LIVS In Plain View

LONG ISLAND VETERINARY SPECIALISTS

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163 SOUTH SERVICE ROAD ▪ PLAINVIEW, NEW YORK 11803
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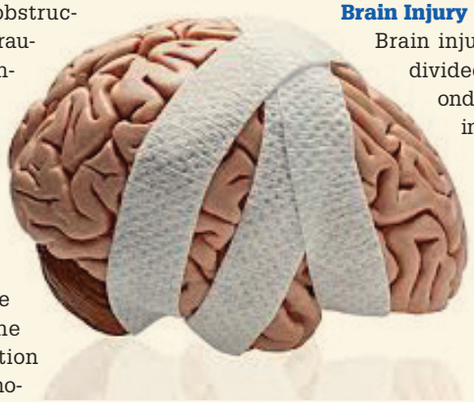
Inside This Issue

Traumatic Brain Injury (Part I)

Ann Bilderback, DVM, DACVIM (Neurology)

Head Injury is a frequent phenomenon in veterinary medicine with the most common causes due to vehicular trauma, missile injury (e.g. gunshot, etc), animal bites, and falls. Controversy exists as to the appropriate therapy for severely brain-injured patients with lack of retrospective and prospective studies pertaining to the treatment of dogs and cats. Consequently, most clinical recommendations for veterinary patients are based on information obtained from human head trauma studies.

It is very tempting when presented with an emergency patient that has severe neurologic signs to rush into evaluating the nervous system. However, traumatic brain injury (TBI) is a multi-factorial disease with possible multi-system trauma and altered hemodynamics necessitating a systematic approach. Initial physical examination should focus on any imminent life-threatening abnormalities and evaluation of vital functions, with the "ABCs" of trauma the primary focus of initial patient assessment. Patients with head injury may have concurrent airway obstruction, pneumothorax, traumatic cardiac arrhythmias, hypovolemic shock, etc., which need to be identified and addressed before evaluating the nervous system. These abnormalities influence not only the interpretation of the neurologic examination but also overall prognosis of the patient. Even a traumatized hypovolemic patient without TBI can appear dull, mentally inappropriate, or tetraparetic due to the hypotensive state but may improve and become BAR and neurologically normal once these abnormalities are addressed.



Pathophysiology of Traumatic Brain Injury

Brain injury can be conceptually divided into primary and secondary injury. Primary brain injury occurs immediately following brain trauma and can include direct parenchymal (brain tissue) damage, such as contusions, lacerations, and diffuse axonal injury, or direct vas-

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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 discover 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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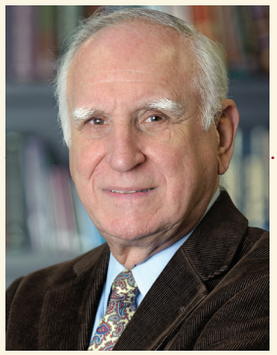
A Note from the Editor



Floodwaters are finally starting to recede from Hurricane Florence, a storm that dumped upward of 35 inches of rain in places and more than 10 trillion gallons across North Carolina, South Carolina, and Virginia. The storm was a 1,000-year rain event, damaging thousands of structures, including toxic animal waste containment sites, which sent bacteria and hazardous chemicals into the water. At least 50 people died as a result of Florence and damages estimates run as high as $22 billion. The Human Society of Missouri rescued a dog left stranded in a flooded house by breaking down the front door of a submerged home to rescue a Maltese terrier who had been floating on a couch inside since the monster storm struck. The rescue was one of many carried out as a result of the storm which left an estimated 5,500 pigs and 3.4 million chickens dead. The Northeast was hardly affected and was able to send rescue teams to the areas involved.

The fall is full of color, and as the seasons change, we greet the new college freshmen at family weekend and the soon to come Thanksgiving holiday break.

Our newspapers and TV channels are replete with opposing commentary on all subjects relating to the White House occupant and Supreme Court nominees; somehow, the US will survive as long as civil discourse is not abridged by those who any label opposing viewpoints "hate speech."

We remain aware of the enormity of the events of 9/11 and on its 17th anniversary, we honor the memory of those lost and remember with pride the involvement of many of the veterinarians from our area especially the members of the LIVS team that answered the call that day. Our hospital administrator, Mr. Brian McKenna and the chief of staff, Dr. Dominic Marino responded at the request of the NYPD and were serving the canine contingent at "ground zero" for 2 days before FEMA arrived. This year, many took part in the run through the tunnel to honor firemen and others who dropped everything to head to the city on that day.

LIVS is looking forward to its upcoming Platinum Series CE on October 2nd at the Crest Hollow Country Club. The topics are varied and timely. Details were mailed out and the spaces have already been filled. Drs Sapienza, Tumulty and Umstead will present.

We are pleased to continue the extend hours for consultation in all our departments to serve our clients more efficiently. Appointments can be made through our telephone receptionists at 516-501-1700.

We welcome your feed-back, e-mailed to lmarino@livs.org.

Leonard J. Marino, MD, FAAP, LVT

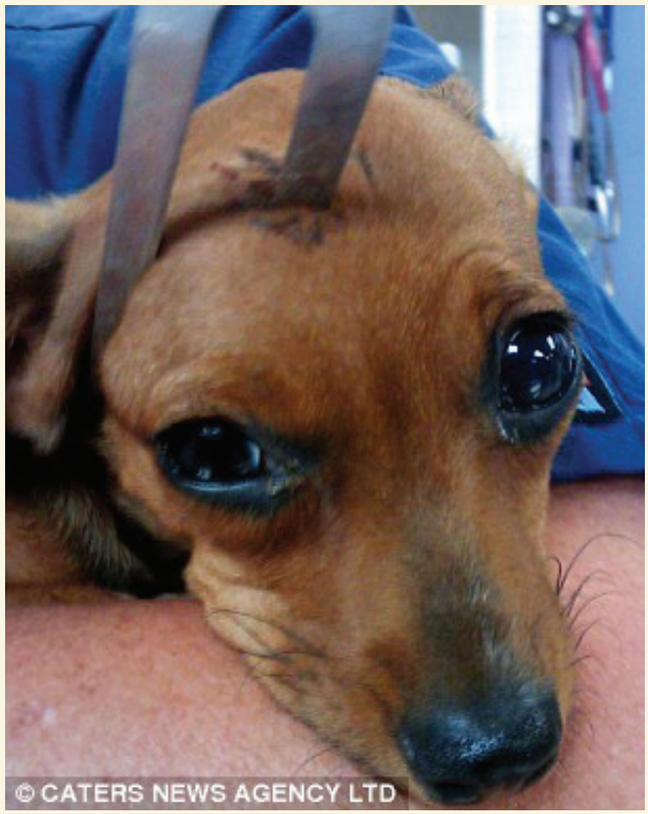
Traumatic Brain Injury (Part I)

➤ *Continued from Front Cover*

cular damage, such as intracranial hemorrhage, vasogenic edema or decreased perfusion. Primary brain injury can initiate biochemical processes that result in secondary injury including energy (ATP) depletion, lactic acidosis, intracellular sodium and calcium accumulation, inflammation and oxygen free radical production, just to name a few. Secondary injury can occur within minutes to days after initial trauma and can occur in already severely damaged brain tissue or relatively normal brain tissue unaffected by the primary TBI. A major determinant of secondary brain injury is compromised oxygen delivery to the brain which results from a combination of intracranial changes and systemic disturbances (e.g. hypotension and hypoxemia).

Both primary and secondary brain injury contributes to increased intracranial pressure (ICP), i.e. the pressure exerted between the skull and the intracranial tissues and fluid. Increased ICP can be dangerous, even fatal, due to the fact that the brain is surrounded by a non-compliant skull which contains the brain (80%), CSF (10%), and blood (10%). Any changes in intracranial volume (e.g. edema, hemorrhage, tumor, etc) results in increased ICP if there are no open skull fractures present. Increased ICP can result in decreased perfusion to the brain, leading to decreased brain oxygenation and nutritional support. When ICP is present, the body's goal is to decrease ICP by first decreasing intracranial CSF volume, then decreasing intracranial blood volume, and by finally displacing intracranial brain volume via herniation of brain tissue out of the back of the skull, through the foramen magnum, which can ultimately be fatal.

The first veterinarian that the TBI patient 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 LIVS, the goals 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. Lowering increased ICP reduces the risk of herniation and improves cerebral perfusion. It is imperative to maintain adequate cerebral perfusion by controlling intracranial pressure, reducing cerebral metabolism and maintaining adequate systemic blood pressure. In order to achieve and maintain intracranial stabilization, stabilization of the patient's cardiovascular and respiratory systems must first be addressed.



Controversy exists as to the appropriate therapy for severely brain-injured patients with lack of retrospective and prospective studies pertaining to the treatment of dogs and cats.

ABCs of Trauma

The basics of Airway, Breathing and Circulation must be evaluated and addressed first in order to prevent further insult to the already injured brain. In human TBI, hypovolemic shock and hypoxemia are strongly associated with elevated ICP and increased mortality. Hypotension, hypovolemia and hypoxemia need to be addressed immediately.

Respiratory

Patency of airway and breathing should be assessed immediately. If partial or complete airway obstruction is present, endotracheal intubation or tracheostomy should be considered. Pneumothorax and pulmonary contusions need to be addressed immediately if present. If pneumothorax persists or returns despite thoracocentesis, a chest tube should be placed. Not only can the presence of a pneumothorax result in hypoxia, but decreased jugular venous return from the intracranial cavity to the thorax due to the pneumothorax can result in increased ICP.

Hypoxia is a concern is any critical patient and supplemental oxygen is indicated in the initial management of patients with TBI, even if no obvious respiratory compromise is evident. Supplemental oxygen can be provided in several ways including flow by, oxygen mask, nasal oxygen, endotracheal intubation, transtracheal oxygen catheter or an oxygen cage. Oxygen masks should be loosely fitted to avoid rebreathing carbon dioxide. When using nasal oxygen, the tip of the catheter should not pass the medial canthus (corner of the eye); if a skull fracture is present in this area the tip of the catheter may enter the cranial cavity if passed too far. Avoid oxygen cages if possible due to risk of hyperthermia and limitations to patient monitoring. Most cases of severe TBI require constant monitoring and evaluation resulting in constant opening and closing of the oxygen cage. As a result, the patient's oxygenation is compromised due to frequent opening/closing of the oxygen cage or monitoring/evaluation of the patient is compromised due to infrequent opening/closing of the oxygen cage in an attempt to preserve patient's oxygenation. If arterial blood gas is available, PaO2 should be >90 mmHg in dogs and >100 mmHg in cats.

It is important to keep in mind that carbon dioxide (CO_2) is a potent cerebral vasodilator. Carbon dioxide should be maintained at the low normal range (venous PCO_2 = 40-45 mmHg; arterial PCO_2 = 35-40 mmHg). Hyperventilation leads to decreased CO_2 (hypocapnea) resulting in vasoconstriction. Hypoventilation leads to increased CO_2 (hypercapnea) resulting in

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Traumatic Brain Injury (Part I)

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vasodilation. Even mild increases in CO2 due to hypoventilation can lead to intracranial vasodilation and further increase ICP. Conversely, hyperventilation resulting in decreased CO2 can cause intracranial vasoconstriction, which in turn can decrease cerebral blood flow and cerebral perfusion, resulting in further cerebral ischemia. If intervention is necessary this can be accomplished by manual or mechanical ventilation with ventilation rates of 10-20 breaths per minute to try and maintain PCO2 in the low normal range. The goal is to try and maintain a balance - not worsen already existing increased ICP by hypoventilation and not worsen cerebral perfusion by hyperventilation.

Circulatory

Maintaining normal blood pressures and adequate hydration status with TBI is imperative and should be restored to normal levels ASAP. Systemic hypotension can result in inadequate cerebral blood flow and cerebral perfusion pressure leading to cerebral ischemia. Additionally, autoregulation of cerebral blood flow may also be impaired resulting in blood flow to damaged brain regions dependent on systemic blood pressure.

If a patient with TBI is hypotensive, volume resuscitation goals should be aggressive. It was previously believed that aggressive volume resuscitation for hypotensive patients with TBI would aggravate intracranial edema. Consequently, volume resuscitation used to be limited in order to dehydrate the patient as this was believed to decrease intracranial interstitial volume. This theory is incorrect and there is no longer any debate as to the disastrous consequences with TBI if hypotension persists. In people, hypotension is a reliable predictor of sustained ICP elevations and increased mortality. With TBI, systolic blood pressures < 90 mmHg is considered hypotension. The goal is to maintain an arterial blood pressure of 80-100 mmHg or systolic blood pressure of 100-120 mmHg.

Volume resuscitation can be achieved with isotonic crystalloids, synthetic colloids, hypertonic saline, and/or blood products depending on concurrent trauma present (hemoabdomen, etc). Hypotensive TBI patients can be treated with isotonic crystalloids. If hypoglycemia is not present, avoid glucose containing fluids to avoid hyperglycemia with TBI. The typical amount administered is 1/4 shock dose (shock dose = 90 ml/kg in dogs, 60 ml/kg in cats) repeated to effect allowing at least 5 minutes between boluses to reevaluate vital signs. The goal is to attain euvolemia and avoid hypervolemia since hypervolemia can result in leakage of intravascular fluids into the intracranial interstital space and elevate ICP. For euhydrated patients, synthetic colloids can be used to restore normal blood pressure and can be used in conjunction with mannitol and/or hypertonic saline to try and reduce elevated ICP. This approach does not work as well in the dehydrated patient where isotonic crystalloids should be used instead. If volume resuscitation is unsuccessful, vasopressor support is indicated (norepinephrine, vasopressin).

Once euvolemia and appropriate ventilation/oxygenation of the patient is attained, the neurologic status of the patient should be assessed. Please see Traumatic Brain Injury (Part II) for further discussion regarding neurologic assessment, specific treatment for elevated ICP and prognosis. ❑

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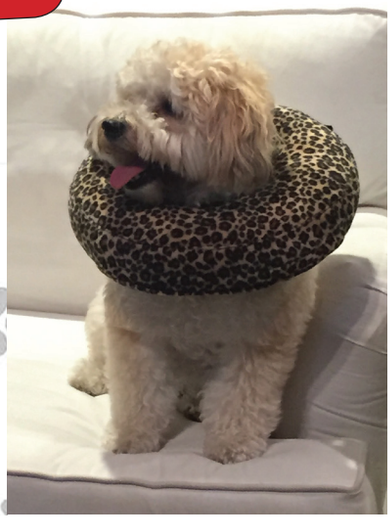

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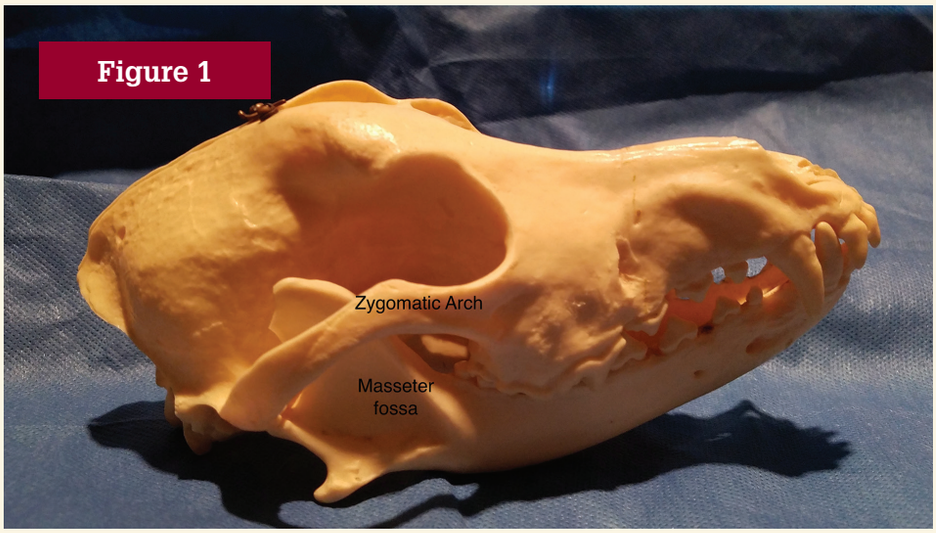
Temporomandibular Joint Disorders in Small Animals

Catherine Loughin, DVM, Dip. ACVS, Dip. ACCT, Staff Surgeon

The temporomandibular joint (TMJ) is a condylar joint, which is the union of two or more bones of the body. At the TMJ the condyloid process articulates in the mandibular fossa of the temporal bone. This fossa generally conforms to the articular surface of the condyloid process (**Figures 1** and **2**). A complete meniscus is between the articular surfaces, making the TMJ a bicondylar joint. The meniscus attaches laterally to the condyle and medially to the temporal bone by ligamentous extensions. Capsular attachments to the meniscus are found circumferentially. The lateral aspect of the capsule is supported by the lateral temporomandibular ligament. This ligament originates from the posterior ventral aspect of the zygomatic arch and inserts on the lateral aspect of the condyloid process and retroarticular process.



The TMJ's movement if more complicated than a simple hinge joint. The mandible rotates about a mutual transverse axis that passes through the center of each condylar process. The medial aspect of the process is firmly set in the fossa throughout the range of motion. The lateral aspect of the process moves in a rostral and ventral curvature during mouth opening and is displaced from the fossa. The masseter, temporalis and pterygoid muscles hold the condyle rigid in the fossa. A small amount of lateral movement occurs during mastication and is limited by normal occlusion.

Anomalies of the TMJ result in the inability to completely open or close the mouth as well as a lateral shift and/or caudal displacement of the mandible. Reasons animals cannot completely close their mouth include: oral foreign body, TMJ luxation or subluxation, mandibular fracture, TMJ dysplasia, open-mouth locking, and trigeminal neuropraxia. Reasons animals cannot completely open their mouth include: TMJ ankylosis from trauma or neoplasia; masticatory muscle myositis; extension of ear inflammation with fibrosis and pain; tympanic bulla neoplasia with fibrosis and pain; and craniomandibular osteopathy.

Physical exam accompanied by skull radiographs or computed tomography (CT) if available, is the best way to diagnose a TMJ disorder. The surgical procedures that are used in the treatment of TMJ conditions include: 1) condylectomy 2) condylectomy combined with caudal mandibulectomy with or without segmental zygomatic arch excision and 3) zygomatic arch partial excision or partial arch excision combined with height reduction of the coronoid process. Below are specific TMJ disorders with their corresponding treatment options.

Intermittent Open Mouth Jaw Locking

This disorder has been associated with TMJ joint dysplasia which then causes joint subluxation and contralateral shifting of the mandible. For example, subluxation of the right TMJ causes the mandible to shift to the left, resulting in contact between the left zygomatic arch and left coronoid process. The interference with coronoid process motion prevents the mouth from closing. Locking does not last long (few seconds to 30-60 minutes) and is corrected when the mouth is opened wider allowing the coronoid process to separate from the arch. This abnormal contact may also result in intermittent oral sensitivity without open mouth locking. Locking episodes occur randomly and usually increase in frequency over time. The majority of animals can spontaneously reduce the lock, but when they are unable then sedated manual manipulation is necessary to restore normal mandible range of motion.

A CT scan should be obtained when available, otherwise a TMJ radiographic series can be acquired. Lateral-oblique views may reveal unilateral signs of joint dysplasia or radiographically normal joints.

Manual reduction of open mouth locking is accomplished by opening the mouth further, pressing the laterally displaced coronoid in a medial direction then closing the mouth. It is possible that manipulation of the mandible may not cause locking. But, the TMJ laxity is often apparent on manipulation and the coronoid process may be displaced laterally to contact the ventral aspect of the arch then slip medially when manipulation is stopped. Neither of these findings are present in normal animals.

Elimination of locking episodes is accomplished by removal of a portion of the involved zygomatic arch. Bilateral surgery may be needed if locking can be produced on both sides by manual manipulation. In brachycephalic breeds locking episodes are associated with the rostral aspect of the coronoid process con-

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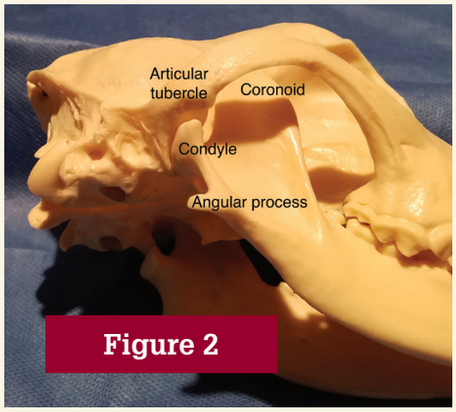

Traumatic Brain Injury (Part I)

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tacting the caudal maxilla. Treatment is surgical reduction of the height of the coronoid process on one or both sides.

Temporomandibular Joint Ankylosis

Ankylosis is immobility of a joint as a result of fibrosis or bony union secondary to disease, injury, or surgery. The clinical result of TMJ ankylosis is moderate to severe reduction in the patient's ability to open the mouth. Common causes are caudal mandibular and/or zygomatic arch fractures and fibrosis from associated soft tissue trauma; and fibrous or calcified adhesions between the arch and coronoid process secondary to trauma, infection, or tumors of the caudal mandible or zygomatic arch.

Most common findings in the history or physical exam include: craniofacial trauma, ear infection, weight loss, inability to completely open the mouth, asymmetry in the regions of the TMJ(s) may be noted due to a neoplasia or fibrous tissue mass. Other findings may include dehydration, atrophy of masticatory muscles, and craniofacial asymmetry. Resulting facial deformities in the immature animal may include mandibular brachygnathia (bilateral ankylosis) or unilateral jaw shift (unilateral ankylosis).

Physical exam, skull radiographs or CT imaging are needed to diagnose TMJ ankylosis. CT with contrast is very beneficial for indicating the extent of the ankylosis. Imaging findings may include TMJ exostosis, joint deformity, irregular bone contours of the caudal mandible and/or zygomatic arch, and soft tissue or bony mass lesions.

Surgical excision of the bone and soft tissues involved is performed with the goal of restoring a more normal range of motion and preventing recurrence of the ankylosis. Ankylosis in the immature animal should be corrected as soon as possible to minimize resultant facial deformity.

Mandibular Condyle Fractures

These fractures may be associated with ipsilateral TMJ subluxation or luxation; other mandibular fractures; fractures of the retroarticular process or fossa of the temporal bone; or a contralateral TMJ subluxation or luxation. Fractures usually occur though the condyle base or neck; however, the condyle itself may be fractured.

Common exam findings include lateral shifting of the jaw toward the affected side and inability to completely close the mouth. A CT scan is best in determining the extent of condyle and fossa fractures. Other cranial bone injuries may be present.

Many condylar fractures that are minimally displaced are managed conservatively with repair of any concurrent mandibular fractures. If normal occlusion is maintained, the patient is fed gruel for two weeks. If occlusion cannot be maintained, then immobilization with a tape muzzle, inter arcade wiring or dental bonding is required. Severely displaced condyle fractures may be primarily repaired, but if necessary, condylectomy results in adequate mandibular function.

Potential complications include malunion, nonunion, degenerative joint disease, and TMJ ankylosis secondary to fibrosis. Condylectomy will preserve mandibular function. Condylectomy with partial mandibulectomy may be needed for treatment of resulting ankylosis.

The TMJ is not a common joint afflicted with disease, but when necessary treatment is available. Sedated manipulation of the jaw/joint as well as radiographs or when available, a CT scan will diagnose the abnormality. Sometimes conservative management will resolve the issue, but if not referral for more advanced surgical correction is recommended. ❑

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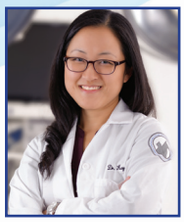

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