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VETERINARY REFERRAL NEWS FROM ANGELL ANIMAL MEDICAL CENTER

SURGERY

Primary Hyperparathyroidism: An Endocrinopathy for Surgeons

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Overview of Primary Hyperparathyroidism

Dogs and cats have two parathyroid glands that are intimately associated with each of their thyroid glands. Generally, the cranial parathyroid gland is present on the external surface of the thyroid gland, and the more caudal parathyroid gland is located within the thyroid parenchyma. These parathyroid glands are responsible for producing parathyroid hormone (PTH), a crucial regulator of calcium levels.

Calcium homeostasis is primarily driven by PTH and calcitonin. PTH is released from Chief cells within the parathyroid glands. It causes a net increase in serum calcium by increasing calcium absorption in the kidneys and gastrointestinal tract, and by freeing calcium from the skeleton. PTH stimulates the kidneys to increase resorption of calcium from urine within the distal convoluted tubule and ascending loop of Henle. In the gastrointestinal tract, PTH stimulates the inactive form of Vitamin D (cholecalciferol) to become active (calcitriol), resulting in increased calcium absorption in the small intestine. In addition to increasing the

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NEUROLOGY

Canine Cognitive Dysfunction (CCD)

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Introduction

Canine cognitive dysfunction (CCD) is a common neurodegenerative disease in geriatric dogs (i.e., older than 8 years old), with an estimated prevalence of 14%–35% in the pet canine population. The risk of CCD exponentially increases with age, such that the odds of developing CCD increase by 52% with each additional year of age. CCD has no known breed disposition, although it is more commonly recognized in smaller breed dogs due to their generally longer life expectancy. CCD shares both a similar clinical course and pathological findings with Alzheimer's disease (AD) in humans, making CCD an ideal model for AD research. Clinical signs of CCD include changes in sleep-wake cycles, disorientation, decreased

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body's retention of calcium from urine and ingesta, PTH also increases osteoclast activity to mobilize calcium stores within bone. In healthy animals, PTH increases serum calcium levels, and calcitonin balances the effect of PTH to prevent hypercalcemia by increasing excretion of calcium from urine, decreasing absorption from the GI tract, and inhibiting osteolysis.

In pets with primary hyperparathyroidism (PHPT), calcium homeostasis becomes dysregulated by excessive production of PTH from one or more parathyroid glands due to hyperplastic or neoplastic changes to the gland(s). The majority of patients are affected by parathyroid adenomas; however, parathyroid hyperplasia and carcinomas are also seen. The gold standard treatment of PHPT is surgical excision of the offending parathyroid gland(s).

Diagnosis

PHPT should be considered as a differential diagnosis for any dog or cat presenting with clinical signs of hypercalcemia, such as polyuria, polydipsia, lethargy, muscle weakness, weight loss, decreased appetite, shaking/tremors, or urinary tract signs associated with calcium-containing urolithiasis. Interestingly, approximately one-third of patients have no clinical signs of disease, and suspicion for the disease is brought on only by detection of hypercalcemia.[1] PHPT is diagnosed via blood work, with or without accompanying cervical imaging. Patients with PHPT will have a high ionized calcium level, accompanied by either a normal or elevated PTH value, and a normal parathyroid hormone-related protein (PTHrP) level. A normal PTH value is considered pathologic when seen in conjunction with hypercalcemia because an elevated calcium value should result in suppression of PTH. Patients with an elevated PTH and low ionized calcium are diagnosed with secondary hyperparathyroidism, which occurs as a result of a calcium-deficient diet or renal disease. Patients with a high PTHrP should be further evaluated for alternative neoplastic causes of hypercalcemia, such as lymphoma, mammary carcinoma, multiple myeloma, and apocrine gland anal sac adenocarcinoma (AGASACA). Although agreement between cervical ultrasound and intraoperative findings regarding the number and side of affected glands is reported to be just 65.9% and 72.3% respectively, it is valuable to confirm the presence of a nodule prior to pursuing surgery, as 3% – 6% of dogs are suspected to have accessory parathyroid tissue within the cranial thorax.[2] CT scans can also be used to confirm the suspicion of one or more parathyroid nodules prior to surgery and are particularly helpful in cases where ectopic parathyroid tissue is suspected. Nuclear scintigraphy has been evaluated for use in identifying parathyroid disease in dogs, but has not proven to be useful.[3]

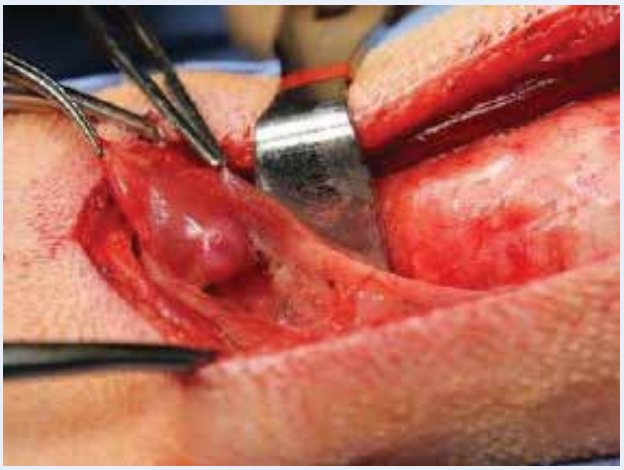
Treatment

Surgical excision of the offending parathyroid tissue is the mainstay of treatment for PHPT, with the majority of patients achieving resolution of hypercalcemia within 48 hours postoperatively.[4] Alternative options for management of parathyroid disease, including percutaneous ultrasound-guided chemical (ethanol) and heat ablation, have been evaluated in a handful of studies; however, outcomes are less successful than those reported with surgical intervention.[5] Medical management with cinacalcet may be an option for the rare dogs who are not surgical candidates or who have refractory hypercalcemia postoperatively; however, evidence is limited, and surgery remains the best option for definitive treatment.[6]

Following surgery, the remaining, or non-pathologic, parathyroid glands are generally atrophied as a result of chronic excessive PTH production by the parathyroid lesion(s). However, once the source of excessive PTH production is removed, the remaining parathyroid glands regain size and function as hypercalcemia resolves. In surgery, normal parathyroid glands cannot be seen or felt, and any nodular changes noted on the thyroid glands should be excised (Image 1, top right).

IMAGE 1

› Intraoperative photo of a parathyroid adenoma on the right internal parathyroid gland of a dog



Prophylactic calcitriol supplementation is recommended by some surgeons as an effort to avoid clinically significant hypocalcemia postoperatively; however, the literature to support this practice is mixed.[4,7,8] Calcitriol has been shown to reach peak serum concentrations within three to six hours, but it takes approximately 48 hours to see an effect on calcium concentrations.[4] Therefore, if clinicians plan to use calcitriol to prevent a rapid drop in calcium postoperatively, the medication should be started approximately two days prior to surgery. Calcitriol is typically initiated at a dose of 20 ng/kg/day and can then be gradually tapered over a period of two to four weeks postoperatively.[4] Unlike unsupplemented patients, it is important to note that patients receiving calcitriol are unlikely to become normocalcemic within 48 hours postoperatively. These patients may experience an increase, no change, or only a mild decrease in calcium within the first 48 hours following surgery; however, they should achieve and maintain normocalcemia as the calcitriol is tapered.

While the need for prophylactic supplementation with calcitriol is questionable, patients who develop clinical signs of hypocalcemia postoperatively must be supplemented. Emergency supplementation with intravenous calcium and electrocardiogram monitoring should be performed in patients who develop seizures, muscle tremors, focal muscle twitching, and facial pawing. Following resolution of clinical signs, the patients should be transitioned to calcitriol and gradually tapered off the medication with serial monitoring of ionized calcium. Most patients ingest enough calcium from well-balanced commercial diets, but supplementation with calcium carbonate (20–50 mg/kg/day) can be considered in patients with postoperative ileus or anorexia. While supplementation is tapered, serial calcium monitoring is essential. The frequency of calcium monitoring depends on the individual patient's calcium status, but it should be checked at least once a week. Meanwhile, owners should diligently monitor for clinical signs of hypocalcemia at home. Clinicians should aim to taper supplements at a pace that maintains serum calcium at the low end of the normal reference range, in an effort to prevent clinical signs of hypocalcemia while concurrently stimulating the function of the atrophied parathyroid glands.

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Summary

PHPT is an important differential for patients with hypercalcemia. The diagnosis is confirmed by an elevated serum calcium with either a normal or elevated PTH and normal PTHrP. Surgical excision of the offending parathyroid lesion typically results in resolution of hypercalcemia within 48 hours.

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↘ Angell Appointment Availability as of January 12, 2026

	Angell in Boston	MSPCA-Angell West in Waltham
Avian & Exotic Medicine	1 day	5 days
Behavior	1 week	1 week
Cardiology	6 weeks 1-2 weeks: Interventional Cardiology Procedure consult	10 weeks: new client 7 weeks: recheck
Dentistry	11 weeks	N/A
Dermatology	6 ½ months	7 months (non-urgent) 1 week (urgent recheck) 2 weeks (recheck)
Grooming & Healthy Boarding *for Angell patients*	1 ½ weeks (Grooming) 1 day (Boarding) *New service for patients who have used non-Emergency/non-Urgent Angell services in the past year*	N/A
Internal Medicine	1 ½ weeks ** IM AUS only available in Waltham. Outpatient AUS in Boston via limited to patients referred by Angell doctors to Diagnostic Imaging service*	3 days: IM consult 2 days: IM outpatient abdominal ultrasound (referred stable patients)
I-131	1 ½ weeks *(Treatment within 2-4 weeks)*	N/A
Neurology	1 day	N/A
Oncology	1 week: Medical Oncology 2 weeks: Surgical Oncology 3 weeks: Radiation Therapy	N/A
Opthalmology	2 weeks	N/A
Surgery	1 week: TPLO consult 1 week: Consult for soft tissue surgery	2 days: Orthopedic consult 3 days: Consult for soft tissue surgery
Urgent Care (Mon.-Fri.)	N/A	Same-day appointments

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Visit **angell.org/availability** to add your email to the list.

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interaction with owners, anxiety, and loss of house-training/learned behaviors. Despite the pervasiveness of CCD in the canine population, treatment options remain limited. Like AD, there is unfortunately no cure for CCD at this time.

Pathophysiology of Disease

The pathophysiology of both CCD and AD is multifaceted and complex, involving cerebrovascular disease, neuritic plaques in the brain parenchyma, and protein aggregation. Beta-amyloid (Aβ) plays a central role in the pathogenesis of CCD. Aβ is a neurotoxic and insoluble protein that forms aggregates or extracellular plaques (neuritic plaques) in the brain around neurons and blood vessels. These plaques affect neuronal signaling and activate glial cells, which, in turn, leads to neuroinflammation and subsequent cell loss and dysfunction. In addition to Aβ aggregates, intraneuronal accumulation of tau protein also occurs, which may contribute to axonal transport dysfunction and neuronal death. Other contributing processes for cognitive impairment observed in CCD include cholinergic dysfunction, neuronal mitochondrial dysfunction, impaired neuronal glucose metabolism, and neuronal hyperexcitability.

Clinical Signs and Diagnosis

Dogs with CCD typically present with a chronic progressive history of forebrain dysfunction, characterized by confusion, anxiety, a change in sleep-wake cycle, and decreased interaction with owners. Owners may report observing pacing at night, hearing loss, and excessive vocalization. Upon examination, abnormal/inappropriate mentation and compulsive circling are commonly observed. Transient vestibular episodes and/or a new history of seizure activity may also be noted. Diagnosis of CCD is a diagnosis of exclusion, typically based on signalment, history, and clinical signs. Other medical conditions that may exacerbate clinical signs should be excluded, namely, hypertension, metabolic disease, pain, and brain tumors. To further aid in CCD diagnosis, veterinarians may use questionnaires for screening and evaluating the severity of the disease. These questionnaires include the Canine Dementia Scale (CADES) and Canine Cognitive Dysfunction Rating Scale (CCDR).

MRI may be used for the diagnosis of CCD; however, it is infrequently performed due to concerns from owners regarding general anesthesia, the cost of the procedure, and the low likelihood that an MRI diagnosis will significantly impact the treatment plan. MRI findings in dogs with CCD are consistent with brain atrophy and include ventricular enlargement, widening of the cerebral sulci, and an interthalamic adhesion thickness of 5 mm or less. Other MRI findings in CCD include cerebral microhemorrhages and leukoaraiosis. Leukoaraiosis is a term used to describe abnormal changes in the white matter of the brain, particularly near the lateral ventricles, and is thought to be secondary to vascular abnormalities. Despite the described MRI findings for CCD, a definitive diagnosis can only be obtained postmortem on histopathology.

Treatment

Though there is no cure for CCD, there are treatment options available to improve the quality of life for the pet and owner by slowing cognitive decline.

Diet and Supplements

1. **Exogenous antioxidants** (i.e., Vitamin B, Vitamin C, Vitamin E) have been used to mitigate oxidative damage. Together with mitochondrial cofactors (i.e., L-carnitine, DL-α-lipoic acid), they have been shown to increase cognitive function in aged dogs, though they are unable to reverse neuron loss.
2. **Docosahexaenoic acid (DHA)** is an omega-3 fatty acid in the brain that plays a role in neuroinflammation, neuroprotection, and synaptic health and is vital for brain function.
3. **Medium chain triglycerides (MCTs)** offer an alternative energy source for the brain in CCD patients who suffer from impaired neuronal glucose metabolism via ketone bodies. They have been shown to improve cognitive function in aged dogs. To obtain the highest dose of MCTs, MCT oil made from coconut oil may be a suitable option.
4. **Commercially available diet options** for CCD include Hill's Prescription Diet b/d and Purina Neurocare.

Pharmaceuticals and Nutraceuticals

1. **L-deprenyl (Selegiline)** is the only FDA-approved treatment for CCD. It is an irreversible monoamine oxidase B inhibitor (MAOI) that is believed to improve cognitive function by restoring dopamine balance, decreasing free radicals, and increasing catecholamine levels. It may take a month to see positive results.
2. **Nicergoline** is an α-adrenergic antagonist that increases blood flow to the brain and has been used to treat cognitive and behavioral disorders in humans, and is used in dogs with CCD.

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3. **Levetiracetam (Keppra)** is an anti-seizure medication that has been shown to improve cognitive function in AD by decreasing neuronal hyperexcitability.
4. **Apoaequorin** is a calcium-buffering protein derived from jellyfish that has demonstrated improvement in cognitive function in dogs by decreasing intracellular calcium dysregulation.
5. **Antidepressant**s (i.e., Fluoxetine, Clomipramine) and **anxiolytics** (i.e., Gabapentin, Pregabalin) may also be used to address clinical signs of anxiety and aggression that may occur as sequelae of CCD. The use of Selegiline with antidepressants should be avoided due to concern for serotonin syndrome.

Cognitive Enrichment

1. **Exercise and social interactions** can improve cognitive function by improving neuronal plasticity and hippocampal atrophy. This may include regular walks, new toys, and even physical rehabilitation.

Chinese Herbs and Acupuncture

1. **Single Chinese herbs and herbal formulas** have been shown to improve cognitive function via several processes, including antioxidant activity, anti-inflammatory activity, improved blood flow to the brain, and decreasing Aβ in the brain. These include Ginkgo biloba (Bai Guo), Ginseng (Ren Shen), Crocus sativus (Zang Hong Hua), among others.
2. **Acupuncture** may be a therapeutic option for CCD based on promising findings in AD. Acupuncture is believed to enhance neuroplasticity and enhance hippocampal activity.

Conclusion

Canine cognitive dysfunction (CCD) is a common disease in veterinary medicine and is likely to become increasingly prevalent as the life expectancy of our pet dogs continues to increase. Further research is warranted to improve early detection and therapeutic intervention. CCD's similarities to AD make it an ideal model for AD. Hopefully, the future will hold more targeted therapies and preventative strategies for both conditions.

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The Most Recent RECOVER Guidelines for Advanced Life Support: Key Updates

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Introduction

The Reassessment Campaign on Veterinary Resuscitation (RECOVER) Initiative is a collaborative effort to improve outcomes for dogs and cats experiencing cardiopulmonary arrest (CPA). Spearheaded in 2010 by a group of veterinary specialists, it focuses on two primary objectives: to devise clinical guidelines on how to best treat CPA in dogs and cats, and to identify knowledge gaps where more research is needed. These knowledge gaps are essential targets for future studies and clinical trials to refine and improve CPR practices in veterinary medicine. The RECOVER committee developed standardized, evidence-based clinical CPR guidelines that were first published in 2012 in the *Journal of Veterinary Emergency and Critical Care,* marking the first consensus-based guidelines for small-animal CPR. A 2024 update was released, reflecting new research, expert opinions, and clinical insights over the past decade. Here we will discuss some of the key changes highlighted in the latest RECOVER Guidelines.

Epinephrine Administration

Historically, high-dose epinephrine (0.1 mg/kg) could be considered after prolonged CPR (i.e., CPR > 10 minutes duration) because of evidence that this dose was associated with a higher rate of return of spontaneous circulation. Despite this evidence, no consistent long-term survival or functional outcome effect was demonstrated. Although high-dose epinephrine has been associated with increased frequency of return of spontaneous circulation (ROSC) in people, it has also been associated with decreased frequency of survival to discharge and with worse neurologic outcomes. The latest recommendation is that high-dose epinephrine should no longer be considered at any time during CPR in dogs and cats. Instead, a standard dosing of 0.01 mg/kg should be administered every three to five minutes.

Atropine During CPR and Dosing Interval

Atropine is recommended to prevent CPA in patients with bradycardia secondary to high vagal tone. The RECOVER 2012 CPR Guidelines also suggest that it can be considered during CPR in dogs and cats with non-shockable arrest rhythms, particularly in animals with high vagal tone as a suspected trigger for arrest.

However, atropine has been removed from human CPR guidelines, and the evidence is primarily supportive of atropine as part of the treatment of severe bradycardia, rather than as part of CPR. The RECOVER committee investigated whether atropine is beneficial in dogs and cats with high vagal tone preceding CPA.

The evidence surrounding the potential benefit of atropine during CPR for patients with non-shockable arrest rhythms is conflicting and extremely limited. Although the majority of studies showed no difference in outcomes in these patients with administration of atropine, one observational study in humans demonstrated an association between atropine administration and reduced likelihood of survival to discharge and one experimental dog study showed a potential benefit. The RECOVER committee found convincing evidence that higher doses of atropine were associated with worse outcomes than placebo control.

The RECOVER Guidelines recommend the use of atropine (0.04 mg/kg IV or IO) in dogs and cats with bradycardia causing hemodynamic compromise to attempt to prevent progression to CPA. They also recommended against administering repeated doses of atropine during CPR for dogs and cats with non-shockable arrest rhythms and that, if given, it should be administered as early as possible.

Defibrillation Protocols

There are two types of defibrillators available. Monophasic defibrillators deliver current in one direction between the paddles and across the patient's chest. In contrast, biphasic defibrillators deliver current in one direction before reversing polarity and delivering a current in the opposing direction. Biphasic defibrillators have been shown to successfully defibrillate patients at a lower energy output, leading to less myocardial damage, and are recommended over monophasic devices. Dosing for monophasic defibrillators begins at 4–6 J/kg, whereas biphasic defibrillation dosing starts at 2–4 J/kg. Multiple studies show improved neurologic outcome, survival to discharge, and ROSC with biphasic defibrillation compared to monophasic. Many experimental studies in pigs and dogs show improved hemodynamics and decreased myocardial injury with biphasic. For patients with shockable rhythms, if a shockable rhythm persists after the initial defibrillation, it's recommended to double the defibrillation energy dose starting with the second shock and maintain this dose for subsequent shocks.

Antiarrhythmic Protocols

Antiarrhythmic drug therapy is an adjunctive treatment for shockable rhythms. Antiarrhythmic medications, such as amiodarone for cats and lidocaine for dogs, should also be considered. Current veterinary and human CPR guidelines suggest that lidocaine may improve outcomes in patients with refractory shockable rhythms that do not respond to initial defibrillation. Further evidence in dogs suggests that lidocaine may increase the defibrillation threshold when a monophasic defibrillator is used. In contrast, a more recent study in pigs indicated that this increase in defibrillation threshold does not occur with biphasic defibrillation.

The latest guidelines recommend that lidocaine be administered to dogs with pulseless ventricular tachycardia or V-fib after an initial shock dose. Amiodarone may be used, if lidocaine is not available. They also recommend against lidocaine in cats and recommend amiodarone instead. The use of intravenous lidocaine in cats is controversial since they have a reported sensitivity to its central nervous and cardiovascular effects.

CPR Drug Dosing for Dogs and Cats

This chart summarizes the doses of drugs that should be used during CPR. As part of CPR preparation, a drug dose chart should be in plain view in the hospital's ready area.

The Most Recent RECOVER Guidelines for Advanced Life Support: Key Updates

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CPR Dosing Chart for Dogs and Cats

RECOVER

	DRUG	DOSE	Weight (kg)	2.5	5	10	15	20	25	30	35	40	45	50
				mL	mL	mL	mL	mL	mL	mL	mL	mL	mL	mL
Arrest	Epinephrine (1:1000; 1mg/mL)	0.01 mg/kg		0.03	0.05	0.1	0.15	0.2	0.25	0.3	0.35	0.4	0.45	0.5
Arrest	Vasopressin (20 U/mL)	0.8 U/kg		0.1	0.2	0.4	0.6	0.8	1	1.2	1.4	1.6	1.8	2
Arrest	Atropine (0.4 - 0.54 mg/mL)	~ 0.05 mg/kg		0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
Anti-Arrhythmic	Amiodarone (50 mg/mL)	5 mg/kg		0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
Anti-Arrhythmic	Lidocaine (20 mg/mL)	2 mg/kg		0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
Anti-Arrhythmic	Esmolol* (10 mg/mL)	0.5 mg/kg		0.13	0.25	0.5	0.75	1	1.3	1.5	1.8	2	2.3	2.5
Reversal	Naloxone (0.4 mg/mL)	0.04 mg/kg		0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
Reversal	Flumazenil (0.1 mg/mL)	0.01 mg/kg		0.25	0.5	1	1.5	2	2.5	3	3.5	4	4.5	5
Reversal	Atipamezole (5 mg/mL)	100 µg/kg		0.06	0.1	0.2	0.3	0.4	0.5	0.6	0.7	0.8	0.9	1
Monophasic Defibrillation	External Defib (J)	4 - 6 J/kg		10 J	20 J	40 J	60 J	80 J	100 J	120 J	140 J	160 J	180 J	200 J
Monophasic Defibrillation	Internal Defib (J)	0.5 - 1 J/kg		2 J	3 J	5 J	8 J	10 J	15 J	15 J	20 J	20	20 J	25 J
			Weight (kg)	2.5	5	10	15	20	25	30	35	40	45	50

*Administer esmolol 0.5 mg/kg IV or IO over 3-5 minutes followed by a CRI at 50 mcg/kg/min

Please note: The above guidelines apply to non-neonatal patients. In August 2025, the RECOVER Initiative released a separate set of guidelines specifically for newborn dogs and cats.

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↘ Grooming and Healthy Boarding Services at Angell

The MSPCA-Angell now offers in-house grooming and healthy boarding for Angell clients. The new space—built alongside our expanded Critical Care Unit on the second floor—offers pets a safe, comfortable environment and owners peace of mind.

Started in 2025, these services provide the exceptional care for which Angell is known. Having a veterinary hospital on-site sets us apart and, while the hospital is separate from these new areas, medical support is available if needed. Healthy Boarding is committed to providing canine and feline guests with enrichment and personalized attention, while grooming is focused on creating a positive wellness experience for pets and their owners.

Clients who have visited non-emergency/non-urgent Angell services in the past year are eligible to book and can take a virtual tour of the facility on angell.org/boarding.

angell.org/boarding | angell.org/grooming | angell.org/referrals | 617-541-5082

Brielle Friedman, Manager of Boarding and Grooming, with Nova (left) and Vina (right).

↘ Stable Fracture Cases Bypass Emergency, Direct to Angell West Surgery

The MSPCA-Angell West Surgery team is now available to see stable fracture cases as emergency add-on appointments in the mornings Monday through Friday. These patients can be booked from their appointments for surgery on the same or following day. This enables patients and clients to be seen directly by the Surgery team, rather than having care facilitated through the Emergency service. Angell West has recently added a Ziehm Solo FD c-arm, which provides intra-operative imaging and allows surgeons to perform minimally invasive approaches to fractures, improving outcome and recovery. Primary Care veterinarians can refer these cases via the online referral form, uploading a case summary and any available radiographs with it.

angell.org/west-surgery

↘ Emergent or Urgent Care at MSPCA-Angell West

24/7 Emergency Care

The Emergency & Critical Care service at the MSPCA-Angell West (Waltham, MA) is available 24/7 for clients whose pets need immediate medical care for life-threatening trauma or disease.

Referring veterinarians may alert staff to an incoming case by calling **781-902-8400 | angell.org/emergency.**

Same-Day Urgent Care Appointments

For non-emergent cases, the Urgent Care service at Angell West offers same-day appointments for dogs and cats. They are available Monday through Friday, 8am–6pm by calling **781-902-8400**. Urgent Care appointments are also available through the Angell West Avian and Exotic service by calling **617-989-1561 | angell.org/urgent.**

↘ Outpatient Ultrasound

Angell West (Waltham) provides outpatient abdominal ultrasounds for stable dogs and cats. A referral form, available at **angell.org/ultrasound,** is required before scheduling.

This service is not for sick patients needing hospital admission. If we identify urgent issues (e.g., intestinal obstruction, free air, or hemorrhagic effusion) during the ultrasound, we will consult with you and expedite emergency admission if requested.

Fine-needle aspirates or cystocentesis are not performed during outpatient ultrasounds. For these procedures, please refer the patient for an Internal Medicine appointment, where an estimate and same-day scheduling are available.

For anxious patients, pre-visit oral sedation should be prescribed by the referring veterinarian; injectable sedation requires referral to the Emergency or Internal Medicine service.

The service has a 60-lbs weight limit for dogs due to the need for sedation in larger dogs. Dogs exceeding this limit should be referred for an Internal Medicine appointment, where sedated ultrasounds can often be performed on the same day.

angell.org/ultrasound

Radiographic Evaluation of the Lower Urinary Tract

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Lower urinary tract disease is a common presentation for dogs and cats in all areas of companion animal care. Common causes of lower urinary tract disease include urolithiasis, urinary tract infection, or neoplasia. This article will focus on radiographic evaluation of the lower urinary tract — urinary bladder, urethra, canine prostate — especially as commonly encountered in a general practice setting. This article will not cover positive or negative contrast urinary tract procedures; these procedures are more appropriately performed in an emergency or specialty setting with videofluoroscopy.

Normal Anatomy

The urinary bladder can exhibit a wide range of sizes in normal patients. When empty, the bladder is often not visible at all, and when distended, it can occupy nearly the entire caudal abdominal volume. There is no defined upper limit to the size of a normal urinary bladder, although it is rare for the bladder to extend cranially to the umbilicus. In dogs, the bladder can extend slightly into the pelvic canal, while in cats, it is usually entirely abdominal in location.

In intact male dogs, the prostate gland is usually visible as a smooth, round soft tissue opacity at the pelvic inlet. A normal prostate is generally less than 70% of the height of the pelvic inlet (defined as a line drawn between the cranioventral tip of the sacrum and the pubic brim).[1] In neutered male dogs, the prostate is typically not visible radiographically.

The urethra is typically not visible radiographically but extends directly caudal from the bladder through the pelvic canal. In cats and female dogs, the urethra terminates caudal to the pelvis in the perineal region, while in male dogs, the urethra courses ventrally after emerging from the pelvic outlet, extending through the penis ventral to the os penis (Figure 1).

FIGURE 1

› Lateral radiograph of an intact male dog with an indwelling urinary catheter, delineating the course of the normal urethra (magenta arrowheads) as well as a normal-sized prostate gland (green arrows). The patient also has numerous calculi in the bladder and urethra.

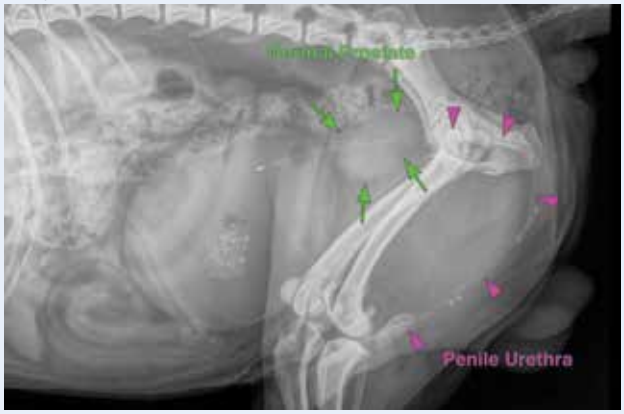
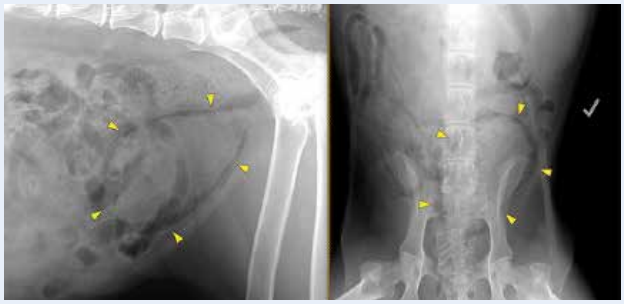


FIGURE 2

› Lateral and ventrodorsal radiographs of a diabetic patient with emphysematous cystitis. Note the irregular peripheral gas lucencies associated with the bladder, indicating intramural emphysema secondary to infection with gas-producing bacteria (yellow arrowheads).



Bladder Abnormalities

The most common bladder abnormalities encountered in canine and feline patients are cystitis, urocystolithiasis, or neoplasia. Cystitis could be infectious or sterile/idiopathic, but generally, there is no radiographically detectable sign of cystitis. One exception is emphysematous cystitis, which is most commonly seen in patients with diabetes mellitus, where glucosuria predisposes to infection with anaerobic gas-producing bacteria. As the infection is often found within the bladder wall, radiographically, there can be an irregular gas rim associated with the bladder (Figure 2).

Classically, urinary calculi have been divided into those that are of a mineral composition and therefore radiopaque, versus those that are non-mineral and therefore radiolucent. The radiopaque composition calculi are calcium oxalate, calcium phosphate, or struvite, while the radiolucent stones are urate or cystine. A few recent studies have shown, however, that on modern digital radiography systems, urate and cystine stones are often detectable. One study in an in vitro

FIGURE 3

› Lateral radiographs without (A) and with (B) lateral compression of the bladder with a wooden paddle in a patient with granular mineral cystic calculi (yellow arrowheads). While the mineral calculi are visible at the center of the bladder without compression, there are multiple intestinal segments also superimposed on the bladder. Paddle compression displaces the intestinal segments away from the bladder, allowing for definitive identification of calculi within the bladder (unfortunately, the paddle we use at Angell is somewhat old and dirty, resulting in some additional mineral opaque streaking).

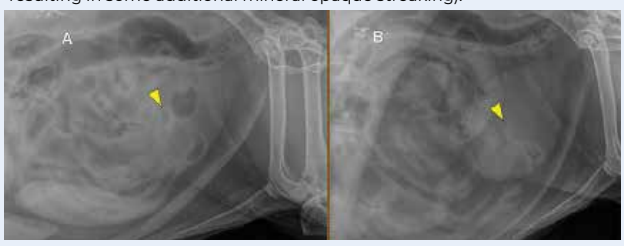


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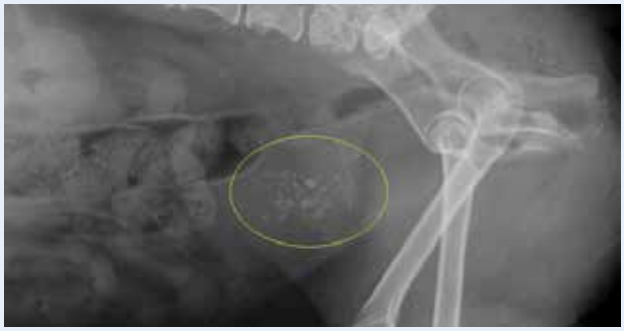
FIGURE 4

› Lateral radiograph in a patient with a large mineralized bladder transitional cell carcinoma (yellow circle). Note the irregular shapes of the mineral opacities, which are more typical of dystrophic mineralization rather than urolithiasis. Ultrasound or CT is generally necessary to differentiate a mineralized mass from urolithiasis, however.

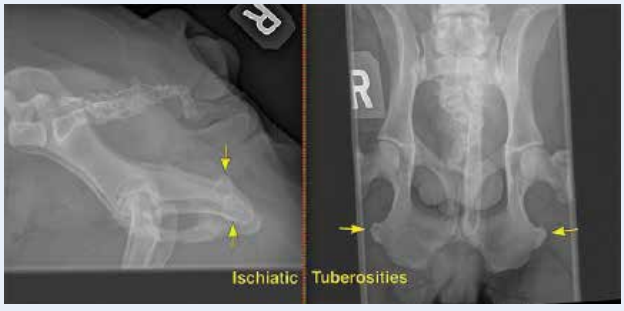


setting using phantoms found that nearly all urate and cystine stones larger than 1 mm are visible radiographically.[2] A second study in a clinical setting found that 85% of urate and 92% of cystine stones were visible on radiographs before stone removal.[3]

Bladder calculi can range from amorphous sediment to tiny sand-like foci to variably spiculated or smooth stones, which occur singly or in clusters. Care should be taken not to over-interpret the presence of mineral calculi in the urinary bladder if there are also intestinal segments (especially the colon) superimposed on the bladder. A useful technique for isolating the bladder during a lateral radiographic exposure is to apply compression over the bladder using a radiolucent wood or plastic paddle (the hand holding the paddle should be in a leaded glove, of course). The compression will push intestinal segments away from the bladder, and the mineral calculi should be visible through the radiolucent paddle (Figure 3, on page 10). The overwhelming majority of cancerous bladder lesions are transitional cell carcinoma (TCC). While squamous cell carcinoma, adenocarcinoma, and various soft tissue sarcomas are also reported, in my experience, these are vanishingly rare. TCC most commonly arises from the region of the trigone, along the dorsal aspect of the bladder neck. However, it can arise from anywhere along the mucosal surface of the lower urinary tract. Mineralization significantly raises the degree of concern for malignancy. Radiographically, most bladder mass lesions are not detectable unless they are mineralized, since fluid and soft tissue have the same opacity (Figure 4).

FIGURE 5

› Lateral and ventrodorsal radiographs in a female dog. On lateral views, the ischiatic tuberosities (yellow arrows) create distinct round mineral opacities that superimpose with the course of the urethra.



Urethral Abnormalities

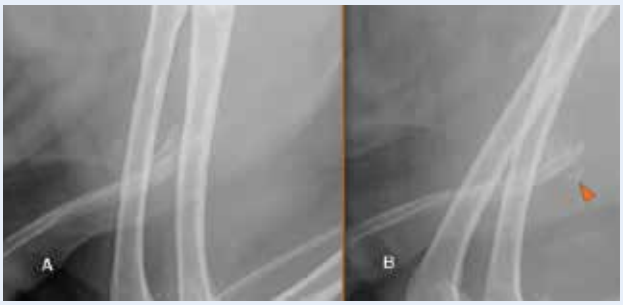
Similar to the bladder, the most commonly encountered abnormalities of the urethra include urethrolithiasis, urethritis, and urethral neoplasia. Generally speaking, urethritis and urethral neoplasia will not be detectable on plain radiographs. While urethral TCC does occur, clinical signs of stranguria would typically begin long before the mass is large enough to exhibit mineralization.

FIGURE 6

› Lateral radiographs of a patient with pelvic limbs neutral (A) and with one leg raised/abducted (B). A round mineral calculus is present in the bladder, visible on both radiographs (green arrowheads). A smaller round mineral calculus is present in the distal pelvic urethra, visible only with the leg raised (magenta arrow).

FIGURE 7

› Lateral radiographs of a male dog with the pelvic limbs neutral (A) and flexed (B). An oblong mineral calculus in the penile urethra is obscured by the femurs on the neutral view but revealed with the femurs positioned more cranially (orange arrowhead).



Urethral calculi can be found along any location of the urethra, from the proximal portion cranial to the pubis, to the intrapelvic portion of the urethra, to the more distal part of the urethra caudal to the pelvis in cats and female dogs, and along the entire length of the penile urethra in male dogs. In dogs, it is important not to mistake the ischiatic tuberosities of the pelvis for large urethral calculi on lateral projections (Figure 5). It can also be helpful to obtain a lateral radiograph with one leg raised/abducted to reduce superimposition in the pelvic canal (Figure 6). In male dogs, the most common location for urethral calculi is at the level of the proximal portion of the os penis, as the os penis creates a relative resistance to urine flow. It is usually recommended to obtain lateral views of the penile urethra in male dogs with the pelvic limbs at different degrees of flexion to ensure urethral calculi are not being obscured by the femurs or stifles (Figure 7). Sesamoids in the stifle region (fabellae and popliteal sesamoids) can also be superimposed with the urethra, perfectly mimicking calculi. Having at least two radiographs with the legs in different positions ensures that sesamoids are not misinterpreted as urethral calculi (Figure 8).

Prostatic Abnormalities

Although the prostate gland is not technically part of the urinary system, it completely encircles the proximal urethra in male dogs. It is therefore often

CONTINUED FROM PAGE 10

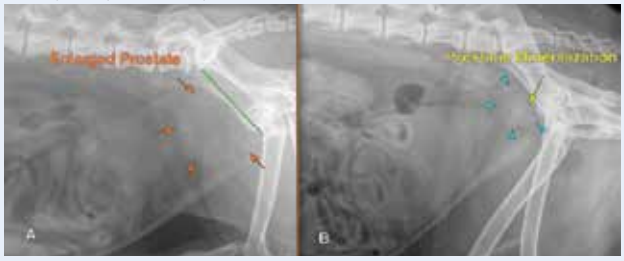
FIGURE 8

Lateral radiographs of a male dog with the pelvic limbs flexed (A) and neutral (B). With the legs bent, the stifles are superimposed with the penis, and a fabella is perfectly positioned to mimic a urolith in the penile urethra (yellow arrow). Note that this patient also has several cystic calculi, some of which are of a similar size and shape to the fabella (blue circles).

implicated in lower urinary tract abnormalities. As noted above, a normal prostate is generally considered to be less than 70% the height of the pelvic inlet. It should be noted, however, that the prostate gland normally increases in size with age in intact male dogs, and there is no strict pathologic delineation between a normal prostate and benign prostatic hyperplasia (BPH). It should be presumed that all intact males will develop BPH given enough time, and prostatomegaly itself should not be viewed as pathologic unless correlated with clinical symptoms. Generally speaking, there is no radiographic way to differentiate BPH from prostatitis or prostatic abscessation (Figure 9).

FIGURE 9

(A) Lateral radiograph of an intact male dog with prostatomegaly. There is a smooth, round soft tissue opacity at the pelvic inlet (orange arrows), which measures approximately 85% of the height of the pelvic inlet (green line). This patient has benign prostatic hyperplasia. (B) Lateral radiograph of a castrated male dog with an enlarged prostate (blue arrowheads) that exhibits dystrophic mineralization (yellow arrow). This patient has prostatic transitional cell carcinoma.



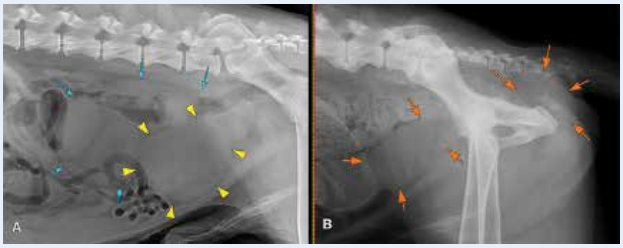
Neutered male dogs have a higher risk of prostatic neoplasia than intact male dogs. The most common forms of prostatic neoplasia are TCC or prostatic adenocarcinoma. The prostate gland is typically not visible at all in neutered male dogs; anecdotally, they usually measure between 1 and 2 cm in thickness. Any rounded soft tissue structure in the location of the prostate in a neutered male dog should raise some concern for prostatic neoplasia. Similar to bladder masses, mineralization of the prostate raises significant concern for cancer, with one study showing 100% positive predictive value for malignancy in mineralized prostate lesions in neutered males (Figure 9).[4]

Prostatic Cysts

In intact male dogs, prostatic cysts can develop either within the parenchyma or adjacent to the parenchyma. Intraparenchymal cysts, previously called retention cysts, are thought to arise from obstruction of prostatic ducts due to BPH, resulting in the accumulation of prostatic secretions. Extraparenchymal cysts, previously called paraprostatic cysts, are classically believed to be formed from Müllerian duct remnants, although this theory remains unproven. Intraparenchymal prostatic cysts are generally not detectable radiographically.

FIGURE 10

Lateral radiographs in two male dogs with extraparenchymal (a.k.a. paraprostatic) cysts. (A) This patient exhibits large (blue arrows) and small (yellow arrowheads) ovoid soft tissue opacities in the caudal abdomen, creating the impression of two urinary bladders. Note that without mineralization of the cyst wall, it is not possible to differentiate radiographically between the bladder and the prostatic cyst. (B) This patient exhibits a large extraparenchymal prostatic cyst, which has faintly mineralized walls (orange arrows). Note that extraparenchymal prostatic cysts can extend caudally through the pelvic canal, causing symptoms of straining to defecate and/or perineal swelling.



Extraparenchymal prostatic cysts can be pretty small and subclinical, but they can also enlarge to quite a dramatic size (Figure 10). Radiographically, extraparenchymal cysts can resemble a moderately full urinary bladder, thus giving the radiographic impression of two urinary bladders. The walls of extraparenchymal prostatic cysts can also exhibit thin mineralization. The cysts are usually located cranial to the prostate, but they can also project caudal to the prostate into the perineal region. Clinical signs can therefore include fecal retention. Complete surgical removal of extraparenchymal cysts +/- castration is the treatment of choice.

Summary

Radiographic evaluation of the lower urinary tract remains a core competency for veterinarians in modern companion animal primary care practice. Detection of radiopaque urolithiasis is shown to be improved in modern digital radiography systems, with up to 85–100% of urate and cystine stones—previously thought to be radiolucent—being visible on plain radiographs. Radiographs of the urethra with the legs in different positions may be necessary for accurate detection of calculi. Mineralized lesions in the bladder wall and prostate (especially in neutered male dogs) raise significant concern for malignancy and warrant further investigation. Extraparenchymal prostatic cysts in male dogs can present with lower urinary symptoms or signs of constipation, and surgical treatment is preferred.

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Managing E. cuniculi in Rabbits: From Diagnosis to Supportive Care

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Encephalitozoon cuniculi is a spore-forming, single-celled parasite, most closely related to fungi. In the United States, an estimated 25%–80% of the rabbit population is seropositive, and antibodies have been found in dogs, cats, birds, non-human primates, and humans. The parasite has a predilection for the central nervous system, kidneys, and ocular tissue, but it can also be found in the liver, lungs, and heart. The parasite can infect non-human primates and immunocompromised humans, causing disease in them and rabbits.

The parasite replicates within mammalian cells until the cells rupture, and the infective spore is shed into the environment mainly through urine, but also in feces and respiratory secretions. Rabbits can shed the parasite for one to three months after initial infection, then intermittently throughout life. Vertical transmission has also been reported. *E. cuniculi* can survive up to four weeks on dry surfaces, though both 0.1% bleach (10-minute contact time) and 70% ethanol (30-second contact time) kill it effectively.

Clinical Signs

Clinical signs of *E. cuniculi* are varied and depend on which body system(s) the parasite affects. There are four major manifestations that we will discuss below.

Neurologic

The neurological form of *E. cuniculi* is likely the most "classical" presentation of the disease. Clinical signs often include ataxia, torticollis, head tilt, and circling. Hind limb paresis and stroke-like events have also been reported. An inability to stand or lack of appetite have been linked with a worse prognosis, but a down rabbit can still recover. So, while it is important to relay the prognosis to owners, it is still reasonable to try treating these cases. Some neurological defects can be permanent, but many rabbits recover well or learn to compensate for these deficits.

Renal

E. cuniculi can cause both acute and chronic kidney injury. Many rabbits with this manifestation present signs similar to a urinary tract infection, including declining litter box habits, urine scald, and urinary incontinence. Bladder atony can also be seen, but this is usually secondary to spinal cord disease rather than primary kidney infection.

Even severe azotemia can respond very well to treatment in cases of acute kidney injury, so don't necessarily recommend euthanasia based on initial values. A more important indicator is if there are changes in values, in response to treatment. Improvement or resolution during and after treatment generally carries a good prognosis.

Gastrointestinal

E. cuniculi is commonly overlooked as a potential cause of rabbit gastrointestinal syndrome (GI stasis). The parasite has been shown to affect both stomach and intestinal motility secondary to spinal cord disease and neuropathy associated with GI innervation. Any rabbit with recurrent GI stasis should be tested for E. cuniculi to rule it out as a contributing factor.

Ocular

The globe is another common site of E. cuniculi infection. The parasite invades ocular tissue and can cause both uveitis and intraocular/lens masses, often white or yellow. *E. cuniculi* cataracts, caused by an influx of white blood cells into the lens, often look "chunkier" than typical cataracts and are usually unilateral. However, bilateral cases can occur and appear at different times, rather than concurrently. Once a cataract develops, changes cannot be reversed, but vision loss is usually tolerated well in rabbits.

Diagnosis

Several diagnostic tests exist for *E. cuniculi.* The University of Miami's serology panel is the gold standard and has a high detection rate.[1] The panel includes IgG and IgM titers and C-reactive protein (CRP) levels.

IgM is produced in early infection, and for *E. cuniculi,* is detectable for four to seven days. Elevations are frequently seen in initial infections, but not necessarily in subsequent flare-ups after seroconversion occurs, meaning that a normal IgM titer does not mean that a rabbit is *E. cuniculi* negative. The body begins to produce IgG antibodies after seven to 14 days from the initial infection and can continue to produce IgG antibodies for months to years. This is the most commonly elevated value seen in positive cases. CRP is an acute-phase protein in

FIGURE 1

› Typical torticollis/neurological presentation of a rabbit with E. cuniculi.

Managing E. cuniculi in Rabbits: From Diagnosis to Supportive Care

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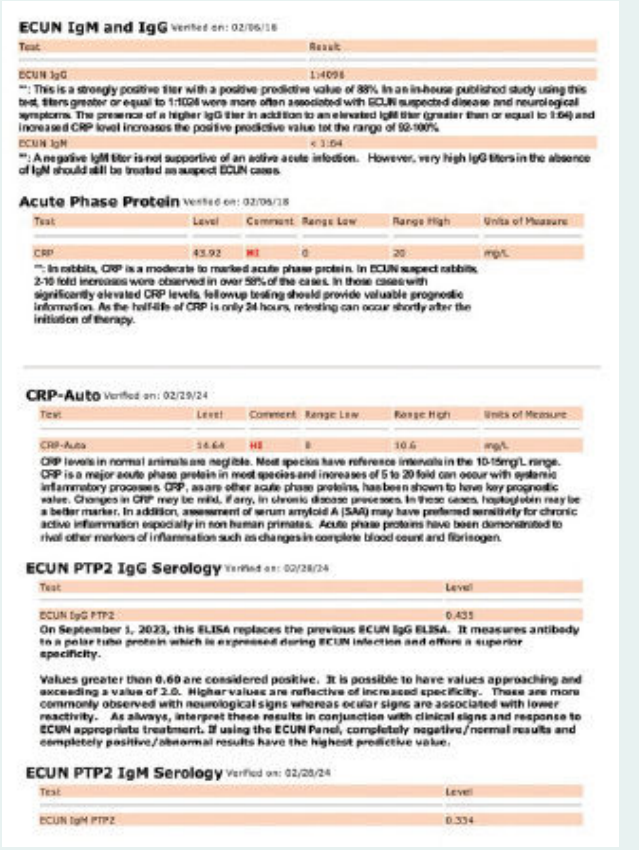
rabbits and can be a good indicator of an active flare-up. However, CRP can be elevated due to any acute inflammatory event, and elevated CRP values are seen in almost every GI stasis case.

PCR testing for urine, cerebrospinal fluid (CSF), and lens material also exists. Urine and CSF PCR are unreliable with high rates of false negatives due to intermittent shedding of the parasite in these fluids. Lens PCR is reliable in ocular cases, but is not a common tissue submitted in clinical practice.

FIGURE 2

› Positive (top) versus Negative (bottom) E. cuniculi titers. Note the elevated CRP levels in the negative case.



ECUN IgM and IgG Verified on: 02/06/18

Test	Result
ECUN IgG	1:4096

**: This is a strongly positive titer with a positive predictive value of 88%. In an in-house published study using this test, titers greater or equal to 1:1024 were more often associated with ECUN suspected disease and neurological symptoms. The presence of a higher IgG titer in addition to an elevated IgM titer (greater than or equal to 1:64) and increased CRP level increases the positive predictive value tot the range of 92-100%.

ECUN IgM	< 1:64

**: A negative IgM titer is not supportive of an active acute infection. However, very high IgG titers in the absence of IgM should still be treated as suspect ECUN cases.

Acute Phase Protein Verified on: 02/06/18

Test	Level	Comment	Range Low	Range High	Units of Measure
CRP	43.92	H1	0	20	mg/L

**: In rabbits, CRP is a moderate to marked acute phase protein. In ECUN suspect rabbits, 2-10 fold increases were observed in over 58% of the cases. In those cases with significantly elevated CRP levels, followup testing should provide valuable prognostic information. As the half-life of CRP is only 24 hours, retesting can occur shortly after the initiation of therapy.

CRP-Auto Verified on: 02/29/24

Test	Level	Comment	Range Low	Range High	Units of Measure
CRP-Auto	54.64	H2	0	10.6	mg/L

CRP levels in normal animals are negligible. Most species have reference intervals in the 10-15mg/L range. CRP is a major acute phase protein in most species and increases of 5 to 20 fold can occur with systemic inflammatory processes. CRP, as are other acute phase proteins, has been shown to have key prognostic value. Changes in CRP may be mild, if any, in chronic disease processes. In these cases, haptoglobin may be a better marker. In addition, assessment of serum amyloid A (SAA) may have preferred sensitivity for chronic active inflammation especially in non human primates. Acute phase proteins have been demonstrated to rival other markers of inflammation such as changes in complete blood count and fibrinogen.

ECUN PTP2 IgG Serology Verified on: 02/28/24

Test	Level
ECUN IgG PTP2	0.435

On September 1, 2023, this ELISA replaces the previous ECUN IgG ELISA. It measures antibody to a polar tube protein which is expressed during ECUN infection and offers a superior specificity.

Values greater than 0.60 are considered positive. It is possible to have values approaching and exceeding a value of 2.0. Higher values are reflective of increased specificity. These are more commonly observed with neurological signs whereas ocular signs are associated with lower reactivity. As always, interpret these results in conjunction with clinical signs and response to ECUN appropriate treatment. If using the ECUN Panel, completely negative/normal results and completely positive/abnormal results have the highest predictive value.

ECUN PTP2 IgM Serology Verified on: 02/28/24

Test	Level
ECUN IgM PTP2	0.334

Treatment

While a small number of rabbits are thought to clear the infection, the majority are considered to remain chronically infected. Treatment is focused on controlling active infection/flare-ups until the immune system can wall it off. Chronic, uninterrupted treatment is generally not recommended, as it increases both the risk of resistance and the risk of bone marrow suppression in rabbits.

Benzimidazoles are the cornerstone of treatment for active flare-ups of *E. cuniculi*. Their mechanism of action against the parasite is still not entirely understood, but theorized to involve potential inhibition of beta tubulins, a key component of the eukaryotic cytoskeleton. There is a higher risk of side effects due to the mechanism, and rabbits seem to be more sensitive to side effects than other species. Reversible bone marrow suppression is the most common side effect of treatment with benzimidazoles, and a CBC should be checked before and then every two weeks during treatment. If heteropenia or leukopenia is found, treatment should be discontinued. While rare, hepatotoxicity and GI upset are also potential side effects.

Fenbendazole, oxibendazole, albendazole, and thiabendazole have all been used to treat *E. cuniculi* in rabbits, and there is still debate over which to use. Research shows that albendazole and fenbendazole reduce *E. cuniculi* loads, while less research supports the use of oxibendazole and thiabendazole. Fenbendazole, though, has been shown to have higher rates of bone marrow suppression and teratogenicity.[2] It is important to choose a benzimidazole with which the clinician feels comfortable and understands the risks and benefits.

In addition to treating the *E. cuniculi*, the clinician should offer supportive care for signs and secondary issues, like GI stasis. Meloxicam helps decrease inflammation from both the disease itself and inflammation secondary to the die-off of organisms. It should not be given in cases of acute or chronic kidney injury. Meclizine is an antihistamine used to treat vertigo in humans, and anecdotal evidence suggests that it can help in vestibular cases of the disease. Cerenia or metoclopramide can be helpful in their anti-nausea effects. Subcutaneous fluids, pain management, and nutritional support should be remembered in GI stasis cases, and padding/bumpers with wickaway material for bedding can help support down rabbits with severe torticollis.

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Jaw Fractures: A Dentist's Point of View

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Introduction

Dogs and cats often present for dentistry services due to routine periodontal disease, endodontic infection, and tooth resorption. However, another cause for presentation can include jaw fractures. The most common cause of jaw fractures is trauma, such as bite injuries, being hit by a car, blunt force trauma, or falls. Jaw fractures can also occur secondary to iatrogenic reasons (surgical complications +/- pathology involved) or primary pathologic causes due to tumor or severe periodontal bone loss.

For bone fracture healing to occur, the area must undergo a similar process to soft tissue healing with phases of inflammation, removal of organic debris, cellular proliferation, and formation of granulation tissue. In the presence of necrotic bone, osteogenic precursor cells must transform the granulation tissue into callus and eventually into bone. There are two types of healing for bone fractures: either indirect or direct bone healing. In indirect healing, mechanical instability of the fracture, resorption of the fracture ends, and callus formation occur. In contrast, with direct bone healing, there is close apposition of the fracture segments and mechanical stability, which allows the fracture ends to make direct contact and bridging of the bone.

FIGURE 1

› Three-dimensional image produced from a CT scan demonstrating a favorable fracture of the right caudal mandible in a canine patient. This fracture was a result of a dog bite injury.

Further breakdown of direct bone healing can be categorized into contact healing and gap healing. Direct bone healing occurs when there is anatomic reduction and absolute rigid fixation. In this situation, bone is the only type of connective tissue to form between the fracture segments during healing. Here, there is quicker function and none of the potential consequences that can occur with large callus formation.

Indirect bone healing begins with the inflammatory phase of fracture repair, which occurs as a result of ruptured blood vessels and a torn periosteum. Ischemic necrosis of the bone ends occurs a few millimeters from the ends

FIGURE 2

› Same patient as Figure 1. Three-dimensional image produced from a CT scan. Lingual view of fracture.

of the fracture segments. Osteogenic cells will be activated, and a vascular response will begin. Fibroblast proliferation occurs in the blood clot, and a cellular infiltrate with neutrophils and macrophages accumulates. The debris in the fracture region will be removed by these cells. Additional fibroblasts, chondroblasts, osteoblasts, and osteoclasts will result from differentiation. The proliferating cells then form a callus, a fibrous matrix of collagen that bridges the fracture segments. Granulation tissue replaces the initial hematoma, and during the reparative phase, the callus matures. Fibrocartilage will form and eventually undergo mineralization. Endochondral ossification

FIGURE 3

› Same patient as Figures 1 and 2. Three-dimensional image produced from a CT scan. Ventral view.

DENTISTRY

Jaw Fractures: A Dentist's Point of View

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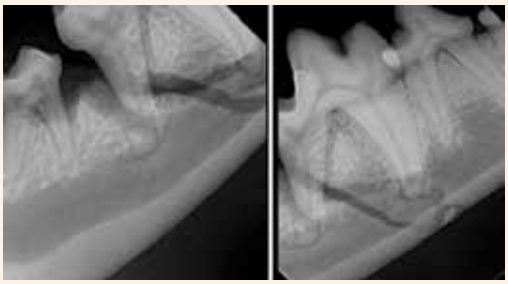
FIGURE 4

› Same patient as Figures 1, 2, and 3. Dental radiographs of mandibular fracture at the level of the mandibular first molar (409). The injury involves a fracture of the distal root of the tooth as well as a disruption of the bone at the apical region of the mesial root. There is a periodontal disease associated with the second molar (410) unrelated to the injury.



will then replace this with bone, resulting in a healed fracture. For both fracture types, an increase in strength will occur as the bone remodels and returns to its original shape. Woven bone is replaced with lamellar bone in this process.

Although the mandible does not have a medullary canal, it heals similarly to other long bones. The bones of the maxilla and face, however, have a thin lamina and greater bone surface area per unit volume exposed to soft tissues. The facial bones thus have increased proximity to the vascular supply of the soft tissues, which is beneficial for rapid bone healing. A major difference in healing between maxillofacial fracture healing versus long bones is the presence of teeth and the mandibular canal. The teeth make fixation more challenging, as they occupy a large volume of the mandible and can create difficulty with applying rigid stabilization along the tension-band side of the bone, where biomechanics dictate the preferred placement of implants. The dorsal 2/3 of the mandible is occupied by the tooth roots, and the ventral 1/3 houses the mandibular canal. Additionally, in comparison to humans, the dorsoventral mandibular height is drastically more limited. Our pet patients have a larger tooth root volume compared to humans, which limits the surfaces available for implant placement.

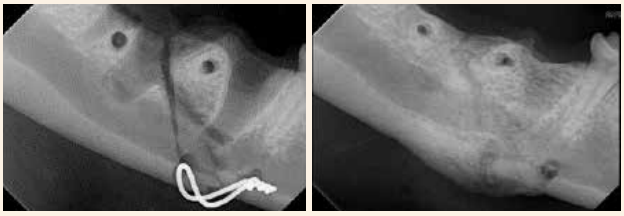
One of the primary surgical goals for repairing maxillofacial and mandibular fractures in patients is to restore proper dental occlusion. In restoring normal occlusion, this subsequently ensures appropriate fracture reduction. If this is not achieved, the resulting malocclusion can adversely affect function and can also cause undesirable leverage forces against the fixation device.

When considering mandibular fractures, there are two general categories: favorable and unfavorable fractures. This is based on the direction of the fracture line, where favorable fractures run from dorsocaudal to ventrocranial. Conversely, unfavorable fractures progress from dorsocranial to ventrocaudal. The concept is derived from the forces the muscles of mastication apply to the mandible. The masseter, temporal, and medial pterygoid muscles are responsible for closing the mouth in the normal situation. They will also "lift" the caudal mandible, where the muscles insert on the body of the mandible and ramus. On the other hand, the small digastricus muscle, with its insertion on the angle of the mandible, is responsible for opening the mouth. Thus, in favorable fractures, the muscles of mastication compress the fracture segments together and "assist" with bony healing. In unfavorable fractures, the masticatory muscles cause distraction of the fracture segments.

Maxillary fractures may not require stabilization because, in the maxilla, forces exerted on the upper jaw are different compared to the mandible, and the maxilla is subject to less strain. There is a "frame" or "box" structure that provides anatomic buttresses and distributes masticatory forces to the

FIGURE 5

› Same patient as Figures 1, 2, 3, and 4. Figure A shows the intraoperative placement of an intraosseous wire. Pilot holes have been created for the dorsal wire, aiming for wire positioning that is approximately perpendicular to the fracture line. The ventral wire is in a "figure 8" on the ventral aspect of the mandible. Figure B is two months after fracture repair, and intraosseous wires are removed. Union between the bone segments is present, along with callus formation along the ventral mandible.



head. Maxillary fractures may not require stabilization unless the buttresses are disrupted.

If fractures are not properly fixed, maxillofacial fractures can result in cavitation, associated soft tissue complications, delayed or non-union healing, and sequestrum formation, in addition to contracture of fibrous connective tissue, which can lead to deformities. Restoring normal anatomy can alleviate pain and discomfort and permit more rapid healing.

FIGURE 6

› Intraoral view of an anesthetized feline patient that suffered unwitnessed trauma (fall or crushing injury). Note soft tissue trauma in the symphyseal region and bruising lingual to the left mandible.

Methods for Fracture Repair

Fracture repair can involve various techniques. For simple separation of the mandibular symphysis or parasymphyseal fractures, cerclage wire placed around the rostral mandible caudal to the canine teeth is often the simplest method of stabilization. Another technique that can be performed is to place an interdental wire and composite resin as a splint spanning from one canine tooth to the contralateral side.

Intraoral Splints

For other fractures in the body of the mandible or involving the maxilla, interdental wiring and composite resin splints are often the least invasive fracture repair technique, as they do not require exposure of the bone or disruption to the periosteum. They can allow normal function and masticatory forces after placement. This technique can involve either forms of cerclage wiring or interdental wiring, along with self-hardening composite resin, which can be bonded to the teeth. The composite resin adds additional strength to the splint and covers any sharp wire twists.

Interdental wire can be placed with several patterns, which are indicated by fracture location and operator preference. The wire is anchored around and

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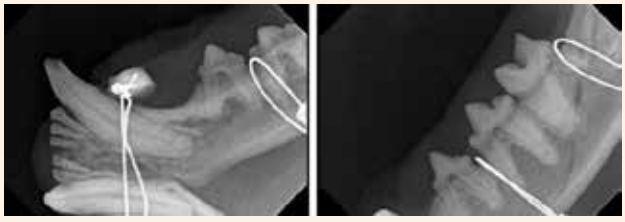
FIGURE 7

› Same patient as Figure 6. Dental radiograph of the left mandible with a favorable fracture. Fracture has disrupted the bone around both apices of the molar tooth (309).

between the crowns of the teeth, utilizing the teeth as anchor points and allowing for fracture fragment alignment and fixation. Following the placement of the wire, a portion of the tooth surface is treated with an acid etchant and a bonding agent to ensure that the composite material adheres. Some materials can be used without the need for bonding agents, and familiarity with the products being used is crucial. This combination of integrating interdental wiring and a composite splint increases the strength of the splint, similar to how rebar reinforces a concrete structure, helping it withstand bending, cracking, or other forces. The use of both interdental wiring and composite splinting has been shown to be more effective than either method alone.

FIGURE 8

› Same patient as Figures 6 and 7. Demonstrating fixation techniques for mandibular fracture and symphyseal separation. Two cerclage wires were placed around the left mandible along the oblique fracture. An intraoral technique for symphyseal wiring was performed with a wire twist located intraorally distal to the left mandibular canine tooth (304). A bead of composite was placed over the wire twists to prevent trauma to soft tissues.



Once fracture healing has occurred, the splint is removed by sectioning the composite resin, transecting the wire, and removing the splint in segments. Maintaining as many healthy teeth as possible is crucial for intraoral splinting, as these are part of the fracture stabilization method. A splint will ideally incorporate at least two to three teeth rostral and two to three teeth caudal to the fracture line. Teeth compromised by severe periodontal disease are not useful for intraoral splinting. Thus, intraoral splinting will be best when there are periodontally sound teeth, a lack of a bony defect, and minimal comminution of the fracture.

Intraosseous Wiring

Intraosseous wiring can be beneficial to use as a small implant in situations where other types of fixation, such as plating, are too large or difficult to apply. The wires placed aim to draw the fracture segments together, resulting in dynamic compression that brings the bone into proximity, allowing for healing. This technique requires that fractures are simple (non-comminuted),

FIGURE 9

› Same patient as Figures 6, 7, and 8. An esophagostomy feeding tube is in place to facilitate feeding. Modified labial button technique performed to limit movement of the mouth in the short term to support healing. The buttons and suture can be removed on a compliant conscious patient (+/- pre-visit sedation) or after receiving injectable sedation in the hospital.

the segments interdigitate well, and the nature of the fracture itself is relatively stable. Intraosseous wiring should not be used in fractures where there are gaps or comminution of the fracture fragments. Although the use of intraosseous wiring draws the fracture segments to one another, the bone can still have rotational force because, naturally, the pilot holes in the bone will be slightly larger in diameter than the wire itself. Two regions of fixation are preferred to prevent torsion and shearing forces. The more dorsal of the wires should be placed close to the alveolar margin, where tensile stress is natural in the mandible, and ideally perpendicular to the fracture line. To avoid damaging tooth roots, pilot holes can be placed between teeth or in the furcational bone. The second stabilization wire can be placed parallel to the ventral margin of the mandible. The wires will often become covered with bone and can remain in place. However, if the wires become loose or erosion of the oral mucosa over the wires occurs, the wire should be removed.

Plating

For plating techniques, miniplates and locking plates are used due to their small size. The benefit of plating fractures is that with appropriate use, the rigid fixation can create anatomic realignment and permit primary bone healing.

The mandible will require two plates, utilizing a tension-band principle, where one plate is positioned near the alveolar margin and the second plate helps to neutralize shearing or torsional forces further. The plates should be placed parallel to one another. Plate fixation can be more difficult in smaller breeds of dogs, where the tooth root-to-mandible height ratio is greater than in larger breeds. Sometimes in these cases, a single large plate along the buccal aspect of the ventral mandible may be reasonable. Penetration of tooth roots will result in damage to the vascular supply of the tooth and eventual pulp necrosis, and therefore should be avoided. A penetrating screw into a tooth can create bacterial access to the tooth and eventual endodontic disease. A combination of techniques can be used if placement of a plate along the alveolar margin is not feasible; an intraosseous or interdental wire can be used.

Significant defects may be present in certain fractures, and for these, a bridging plate can provide stability in combination with either cancellous bone grafting or a cortical bone graft. Cancellous grafts can stimulate bone healing in the presence of gaps or comminuted fractures. The cortical bone

DENTISTRY

Jaw Fractures: A Dentist's Point of View

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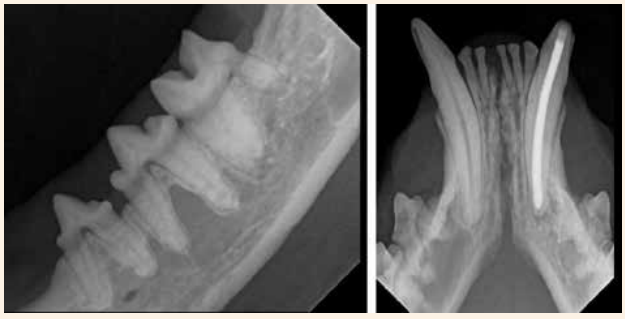
FIGURE 10

› Same patient as Figures 6, 7, 8, and 9. Presenting two months post-fracture repair. Symphyseal wire with composite bead is visible distal to the mandibular canine tooth (304). The rostral cerclage wire around the mandible between the third and fourth premolars (307, 308) is visible. Not in view is the cerclage wire caudal to the molar (309).

graft acts as a strut and can share some of the support of the fixation device. For placement of the plate, there should be at least three screws on each side of the fracture. Repair using plates can be difficult in regions where the bone is very thin, such as the maxilla and mandibular ramus. Screw length is crucial to prevent perforation of the oral mucosa, which can lead to infection or further trauma to intraoral soft tissues. Referral to a veterinary school or university is often recommended if mini plating is desired for severe maxillofacial injuries.

FIGURE 11

› Same patient as Figures 6, 7, 8, 9, and 10. Follow-up eight months later, at which time the mandibular molar (309) was extracted due to initial damage to the apical blood supply to this tooth. The tooth was not extracted at the two-month visit when the wires were removed to avoid iatrogenic damage to the recently healed fracture. Root canal therapy was performed two months post-fracture, when the wires were removed due to pulp exposure resulting from the initial injury. A well-healed fracture and symphysis are present.

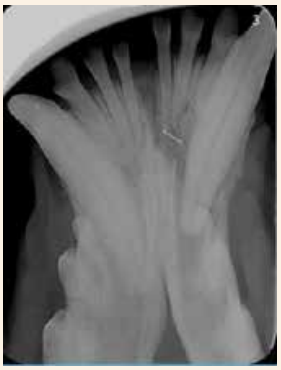


Maxillomandibular Fixation

Maxillomandibular fixation (MMF) involves connecting the maxillary and mandibular arcades with columns of composite spanning between the maxillary canine teeth to the mandibular canine teeth. This form of interdental bonding enables the alignment of occlusion and the stabilization of the jaw. Still, it is not suitable for cases where there are gap defects or comminution of the fracture. Generally, this procedure is reserved for fractures caudal to the regions of the jaw containing teeth or for injury to the temporomandibular joint (TMJ). These patients may be able to lap up gruel, but esophagostomy tube placement should be performed before interdental bonding. Care must be taken to consider the time frame during which a patient is placed on MMF to prevent functional ankylosis of the TMJ.

FIGURE 12

› Dental radiograph of a canine patient with a fracture of the left rostral mandible and extrusive luxation of the left mandibular canine tooth (304).



A modified technique of MMF using labial buttons and sutures can keep the mouth in a mostly closed position while still permitting some movement of the TMJ. This reduces some of the risk of functional ankylosis, although it still requires consideration of the duration of treatment.

Muzzle Coaptation

Nylon muzzles and tape muzzles serve to limit the mouth opening and maintain the pet's occlusion, while also providing support to the ventral mandible as a sling. This is the least invasive method for jaw fracture treatment, but it will have the most significant risk for healing complications, most notably malocclusion. When using a form of muzzle as a coaptation, the technique must ensure that the crowns of the canine teeth overlap by 30%–50% of the crown height, thereby maintaining interdigitation of the teeth and ensuring they remain in occlusion.

FIGURE 13

› Same patient as Figure 12. Intraoral splint comprised of interdental wire and composite resin placed following the extraction of the left mandibular incisors (301, 302, 303) and the reimplantation of the luxated canine tooth (304).

Mandibulectomy or Maxillectomy

Partial jaw excision is a last resort or salvage procedure to address particular situations where there is poor quality or quantity of bone to permit healing, or if financial constraints prevent consideration of surgical repair. These procedures remove a fractured segment of the jaw to avoid bone-to-bone contact and the associated pain. These procedures often result in a malocclusion, particularly in the mandible, where mandibular drift can occur.

Conservative techniques for non-invasive fracture management can range from tape muzzles to nylon muzzles to interdental wire and composite splints. These non-invasive techniques will reduce the risk of iatrogenic trauma to the tooth roots and neurovascular structures of the mandible. They will avoid disruption to the fracture hematoma, eliminate surgical trauma to

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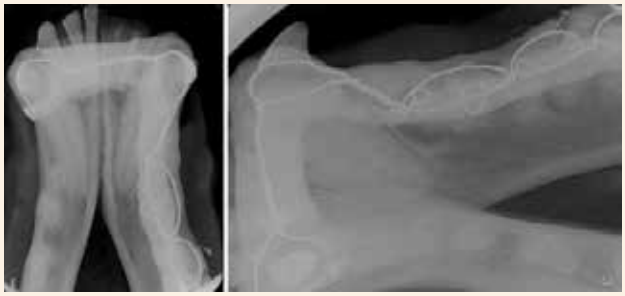
FIGURE 14

› Same patient as Figures 12 and 13. Dental radiographs two months following fracture repair. Interdental wiring and composite splint before removal. The previously luxated canine tooth was treated with root canal therapy at this visit, following splint removal.



the periosteum, and decrease the risk of additional bacterial contamination at the fracture site.

Disadvantages of non-invasive techniques include difficulty with reduction and apposition, as these techniques create a semirigid fixation that allows for micromotion. The non-invasive methods are not the primary choice for severely comminuted or unstable fractures. Often, non-invasive techniques are preferred for young patients, particularly those with deciduous or mixed dentition, as plating can cause damage to developing tooth buds, and exposure of the periosteum may interfere with skeletal growth. Edentulous patients can consider the placement of an intraoral splint and cerclaging to the mandible; however, open reduction and internal fixation may be a better treatment option. Non-rigid muzzle coadaptation and MMF can lead to delayed healing, excessive callus formation, and non-healing.

The decision on whether a tooth is involved in the fracture line and whether extraction of the tooth is required can also influence the decision-making process for the fracture repair method. Previous studies have demonstrated, for example, that the presence of the first molar tooth in canine mandibular fractures affects the strength of an intraoral splint. Regardless of the technique used for fracture repair, teeth at the fracture line or rostral to the fracture line should be assessed in six to 12 months to ensure they remain vital, as the blood supply may have been compromised at the time of injury.

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ANESTHESIA

Ketamine Revisted: Are There Any Absolute Contraindications?

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Introduction

Ketamine is one of veterinary medicine's most critically important drugs. Its utility for sedation, anesthesia, and analgesia has been long recognized in both general and specialty practice. As with all drugs, our understanding of ketamine and its optimal use continues to evolve. There is growing evidence that ketamine can be included in part of a thoughtful treatment plan for patients where it previously may have been considered risky. This emerging body of evidence invites us to revisit long-standing beliefs about when ketamine is "safe."

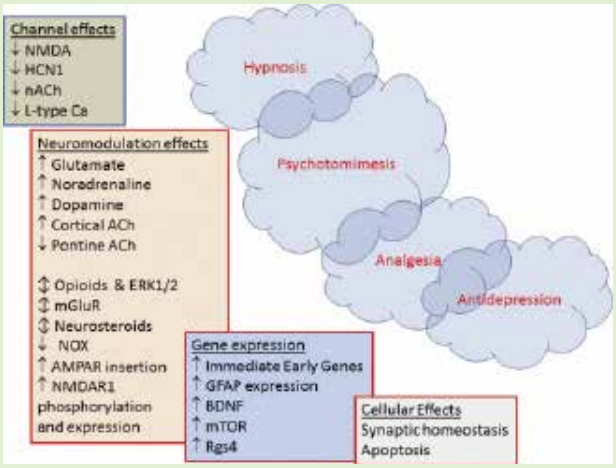
Physiology and Clinical Use

Significant progress has been made in understanding the mechanisms of unconsciousness; however, questions persist about how anesthetics ultimately "work."[1] Ketamine's effects are attributed to inhibition of glutamatergic signaling via antagonism of the N-methyl-D-aspartate (NMDA) receptor, although this is just one of many mechanisms of action.[2] Certainly, our understanding of ketamine's molecular pharmacology is far from complete.

Clinically, ketamine has effects that are desirable in many situations. Relative to other induction agents, such as propofol and alfaxalone, ketamine preserves respiratory drive and laryngeal reflexes, thus reducing the risk of apnea, hypoxemia, and aspiration; it can also promote bronchodilation in some cases. Ketamine is also a powerful and unique analgesic, whereas propofol, etomidate, and alfaxalone have no analgesic properties. Hemodynamically, ketamine acts as a sympathomimetic and can promote stability through an increase in heart rate and blood pressure, which can ameliorate the bradycardia and hypotension associated with anesthetic agents. This is in contrast to propofol, which, despite its own merits, invariably causes dose-dependent hypotension. Ketamine can be administered through almost any route lending to its versatility in situations where intravenous access is not straightforward, such as fractious animals or wildlife. Additionally, ketamine is a "dissociative" anesthetic, causing fragmentation of the conscious experience, not total abolishment of it. This makes ketamine especially useful for animals who may otherwise be difficult to control, providing restraint, amnesia, and analgesia without the downsides of excessive depth.

Intracranial Pressure (ICP)

Historical wisdom states that ketamine increases ICP. As a result, ketamine has been considered contraindicated for patients with possible intracranial disease, such as encephalitis or neoplasia. The basis for this attitude stems from a series of papers from the 1970s where direct measurement of ICP in humans undergoing ketamine-facilitated procedures demonstrated an increase in ICP.[3,4,5] Recent work with traumatic[6,7] and non-traumatic[8] brain disease has challenged this attitude, arguing that ketamine does not increase ICP, especially when multiple drugs and mechanical ventilation are employed; ketamine may even decrease ICP in some cases. Furthermore, no studies have documented a link between ketamine and negative clinical outcomes linked to increased ICP. For patients in which there is concern for elevated ICP, ketamine may still be appropriate, especially if the patient has other comorbidities that make it less ideal to use agents like propofol or alfaxalone, or if a painful procedure is being undergone. Additionally, for patients with truly elevated ICP, adequate mean arterial pressure (MAP) is critical to maintain cerebral perfusion. Ketamine may be beneficial in these scenarios, as it can support cardiac output.



Seizure Disorders

Ketamine has classically been believed to be epileptogenic, and this fuels fear of using ketamine in patients with a known seizure history. However, the 2023 ACVIM Consensus Statement on the management of status epilepticus recommends ketamine as a third-line antiepileptic agent.[9] It has been recently argued that there is no sound reason to withhold the benefits of ketamine from human epileptics; however, combination with a benzodiazepine or propofol may enhance safety.[10] Overall, the historically entrenched idea that ketamine is epileptogenic has been largely overturned, and this is particularly true in emergency management of an actively seizing patient. Ketamine is now routinely administered (per the 2023 guidelines mentioned above) to dogs presenting with seizures refractory to benzodiazepines and maintenance antiepileptics.

Ophthalmic Disease and Intraocular Pressure (IOP)

Ketamine is generally thought to increase IOP in small animal patients, and thus, is sometimes avoided for ophthalmic procedures or for patients with underlying ocular disease. This concern, similar to ICP, stems from early studies in humans with significant limitations.[11,12] Indeed, multiple studies have documented that ketamine has the potential to cause a clinically relevant increase in IOP in dogs; notably, these effects can be blunted when multiple

ANESTHESIA

Ketamine Revisted: Are There Any Absolute Contraindications?

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medications are used for anesthesia.[13,14] However, a recent paper demonstrated that propofol induction caused comparable increases in IOP in healthy dogs when compared to ketamine-diazepam inductions.[15] Additionally, a 2023 paper compared inductions with propofol, alfaxalone, and ketamine (each with midazolam) in cats, where IOP was actually increased relative to baseline in the propofol group, but not the ketamine or alfaxalone groups.[16]

In cases where acute elevations in IOP would be detrimental (e.g., underlying glaucoma, corneal surgery, phacoemulsification), ketamine should be used with caution. Ketamine can be considered for patients undergoing ophthalmic procedures where modest increases in IOP (if they occur) would be clinically irrelevant, such as non-glaucomatous enucleation (or second eye glaucomatous enucleation) or periocular/eyelid surgery.

Cardiovascular Disease and Arrhythmogenesis

One of the advantages of ketamine is that it supports hemodynamic stability and can even increase cardiac output in some cases; however, some conditions, particularly arrhythmogenic heart disease and hypertrophic cardiomyopathy (HCM), are often cited as reasons to avoid ketamine. First, it is important to note that there is a wide spectrum of heart disease; many dogs with mitral valve degeneration, for example, will never experience any relevant sequelae, such as left atrial dilation. Therefore, the suitability of ketamine depends mightily on the type and severity of the underlying cardiac disease. For patients where tachycardia or increased sympathetic tone would be undesirable, such as cats with HCM or animals with a known tachyarrhythmia, caution is advisable. Similarly, caution is advisable for patients with arrhythmias secondary to systemic disease, such as hemoabdomen. However, in many cases, ketamine can be incorporated at judicious doses for patients with some cardiac diseases.

Conclusion

When considering ketamine, three bedrock principles of pharmacology and anesthesia are essential:

1. No drug is benign.
2. *Sola dosis facit venenum* — only the dose makes the toxin.
3. There is no “safe” anesthesia or “safe” drug — only safe anesthetists.

Ketamine is a powerful, versatile, and reliable medication, but it is not without its limitations. It is irreversible, it is a controlled substance, and our understanding of ketamine is incomplete. However, the key is that all drugs have their advantages and disadvantages. Discussed above are some clinical situations where ketamine might be used with caution, but, in reality, ketamine, like every drug, should always be used with some degree of caution. No medication should be prescribed with impunity. Anesthesia is a balance, and refining how we incorporate ketamine into this balance will help us continue to improve patient care.

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6:15pm–8:45pm
Live Webinar
2 CE Credits *(pending RACE approval)*

PLEASE VISIT ANGELL.ORG/CE FOR UPDATES ON SPEAKERS AND TOPICS.

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