LIVS In Plain View

LONG ISLAND VETERINARY SPECIALISTS

MAY/JUNE 2019
VOLUME 12 ▪ ISSUE 3

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Inside This Issue

Metabolic Encephalopathies (Part I): It's More Than Just HE

Ann Bilderback, DVM, DACVIM (Neurology)

Metabolic encephalopathy is a clinical syndrome resulting from disorders of metabolism. Due to the extremely high metabolic demands of the brain, systemic abnormalities that interfere with its energy metabolism may result in clinical signs of encephalopathy. The majority of metabolic diseases lead to diffuse, symmetrical forebrain signs because the cerebral cortical neurons are the most susceptible to dysfunction of energy metabolism. Clinical signs can be acute or chronic in onset and signs may wax and wane. Although the most well known metabolic encephalopathy is hepatic encephalopathy, there are numerous other metabolic causes of encephalopathy. Treating the clinical signs of encephalopathy in these patients generally depends on treating the underlying metabolic disease. This article is meant as an introduction to the various causes of metabolic encephalopathies; an in-depth discussion, including diagnosis and treatment of the underlying disorder, is beyond the scope of this article.

Hepatic Encephalopathy

Hepatic encephalopathy (HE) is a complex neurological condition that can occur secondary to acute or chronic liver disease. One function of the liver is to filter out potentially toxic substances from the gastrointestinal tract, via the portal venous system, so that these toxins do not gain access to the general circulation. If these toxins gain access to general circulation, clinical signs of encephalopathy can result. The pathogenesis of HE is complex and not clearly understood with several possible causes and mechanisms including:

- gut-derived toxins (such as ammonia, indoles, and short-chain fatty acids) that reach systemic circulation and cause neurotoxicity;
- alteration in brain neurotransmitter balance and/or production of "false" neurotransmitters due to increased levels of aromatic amino acids in general circulation;
- and circulating benzodiazepine-like substances that act on brain GABA receptors

Ammonia is the only HE toxin that can be measured clinically. Elevated ammonia is an important cause of HE, however, the role of ammonia in the pathogenesis of HE is controversial. Although elevated blood ammonia level is characteristic of HE, the degree of correlation between HE severity and blood ammonia concentration is variable. Not all animals

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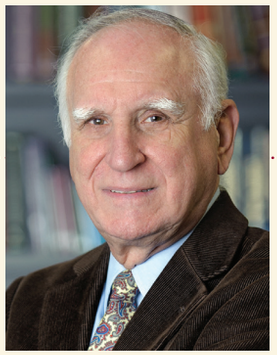
A Note from the Editor



The arrival of pollens, weeds, dust and other allergens has made breathing a difficult exercise for many, our pets included. The Dermatology department has maintained extended hours to offer the services of LIVS to clients and referring veterinarians and is available to consult in cases that need direction and appropriate allergic management.

Graduation ceremonies and other outdoor events mean much will be prepared on grills so our pets will be doing their usual begging for tasty bits of BBQ'd foods, most of which are not completely compatible with canine metabolic processes.

Summer seems to bring on more accidents, rashes, ingestions, gastrointestinal disruptions with subsequent dehydration and injuries of many kinds. LIVS is always open for any emergencies that may arise and our extended hours remain as before with each service ready to serve the needs of our clients and those patients referred to LIVS.

We remain aware that many plants are toxic to pets and one extremely toxic to cats is the lily- the whole plant, leaves, flowers, pollen and even the water in the pot, leading to kidney failure. Dogs get serious digestive disruptions but it is not usually fatal. Something that has puzzled me is that when fresh water is provided to pets, some still seem to find the overflow in the pot base quite attractive although contaminated with fertilizer runoff.

A recent increasing trend in feeding pets freshly prepared foods including raw meat is of some concern to veterinarians as the U.S. FDA says raw pet food has a high risk of bacterial contamination. The agency is also investigating a possible link between a grain free diet containing peas, lentils and potatoes with elevated levels of heart disease in dogs….established pet food companies are backed by decades of research while the verdict is still out on fresh food as so many essential nutrients need to be considered. Although sales of fresh pet food jumped 70% in the last 3 years, it's still a fraction of the 25 billion dollar U.S. pet food market.

Dr. John Sapienza, director of Ophthalmology is again hosting a visitor, this time one from Argentina, Ms. Maria Paula Fulgenzi, who is in her last year in the veterinary school at the University of Buenos Aires.

LIVS sponsored a CE event held at the Crest Hollow Country Club on May 21st for about one hundred veterinary technicians. It was a successful blend of learning, food, conviviality and dessert. The evening began at 6pm, with a cocktail hour and lectures stated at 7pm. It was another well attended and professionally rewarding LIVS event.

Dr. Meghan Umstead has extended hours to offer services to our clients and referring veterinarians and is available to consult in cases that need direction and appropriate allergic management. The Internal Medicine Department under Dr. Joshua Tumulty's direction, has expanded appointment availability for elective and emergency internal medicine consultations and ultrasound evaluations Monday through Saturdays. Dr. Curtis Dewey, associate professor and section head of Neurology/Neurosurgery at the College of Veterinary Medicine at Cornell is at LIVS regularly for consultation. Appointments can be made at 516-501-1700. Feel free to contact any of the aforementioned staff members about how they may be of service.

As before we welcome all comments, please submit them to lmarino@livs.org

Leonard J. Marino, MD, FAAP, LVT

Metabolic Encephalopathies (Part I): It's More Than Just HE

➤ *Continued from Front Cover*

with HE will have elevated blood levels of ammonia and blood levels do not always correlate with neurologic dysfunction. This is an important point to remember as some people (including veterinarians) mistakenly believe that if ammonia levels are normal, the patient does not have HE. That is incorrect – a patient with HE can have normal ammonia levels.

Ammonia is primarily synthesized in the gastrointestinal tract and transported to the liver via the hepatic portal vein; it is also released into circulation secondary to muscle damage. In animals with normal hepatic function, the majority of ammonia is metabolized via the urea cycle into urea which is then excreted by the kidneys. If ammonia is not appropriately metabolized by the liver, it will enter the general circulation and into brain tissues. The brain lacks an effective urea cycle so has limited capacity to metabolize and remove ammonia. Ammonia is mainly metabolized to glutamine which is degraded to glutamate, an excitatory neurotransmitter which is a direct neurotoxin when present in excess and is one factor in the development of HE.

The clinical signs of HE include abnormal behaviors (pacing, head-pressing, inappropriate mentation, etc), changes in mentation (dull to comatose), visual deficits, and, less commonly seizures. Ptyalism can also be a common clinical sign in cats. Clinical signs can wax and wane, can be intermittent, and do not have to correlate with having recently eaten a meal. The clinical signs can be noted at any point of the day, regardless of when a meal was ingested.

The most common cause of HE is portosystemic shunts (PSS); however, HE can be present with microvascular dysplasia (MVD) or liver failure from any cause. Although the most common PSS/MVD breeds are small or toy breed dogs, PSS/MVD and HE should be considered in any puppy, kitten or young adult, including large breed dogs, presenting neurologically inappropriate.

Treating the underlying cause of HE is the key to controlling the clinical signs of HE with prognosis dependent on the underlying cause and severity of neurologic signs. In addition to treating the underlying cause, it is necessary to stop the seizures (if present) and reduce serum levels of neurotoxic metabolites usually centered around reducing serum ammonia levels (lactulose, antibiotics, and low protein diet). For patients with seizures, controversy exists over the use of benzodiazepines (i.e. diazepam) for the treatment of HE-associated seizures due to hepatic metabolism, although it remains the first-line drug of choice to stop an actively seizing patient with some opting to use it at a lower dose. Anticonvulsant therapy should be initiated, preferably one that does not affect or depend on hepatic metabolism, therefore avoiding

phenobarbital in these patients. Potassium bromide has historically been the first choice in these patients, however, due to side effects and time to reach therapeutic blood levels, levetiracetam (aka Keppra) has become a better option. Reaching therapeutic blood levels rapidly when used intravenously, levetiracetam has minimal side effects, although sedation in some patients has been noted. For those patients that continue to seize, a propofol CRI should be considered.

Renal Encephalopathy

Renal-associated encephalopathy may be secondary to uremic encephalopathy (UE), dialysis dysequilibrium syndrome (DDS), or posttransplantation encephalopathy (PTE). DDS is believed to be caused by an osmotic gradient between the brain and the extracellular fluid environment due to overly rapid hemodialysis which results in cerebral edema. PTE has been described in cats after renal transplantation with uncontrolled hypertension believed to be a major factor in the resulting encephalopathy.

Uremic encephalopathy (UE) may be present with acute or chronic renal failure but is believed to be more severe with acute renal failure. Patients with UE are typically in end-stage chronic renal failure or are anuric due to an acute renal insult. Renal failure results in the accumulation of numerous organic substances that possibly act as uremic neurotoxins, but no single metabolite has been identified as the sole cause of uremia and the degree of azotemia correlates poorly with the degree of neurologic dysfunction. The pathophysiology is complex and poorly understood with many contributing factors thought to be involved: metabolite accumulation, hormonal disturbances, metabolic abnormalities, and neurotransmitter imbalances may all contribute. Circulating neurotoxins, such as high levels of parathyroid hormone, can have a direct toxic effect on neurons. Ionic imbalances, especially hypercalcemia, can lead to neuronal mineralization. Hyperosmolality can result in cerebral neuronal dehydration. Hypertension can lead to tortuosity, necrosis of cerebral vasculature, hemorrhages, cerebral edema, and uremic vasculitis can affect cerebral blood vessels. Acid-base imbalance can occur with renal failure resulting in acidosis depressing cerebral function. Fluid and electrolyte disturbances, including hypercalcemia, hypo- and hypernatremia, and hypo- and hyperosmolality, are common in patients with renal failure which may contribute to encephalopathy. And uremia can result in neurotransmitter imbalance in the brain. It is also important to keep in mind that drug metabolism and excretion may also be altered due to renal failure resulting in drug accumulation and subsequent encephalopathy.

Clinical signs of renal-associated encephalopathy are similar regardless of whether the cause is due to UE, DDS or PTE. Typical encephalopathic signs include behavior changes (e.g. dementia), change in mentation (dullness to

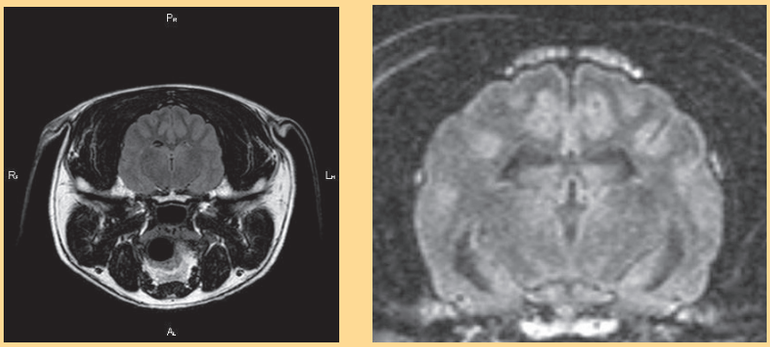


Figure 1: *Brain MRI at level of thalamus (FLAIR sequence). A) Normal Brain. B) 6.5 yr MN Cavalier King Charles Spaniel with liver failure. MRI reveals bilaterally symmetrical hyperintensity (brightness) in the gray matter of the cerebral cortex consistent with cortical laminar necrosis confirmed at necropsy. These changes were most likely secondary to HE or hypoglycemia present in this patient.*

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Metabolic Encephalopathies (Part I): It's More Than Just HE

➤ *Continued from Page 4*

comatose), and seizures. Muscle tremors, generalized weakness, and irregular respirations may also be present.

Diagnosis of renal-associated encephalopathy is based on signs of encephalopathy in patients with renal failure and no other cause of brain disease. Patients that develop neurologic signs soon after hemodialysis or renal transplantation is evidence for DDS and PTE, respectively.

As with most metabolic encephalopathies, treating the underlying cause is the best way to address the encephalopathy. Treatment of renal-associated encephalopathy depends primarily on reversing or managing the underlying renal disease. If acid-base or electrolyte disturbances are present, they should be corrected. If the patient is seizing, anticonvulsant therapy should be initiated. In most cases, the encephalopathy can be improved or may resolve with control of uremia. It is best to avoid the development of PTE by managing hypertension in cats before and after renal transplantation as these cats tend to have severe neurologic signs and a high mortality rate.

Hypoglycemic Encephalopathy

Although the brain is only 2% of total body mass, it utilizes 25% of total body glucose due to its absolute requirement for glucose, high metabolic rate and limited local storage resulting in continuous transport of glucose into the brain. Glucose enters the brain via a noninsulin-dependent transport mechanism that requires a minimum blood glucose to operate effectively. Encephalopathic signs of hypoglycemia often results when blood glucose levels are less than 45 mg/dl due to neuronal energy depletion.

The severity of clinical signs present with hypoglycemia depends on the rate of decline of blood glucose levels, the degree of hypoglycemia, and duration of hypoglycemia. With rapid, sudden decreases of blood glucose one may see dilation of pupils, tremors, irritability, and vocalization. With slow decreases in blood glucose, encephalopathic signs become more evident with behavior changes, mentation changes (dull to comatose), seizures and/or visual impairment present.

Diagnosing hypoglycemic encephalopathy is based on the presence of clinical signs concurrently with hypoglycemia and whose neurologic abnormalities improve or resolve with normoglycemia. Once hypoglycemic encephalopathy has been diagnosed the underlying cause must be identified in order to try and prevent future episodes of hypoglycemic encephalopathy. Hypoglycemia can result from many causes including hyperinsulinemia (endogenous or iatrogenic), sepsis, liver failure, hypoadrenocorticism, neoplasia, age/breed-related (e.g. neonatal, toy breed, hunting dogs), or intoxication (e.g. xylitol toxicity). In the meantime, hypoglycemia can be addressed with intravenous dextrose, prednisone, and/or frequent small meals fed throughout the day. ❑

This is just part I of a two part series discussing metabolic encephalopathies with hepatic, renal and hypoglycemic encephalopathies introduced so far. To learn about electrolyte- and endocrine-associated encephalopathies, as well as other causes of metabolic encephalopathies, please refer to Metabolic Encephalopathies (Part II): It's more Than Just HE

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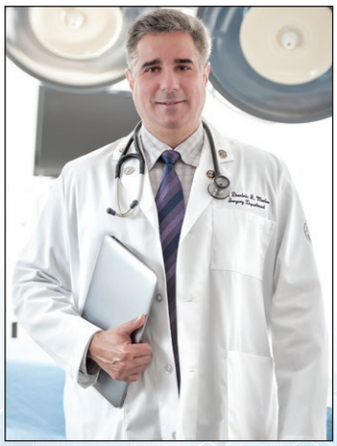

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Prosthetics for Small Animals

Catherine Loughin, DVM, DACVS, DACCT

We as veterinarians have recommended limb amputation for birth defects, severe traumatic injuries or tumors of the limbs. Dogs and cats can usually navigate on three limbs and there is no social stigma associated with limb loss; therefore, amputation has been a good option for most animals. However, studies involving limb amputated animals have revealed secondary conditions that occur due to the changes in stance and gait. These include ligament breakdown causing carpal or tarsal collapse; cranial cruciate injury; chronic neck and back pain; weight gain; and muscle pain syndromes. Such issues can affect the quality of life for an animal and result in euthanasia. Also large and giant breed dogs or animals with concurrent osteoarthritis are not always good candidates for amputation. Prosthetics offer the option of lower joint level amputations, including partial paw, instead of amputations at the hip or shoulder joints.

In recent years the technology used to construct prosthetic limbs for humans has been applied to animals. Prosthetics are made using thermosetting laminate plastic and closed cell thermo-foams, providing a snug fit for weight bearing comfort and functional use. Prosthetic paws have a rocker bottom surface for a smoother gait, and a rubber grip on the bottom that allows for good traction on slippery surfaces and longer use of the prosthetic paw. Custom colors and cosmetic designs are available (**Fig 1**). Current technology provides a prosthetic limb for the front or hind limb so long as 40 to 50% of the radius/ulna or tibia/fibula are present.

Most animals adapt very well to the use of a prosthetic limb. Familiarizing the animal to the prosthetic limb, learning to walk properly in the limb as well as learning to negotiate the environment is necessary in the early stages. The residual limb must be checked daily for skin irritation or wounds. Excessive activity while wearing the prosthetic limb must be monitored. The prosthetic limb must be kept clean and in good working condition.

Human beings receiving a prosthetic limb undergo professional rehabilitation, and it is important for animals with prosthetic limbs as well. Most animals quickly adapt to a prosthetic limb, but there is a learning period. During this time the animal will need to learn to recognize the ground through the prosthetic, learn to step up and over obstacles, sit, lay down, get up, stairs, getting in and out of vehicles, and manage on different surfaces (ground, carpet, hardwood floor, etc.). Rehabilitation

Figure 1: *Prosthetic for a left hind tarsus.*

Figure 2: *Left hind limb paw at time of presentation.*

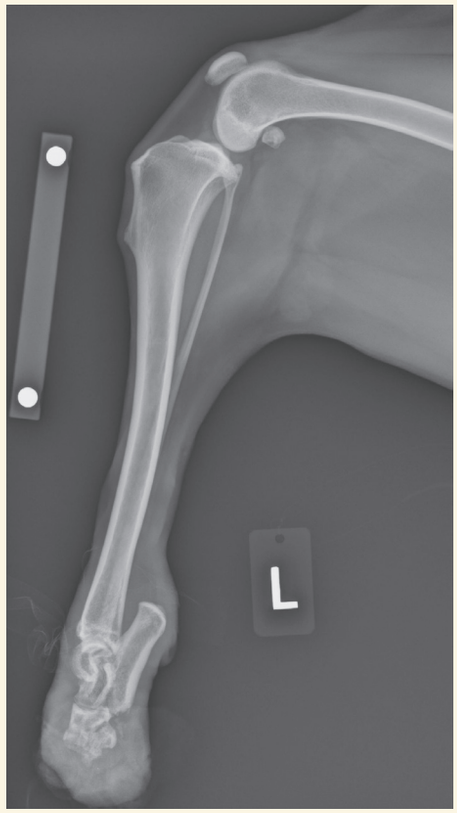

Figure 3: *Radiographs of lateral left tarsus after amputation of the paw.*

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Contact Dr. Dominic J. Marino or Dr. Patrick Roynard at 516-501-1700 or email epilepsystudy@livs.org for more information on a new treatment option.

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Client Liaison

➤ *Continued from Page 8*

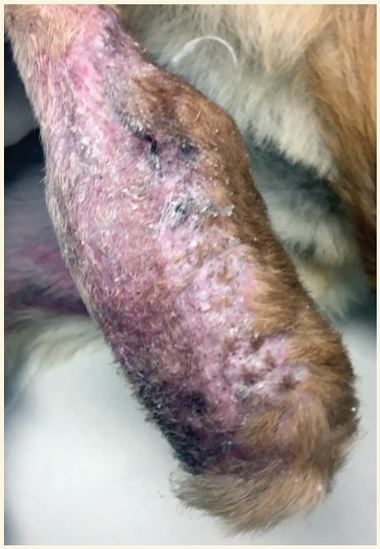
will also take into consideration the secondary muscle strain and weakness, and focus on pain relief and strengthening. Maintenance of the prosthetic is required for life, and the company that makes the prosthetic will work with you and the owner to make sure fit and longevity of the prosthetic are retained.

Case

Lulu is a 1 year-old female spayed mixed breed dog. She was hit by a car and sustained a skin avulsion to the left thorax and abdomen, degloving injury right medial hind limb (including shearing from the stifle to the metatarsals), and traumatic amputation of the distal paw on the left hind limb (**Fig 2**). She was treated for shock and once stabilized her wounds were addressed. The left hind paw was hanging by the skin with no intact nervous nor blood supply, and therefore needed to be removed. This left a stump at the tarsometatarsal joint (**Fig 3**). Both limbs and the thorax/abdominal wound were managed as open wounds with daily bandage changes until healthy tissue enabled closure. Due to the age of the dog, her high activity level, and damage to the right tarsus a prosthetic was considered for the left hind limb.

The left thoracic/abdominal wound needed a vacuum system to assist with primary closure and took 2 weeks before an H-plasty was used for closure. The medial side of the right hind limb grew healthy granulation tissue, but due to the bone shearing and loss of ligament, the right tarsus was fused with a medial pantarsal arthrodesis plate 11weeks after the injury. The left hind limb stump was surgically debrided, medicinal honey was used for the anti-bacterial and anti-inflammatory properties, and alginate dressings were used until healthy granulation tissue was noted. The wound was then slowly closed to stretch the skin and cover the stump. This took 12 weeks. During this time a metasplint fitted with 3 stockinette donuts covered in tape was used as a temporary prosthetic to allow Lulu to use both hind limbs for ambulation during the healing period (**Fig 4**)

A mold of the left hind limb stump was made by us and sent a company certified to make a prosthetic. It took 6 weeks for the prosthetic to be made and mailed to the owner. We made sure the prosthetic fit Lulu, and then had her walk around the hospital (**Fig 5**). Due to the 9 weeks Lulu spent in the handmade splint, she was able to acclimate to the prosthetic quickly. A 3-ply sock needs to be placed over the stump before the prosthetic is velcroid into place. The owner followed instructions for acclimation and has purchased several socks so they can be laundered regularly. As Lulu gets more comfortable with the prosthetic and her skin has toughened, we hope that she will be able to go for walks with her owner and run across the yard again. ❑



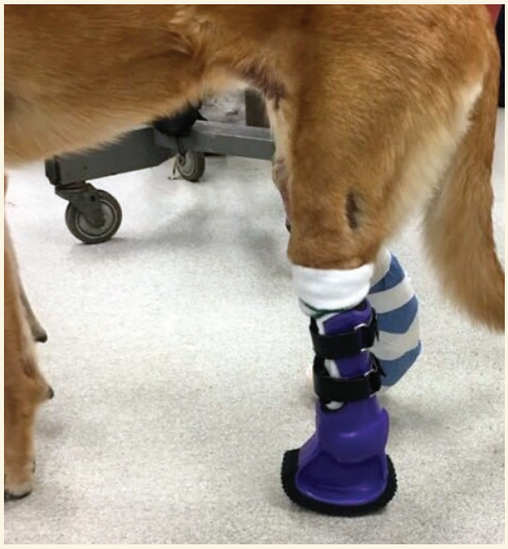
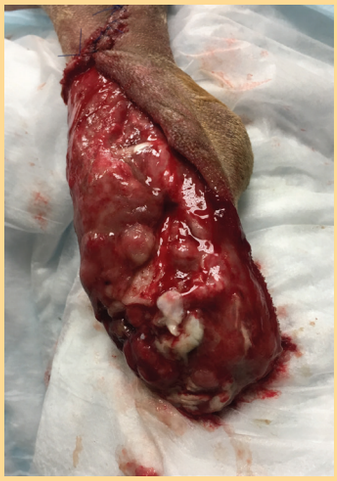
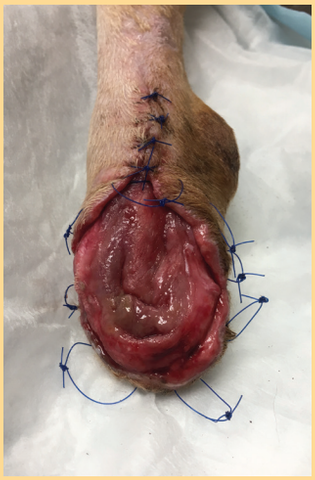
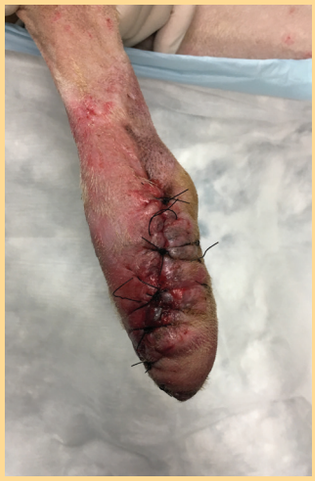


Figure 5: *The stump day of prosthetic fitting (A) and Lulu is prosthesis (B).*







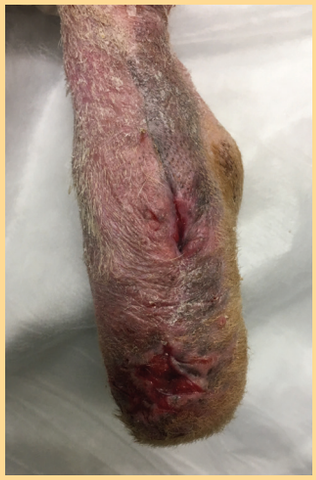


Figure 4: *Progression of the left hind stump. A) 7 day post trauma, B) 36 day post trauma, C) 66 day post trauma, and D) 75 day post trauma.*

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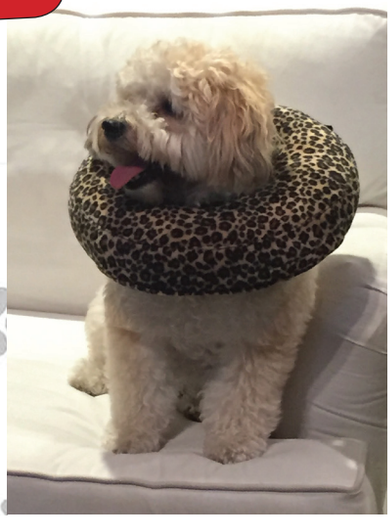

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Long Island
Veterinary Specialists

Where You Refer Your Patient First Makes All The Difference

163 South Service Road, Plainview, NY 11803

LIVS is the only facility on Long Island with a Veterinary Behavior Department committed to helping your client communicate with their pet in a more positive and productive way to manage behavior issues.

Dr. Sabrina Poggiagliolmi, DVM, MS, DACVB, treats pets with behavior issues such as:

- Aggression directed towards both humans and other pets
- Destructive behavior when left unattended (such as at home during the day)
- Excessive barking (or excessive vocalization)
- Compulsive disorders and/or Self Destructive Behavior
- Inappropriate elimination
- Fears and phobias