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Traumatic Brain Injury (Part II)

Ann Bilderback, DVM, DACVIM
(Neurology)

Neurologic Assessment

With traumatic brain injury (TBI), once eu-
volemia and appropriate ventilation/oxygen-
ation of the patient is attained the neurologic
status of the patient should be assessed. A
complete neurologic examination should be
performed, if possible, in order to neurolocalize
the areas of concern and to monitor neurologic
progression. A patient with TBI and obvious
intracranial signs may also have concurrent
spinal cord injury (IVDD, fracture, hemorrhage,



etc) which can be easily overlooked when one
is focused on the TBI. Establishing a neuro-
logic baseline is also important in order to
monitor neurologic progression, with repeat
assessment every 30 minutes until neurologi-
cally stabilized. Changes in mentation can be
the most useful indicator of severity and pro-
gression of intracranial disease. However, for
patients presenting comatose from TBI there
is no reliable guide to intracranial function.

After systemic assessment and stabiliza-
tion, diagnostics focusing on the neurologic



system are recommended. Advanced imaging
of the head is often indicated in order to as-
sess intracranial status (e.g. hemorrhage, skull
fractures, etc) and assess the need for surgery,
especially in those patients that fail to respond
to aggressive medical therapy or deteriorate
after initially responding to such therapy. Skull
radiographs are unlikely to reveal clinically
useful information in cases of severe TBI. Skull
fractures may be evident, however, intraparen-
chymal injury (hematoma, edema, infection,
abscess, etc) affecting brain tissue will not be
visible on radiographs.

If general anesthesia is not contraindicated
for systemic injury, computed tomography (CT)
or magnetic resonance imaging (MRI), both
available at Long Island Veterinary Specialists,
is recommended in order to assess intracranial
status. MRI is superior to CT for assessing
intraparenchymal injury and the caudal brain
(cerebellum, brainstem, herniation, etc) (see

Continued on Page 4 ►



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LIVS is proud to welcome:



Dr. Kimberly Golden - Internal Medicine

Dr. Golden is a native New Englander and is very excited to be back in the northeast. She developed a love for animals at a young age. Dr. Golden enjoys forming long-standing relationships with clients while working together to determine the best treatment plan tailored to each individual patient.

She obtained her Doctorate of Veterinary Medicine from Ross University, School of Veterinary Medicine in St. Kitts, West Indies. It was during her clinical year of veterinary school at the University of Florida where she discovered a keen interest in internal medicine. She continued her education with a 1-year small animal rotating internship at Bay Area Veterinary Specialists in California and then an

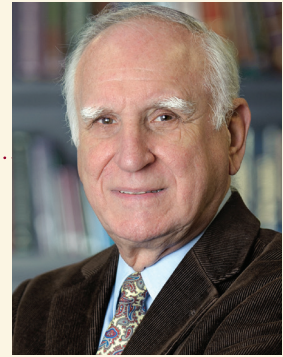
internal medicine specialty internship at Memphis Veterinary Specialists. She then completed a 3-year internal medicine residency at BluePearl Veterinary Partners in Michigan. After completing her residency, she worked in San Antonio, Texas before moving back up north.

Dr. Golden has clinical interests in all aspects of small animal veterinary internal medicine with special interests in feline medicine, infectious diseases, immune-mediated disease, and gastroenterology.

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A Note from the Editor



We have entered fall, made it through Halloween and are preparing for Thanksgiving.

It's halfway through the presidential term and the news in our country is replete with people acting out their anger by sending mail bombs to people of differing political views and shooting congregants at houses of worship. It appears that rage, not discussion is the newest means of expression; each faction certain of its correctness with dissent being described as mob action and hate speech. Elections were ways of expressing agreement or dismay with the party in power yet we are now witnessing intimidating scenarios that place expression and speech in danger of extinction. "Under democracy one party always devotes its chief energies to trying to prove that the other party is unfit to rule - and both commonly succeed, and are right." — H.L. Mencken.

LIVS welcomed a new addition to its Internal Medicine Department recently, Dr. Kimberly Golden and she is presently actively participating in its daily functions. A two week visit to the Ophthalmology Department was completed by Dr. Sebastian Solzano from Ecuador who observed the activities of that specialty.

A recent report from Johns Hopkins Hospital revealed that children recovering from cancer who were in contact with therapy dogs and who spent longer intervals with them, had a six fold increase in superbug infections compared to those who spent shorter periods interacting with the dogs. The "cure" was to wash the dogs before bringing them to the hospital and using special wipes after contact. Samples from 40% of healthy dogs showed that they carried MRSA and 10% of the children in contact with them exhibited the superbug. Whether covered in fur, scales or feathers, animals have the potential to make people sick, immunocompromised individuals especially.

In Prospect Park, two raccoons have recently tested positive for canine distemper virus (but not for rabies). It can spread to unvaccinated canines but not to humans. The same virus was found in raccoons in Pelham Bay Park and in Central Park as well. The raccoons act lethargically, have runny noses and appear confused or act aggressively. Pooches should be kept on leashes and be vaccinated.

Our extended hours for consultation at LIVS, including our newest, the Behavior Department, led by Dr. Sabrina Poggiagliolmi — "Dr. Sabrina" — is designed to serve our clients more efficiently. Appointments can be made through our telephone receptionists at 516-501-1700.

On a regular basis, Dr. Curtis Dewey, associate professor and section head of Neurology/Neurosurgery at the College of Veterinary Medicine at Cornell is here at LIVS for consultation. Appointments can be made also at 516-501-1700.

We continue to welcome input and opinions which can be directed to the editor at lmario@livs.org.

Leonard J. Marino, MD, FAAP, LVT

Traumatic Brain Injury (Part II)

► Continued from Front Cover

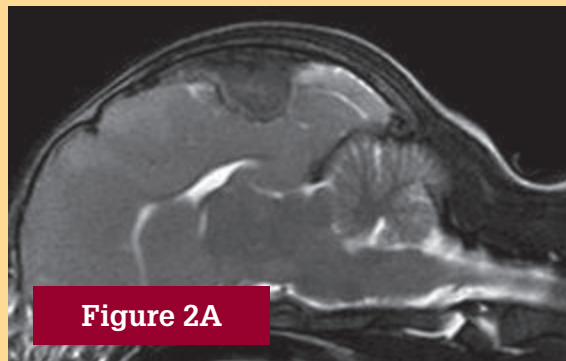


Figure 2A

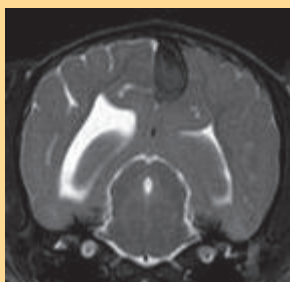


Figure 2B

Figure 2: Brain MRI of an 8 wk old MI Yorkshire Terrier with meningeal hemorrhage secondary to head trauma resulting in compression of the parietal lobe. Patient recovered well with supportive care. T2-weighted sagittal (A) and transverse (B) images.

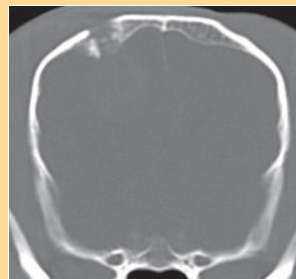


Figure 3A

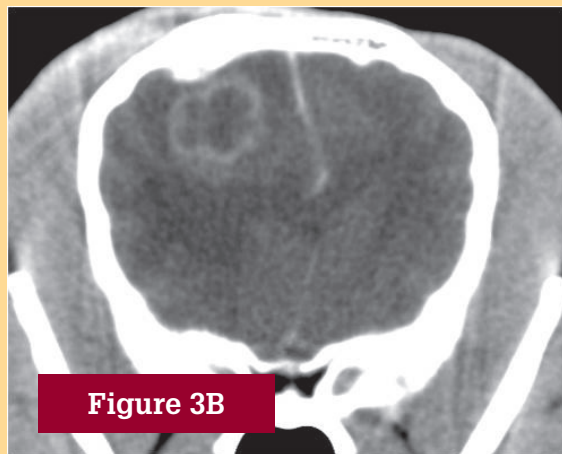


Figure 3B

Figure 3: Brain CT of a 10 yr old FS mixed breed dog with progressive neurologic worsening due to an intracranial abscess secondary to a dog bite. (A) Pre-contrast at level of thalamus reveals a defect in the right parietal bone secondary to the bite wound. (B) Post-contrast at same level reveals contrast enhancing lesion (abscess) in the parietal lobe ventral to the bony defect visible in (A).

Figure 2). However, CT is ideal for identifying skull fractures (see **Figure 3**). Furthermore, if general anesthesia is a concern due to systemic injuries a CT can be obtained more quickly than an MRI which decreases anesthesia time. There is also increased ability to closely monitor the patient with standard monitoring equipment in CT which can be a limitation with MRI due to the magnetic field. When considering anesthesia, halothane should be avoided as it has the most profound cerebral vasodilatory effect compared to other inhalant anesthetics which can result in increasing intracranial pressure (ICP)

Hyperosmolar Therapy

Treatment for TBI includes medical and sometimes surgical management to prevent or reduce increased ICP. Hyperosmolar therapy is one of the most effective treatments for cerebral edema following TBI with mannitol the

cornerstone of management of increased ICP. Indications for hyperosmolar therapy include neurologic deterioration over time, risk for herniation, and poor neurologic assessment on initial presentation. The most immediate (within minutes) and profound effect on ICP is due to the effect of mannitol in decreasing blood viscosity which allows for vasoconstriction of brain vasculature and improved cerebral perfusion at lower brain blood volume effectively decreasing ICP. The osmotic gradient created by a hypertonic solution such as mannitol also moves water from the cerebral intracellular and interstitial spaces into the capillaries, thereby decreasing the volume of the intracranial compartment and lowering ICP within 15-30 minutes of administration. Mannitol is recommended as an intravenous (IV) bolus of 0.5-1.5 g/kg over 5-15 minutes and can be repeated every 3-6 hours or more frequently depending on the patient's neuro-

logic status. Hypertonic saline (HTS) has the similar osmotic effect as mannitol and may be used as an alternative or in addition to mannitol therapy. HTS is recommended at 7.5% with 4-5 ml/kg IV over 5-10 minutes.

When considering hyperosmolar therapy, it is important to assess hydration status and electrolytes prior to initiating treatment and with repeated use. Treatment should be avoided in hypernatremic patients as this can worsen hypernatremia which in itself can cause neurologic deterioration. HTS should also be avoided in hyponatremic patients as this can result in a rapid rise in serum sodium concentrations and delayed neurologic sequela (e.g. central pontine myelinolysis). Serum osmolality should be maintained <320 mOsm/L (normal 295 mOsm/L) due to risk of renal failure secondary to renal vasoconstriction. In euvoletic patients with increased ICP, both mannitol and HTS can have beneficial effects. If the patient is not responding to one, s/he may respond to the other. However, HTS is preferable to mannitol for treating patients with increased ICP and hypotension due to hypovolemia because HTS does not cause rebound hypotension which is a possible complication with mannitol therapy due to urinary osmotic diuresis.

Head Elevation

It is recommended that recumbent patients with increased ICP be positioned with their head elevated 15-30 degrees above the heart. By facilitating venous drainage from the brain (cerebral outflow) which reduces cerebral volume, ICP can be decreased and cerebral perfusion can be increased without deleterious changes in cerebral oxygenation. Elevating >30 degrees is not recommended as this may cause a detrimental decrease in cerebral perfusion. Placing the patient on a slant board to elevate the head 15-30 degrees above the heart is preferred, rather than placing a pillow or towel under the patient's head/neck. The latter can result in compression of the jugular veins and impede jugular venous flow, which will impede venous drainage from the brain (cerebral outflow), and subsequently increase ICP. As a result, jugular lines and neck wraps should also be avoided.

Anticonvulsive Therapy

Seizures are a common sequela in people after TBI with a reported incidence rate of up to 54% and 86% risk of having additional seizures within 2 years. Although controlled trials in people indicate that the prophylactic use of

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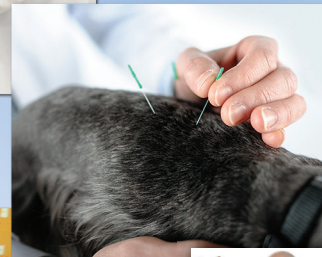
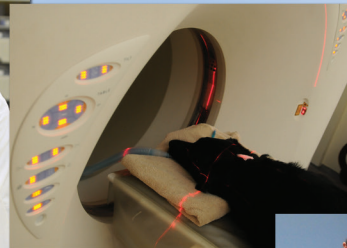
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Traumatic Brain Injury (Part II)

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antiepileptic drugs (AEDs) reduces the overall risk of seizures within 7 days of TBI, it has no effect on the risk of seizures >7 days after TBI. In veterinary patients, the use of AEDs in patients that have not yet seized is controversial with most neurologists tending not to initiate AED unless the patient has seized. If seizures do occur, AED is warranted with many options including phenobarbital, potassium bromide, zonisamide, and levetiracetam, to name a few. Compared to phenobarbital and potassium bromide, zonisamide and levetiracetam reach therapeutic blood levels faster and are minimally sedating making them less likely to interfere with neurologic assessment. Consequently, levetiracetam (60 mg/kg IV bolus loading dose, 20 mg/kg IV or PO TID) and zonisamide (5-10 mg/kg PO BID) are the author's first choice AEDs for TBI. Seizures can be the most significant chronic complication of TBI with onset not always occurring at the time of injury but can occur up to 2 years following TBI.

Control Hyperglycemia

Hyperglycemia >200 mg/dL has been associated with increased mortality in people with severe TBI. The presence of excess glucose in the ischemic brain results in anaerobic glycolysis and subsequent increased lactic acid which contributes to acidosis and cellular and local tissue damage. As a result, it is recommended to avoid hyperglycemia in patients with head trauma (i.e. avoid glucose containing intravenous fluids, unnecessary glucocorticoid usage, etc).

Furosemide

Furosemide was previously thought to have a synergistic effect of reducing cerebral edema when used with mannitol. However, recent studies suggest that furosemide does not reduce cerebral edema alone or in combination with mannitol. Due to potential deleterious effects on fluid and electrolyte imbalance, the use of furosemide cannot be recommended in the treatment of cerebral edema and should be reserved for concurrent disease (e.g. pulmonary edema, oligo-anuric renal failure, etc).

Corticosteroids

Corticosteroids are very commonly used by veterinary neurologists. However, they should be used with caution for patients with TBI. Clinical trials in people demonstrate a significantly increased mortality with high doses of MPSS (methylprednisolone sodium succinate). If ischemia is present, corticosteroids can perpetuate neuronal damage. Corticosteroids can also induce hyperglycemia and, as previously

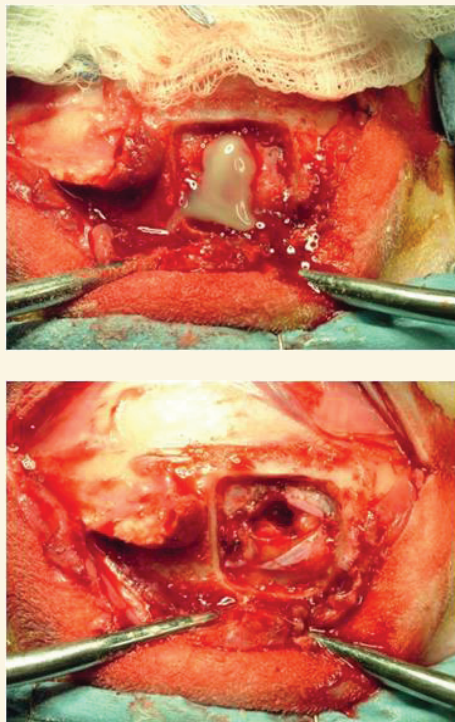


Figure 4: Craniectomy of dog from Figure 3. (A) Mucopurulent discharge (center of image) exposed upon removal of the parietal bone. (B) Meningeal and parietal lobe defects were revealed once the mucopurulent discharge was removed. The patient recovered well and died from unrelated causes 4 years post-craniectomy.

discussed, hyperglycemia should be avoided with TBI. Some try to use corticosteroids to treat increased ICP, however, the onset of action is delayed and may be too long to be helpful in acute elevation of ICP. Consequently, corticosteroids are not recommended for the treatment of TBI due to the lack of evidence of any beneficial effects, possible detrimental effects in people and general side effects of corticosteroids in dogs and cats.

Decompressive Craniectomy

Decompressive craniectomy, removing a portion of the skull solely for the purpose of relieving increased ICP to allow time for increased ICP to resolve, is controversial in both humans and veterinary patients. In people, it is considered within 12 hours of TBI in those patients who are refractory to medical therapy. Craniectomy reduces ICP by 15% in dogs and people, durotomy (incising the meninges) decreases ICP by an additional 65%, but these benefits may be short lived and may not persist into the postoperative period. The value of craniectomy solely as a decompressive surgery in dog and cats with TBI is not known, especially due to the limitations of monitoring ICP effectively in our veterinary patients. However, surgery

should be considered in dogs and cats for TBI in the following circumstances: evacuation of hematomas displacing brain parenchyma, open skull fractures or fracture encroaching on brain parenchyma, retrieval of bone fragments or foreign material lodged in brain parenchyma, or for bite wounds with penetration of the skull (see **Figures 3 and 4**).

Nursing Care

One of the most significant aspects of TBI treatment is supportive and nursing care. Throughout the management of an animal with TBI, intensive supportive care is essential. Factors such as frequent turning, physical therapy, management of nutrition including parenteral nutrition if needed, preventing (or treating) aspiration pneumonia and pressure sores, and attention to bladder and bowel function are of utmost importance in preventing complications commonly encountered in recumbent animals. For most patients, 24-hour ICU care is required for some period after the TBI for the most beneficial outcome. At Long Island Veterinary Specialists, with a team of critical care nurses, experienced specialists, and rehabilitation therapy program all working together, we are here to help with even the most critical TBI patient.

Prognosis

The first veterinarian that the TBI pet encounters after the traumatic incident will likely dictate the eventual outcome of the patient. If the patient survives the initial TBI, factors associated with neurologic deterioration include progressive edema, continued hemorrhage or infection. Whether the patient remains in your care or is referred to Long Island Veterinary Specialists, the main goal of therapy with TBI is to treat intracranial edema, avoid increased ICP and manage increased ICP in order to alleviate brain swelling and prevent damage to vital brainstem structures. For a patient whose only presenting issue is traumatic brain injury, who is otherwise stable (cardiovascular, respiratory, etc), do not make prognosis based on initial neurologic appearance of the patient, even for those that may be comatose. Although prognosis will depend on the primary brain injury and neurologic progression over time, many dogs and cats can recover from TBI with appropriate treatment and supportive care. Unlike people, dogs and cats can function well and maintain a good quality of life despite severe TBI and significant compromise to brain parenchyma. If you have any questions regarding traumatic brain injury or any other neurologic disease, do not hesitate to contact the Neurology Service at LIVS. ■



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LIVS Neurology Department:



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Keys to Evaluating Forelimb Lameness in Juvenile Dogs

Matthew J. Morgan, DVM

Juvenile forelimb lameness cases can be challenging and frustrating for many veterinarians. There are numerous differential diagnoses, but when you're looking for "horses rather than zebras," the list becomes much shorter. This article will address key steps to consider during your initial evaluation as well as four common disease processes you will likely come across.

There are several key steps that we consider during the initial presentation. The first and often overlooked step is to take into account the dog's breed and age when developing our differential diagnosis list. Next consider onset and duration of clinical signs. Was this an acute lameness or a more gradual onset? Observe the dog's gait and attempt to characterize the lameness. Observe the animal's gait at a walk and run. Palpate the limbs in standing (weight-bearing allows for better assessment of joint effusion) and recumbent positions. Is the patient offloading when standing? Remember the old phrase "down on sound" when evaluating the patient moving toward you. For patients with a mild forelimb lameness I

bone or joint localized. For good quality orthopedic radiographs patients usually need to be sedated for proper positioning. It is a good idea to obtain radiographs of both limbs for comparison and to help identify subtle changes.

Following a good history, thorough orthopedic examination and properly positioned radiographs your differential list should be getting shorter and shorter. In the next section I will go over the four most common juvenile forelimb lameness conditions seen.

Panosteitis

Lameness localized to the limb in general could be panosteitis, which affects the long bones of large- and giant-breed dogs at 5 to 18 months of age. It results in acute lameness and pain on palpation of the long bones. Its cause is largely unknown.

Panosteitis is characterized by endosteal new-bone formation, giving affected long bones the radiographic appearance of an increased and blotchy density of the medullary canal. The condition can affect multiple bones concurrently or sequentially. Treatment includes supportive care, analgesics and time. Panosteitis tends to be self-limiting but can recur, although it is unlikely to do so in the same limb.

Hypertrophic osteodystrophy

Localization to the distal metaphysis of the long bone in large- and giant-breed dogs that are 3 to 5 months old should lead you to consider hypertrophic osteodystrophy (HOD), an uncommon disease of unknown etiology characterized by marked necrosis, inflammation and hemorrhage in the metaphysis

adjacent to the physis. These changes lead to the radiographic appearance of a pseudo or double physis seen at the distal aspect of the long bones and are pathognomonic for HOD.

Affected dogs exhibit acute onset of swelling over the distal limbs, severe pain, anorexia and fever. The standard treatment for HOD includes supportive care, nutritional support and analgesics. Peracute and severe cases may benefit from a short course of corticosteroids. Antibiotics may be used to treat secondary infections (a specific infectious cause for

HOD has not been identified, nor has a specific pathogen been isolated in these cases). Mild cases often spontaneously resolve. HOD is a self-limiting disease, and recurrence is rare.

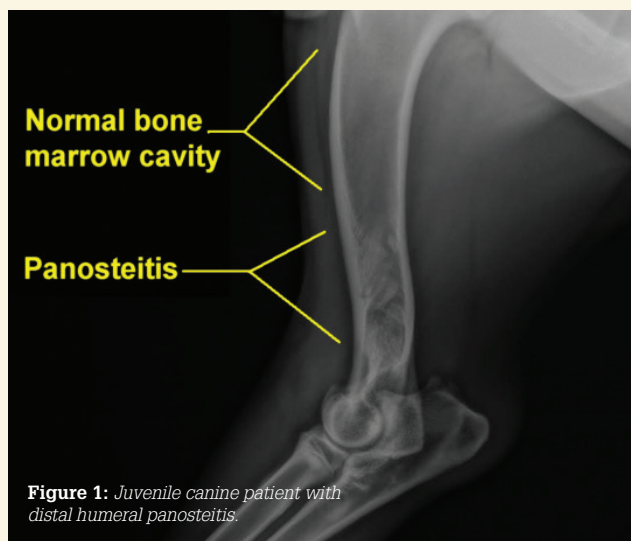
Keep in mind that, as with panosteitis, it is unlikely HOD will recur in a single limb. So if recurrent single-limb lameness is noted in a young dog, further evaluation for other possible causes of the lameness is warranted.

Elbow dysplasia

Lameness localized to the elbow joint is usually the result of elbow dysplasia. The term dysplasia is used to define osteoarthritis of the elbow resulting from one or more of the following: incongruence, ununited anconeal process, fragmented coronoid process or osteochondritis dissecans (OCD) of the humeral condyle. Elbow dysplasia is a polygenetic heritable disease in which one proposed pathogenesis includes incomplete ossification or bony fusion of the anconeus or medial coronoid process to the rest of the ulna. Premature distal antebrachial growth plate closure also can lead to joint incongruence and abnormal wear on articular surfaces.

Elbow dysplasia is common in Rottweilers, Bernese mountain dogs, German shepherds, Labrador and golden retrievers and Newfoundlands 5 to 10 months old. Signs include intermittent or persistent weight-bearing forelimb lameness, a head-bobbing gait, decreased range of motion and joint effusion. Affected dogs often have an elbows-out stance. Most dysplastic dogs have bilateral disease. Radiography and computed tomography are used for diagnosis.

Treatment can be medical or surgical but often involves both. Medical management consists of weight control, controlled exercise and administration of NSAIDs, analgesics and chondroprotectives. Surgical management includes either open or arthroscopic exploration of the joint, removal of fragments and debride-



have found that video recording a patient in slow motion can be a big help when looking for a subtle head bob. Next perform a thorough orthopedic exam in both standing and lateral recumbency to localize the lameness. It is common for puppies, especially in an excited state, not to react to a painful stimulus on palpation. Evaluation of the patient's gait after holding a joint (carpus, elbow, shoulder) in flexion for 20-30 seconds can help with localization.

Following a good orthopedic examination obtain orthogonal radiographic view of the



Continued on Page 10 ►

Keys to Evaluating Forelimb Lameness in Juvenile Dogs

► Continued from Page 9



Figure 2: Canine patient with hypertrophic osteodystrophy. The white arrow is pointing to the radiographic appearance of a pseudo or double physis seen at the distal aspect of the long bones and are pathognomonic for HOD.

ment of fibrillated or malacic cartilage. It is imperative that surgical treatment be followed with good medical management.

All dogs with elbow dysplasia develop arthritis over time and eventually demonstrate some degree of lameness. All will require medical arthritic management for their lifetimes. Surgery is most helpful when dogs are young and still growing and have minimal or no arthritis. Arthroscopy provides a minimally invasive means to explore the joint and treat

several components of elbow dysplasia with generally greater success than conventional surgical techniques.

Osteochondritis dissecans

The most common cause of forelimb lameness localized to the shoulder is OCD, which results from a disturbance of articular or epiphyseal cartilage growth characterized by slow ossification of deep zone cartilage, leading to thickened, poorly nourished articular cartilage sus-

ceptible to trauma experienced with normal weight-bearing. A dissection lesion develops between the subchondral bone and articular cartilage, resulting in the characteristic flap.

OCD affects large-breed dogs, including Labradors and Golden retrievers, Rottweilers, Saint Bernards, German shepherds and Bernese mountain dogs, that are 5 to 10 months old. Most dogs with shoulder OCD have bilateral disease. Signs include forelimb lameness, adduction of the elbow, muscle atrophy over the scapula and pain with flexion and extension of the shoulder. The typical radiographic appearance is that of flattening and sclerosis of the caudal humeral head.

Treatment involves debridement of the cartilage flap and fragmented, malacic cartilage, followed by debridement of the lesion to the subchondral, bleeding bone via an open arthrotomy or arthroscopy. Arthroscopy provides a minimally invasive means to explore the entire shoulder joint, remove the cartilage flap and debride the cartilage defect with generally more rapid recovery than conventional surgical techniques. The prognosis for a dog with an appropriately treated shoulder OCD is good, and clinically significant osteoarthritis in the future is uncommon. □

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The LIVS Surgery Department is staffed by a team of specialty-trained doctors and technicians whose main goal is to provide the best possible outcome for your patient's surgical problem. Our surgeons have years of experience, and are nationally and internationally recognized as leaders in veterinary surgery who are not just adept at performing the latest surgical procedures; they also have developed some of these procedures.

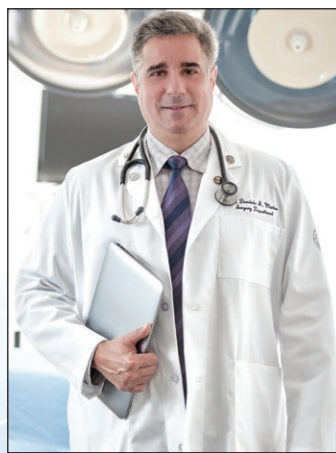
In addition to a wealth of expertise and training, our surgery department has the most advanced, state-of-the-art equipment in order to provide the best surgical management of our patients' conditions. Most notably: cavitron ultrasonic aspirator (CUSA); arthroscopic and laparoscopic towers for minimally-invasive surgery; total hip replacement instrumentation; and stereotactic MRI assisted brain biopsy equipment.



Dr. Curtis W. Dewey
LIVS Since 2000



Dr. Catherine A. Loughin
LIVS Since 2002



Dr. Dominic J. Marino
LIVS Since 1998



Dr. Patrick F. Roynard
LIVS Since 2010



Dr. Fernando J. Leyva
LIVS Since 2011



Dr. Matthew J. Morgan
LIVS Since 2013



Dr. Robert E. Waddell
LIVS Since 2001

Appointments 6 days each week • 24 hour emergency surgery service



**Long Island
Veterinary Specialists**

Where You Refer Your Patient First Makes All The Difference

**Call 516-501-1700 or visit livs.org to refer
your patient to the LIVS Surgical team.**

163 South Service Road, Plainview, NY 11803



Long Island Veterinary Specialists

Where You Refer Your Patient First Makes All The Difference

163 South Service Road, Plainview, New York 11803



Behavior Therapy



To refer your clients to Dr. Poggiagliolmi, call
516-501-1700 or visit www.livs.org



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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