to uveitis, glaucoma,[2] retinal detachment, hyphema, cataract, reduced vision and blindness from sequelae. Uveitis likely occurs from lens motion stimulating inflammation of the iris, ciliary body, and even corneal endothelium in full anterior luxation. Glaucoma can occur as a sequel to uveitis (either acute or chronic buildup of inflammatory membranes or pre-iridal fibrovascular membranes that occlude the filtration angle). Glaucoma can also occur due to a primary obstruction of aqueous humor filtrate through the filtration angle. Dogs are generally at much higher risk of glaucoma than cats, due to their disparate ratios of lens to anterior chamber volumes, and a lens in the anterior chamber leaves little space for fluid to flow through the pupil out the angle. Additionally, anterior lens movement typically brings the vitreous forward, which can become entrapped in the pupil. Retinal detachment can occur from the remaining lens zonular attachments at the peripheral retina exerting more pulling force on the retina as the lens begins to luxate. This, or direct irritation and inflammation of iris vasculature, can lead to hyphema. A cataract forms in the lens as the typically nourishing aqueous humor alters in flow around the lens, reducing nutritional support to the lens. Reduced vision occurs from altered refraction/focusing of light onto the retina, or from any of the above sequelae, which can all, of course, lead to complete blindness.

Part two will discuss the medical and surgical management of lens instability in the dog and cat. To view part two on our website, please visit **angell.org/lens2**.

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For more information about Angell's Ophthalmology service, please visit **angell.org/eyes**. Dr. Coster can be contacted at **617-541-5095** or **ophthalmology@angell.org**.

Canine Hyperadrenocorticism

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Canine hyperadrenocorticism (HAC) is an endocrine disease affecting primarily older dogs with 80–85% of cases caused by pituitary-dependent disease (PDH); the remainder is due to an adrenocortical tumor (AT). There is a slightly increased predisposition for females (55–65%), and larger breeds may be affected more frequently with an AT.

We are all familiar with the common clinical signs of HAC as well as laboratory findings, so those will not be discussed in this article. If a single sign compatible with HAC is present, it is likely to be either polyuria/polydipsia or skin coat changes. Less commonly seen signs include those related to a macro-tumor (behavior change, inappetence, obtundation, aggressiveness, apparent blindness and seizures), though sometimes signs are subtle. About 10-25% of dogs will develop neurologic signs months to years after the initial diagnosis. Other less common signs include anestrus, testicular atrophy, ligament laxity, facial palsy, spontaneous thromboembolism and cortisol-induced insulin resistance. Signs from an invasive adrenocortical carcinoma may include hemorrhage due to vessel invasion or thrombosis of the vessel (ascites, hind-limb edema or paresis). If less common clinical presentations are identified first, a thorough review of the history, physical examination findings and routine laboratory test results may provide additional evidence for HAC.

A recent Consensus statement was published in the Journal of Veterinary Internal Medicine (JVIM 2013; 27: 1292-1304), so the remainder of this article will focus on the Panel recommendations in regards to testing for HAC. Testing should only be considered if there are clinical signs (common or uncommon). Biochemical panel, complete blood cell counts, urinalysis, UPC and blood pressure results by themselves are not indications to test.

No test is 100% diagnostic for HAC. Diagnosis depends on documenting increased cortisol production (ACTH stimulation) or decreased sensitivity of the hypothalamic-pituitary-adrenal axis (HPPA) to the normal negative feedback of steroids administration (low-dose dexamethasone suppression test; LDDST). Reference ranges may need re-evaluation in the future, as we are likely testing sooner and ranges were generated many years ago. If a patient tests negative, repeat testing in 3-6 months may be warranted, especially if signs progress.

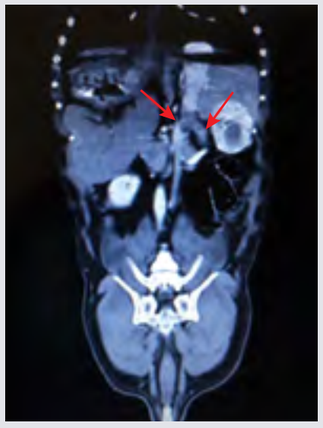
The LDDST is currently considered the screening test of choice unless iatrogenic HAC is a consideration. The cortisol concentration, at 8 hours after dexamethasone administration, is used to diagnose HAC. In the clinical experience of the Panel, normal dogs are usually below or close to the detection limit of the sample (i.e., <1.0). Therefore, if a patient is below 1.4 but not below the detection limit, HAC may still be considered. In addition, an inverse pattern, where the 4-hour cortisol is high but the 8-hour is below the detection limit, may also indicate HAC. Results should be interpreted with caution if the patient is currently taking phenobarbital, as those dogs occasionally will not show suppression.

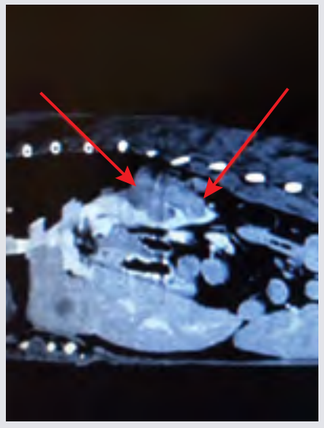
The ACTH stimulation test has a lower sensitivity (range 57-95%) so is considered inferior to the LDDST as a screening test, but it is the test of choice for iatrogenic HAC. Synthetic ACTH is recommended over compounded drug, and intravenous administration is preferred over intramuscular. Progestogens, glucocorticoids, and ketoconazole all suppress the HPAA axis and response to ACTH. Phenobarbital does not appear to affect the test. The urine corticoid : creatinine ratio (UCCR) is a sensitive test (75%-100%) for detection of cortisol hypersecretion but is influenced by endogenous stress and non-adrenal illness. Collection should take place at home, at least 2 days after a veterinary visit, to decrease the likelihood of false positives.

Differentiating tests should only be pursued once there has been a positive on a screening test. The canine ACTH measurement is the most accurate stand-alone biochemical test for differentiation; however, reference ranges

FIGURES 1 & 2

↘ The two images here represent dorsal and saggital views from a CT scan of a patient with bilateral adrenomegaly. Ultrasound was concerning but not definitive for vena caval invasion.

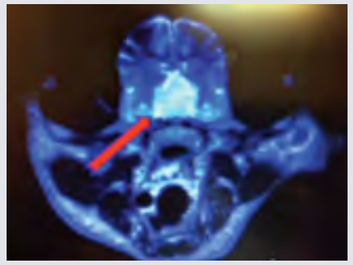




INTERNAL MEDICINE

FIGURE 3

↘ CT image of a pituitary macroadenoma. The patient was displaying behavioral changes and was not eating well each time trilostane was restarted despite a normal ACTH stimulation.



vary by technique and certain assays are less sensitive. In addition, proper sample handling is crucial to accurate results. Discordant results can occur, and many dogs with PDH will have ACTH at the lower end of the reference range, but they should not be lower than the low-end reference range. The high-dose dexamethasone suppression test (HDDST) can be used to differentiate PDH from AT. If suppression occurs, the patient likely has PDH. However, if there is no suppression, it should be kept in mind that about 25% of PDH dogs will not suppress even at the higher dose. A macroadenoma may be more likely in a dog without an AT that does not suppress on the HDDST.

For those patients who cannot be hospitalized, an oral dexamethasone suppression test can be considered. Samples for UCCR are taken on 2 consecutive days, and then 3 doses of dexamethasone (0.1mg/kg PO) are given at 6- to 8-hour intervals and urine collected again the following day. A decrease in the UCCR to <50% of the mean basal values is consistent with PDH. Lack of suppression does not discriminate between PDH or AT. Changes in metabolism of dexamethasone may influence the results of any of the suppression tests.

Imaging must be used in conjunction with hormonal testing and should not be used as the sole modality for diagnosis. Radiographic changes in patients with HAC include abdominal distention, good contrast due to fat deposition, hepatomegaly, bladder distention, mineralization of the bronchi and pulmonary interstitium, and possibly a mass effect or mineralization in the retroperitoneal space. On US, adrenal gland width is considered the most informative parameter but breed and body-size related differences must be considered. US can estimate tumor size and possibly vascular or soft tissue invasion; however, CT scan and MRI are more sensitive for evaluation of invasiveness as well as metastases (see Figures 1 and 2). Metastases, venal cava invasion by a tumor mass, adrenal width (>4cm) or a combination of these findings is highly suggestive of malignancy.

Because radiation therapy or hypophysectomy is the required treatment for macro-tumors, and both are more effective with smaller tumors and in the absence of neurologic signs, the Panel recommends pituitary imaging be considered for all dogs at the time of initial diagnosis (see Figure 3). Of course, if clinical signs suggest a macro-tumor, CT or MRI is also recommended. As pituitary tumors and a cortisol-secreting AT may be present at the same time, some also recommend pituitary imaging with AT.

If a patient does not fit the clinical picture for HAC, testing for occult HAC ("atypical HAC") should not be considered. While a cause and effect relationship between AT sex hormones and clinical signs is well documented, a causative relationship with sex hormones and PDH has not been established. Therefore, an adrenal sex-hormone panel should be considered for patients with inappropriately low cortisol levels on initial screening tests and with adrenal tumors not testing positive for cortisol secretion. Food-stimulated HAC should also be a differential when considering occult HAC, especially if the fasting cortisol concentration is low.

IN CONCLUSION

- Endocrine testing for HAC should only be considered if there are compatible physical exam findings and history. Testing should not be performed based on laboratory changes alone
- The LDDST is considered the screening test of choice
- An endogenous ACTH level or abdominal ultrasound are recommended for differentiating between AT and PDH
- Sex-hormone testing is recommended mainly when an adrenal tumor is present and the initial cortisol testing has returned low

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Tooth Fractures

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Tooth fractures are a common finding when examining the mouths of cats and especially dogs. Most of these fractures expose the pulp canal of the tooth, leading to tooth death and infection. Unfortunately, most dogs and cats are too stoic for their own good and do not overtly show the pain associated with pulp exposure or subsequent root abscess. It is up to us to detect and properly treat these painful, diseased teeth. If the pulp canal of a tooth is exposed, the only proper treatments are extraction or a root canal procedure; "wait and see" is not appropriate (Figure 1).

Canine teeth (and incisors) are frequently damaged from random trauma (HBC, falls, etc.). The most commonly fractured tooth in dogs is the maxillary 4th premolar. This tooth is most often fractured due to chewing trauma from items like bones (ANY actual bone), non-flexible nylon bones, antlers, hooves, and, a recent addition to the bad chew-toy list, yak's milk. These products are often advertised as long lasting. Unfortunately, because they don't break down, the teeth used to chew them, usually the maxillary 4th premolar, do fracture. Since these fractures occur from chewing trauma, rather than random trauma, dogs are likely to fracture both maxillary 4th premolars. A common fracture of the maxillary 4th premolar can be a "slab fracture," fracturing a significant portion of the buccal surface of the tooth and often extending below the gum line to the root structure (Figure 2). Even if the pulp canal is not exposed, these complicated fractures can make it very difficult to realistically keep these teeth without significant periodontal disease developing. Any damage to any tooth at or below the gum line will increase the periodontal disease. The maxillary 4th premolar is one of the teeth most likely to have calculus buildup, making detection of fractures more challenging. Some of these fractures are subtle, so it is important to know the normal shape of the maxillary 4th premolar. This tooth resembles the number four on its side (Figure 3).

The commonly accepted classic sign of an abscessed tooth is facial swelling. Unfortunately, the vast majority of abscessed teeth do not form these externally visible swellings. It can take years, if ever, for the chronic infection to destroy enough bone in the right direction as to form a visible swelling. More common is the formation of intermittent draining tracts as the root abscesses go through cycles of forming and draining. These are called gumboils, or paruluses (or parulises), and are visualized as pink/red bumps on the gum line. They represent the openings of chronic draining tracts from tooth root abscesses and are at the mucogingival margin of the affected tooth (Figures 4 and 5).

FIGURE 1

↘ Fractured canine with pulp exposure.

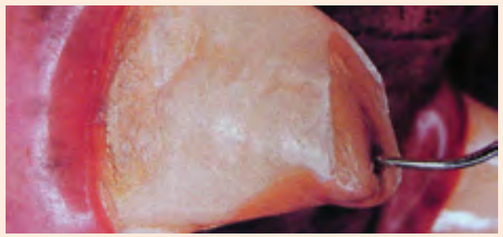


FIGURE 2

↘ 4th premolar slab fracture.

FIGURE 3

↘ Close-up of healthy 4th premolar.

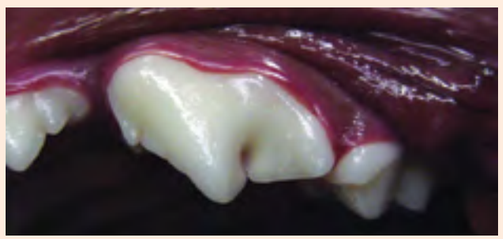


FIGURE 4

↘ 4th premolar fistula.

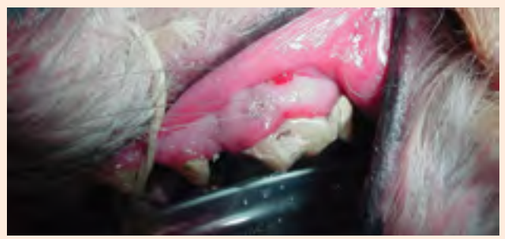


FIGURE 5

↘ 4th premolar abscess radiograph.

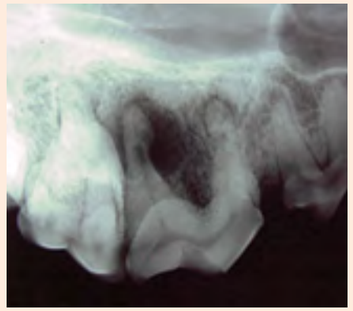
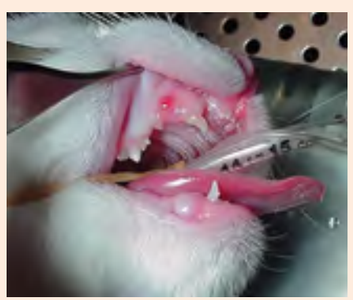


FIGURE 6

↘ Deciduous tooth abscess.

FIGURE 7

↘ Deciduous canine tooth abscess in a kitten.



Fractures of deciduous teeth are also often neglected with the idea that the tooth will eventually fall out anyway. Normal exfoliation (loss) of a deciduous tooth is due to resorption of the root by odontoclasts present in the tissue above the crown of forming, unerupted permanent teeth. Any pulp-exposed tooth is a highway for infection. At the end of this highway of infection of a fractured deciduous tooth, is the forming, unerupted adult tooth. Proper formation and eruption of the permanent tooth can be affected. The deciduous tooth will indeed eventually fall out, but due to erosion of the root and supporting structures from infection. I consider fractured deciduous teeth to be an urgent problem, and they should be extracted promptly to prevent damage to the underlying permanent tooth (Figures 6 and 7).

All too often, our patients suffer silently with abscessed teeth. As we head into National Pet Dental Month, don't miss the opportunity to spot these teeth and treat them properly or, even better, educate clients on what NOT to give their dogs to chew and prevent the majority of tooth fractures.

For more information, please contact Dr. Rosenblad at **dentistry@angell.org** or **617-524-5643**.

Diagnosis of Multiple Myeloma: Bence-Jones Proteins

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DEFINITION AND PATHOPHYSIOLOGY

Bence-Jones proteins, also referred to as M-proteins, myeloma proteins, paraproteins and free immunoglobulin light chains, are a component of immunoglobulin produced in excess by B cell-derived clonal cell populations. The specific immunoglobulin component is unassociated kappa or lambda light chains. Multiple myeloma, and some extramedullary plasma-cell tumors and chronic B-cell lymphocytic leukemia, can produce Bence-Jones proteinuria. Multiple myeloma is a clonal proliferation of malignant plasma cells, typically arising in the bone marrow, that produce an immunoglobulin or component of immunoglobulin. Because of their small size (MW=22,000-44,000), Bence-Jones proteins can readily pass from the blood through the normal glomerular fenestrations of the kidney into the urine. Bence-Jones proteins were named after the English physician Henry Bence Jones, who described their ability to precipitate when urine is heated to 45-70°C, then redissolve when urine is further heated to near boiling.

CONSIDERATIONS FOR DIAGNOSIS OF MULTIPLE MYELOMA

Two of the four following criteria are generally required for diagnosis of multiple myeloma:

- Radiographic evidence of osteolytic bone lesions
- >20% plasma cells in bone marrow aspirates or biopsy specimens (Figure 1)
- Demonstration of monoclonal or biclonal gammopathy with serum electrophoresis
- Demonstration of Bence-Jones proteinuria

More than 50% of dogs and cats with multiple myeloma may have light-chain proteinuria. A diagnosis of multiple myeloma cannot be excluded based on a negative Bence-Jones protein result for the following reasons: 1) secretion may be intermittent or the concentration too low to be detected in a single urine sample; 2) myeloma cells may be secreting intact immunoglobulin molecules rather than free light chains; and 3) non-secretory types of multiple myeloma (rare) do not produce Bence-Jones proteinuria.

LABORATORY TESTS FOR BENCE-JONES PROTEINS

Bence-Jones proteins are not detectable via urine protein dipsticks. Detection of Bence-Jones proteinuria requires sophisticated tests, such as urine protein electrophoresis, immunoelectrophoresis and immunofixation electrophoresis. The screening test most commonly used is urine protein electrophoresis.

SAMPLE REQUIREMENTS FOR URINE PROTEIN ELECTROPHORESIS

Submit 10ml of urine in a sterile leak-proof container or red-top (serum) tube on ice packs to the testing laboratory. Twenty-four-hour urine collection is ideal but impractical in most practices. Cystocentesis is the preferred collection method, although catheterized samples and clean, free catch are considered acceptable. An early morning urine collection is ideal. Remember to include the method of collection and date/time of collection on the submission form. The urine sample should be kept refrigerated prior to shipping, since the proteins are only stable for approximately two hours at ambient temperature. The sample is stable at refrigerator temperature (2-8°C) for approximately one week or can be stored frozen at -18°C for up to one month.

INTERPRETATION OF TEST RESULTS

A positive test result will appear as a monoclonal spike in the ß or Y protein regions on urine protein electrophoresis. It is important to remember that a negative test result does not exclude a diagnosis of multiple myeloma or B-cell neoplasia, and a positive result requires additional confirmatory testing. Causes of abnormal test results are presented in Table 1.

CONFIRMATORY TESTS

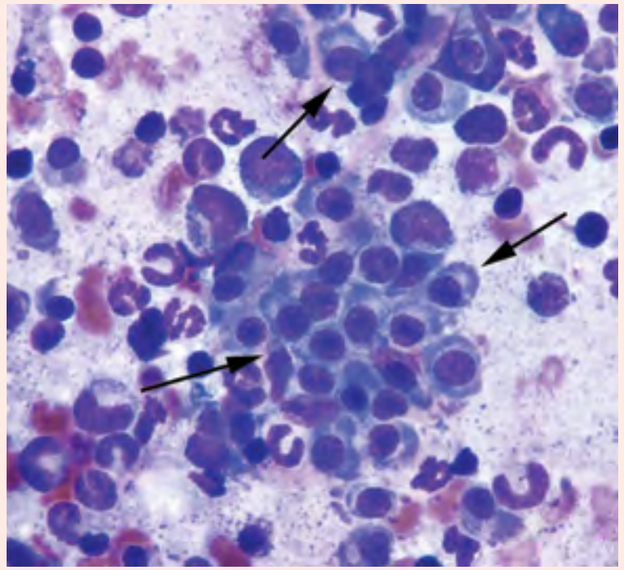
A positive Bence-Jones protein result on urine electrophoresis can be confirmed and further characterized by more sensitive and specific techniques, such as immunoelectrophoresis and immunofixation electrophoresis. Immunofixation electrophoresis distinguishes between kappa and lambda light chains and identifies the heavy chains of IgA, IgM and IgG. The most common problem reported with immunofixation electrophoresis is that it also detects intact immunoglobulins in the urine that are unassociated with Bence-Jones protein. Additional diagnostic tests that may be helpful in confirming a diagnosis of multiple myeloma include serum-protein electrophoresis to evaluate for monoclonal or biclonal gammopathy, radiography to evaluate for osteolytic lesions and bone marrow cytology to evaluate for plasmacytosis (Figure 1).

TABLE 1

POSITIVE RESULT	NEGATIVE RESULT
Multiple myeloma	**Intermittent secretion of light chain**
Extramedullary plasmacytoma	**Light-chain concentration below limit of detection of the assay**
Other B-cell derived neoplasms, such as chronic lymphocytic leukemia	**Non-secretory type of multiple myeloma (rare)**
Chronic lymphoplasmacytic inflammation (rare)	**False Negative:**
False Positive:	• Delay in sample processing • Sample not refrigerated • Bacterial contamination
• Marked proteinuria • ß2-microglobulin interference • Hemoglobin interference from (hemorrhage or hemolysis)	

FIGURE 1

↘ Bone marrow cytology from a 10-year-old dog with multiple myeloma. Note clusters of plasma cells (black arrows) among the hematopoietic precursors. 500x magnification, Wright-Giemsa stain.



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For more information, please contact Angell's Pathology service at **617-541-5041** or **pathology@angell.org**.

2 NEW FUNDED CLINICAL TRIALS AT ANGELL: NOW ENROLLING

Angell Animal Medical Center in Boston is now enrolling dogs for 2 separately funded clinical trials:

ANGELL ONCOLOGY – FUNDED CLINICAL TRIAL FOR T-CELL LYMPHOMA IN DOGS: FREE FLOW CYTOMETRY AND BIOPSY

Prior to enrollment all patients will have samples taken for lymph node biopsy and flow cytometry to confirm T-cell phenotype at no cost to the owner. Once the dog is confirmed eligible (non-indolent, T-cell lymphoma), all procedures and treatments required by the study including chemotherapy, antibody therapy, exam, bloodwork, and other required diagnostics will be fully funded.

To enroll a patient, please call the Angell Oncology service at **617-541-5136** or email **oncology@angell.org** to contact Dr. Ivan Martinez for further directions. To learn more about this study, including eligibility requirements, please visit **angell.org/lymphoma**.

ANGELL INTERNAL MEDICINE – FUNDED CLINICAL STUDY TO INVESTIGATE THE CLINICAL EFFICACY OF A NEW ANTI-DIARRHEA DRUG, CROFELEMER

The drug is an anti-secretory compound that has shown promise in treating diarrhea without affecting intestinal motility. Animals accepted into the study will receive up to $1,000 for all costs of treatment and testing.

To qualify, vaccinated and parvo-free dogs must be between 2 months and 12 years of age, have watery diarrhea without blood or mucus, and cannot be on Metronidazole, Albendazole, probiotics, Animopentamide (Centrine), Sufladimethoxine (Albon), or Flagyl.

To enroll a patient, please call Jen McManus in Angell's Internal Medicine service at **617-541-5188** or email **jmcmanus@angell.org** for further directions. To learn more about this study, please visit **angell.org/crofelemer**.

Please visit **angell.org/studies** to view Angell's other ongoing clinical studies.

Continued from page 1

Laparoscopic Ovariectomy and Gastropexy

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OVE is most commonly performed completely through two small 5mm incisions in the abdomen (one just caudal to the umbilicus and one midway between umbilicus and pubis). A vessel-sealing device is used to achieve hemostasis of the ovarian pedicle and to transect the suspensory ligament and uterine horn at the level of the proper ligament of the ovary. The ovaries are removed through the same small incisions and the abdomen and subcutaneous skin layers closed routinely. The patients are monitored overnight for pain control and discharged the following day.

Certain dogs are at high risk for gastric dilatation and volvulus (GDV). GDV is a highly fatal and costly condition, and most dogs will die without rapid emergency and surgery treatment. Mortality can approach between 15-27%.[7] Some common at-risk breeds for GDV include those with a deep chest, large or giant breeds, such as Great Danes, Standard Poodles, German Shepards, Weimeraners, Akitas, Rottweilers, Irish Setters, etc. Other factors that have been evaluated include dogs with a first-degree relative that has experienced GDV. Gastropexy is a permanent adhesion created surgically between the body wall and the stomach to prevent recurrence.

Gastropexy can be performed via laparoscopic-assisted technique prophylactically in order to prevent life-threatening GDV from occurring. This minimally invasive surgical technique can be performed at any time during a dog's life and is highly recommended to be performed at the same time as a spay or neuter in a predisposed dog. One study showed that none of the dogs that had laparoscopic gastropexy performed developed GDV and all had good attachments visualized with ultrasound at follow-up.[7,8] The overall goal of a laparoscopic gastropexy is that it is a very easy technique to perform, it produces a permanent attachment between the antrum and the right abdominal wall, it does not alter gastric function, and it has minimal complications.[8] Also, this very minimally invasive technique may save a pet's life by preventing GDV, as well as preventing a very expensive and stressful life-threatening event.

Ovariectomy and gastropexy can be performed at any age and are most frequently performed simultaneously. Gastropexy can also be performed at the same time as a routine castration for male dogs and only involves one 5mm mid-abdomen incision and a right-sided 5cm incision in the skin. Another commonly recommended laparoscopic technique is laparoscopic cryptorchidectomy. This can be performed for unilateral or bilateral cryptorchid dogs. Lastly, laparoscopic surgery is a fantastic technique for ovarian remnants. Patients with presumptive ovarian remnants benefit immensely from this surgery because of increased visualization and magnification, as well as the less invasive technique with an excellent prognosis.

Postoperative recovery from laparoscopic surgery has been proven to be faster, less painful and less invasive compared with a routine celiotomy. Patients routinely stay overnight for pain management and monitoring and are discharged the following day. They are rested for about 10-14 days with incision care and then returned to normal routine. They have much smaller incisions and less swelling and pain than traditional surgery patients. At Angell, we strive to deliver the most advanced medical care available to our veterinary patients—the same that would be offered to patients in human medicine. We are fortunate to be able to offer many minimally invasive techniques for our pets, which have been proven to be less painful, invasive and have faster recoveries than more traditional techniques.

FIGURE 1

↘ 6-month-old Standard Poodle Chloe Kelley poses for belly rubs less than 1 day following her laparoscopic gastropexy performed by Drs. Meghan Sullivan, Lisa Benson, and Deandra Dill.

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