

# IVG Newsletter

OCTOBER 2011 | VOLUME 11, ISSUE 4



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**Oct. 12 (Mass Vet):** GME & other types of Encephalitis

**Oct. 19 (Bulger):** Regurgitation and Esophageal Disease

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## Coming Soon: MetroWest Veterinary Referral Hospital.

A new member of the IVG Network of Hospitals will be opening in Natick, MA.

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# Vomiting and Anti-emetic Therapy in Veterinary Medicine

**VOMITING CAUSES ALARM AND CONCERN ACROSS** species and is a common presenting complaint in our small animal patients. Causes for vomiting may be mild and self-limiting or associated with serious, potentially life-threatening diseases. While often directly related to the intestinal tract, there are many non-intestinal causes for vomiting that require more extensive investigation for accurate diagnosis and directed therapeutics. An understanding of the physiology behind vomiting and our options for management help us to determine the extent of diagnostics and therapeutics needed for each patient.

## THE REFLEX

The act of emesis is protective in nature, ridding the body of dangerous substances, or responding to various catalysts. Vomiting is a reflex that can be triggered by both neural and hormonal stimuli from many disorders in the body. It involves three stages including nausea, retching and expulsion of gastric contents. Nausea manifests differently in individuals and may be perceived as hiding, depression, yawning, shivering, licking of the lips, licking of objects, or pica (such as grass). Excessive salivation and swallowing allow for lubrication of the esophagus with saliva high in bicarbonate to neutralize the acid of the stomach acid as it is expelled. The esophageal peristalsis slows and the lower esophageal sphincter relaxes as the proximal small intestines begin retrograde motion. Retching, contraction of the abdominal muscles and diaphragm causes increased abdominal pressure and decreased intra-thoracic pressure as the abdominal muscles contract. This pressure change during retching causes the movement of stomach contents up into the esophagus. When the intra-thoracic pressure becomes positive again, the material in the esophagus is expelled out of the mouth as vomit. It is the second phase of vomiting, retching, that we can use to distinguish vomiting from regurgitation. As material is expelled from the esophagus, breathing is inhibited and the glottis closes to prevent aspiration.

## THE PATHOPHYSIOLOGY

Vomiting is initiated by the emetic center of the central nervous system (CNS), a number of nuclei in the

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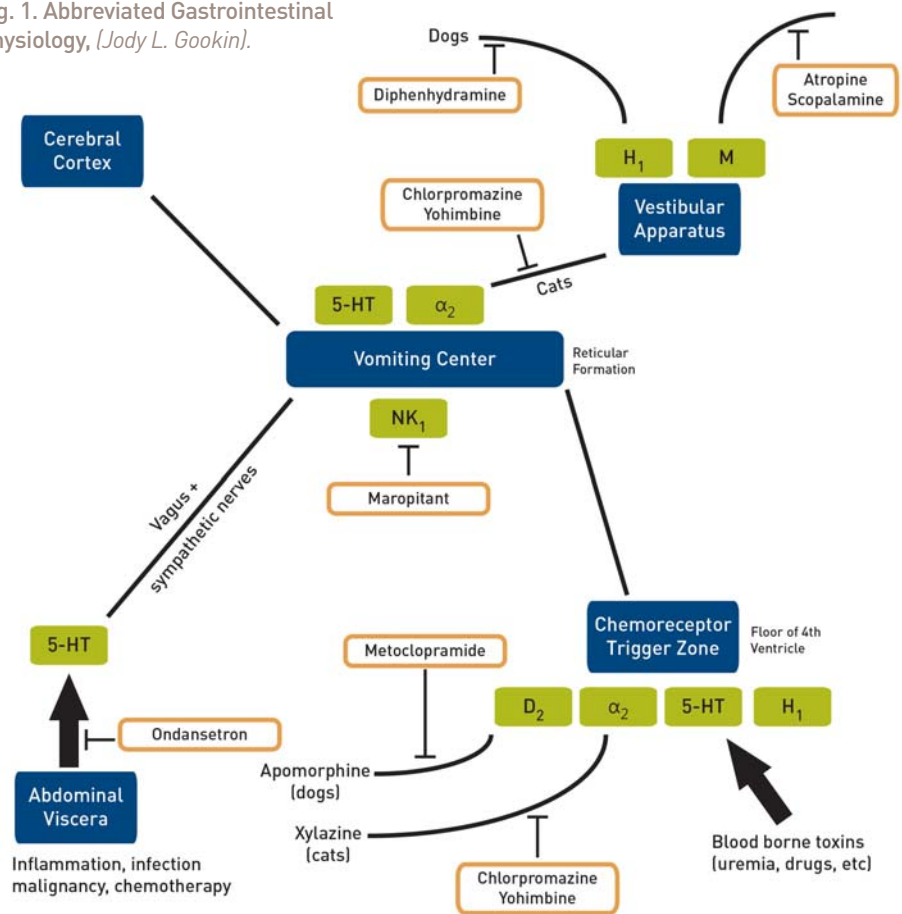
medulla oblongata within the brainstem. These nuclei and associated complex nerve pathways are responsible for organizing and triggering the act of vomiting. Known receptors such as 5HT<sub>3</sub> (serotonergic), α<sub>2</sub> (adrenergic), and NK<sub>1</sub> (neurokinergic) reside in the emetic center. Stimulation of these receptors may occur through neural pathways such as peripheral sensory receptors (sight, smell, taste), including afferent receptors from the gastrointestinal tract, or via vestibular and hormonal activation of the chemoreceptor trigger zone (CRTZ).

Neural activation of the vomiting center begins with stimulation of afferent vagal, sympathetic, vestibular, cerebrocortical, or nearby nucleus tractus solitarius pathways. Peripheral receptors within the body, particularly within the abdominal viscera, activate these pathways. The duodenum, known as the “organ of nausea” has the largest number of receptors. Vagal afferent pathways may be directly stimulated by disorders within the gastrointestinal tract. Smooth muscle within the intestinal tract and vagal afferent neurons contain large quantities of 5HT<sub>3</sub> and NK<sub>1</sub> receptors. The 5HT<sub>3</sub> receptors are stimulated by serotonin, and the NK<sub>1</sub> receptors are stimulated by substance P from enterochromaffin cells in response to inflammation or cytotoxic elements. Additional afferent signals may be through sympathetic nerves sent from receptors located within the kidney, uterus, liver, pancreas, peritoneum, urinary bladder and cardiac vessels. The glossopharyngeal nerve transmits signals from the pharynx and the tonsillar fossa. Disease within the CNS stimulates the emetic center

Fig. 1. Abbreviated Gastrointestinal Physiology, (Jody L. Gookin).

directly via NK<sub>1</sub> cells within the nucleus tractus solitarius pathway. The higher brain centers, the cerebral cortex and limbic systems, are capable of initiating emesis in three ways. Inflammation, hydrocephalus or neoplasia will directly stimulate the vomiting center. Secondly, fear, stress, excitement or pain will cause psychogenic stimulation of the vomiting center. Thirdly, head injuries or increased intracranial pressure will cause traumatic stimulation of the vomiting center. When the emetic center is stimulated by afferent neurons from the gastrointestinal tract, the higher centers of the brain, vestibular apparatus or the CRTZ, it sends signals that coordinate vomiting through efferent motor neurons.

Humeral factors in the blood stimulate the CRTZ, a set of centers located on the floor of the fourth ventricle in the brainstem. The CRTZ does not have a blood-brain barrier, allowing stimulation from chemicals within the circulation. Drugs, uremic toxins, acid-base disorders, electrolytes, infection and metabolic disorders stimulate the CRTZ to transmit signals to the vomiting center. Receptors located within the CRTZ include dopaminergic (D<sub>2</sub>), cholinergic (M<sub>1</sub>), histaminergic (H<sub>1</sub>), serotonergic (5HT<sub>3</sub>), adrenergic (α<sub>2</sub>) and neurokinergic (NK<sub>1</sub>). Cats have less developed D<sub>2</sub> and H<sub>1</sub> receptors in the CRTZ than dogs which explains their minimal reaction to apomorphine and histamine. Cats have increased α<sub>2</sub> receptors when compared to dogs, which makes xylazine, an α<sub>2</sub>-agonist, a very effective emetic in our feline patients. The vestibular apparatus passes through the CRTZ before stimulating the vomiting center. Inflammatory conditions of the pathway, motion sickness or cerebellar lesions stimulate M<sub>1</sub>, H<sub>1</sub> and NK<sub>1</sub> receptors within the vestibular apparatus. Internist Jody Gookin's depiction above is helpful in visualizing the receptors involved in the vomiting center.



## THE CAUSES AND CLINICAL DIAGNOSTIC APPROACH

Obtaining a complete and thorough history is essential to determining how aggressive to be in the diagnostic work up and therapeutic approach. Signalment, history, a description of the vomiting (to rule out regurgitation, gagging, coughing, retching or dysphagia), travel history (for infectious disease), medications history (for side effects, toxicity and NSAID exposure), possible ingestions of toxic substances or foreign bodies, chronicity and concurrent clinical signs are all important to determine an appropriate work up. Vomiting has a tremendous number of causes. Ettinger provides helpful diagnostic flow diagrams for our patients that present with acute onset of vomiting or a chronic history of vomiting. (See figures 2 and 3, over).

## TREATMENT

Control of vomiting is important in preventing dehydration, electrolyte derangements, aspiration pneumonia and allows for return of nutritional support. To treat our vomiting patients most effectively, it is important to first determine the cause of vomiting. Reflexive use of anti-emetics in veterinary medicine often masks the underlying disease and risks delaying appropriate diagnostics and therapeutics. Gastrointestinal infections and toxin elimination may

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# Vomiting and Anti-emetic Therapy in Veterinary Medicine

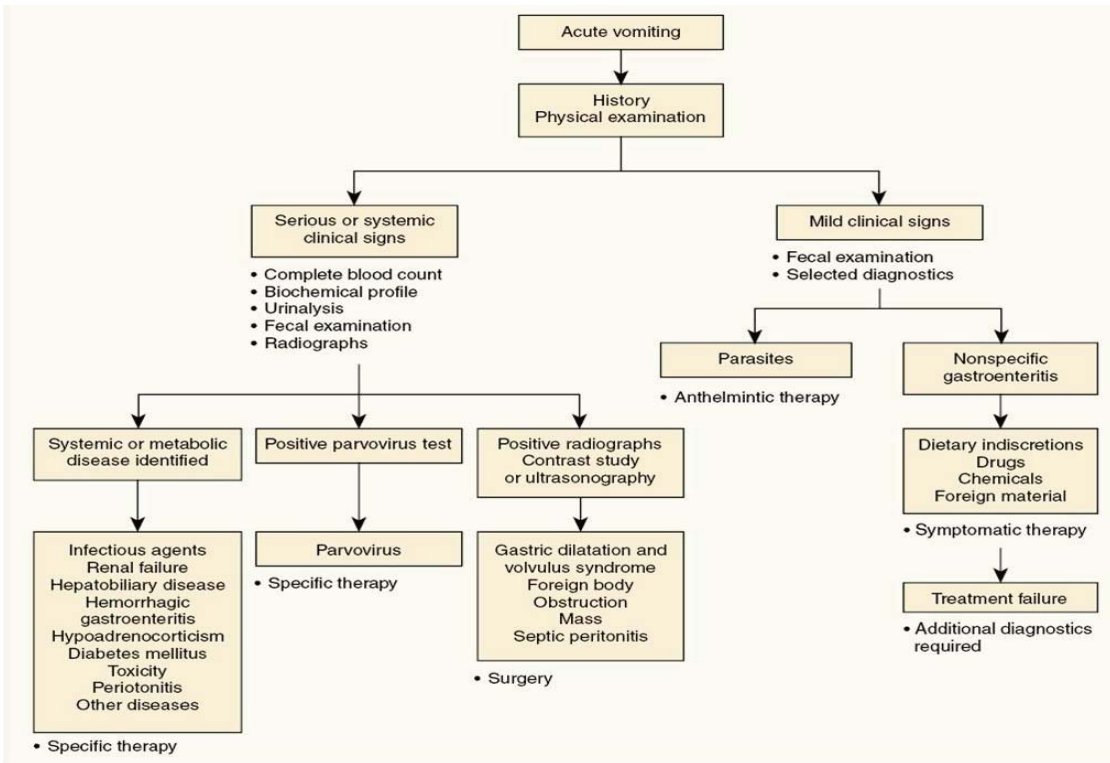
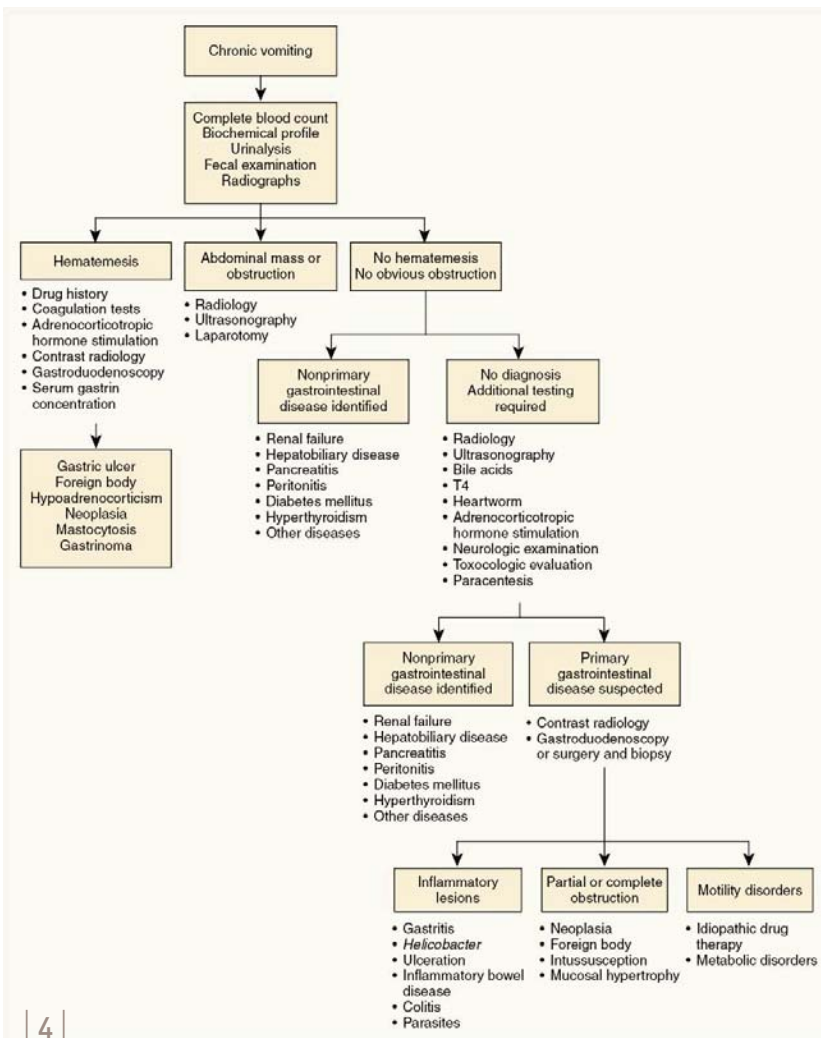


Figure 2: (left): "Algorithm for the diagnosis of acute vomiting," (Ettinger 7th ed.).

Figure 3: (below left): "Algorithm for the diagnosis of chronic vomiting" (Ettinger 7th ed.).



be prolonged by inappropriate anti-emetic therapy that slows gastrointestinal motility. Rational use of anti-emetics is warranted, and an understanding of how anti-emetics work is helpful in making treatment decisions on a daily basis.

## Antihistamines

Antihistamines block cholinergic and histaminic nerve transmission of the vomiting center from vestibular stimulation. This class includes diphenhydramine, dimenhydrinate and meclizine. Used most commonly for motion sickness, these drugs block H<sub>1</sub> receptors. Because cats do not have H<sub>1</sub> receptors in their CRTZ, antihistamines are not effective against vomiting in cats.

## Substituted Benzamides

These drugs work in different ways. Metoclopramide blocks dopamine receptors (and should not be given to patients receiving dopamine) in the CNS and 5HT<sub>3</sub> serotonergic receptors at high doses. It activates 5HT<sub>4</sub> receptors at the same time causing increased lower esophageal sphincter tone and gastric

motility. Cisapride also activates 5HT<sub>4</sub> neuronal receptors causing gastric emptying. Both metoclopramide and cisapride should be avoided in cases where gastrointestinal obstruction is a possibility as the increased motility may lead to perforation.

### **Serotonin Antagonists**

These drugs specifically inhibit 5HT<sub>3</sub> receptors located along the vagus nerve and in the CRTZ. These receptors are stimulated by serotonin released from enterochromaffin cells within the small intestine in response to mucosal damage. This explains why ondansetron and dolasetron are effective at controlling vomiting caused by radiation and chemotherapy in dogs and cats, but are not effective at controlling motion-sickness induced vomiting. Other indications include gastritis, pancreatitis, parvovirus and uremia.

### **Opioids**

Butorphanol has been shown to have some anti-emetic properties by blocking K and  $\Omega$  receptors located within the vomiting center in both dogs and cats.

### **Phenothiazines**

Chlorpromazine and acepromazine have dopamine receptor blocking and histamine receptor properties in the CRTZ at low doses. At higher doses, they have been shown to have anticholinergic properties within the vomiting center. Adverse effects, especially in dogs are common and fluid therapy should be given concurrently to compensate for the vasodilatory effects of these drugs.

### **Neurokinin Antagonists**

Neurokinin (NK<sub>1</sub>) antagonists are now commonly used in veterinary medicine for vomiting. Maropitant acts by inhibiting substance P in the central nervous system. Substance P is the main neurotransmitter involved in stimulation of the vomiting center. Maropitant is therefore effective against both peripherally and centrally mediated vomiting.

### **Other Drugs**

Centrally mediated vomiting may also be controlled by other medications. Yohimbine works in both dogs and cats by pure  $\alpha_2$ -adrenergic-antagonist action. Diazepam is thought to have effect on the vestibular system and controls nausea and vomiting in humans. Studies in

animals models show diazepam may also be effective in our patients. Corticosteroids have anti-emetic effect in people and dogs undergoing chemotherapy. While the physiology is poorly understood, the mechanism is believed to include the activation of receptors in the emetic center within the medulla, especially in cats. Propofol may reduce serotonin concentration in the CRTZ by 5-HT<sub>3</sub> serotonin receptor antagonism and  $\gamma$ -aminobutyric acid activity. This alkylphenol derivative is used for chemotherapy induced vomiting control in people when serotonin antagonists or dexamethasone are not effective. It's anti-emetic properties are uncertain in veterinary medicine. Mirtazapine, an antidepressant piperazinoazepine medication, is also used for chemotherapy induced nausea in humans. It is more commonly used as an appetite stimulant in dogs and cats. It exerts anti-emetic effects by blocking presynaptic  $\alpha_2$ -receptors, blocking serotonin receptors, acting as a strong 5-HT<sub>2</sub> and 5-HT<sub>3</sub> serotonin receptor antagonist, acting as a weak 5-HT<sub>1</sub> serotonin receptor antagonist and by exhibiting H<sub>1</sub>-histamine antagonist activity.

### **CONCLUSION**

Anti-emetic therapy is implemented by many of us daily in veterinary medicine. Determination of the cause of vomiting in each patient is essential for directed and appropriate anti-emetic therapy. A thorough history, physical examination and diagnostics to determine the cause of vomiting must be performed as much as possible for each patient in order to safely and effectively manage vomiting. Correct use of current dosing regimens (found easily in veterinary formularies) should also be verified. In patients where the cause remains undetermined and the vomiting remains refractory, combinations of anti-emetics may be used, but the importance of further diagnostics to obtain a definitive diagnosis must be emphasized.



# Repetitive Strain Injury of the Psoas Muscle in Dogs

**REPETITIVE OVERUSE OF THE PSOAS IS A COMMON** yet underappreciated cause of mobility impairment in the dog. Sustained contraction, fatigue, and spasm of the psoas musculature develop frequently in dogs with a wide range of orthopedic or neurologic disorders as they alter their posture to compensate for painful or dysfunctional limbs. This form of repetitive strain injury (RSI) can be difficult to recognize, yet it is a significant cause of pain and decreased mobility, especially in geriatric dogs.

Strain-counterstrain is a manual therapeutic technique adapted from the field of physical therapy that can be used to treat psoas muscle strain that develops secondary to injury or surgery. It is also a useful method for preservation and enhancement of mobility in older dogs suffering from chronic progressive degenerative joint disease. This article presents an overview of the pathomechanics, diagnosis, and management of psoas RSI in dogs.

## PATHOMECHANICS

The musculoskeletal system is highly interconnected and functionally integrated, and injuries or disorders affecting any one part of this system often lead to secondary problems and malfunction at other sites. For example, the limp that develops in humans following even a mild ankle sprain can lead to flares of secondary lower back pain due to changes in posture and body mechanics at the level of the hip and spine. Physical therapists have long recognized the deleterious changes in posture and movement that develop in humans in response to specific injuries or orthopedic disorders, and a major goal of physical therapy is to limit this “ripple effect” of secondary pain and dysfunction. Dogs also adopt compensatory postures and patterns of movement in response to injury or orthopedic disease, and these changes in basic body mechanics can also lead to problems at distant sites. One of the most common examples of this is psoas RSI that develops in association with pain and dysfunction of a hindlimb.

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Hind limb lameness is the most common form of mobility impairment in the dog. Disuse or offloading of a hind limb results in several characteristic postural changes including low and extended head carriage, elbow abduction, sloping of the topline with elevation of the pelvis above the level of the scapulae, and hunching or “roaching” of the lumbar vertebral column into an abnormally kyphotic conformation (Figure 1). These adaptive changes are designed to shift a dog’s center of gravity towards the forequarters and offload the hind limbs. Lumbar roaching is a consistent postural adaptation to hindlimb lameness; the psoas musculature is one of the primary structures responsible for maintaining the lumbar spine in a roached position.

The psoas system consists of several muscles that originate along the ventral aspect of the cranial lumbar vertebrae and that insert on the pelvis and proximal femur. The major component of the psoas system is the iliopsoas, which inserts on the lesser trochanter of the femur (Figure 2). The iliopsoas is an important flexor of the hip. The psoas system as

a whole also functions to flex the lower spine and draw the pelvis forward under the body. Such lumbar flexion occurs normally during certain gaits such as the gallop. However, in response to hind limb lameness, the psoas musculature is recruited into a postural role and undergoes sustained contraction to maintain lumbar flexion.

## DIAGNOSIS

Two forms of psoas muscle injury are recognized in dogs. The classic form is a sprain of the iliopsoas at its musculotendinous junction. This is usually a painful traumatic injury. Iliopsoas sprain is common in sporting dogs and is often associated with high-intensity activities that subject the hip to forceful extension, such as hard running or certain agility exercises. In contrast, RSI is a more insidious form of injury that is uniquely associated with postural adaptations to hind limb lameness.

RSI refers to a syndrome of muscular pain, spasm, and diminished strength that occur as a result of repetitive activity or constant sustained contraction. In dogs with painful hind limb disorders, persistent forward weight shifting imposes a high work load upon the psoas musculature, and leads ultimately to muscle fatigue and RSI. The psoas muscle is predominantly a fast-twitch muscle, and as such is adapted to cycles of transient forceful contraction and rapid relaxation. As forward weight-shifting posture becomes a chronic condition, the fiber type distribution within the psoas musculature will transition from primarily fast twitch muscle fibers to a combination of slow and fast twitch fibers (adaptive fiber type switching). However, despite this adaptation, the ability of the psoas to function as a postural muscle remains limited.

Psoas RSI is often a clinically subtle condition and may manifest as stiffness after rest, difficulty rising, reluctance to climb stairs or jump into a vehicle, and general exercise intolerance. Some dogs with this condition exhibit pain on deep palpation of the



Figure 1. This dog shows classic hind limb off loading posture. Note the elevated pelvis, low head position, and lumbar roaching.

musculature of the groin; however, more commonly, pain is localized to the mid-body as well as the origins of these muscles on the transverse processes of the third and fourth lumbar vertebrae. This form of strain injury does not cause structural abnormalities within the muscle. Thus, radiography, computed tomography, and MRI of affected muscles are invariably normal.

Diagnosis of this condition is based on physical examination and identification of regions of tight and painful muscle (trigger points) within the psoas system.

Psoas RSI can be difficult to recognize and the pain and dysfunction associated with this condition are often attributed to the primary cause of a given hind limb lameness. For example, psoas RSI is extremely common in dogs with hip dysplasia. Hallmark clinical features of hip dysplasia include stiffness and pain on extension of the hip joint. However, this movement also stretches the psoas musculature; thus, resistance to hip extension may reflect pain both at the level of the hip and the muscles of the lower back. Recognition of these interconnected problems is important since the most effective treatment strategies are those that address both conditions simultaneously.

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## Repetitive Strain Injury of the Psoas Muscle in Dogs

### Iliopsoas anatomy

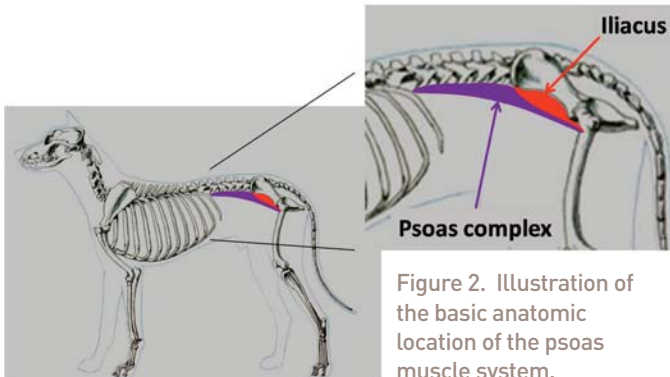


Figure 2. Illustration of the basic anatomic location of the psoas muscle system.




Figure 3. Strain-counterstrain maneuver, a manual therapy technique for treatment of psoas strain injury being performed on a dog.

### MANAGEMENT OF PSOAS RSI

Standard approaches to the treatment of psoas RSI have not been established. In all cases, the primary cause of a given hind limb lameness should be addressed if possible. However, this is often difficult in older dogs, especially those suffering from progressive osteoarthritis of key joints such as the hip, stifle or tarsus. In such cases, treatment of psoas RSI can nevertheless be of value in enhancing the ability of a dog to compensate for ongoing joint degeneration.

Strain-counterstrain is an established manual technique used by physical therapists for treatment of a wide range of human muscle strain injuries. It is an emerging approach to the treatment of psoas RSI in the dog. Muscles affected with RSI become hyper-responsive to elongation and when stretched, undergo vigorous and painful reflexive spasm. Strain-counterstrain involves manipulation of a

portion of the body into a position that maximally shortens a strained muscle. This position is held for a brief period before the body is allowed to gently return to a neutral position, and the process is repeated several times. Cyclic passive shortening of a strained muscle resets the level of tension in the muscle through modulation of the afferent signaling of the muscle spindle apparatus to the central nervous system. With time, this recalibration of tonic muscle tension and responsiveness to stretch stimuli facilitates gradual relaxation, relief of spasm, and improved stretch tolerance. The basic strain-counterstrain maneuver used for treatment of psoas RSI involves flexion and gentle outward rotation of the hip while a dog is relaxed and lying on its side (Figure 3). This technique is simple to perform, and when incorporated into an individualized home-program of daily exercise, can result in meaningful improvements in comfort level and mobility in an affected dog over time.

Our understanding of whole-body adaptations to specific orthopedic ailments in the dog, and how these can lead to secondary syndromes of muscular pain and dysfunction is expanding. Many secondary problems respond well to simple and non-invasive manual treatments. Carefully designed programs of exercise and manual therapy can easily be incorporated into home programs, and can be extremely useful tools for long-term preservation of mobility and quality of life in our canine patients. 

# Intravenous Lipid Emulsion Therapy for the Treatment of Toxicity

OVER THE PAST FEW YEARS, INTRAVENOUS LIPID emulsion (IVLE) therapy has become an exciting and newly recognized treatment for toxicities in human and veterinary medicine. For certain toxicoses, IVLE has been shown to significantly increase survival and speed recovery. Controlled studies in laboratory animal models (rats, dogs, rabbits and swine) have shown that IVLE can be life-saving for certain drug overdoses or toxicities as compared to controls. Controlled studies are not available in humans but there have been many well-documented cases of successful treatment of toxicoses using IVLE.

Although the exact mechanism by which IVLE is effective is still unclear, the theory of a lipid sink has been proposed. The lipids act as a binding agent for lipophilic toxins in the plasma. By this binding, or trapping, the lipids reduce or eliminate the toxins' ability to bind to other molecules, thus rendering them inactive. This theory supports the evidence of efficacy of IVLE therapy in the treatment of different classes of lipophilic toxins.

## INTOXICATIONS

The literature describes the benefits of IVLE on several drug categories. Local anesthetics (lidocaine, bupivacaine) were the first toxins to be experimentally treated with IVLE therapy. Clinical signs of toxicity include CNS depression, ataxia, nystagmus, seizures, bradycardia, cardiac arrhythmias, hypotension and circulatory collapse.

In an experiment in rats, bupivacaine overdose was used to induce cardiopulmonary arrest. IVLE was administered in conjunction with standard cardiopulmonary cerebral resuscitation in rats. Return of spontaneous circulation, normal sinus rhythm and normal blood pressure parameters occurred within minutes. A similar study in twelve laboratory dogs found similar results: the IVLE treatment group exhibited the return of normal hemodynamics while none of the dogs in the control group survived.

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A single case report of IVLE therapy in a cat is available. A cat was treated with subcutaneous lidocaine to facilitate repair of a wound on a limb. Within 30 minutes, the patient was obtunded and recumbent with cardiac arrhythmias and hypotension. Treatment with oxygen and IV fluids were minimally effective. By the conclusion of a 30-minute IVLE bolus, the cat had normal mentation and appeared clinically stable. The cat was discharged the following day with no apparent adverse effects from lidocaine toxicity or IVLE therapy.

The impact of IVLE therapy upon calcium channel blocker toxicities has also been investigated, specifically in connection with verapamil. Clinical signs of calcium channel blocker toxicity include vomiting, bradycardia, pulmonary edema, hypotension, junctional rhythms and AV block. In a study of fourteen laboratory dogs, high dose verapamil was administered. Of the dogs that received IVLE, 100% survived. In comparison, only 14% of the control group survived.

Another documented use for IVLE therapy is for the treatment of macrocyclic lactone parasiticide (ivermectin, moxidectin) toxicity. This toxicity is most common in breeds predisposed to the MDR-1 (multi-drug resistance) gene mutation such as Collies, Australian Shepherds, Long-haired Whippets, McNabs and Silken Windhounds. Clinical

## Intravenous Lipid Emulsion Therapy for the Treatment of Toxicity

Class of Drug	Drugs
Local Anesthetics	Lidocaine, Bupivacaine
Calcium Channel Blockers	Verapamil, Diltiazem, Amlodipine
Beta Blockers	Propranolol
Macrocyclic Lactone Parasiticides	Ivermectin, Moxidectin
GABA Derivative Muscle Relaxant	Baclofen
Pyrethroid	Permethrin
Opioid Antidiarrheal	Loperamide
Non-tricyclic Antidepressant	Bupropion (Wellbutrin®)
SSRI	Sertaline (Zoloft®)
Tricyclic Antidepressant	Clomipramine

Table 1. Drug toxicities likely to respond to IVLE therapy.

signs of toxicosis include vomiting, ataxia, weakness, tremors, and seizures.

A clinical case report of moxidectin toxicity in a puppy demonstrated remarkable response to IVLE therapy. Within five hours of toxin ingestion, the puppy was in severe respiratory acidosis and hypoventilation requiring intubation and positive pressure ventilation. Within two hours of initiation of lipid therapy, the puppy was weaned from mechanical ventilation. A second dose was given approximately 15 hours later and the puppy became alert and ambulatory. The patient was discharged two days after exposure with no remaining neurologic deficits or abnormalities. The timeline to recovery of this case is remarkable considering the terminal elimination half-life of moxidectin is 25.9 days in the dog; reported recovery rates vary from 3-16 days. Although a published report of IVLE to treat ivermectin toxicity is not available currently, anecdotal reports demonstrate varied levels of success.

Please see Table 1 for a list of toxins that may be treated with IVLE therapy. Other lipid-soluble toxins may also be treated with IVLE therapy but to date have not been reported.

### ADVERSE EFFECTS

Few adverse reactions to IVLE have been reported in humans. Risks include fluid overload, IV catheter site phlebitis and hypersensitivity. Although in theory there are other risks associated with lipid administration (pancreatitis, fat embolism, immune suppression), none have been reported in conjunction with the use of lipids to treat toxicoses.

### ADMINISTRATION

IVLE therapy has been reported using 20% Intralipid® or Liposyn II. Either form can be administered through a peripheral IV catheter. Aseptic technique is required due to the risk of bacterial growth within the Intralipid. The bag or bottle should be disposed of within 24 hours of opening.

The current dosage recommendation for IVLE therapy in dogs is a 1.5 mL/kg rapid bolus (5-15 minutes) followed by 0.25 mL/kg/min over 1-2 hours. If clinical signs persist or return, it is recommended to check a serum blood sample for lipemia. If the serum is not lipemic, the initial dose can be repeated. The dose used in the case report for the cat is 1.5 mL/kg bolus over 30 minutes. The dose was not repeated in this case due to resolution of clinical signs.

As with any toxicity, additional supportive care is often necessary. To discuss a specific case and consideration for use of IVLE therapy, consultation with a toxicologist can be helpful. The Animal Poison Control Center of the ASPCA (888-426-4435) or Pet Poison Helpline (800-213-6680) are available by telephone for consultation on cases as needed.

### SUMMARY

IVLE therapy is relatively new to veterinary medicine and shows remarkable promise for the treatment of specific toxicoses. Because of the relative ease of administration, this treatment can be used in a

variety of clinical settings. To date, no adverse effects have been reported although there may be risks to this treatment. Further research is certainly needed to elucidate appropriate treatment protocols and the risks of IVLE therapy, especially with regard to repeat administration. For each case, the clinician will need to weigh the relative risk and benefit to IVLE therapy and determine the best course of treatment. Other supportive measures and decontamination therapies should be implemented as needed as well.



*For further information on the subject of IVLE therapy, please refer to the article in the Journal of Veterinary Emergency and Critical Care, 21 (4), 2011, pp309-320, entitled "The use of Intravenous Lipid Emulsion as an Antidote in Veterinary Toxicology," by Fernandez, Lee, Rahily, Hovda, Brutlag and Engebretsen.*

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