When an animal is presented with spinal pain of a neurological origin, there are five possible differentials for the cause of that pain. They are; intervertebral disk disease, neoplasia (of the spinal cord or surrounding structures), diskospondylitis, trauma, and myelitis / meningitis. Occasionally, other disease processes may fit into a list of differentials for spinal pain based on the signalment of the animal, such as atlantoaxial instability, cervical vertebral malformation / malarticulation, or a caudal brainstem mass, all of which may or may not present with cervical pain. Often, animals with orthopedic disease are mistakenly diagnosed with neurological disease simply because there are no radiographic demonstrable signs. Soft tissue injuries of the muscles, joints, or ligaments may be difficult to diagnose. An animal with bilateral cruciate rupture will have a gait that is very reminiscent of an animal with a neurological dysfunction. The key to differentiating neurological disease from orthopedic disease is to remember that animals with an orthopedic problem will not want to use the limb (limping or lameness), and animals with neurological problems cannot use the limb (dragging or weakness).

One caveat to this generalization is an animal that presents with a nerve root signature. A nerve root signature implies compression or disease of a nerve root, which results in pain and is manifested as lameness of a limb\(^1\). These animals may have severe pain in the axillary or inguinal areas if the compression is due to a nerve root tumor. Range of motion of the cervical spine may be compromised if the compression is due to a
lateralized lesion in the spinal cord (such as disk extrusion / protrusion or neoplasia). The differentiation between orthopedic disease and neurological disease is best accomplished by the demonstration of conscious proprioceptive abnormalities. Most animals even with moderate to severe orthopedic disease will not have conscious proprioceptive abnormalities if their weight is adequately supported while performing the test. Of course severe injuries to the bones, such as compound fractures or crush injuries, may also result in nerve damage.

Once it is determined that the problem is neurological, the next step is to localize the lesion. This allows the clinician to focus in on the area of interest and generate a list of differentials that would likely result in the clinical signs. Based on the list of differentials a group of diagnostic tests may be proposed to reach a definitive diagnosis or at the very least a presumptive diagnosis.

Intervertebral disk disease is the most common neurological dysfunction of the spine presented to veterinarians. It has been reported that it accounts for up to 2% of all diseases diagnosed in dogs. The most common area affected is the cervical spine. Disk extrusion in the cranial thoracic spine is seen but uncommon due to the structural reinforcement of the ventral aspect of the vertebral canal by the interarcuate ligament. The most common site in the thoracic spine is the T12 – T13 intervertebral disk space.

Intervertebral disk disease has been classified as Hansen’s Type I or Hansen’s Type II due to the nature of the lesion. Hansen’s Type I disks are extruded into the spinal cord
and tend to affect small, chondrodystrophic breeds such as the Dachshund, Pekinese, Cocker Spaniel and Lhasa Apso. The disk degeneration is typically complete by two years of age. In these cases the nucleus pulposus has essentially lost the qualities that allow it to absorb a compressive force (hydration). The nuclear material degenerates and transfers the compressive force to the dorsal aspect of the annulus fibrosis (which is thinner than the ventral aspect of the annulus). This constant compression leads to the development of fissures and radial tears in the annulus. Eventually the disk ruptures and the nuclear material along with fragments of the annulus are extruded into the spinal canal. This forceful ejection of material will often contuse the spinal cord. The resulting hemorrhage and malacia that may develop after an extrusion may cause neurological deficits by compressing the spinal cord.

Hansen’s Type II intervertebral disk disease usually affects non – chondrodystrophic breeds of dogs. These dogs are often older as the disk degeneration usually is complete toward the end of middle age. This form of the disease is characterized by the protrusion of the annulus fibrosis into the spinal canal and subsequent spinal cord compression. Due to the chronic nature of this disease the protruding disk will often form adhesions to the dura mater. The breeds commonly affected with this form of the disease are the German Shepherd, Doberman Pinscher, Labrador Retriever, and medium to larger mixed – breed dogs. With this form of degeneration the onset is usually slow and progressive. Depending on the degree of compression and the duration of the clinical signs, it may be difficult to demonstrate spinal pain. Many older animals will present with various degrees of conscious proprioceptive deficits manifested as weakness and pelvic limb
ataxia. Pain may not be demonstrated and they are often diagnosed with degenerative myelopathy.

Degenerative myelopathy is both a pathologic condition (degeneration of the spinal cord) that may result from compression and a disease entity based on degeneration of the spinal cord without any demonstrated structural disease. It is extremely important for the clinician to realize that degenerative myelopathy is a diagnosis by exclusion. With the advent of advanced imaging, such as MRI, many animals that have been diagnosed with degenerative myelopathy based on signalment and clinical signs have gone on to have further diagnostics performed and been diagnosed with, treated and recovered from chronic disk protrusion. Even if degenerative myelopathy is suspected it is very important to rule out structural disease of the spine. When intervertebral disk disease is diagnosed in an older dog, clients should also be warned that even though disk disease is present, degenerative myelopathy might be present and either contributing to the clinical signs alone or in combination with the disk disease. In these cases there are two options for the owner, treat the dog as if it has degenerative myelopathy (vitamins, exercise and aminocaproic acid) or remove the disk and hope to see clinical improvement. If there is no improvement then barring any complications of degeneration secondary to the surgery, a presumptive diagnosis of degenerative myelopathy may be may. While it is a gamble to have the surgery performed, I have personally seen many dogs improve with surgery that were diagnosed with degenerative myelopathy prior to any advanced spinal cord imaging.
A diagnosis of intervertebral disk disease may be confirmed with myelography, computed tomography / myelography (CT myelogram), and magnetic resonance imaging (MRI). MRI is considered the gold standard for imaging the spinal cord and surrounding structures. In addition to providing the best visualization of soft tissue structures, the MR allows the clinician to view the spinal cord in multiple views (axial, sagittal, and coronal). Radiographs may give “footprints” of the disease but should not be used as the sole method to make a diagnosis, especially if surgery is considered. More damage can be done by approaching a disk from the wrong side, or by excessive laminectomy trying to find a disk. Myelography has been used for decades for the diagnosis of intervertebral disk disease, however, these days clients are demanding the best for their pets and where available, MRI will provide the best imaging of the spinal cord. The contrast agent used in myelography to outline the spinal cord is known to be neurotoxic and possible complications of myelography include seizures and further clinical deterioration. In addition, myelography is an invasive procedure resulting in trauma to the spinal cord or surrounding structures. There is no doubt that many animals have benefited from a diagnosis received via myelography, however, as veterinary medicine progresses and the standards of care are raised, many believe that if an MR is not offered when available, a myelogram may be considered malpractice. Certainly the availability of MRI is currently limiting its use, however, today the acquisition of an MR unit is comparable to a high-end ultrasound machine. It is also important to know the limitations of an MRI exam. The first is to realize that the MRI will tell you exactly where the problem is, but not necessarily what the problem is. It will give a person experienced in the interpretation of an MRI exam an idea of what the lesion might be (in many cases of intervertebral disk
disease, almost certainty). A few key rules that I like to remember are that no imaging modality will give a definitive diagnosis and no imaging modality should ever be used to determine the prognosis or possible treatment alone. Imaging should be combined with clinical signs. For instance, an animal with mild hind end weakness or spinal pain, does not necessarily need to have decompressive surgery if clinically the animal is getting better or its quality of life is considered good by the owner.

Once a diagnosis has been made, there are basically two options for an owner. The first option is conservative therapy. The second option is surgical decompression of the spinal cord. Of course the clinical signs of an animal may determine which therapy has the best chance of providing a satisfactory outcome. A scale based on clinical signs may be used to guide a client as to which therapy modality may be best for their pet and the relative prognosis based on the animal’s clinical signs. Remember these are only generalizations.

Grading scale used at Seattle Veterinary Neurology and Neurosurgery for the assessment of spinal injury.

<table>
<thead>
<tr>
<th></th>
<th>Description</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>10</td>
<td>Normal</td>
<td></td>
</tr>
<tr>
<td>8</td>
<td>Pain Only</td>
<td></td>
</tr>
<tr>
<td>6</td>
<td>Paresis (walking)</td>
<td></td>
</tr>
<tr>
<td>5</td>
<td>Paresis (not walking)</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>Plegia (urination and deep pain intact)</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Plegia (urination absent and deep pain intact)</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>Plegia (deep pain absent &lt; 48 hours)</td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>Plegia (deep pain absent &gt; 48 hours)</td>
<td></td>
</tr>
<tr>
<td>0</td>
<td>Myelomalacia</td>
<td></td>
</tr>
</tbody>
</table>

Least Severely Affected

Most Severely Affected
In general, those animals at a grade of 6-10 are treated with conservative therapy. However, many owners are not happy with their animals experiencing pain and in cases of inadequate pain control or the inability to follow the guidelines of conservative therapy, may elect to have surgical decompression performed. In the author’s experience, those animals treated with conservative therapy that are at least a grade 6 or higher, have about a 65% chance of not requiring surgery (35% failure rate). They may not get 100% better, however, the owner will feel that their quality of life does not warrant further treatment. I always caution owners that there is a lack of scientific data that support these guidelines and virtually no long-term follow up on cases that are managed conservatively.

Those animals that fall in the 2–5 graded area are typically those that require surgery to improve. There is much debate as to the appropriate course of therapy based on the clinical status of an animal within this group. Most recommendations are based upon the clinician’s personal experience and knowledge of the outcome of past cases. Many believe for instance that emergency surgery is warranted once deep pain is lost (grade 2). I recommend emergency surgery once the animal loses voluntary movement (plegia, grade 4). It is well recognized that a resolution of clinical signs is directly comparable to the degree of dysfunction, prior to surgery and the point at which surgery is performed. Those animals that have a grade of 3–5 typically will have a 75–85% chance of recovery if surgery is performed. Those that are a grade 2 have about a 50% chance of getting deep pain back (not necessarily voluntary movement). Those animals that are graded as 1 have a less than 5% chance of getting deep pain back and those that have
malacia have 0% chance of improvement. One of the benefits of MR imaging in severe cases of intervertebral disk disease or spinal trauma, is the ability to see changes in the spinal cord consistent with malacia. Clinically, animals with lesions demonstrated in the T3 – L3 spinal cord segments that have lower motor neuron signs to the hind limbs should be highly suspect as developing a descending malacia of the spinal cord. Malacia results from a cascade of events leading to spinal cord necrosis. There is no treatment for it. While it may be suspected via an MRI examination, the only way to definitively diagnose malacia is to see it at surgery and this requires a durotomy. The affected spinal cord will often ooze from the durotomy incision. I recommend that these animals be humanely euthanized as most cases of malacia will ascend the spinal cord and result in respiratory compromise and paralysis within several days. Additionally, spinal cord malacia is extremely painful. The prognosis for recovery when deep pain is lost is also different depending on the injury to the spinal cord. Loss of deep pain secondary to intervertebral disk disease should be differentiated from loss of deep pain secondary to trauma. Those animals that lose deep pain after trauma to the spinal cord (fracture or luxation) have a less than 5% chance of regaining deep pain function if the deep pain was lost <48 hours prior to presentation (as opposed to 50% with intervertebral disk disease).

**Conservative Therapy for Intervertebral Disk Disease**

The hallmark of conservative therapy is confinement. This is akin to bedrest in humans. Many clients have a difficult time contemplating confinement for up to six weeks. I try to explain the problem as if it were they who had an injury. Most people have had a
sprained ankle or torn muscle and can relate to the idea that if the injury is not allowed to heal, or a return to normal exercise is considered before the healing is complete, the injury will not heal and may become worse. They also need reassurance that conservative therapy may work and may save their pet a significant amount of surgery and them a significant amount of money. Conservative therapy is best performed if the client has specific instructions and progress goals to look for in the recovery of their pet. This is best accomplished with specific written instructions. Many owners are concerned with the comfort of their animal, both in terms of pain and interaction with the family. Both of these can easily be achieved. Clients are more likely to follow instructions and compliance is best achieved if the treatment process is clearly defined and written instructions, that they understand, are provided. Below is a sample handout; my clients are provided with if they choose conservative therapy.
Conservative and Post Operative Management

Spinal Injury _____ Surgery Type _____

Your pet, _____, has suffered a spinal injury or has had spinal surgery. To help in rehabilitation, post-injury/post-operative care is extremely important. The following rehabilitation steps should be performed if checked:

☐ **Cage Confinement** – Your animal should be confined to an area that is 3’ x 3’ or less. Airline carriers (crates) work well, however, some animals do not tolerate this type of confinement. A baby playpen is an alternative, allowing your animal to see out and interact with the family. In addition certain expandable dog play pens may be obtained. Try to keep your pet off of slipper floors

Please do not allow any running, jumping or going up or down stairs. If your pet stands on its rear limbs when you approach its confined area or runs back and forth in the confined area, then the area is too large and should be reduced.

Cage confinement is used for rest and to minimize spinal strain. Your pet should be kept in the confined area at all times. You may carry your pet outside to allow for urination and defection 3 to 5 times/day.

Your pet should be confined for _____ week(s). Prior to returning your pet to any exercise, a veterinary reevaluation is necessary.

**Restricted Exercise** – If your pet has had a cervical (neck) problem, a harness should be used. Otherwise, the animal should keep confined to prevent further injury. Exercise restriction should be continued for _____ week(s). If at any time your animal appears to have increasing pain or difficulty walking, stop the exercise **immediately** and **consult** your veterinarian.

☐ **Physical Therapy** can be performed as stated on the accompanying physical therapy sheet.

☐ **Return to Exercise** – Returning your animal to exercise should be performed slowly. If cage confinement has been ordered, return to exercise should only be initiated after a recheck examination by a veterinarian. Otherwise, return to exercise should begin with short (5-10 minutes/day) leash walking for the first 1 to 2 weeks. Depending upon how your animal tolerates this amount of exercise, the distance or time of walking can slowly be increased weekly over the next 1-2 months. If at any time your animal appears to have increasing pain or difficulty walking, stop the exercise **immediately** and **consult** your veterinarian.

☐ **Follow-up Communication** – Please contact us and/or your local veterinarian in _____ days to give a verbal update of your animal’s progress.
Medication Schedule:

**Corticosteroids** (Prednisone) – an anti-inflammatory drug that will help reduce swelling and pain.

Dose _____ by mouth every _____ hours for _____ day(s) then _____ by mouth once a day for _____ day(s) then _____ every other day until gone.

This drug may cause increased drinking and urinating. Concerning side effects include gastrointestinal ulcers (blood in stool or vomit). If these latter side effects are noted, contact your veterinarian immediately for assistance.

**Valium (Diazepam)** – an anti anxiety drug that also helps to provide muscle relaxation. This drug may also be given to help ease of urination.

Dose _____ by mouth every _____ hours for _____ day(s)

This drug may cause sedation. If excessive sedation is noted, contact your veterinarian for assistance.

**Phenoxybenzamine (Dibenzyline)** – a drug that helps to relax the bladders sphincter and ease urination.

Dose _____ by mouth every _____ hours for _____ day(s). Best if given with food.

**Antiulcer drug – (_____)**

Dose _____ by mouth every _____ hours for _____ day(s)

**Antiulcer drug – (_____)**

Dose _____ by mouth every _____ hours for _____ day(s)

**Others**

Drug _____ Dose _____ by mouth every _____ hours for _____ days

Drug _____ Dose _____ by mouth every _____ hours for _____ days

Drug _____ Dose _____ by mouth every _____ hours for _____ days

Comments
Use of Corticosteroids and Management of Pain in Animals with Intervertebral Disk Disease.

There are few topics that have received as much debate in veterinary medicine as the use of corticosteroids in the treatment of intervertebral disk disease. The arguments as to the formulation, dose, and duration of steroid therapy are very diverse. The first point to realize is that the only steroid that has ever been shown to be beneficial in the treatment of spinal cord dysfunction is methylprednisolone sodium succinate (solu medrol). Even in the study where it was demonstrated that solu medrol was effective in improving clinical outcome, there is much debate as to the validity of the study itself, and the clinical relevancy of the measured improvement. The study did demonstrate that there was improvement in motor function only when methylprednisolone was given within 8 hours of the spinal cord injury. If it were given after 8 hours, motor function scores actually decreased. Complications associated with high doses of steroids have been well documented.

In general, I avoid the use of steroids unless I can document that trauma resulted within the previous 8 hours, and then I only give methylprednisolone sodium succinate. The first dose is given at presentation if the animal is not hypotensive. The dose is 30 mg/kg given intravenously over 15 minutes followed by 15 mg/kg IV give at 2 hours and 6 hours after the first dose. The 2nd and 3rd dose are also not given if the animal is hypotensive.

I seldom use oral steroid therapy and then only when combined with cage confinement. A dose of 0.5 mg/kg PO q 12 hours (dogs) and 1.0 mg/kg PO q 12 hours (cats) can be
given for 1-3 days with no need to provide a decreasing dose schedule. This is only used to provide comfort and should not be substituted for cage confinement. Methocarbamol is often used as an adjuvant to prednisone therapy or alone. It is a centrally acting muscle relaxant and does not have any direct effect on skeletal muscles. There is very little documented information on the effects of methocarbamol in dogs. I occasionally use methocarbamol especially in situations where there are muscles tremors or excessive muscle tone secondary to an upper motor neuron lesion. Many clinicians use dexamethasone for the treatment of neurological conditions. Complications associated with dexamethasone use in animals can be severe, and those complications usually outweigh any potential benefits. The primary reason I do not advocate its use is that by the time an animal is presented for surgery, it may already have gastric hemorrhage secondary to poor gastrointestinal perfusion and exacerbated by dexamethasone. These are the animals that may benefit from methylprednisolone sodium succinate and are therefore deprived of the potential benefits of solu medrol because of the presence of ongoing complications secondary to long-term dexamethasone administration.

There are numerous methods advocated for the treatment of spinal pain. I often find that many animals are comfortable once cage confinement is instituted. Some may benefit from 1–3 days of low dose prednisone (0.5 mg/kg PO q 12 hours), while others will be comfortable with confinement alone. I do not see as good of results with animals treated with either carprofen or etogesic.
In those animals that still demonstrate spinal pain or discomfort, despite confinement and low dose prednisone therapy, I find that codeine sulfate is the safest and most effective way of treating spinal pain. The most severe side effect of codeine is CNS depression. It should not be used in animals suffering from head trauma, CNS depression or respiratory depression. I prescribe codeine at 0.5 – 1 mg/kg PO every 6 – 8 hours as needed for pain. The drug is very inexpensive and available in small pill sizes (15, 30 and 60mg).

Codeine with acetaminophen should never be given to animals. Tramadol is another alternative and very effective analgesic. It is dosed similar to codeine and considered very safe.

When spinal pain is intractable and non responsive to traditional analgesics then surgery may be warranted to remove the compressive disease.

**Surgical Treatment of Intervertebral Disk Disease**

Most animals will benefit from surgical decompression of the spine. Those that have a lower prognosis include chronic type II disk protrusions and extrusions that result in sever contusion to the spinal cord. As stated above, I consider an animal that has lost voluntary movement to be a surgical emergency. The most important prognostic factor as to whether or not a dog will improve is the presence of deep pain. Deep pain should be assessed in the medial and lateral digits, as well as, the tail. It is very important to remember that withdrawal of the limb does not constitute either deep pain perception or
voluntary movement. Limb withdrawal is a reflex and will occur even if the spinal cord is severed.

Surgical treatments of intervertebral disk disease include laminectomy, ventral slot, fenestration, chemonucleolysis and laser disk ablation. The most common form of decompression of the thoracic and lumbar spine is a laminectomy. In the cervical spine the most common form of decompression is a ventral slot procedure. A hemilaminectomy is performed on the side that demonstrates the most lateralized aspect of the extrusion or protrusion as best define by advanced imaging techniques. This method, when performed correctly has a low complication rate and high success rate, depending on the severity of clinical signs prior to surgery. It provides excellent visualization of the spinal cord and nerve roots and allows for complete removal of the extruded or protruding disk material.

The ventral slot carries an increased risk of complication including hemorrhage, respiratory and cardiac depression. In addition, because the spinal cord is only partly visualized complete removal of the disk material is difficult to demonstrate. Complications secondary to a ventral slot procedure tend to be severe and can result in the death of the animal either during surgery or several hours after surgery. Respiratory and cardiac depression is thought to occur through injury to the vagosympathetic trunk or tectotegmental tracts of the spinal cord.
Prophylactic fenestration remains a highly controversial topic. There have been many studies both demonstrating the benefit of prophylactic fenestration and no benefit. Additionally, it has been shown that fenestration may actually increase convalescent time. The author does not perform prophylactic fenestration. The only disks that are routinely fenestrated are those that are included within the confines of the hemilaminectomy. This is performed to prevent having to approach a previous surgery site, if a second disk extrusion occurs. The recurrence rate of disk disease varies depending on the study sited (2 – 25%), however, in general it may occur in about 10% of cases. One study demonstrated that manual fenestration only removes 41% of the material. This assessment of course, is highly dependent upon the surgeon performing the fenestration and the method of determining the amount of material removed following fenestration.

Chemonucleolysis has been shown to be both effective and contraindicated in treating intervertebral disk disease\textsuperscript{11-13}. Chemonucleolysis is the chemical dissolution of the nucleus pulposus using various digestive enzymes. Problems that result with this technique are potential anaphylactic reaction to the enzyme, dissolution of the annulus fibrosis and leakage of the enzyme into the spinal canal (control of the reaction). This technique is not beneficial for type I intervertebral disk disease, as there is no way to limit the digestion to extruded material alone. With MRI, however, it may be possible to identify type II disk protrusions and treat those cases with digestive enzymes. Further research in this area is greatly needed, however, it is unlikely that it will replace decompressive surgery as the treatment of choice for intervertebral disk disease.
Alternative Therapies for the Treatment of Intervertebral Disk Disease

Very little is known regarding the effectiveness of chiropractic manipulation of the spine following intervertebral disk disease and acupuncture for the treatment of intervertebral disk disease. While it is well recognized that acupuncture may alleviate spinal pain, removal of the disk material is unlikely. Neither acupuncture or chiropractic manipulation should be substituted for cage confinement. Occasionally acupuncture may inhibit further treatment. For instance, gold beads implanted subcutaneously will create an artifact when the spine is visualized using MR and CT imaging. As medical therapies are continually changing and new modalities are being developed, the application of techniques to improve the quality of life of animals should be critically evaluated. An open mind is all that is necessary as long no harm is done.

References


Introduction

The difference between a successful and an unsuccessful outcome after spinal surgery often has little to do with the technique employed or the disease process that is affecting the patient. Rather, it is often the amount of time that takes place between the initial injury to the spine and the point where the spine is either decompressed or stabilized. While decompression is often the main purpose for spinal surgery, stabilization and obtaining a definitive diagnosis are also common goals of spinal surgery. The decision to pursue spinal surgery or continue with conservative management will depend on several variables. The variables we must consider include: the injury to the spine, the stability of the patient, the goals of the owner, the clinical status and signalment of the patient and the owner’s finances relative to the cost of the diagnostics, treatment and aftercare.

Certain disease processes warrant rapid spinal decompression while in other cases a more conservative approach is desired, always in an attempt to either prepare the patient for the best possible outcome of surgery or in some cases to avoid surgery altogether. Surgery should be considered a last resort. It should be employed when there is continual pain that is unresponsive
to reasonable analgesics, deteriorating neurological status, failure for conservative management to resolve the problem and to obtain a definitive diagnosis.

In all cases where spinal surgery is a consideration, some form of advanced imaging technique has often provided a reasonable diagnosis of the problem or at least directed the surgeon toward a reasonable plan. Advanced imaging techniques utilizing MRI are considered the gold standard of imaging the nervous system. However, with certain disease processes, MRI is often combined with or potentially replaced by CT scanning. Radiographs are often considered part of the minimal database prior to advanced imaging. Radiographs of the spine will rarely be definitive as to a disease process. The often show “footprints” of the disease process, however, in a few cases they can provide a definitive diagnosis.

Time, above all is the one variable that we have the most control over. Once an owner is committed to surgery, it is up to us to direct the patient toward the most appropriate diagnostic technique in the right amount of time. Knowing when to say “now” to spinal surgery is quite often the pivotal point in the healing process.

**Intervertebral disk disease**
Patient stability is rarely a cause for concern in cases of intervertebral disk disease with the exception of patients that are overtly painful, anxious and usually – brachycephalic. Of number one concern is the French Bulldog. French Bulldogs are often affected by Type III (explosive) disk herniations. While it is very important to decompress the spine as soon as possible, this breed seems to be particularly prone to hyperventilation, acidosis and hyperthermia. If not calmed down, they may die simply for metabolic reasons pertaining to a painful or anxious condition. The use of anxietolytics such as alprazolam and sedatives such as acepromazine are indicated if a patient is not responding to analgesics alone. Situations where surgery should occur include:

1. If a patient’s clinical status is deteriorating
2. Pain that cannot be adequately controlled.
3. Instability of the spine
4. Paresis with the inability to walk
5. Paralysis
Type I intervertebral disk disease commonly affects chondrodystrophic dogs and consists of a disk herniation into the spinal canal. The damage to the spine is due to both the amount of time it took the disk to herniated and the consistency of the extruded disk material. A jelly like nuclear herniation that occurs rapidly hits the spine with a large degree of blunt force like a water jet. Occasionally, the force of the herniation can be so severe that disk material is actually injected into the spinal cord. A severely compressed spinal cord that gradually occurred over days may not exhibit any contusion at all if the compression was gradual. The gradual compression is usually due to the highly viscous material that oozes out of a small tear in the annulus fibrosis. One can usually predict what the nature of the herniation is based on the clinical course of the neurological deficits. Gradual worsening of clinical signs over weeks to months usually implies a type II disk herniation or a more viscous type I herniation. Acutely affected dogs have usually experienced a type I or type III disk herniation.

Type II disk disease can affect any breed and typically is found in older patients, although not always. In these cases there is dorsal protrusion of the annulus fibrosis. The spinal cord is usually gradually compressed over weeks to months. The clinical deterioration is also reflected by this gradual compression and is typically chronic in nature.
Type III disk disease is considered a surgical emergency. In these cases there is severe contusion to the spinal cord and even though decompression may occur rapidly, often the cascade of events leading to spinal cord malacia has already begun.

The greatest challenge to the clinician will be to try and determine which case is considered a surgical emergency (surgery should occur within 24 hours), which cases will not benefit from further conservative therapy or are in need of more urgent care (surgery in 24 – 72 hours), and which cases can wait.

In general, I consider all cases that do not have the ability to walk to be a surgical emergency if the patient acutely lost the ability to walk. Patient’s whose neurological status is declining over hours or days should have surgery considered within the next few hours or days. Conservative management can be employed in cases of pain, mild weakness or proprioceptive deficits. Guidelines for conservative management should be well established. Patients should be strictly confined to an airline crate, expandable kennel or bathroom with a baby gate. A child playpen will often work well and is especially useful when turned upside down in order to confine cats. Confinement should be for a minimum of two weeks only being let outside to urinate or defecate and then only with appropriate support and a leash. There should be no unrestricted activity.
Appropriate pain medications and analgesics should also be used. Prednisone at a dose of 0.5 mg/kg every 12 hours and either tramadol or codeine at 0.5 – 2 mg/kg every 8 hours can also be used. Large doses of steroids have not been adequately demonstrated to be useful and are usually contraindicated due to the potential negative side affects of steroids in a recumbent or stressed individual. If after two weeks, the patient is improving then conservative management should be continued for a total of 4 more weeks. After 6 weeks of confinement, exercise can be increased by 25% each week thereafter. If at anytime, during the conservative management clinical signs worsen, then advanced diagnostics should be performed.

There are only two reasons to ever consider an MRI. One would be the piece of mind an owner may get from knowing what is either wrong or not wrong with their pet. Knowing what the disease process is will allow the owner to make better decisions for their pet based on a more refined prognosis. A second reason to perform an MRI would be that the information gained from the diagnostic will move the case onward to surgery. If surgery is not an option (for whatever reason) then the best option is conservative therapy.

The pathological changes that occur to the spine following injury are such that unless the spine is decompressed, the damage to the spine may promote a worsening of clinical signs secondary to
an inflammatory cascade. The release of excitatory neurotransmitters secondary to damaged neurons, the action of inflammatory cells that migrate to the area and ongoing compression to the spine are all contributory factors. The one variable that we have the most control over is compression of the spine. The goals of surgery for intervertebral disk disease are to decompress the spine and to allow an environment where repair can occur. Once a neuron in the central nervous system is dead, it cannot be repaired. However, damaged neurons have the ability, to some degree, to be repaired. At least the outer myelin covering can be repaired by oligodendrocytes that migrate back to an area after the compression has been removed.

Magnetic Resonance Imaging (MRI) is the diagnostic modality of choice for the spinal column. MRI allows for visualization of not only the bone, but the soft tissue as well. It allows the clinician to distinguish between the spinal cord, extradural fat, blood vessels, extruded disk material and in most cases, hemorrhage. The purpose of the MRI is do tell the clinician what level of the spinal cord the injury is at, what side of the spinal cord is most severely affected and to a very high degree, what exactly the problems is (a definitive diagnosis). Accurate localization is very helpful in keeping scan times to a minimum, however, it is equally important that all portions of the spine that could account for the clinical signs are examined. For example, if a dog is localized to T3 – L3 and an obvious disk is identified at L1 – L2, it is very important
to examine all the way up to the level of T3 to make sure there are not more than one problem.

Most of the time, the patient is taken directly from MRI into surgery, following a telephone consultation with the patient’s owner.

The three most common surgical techniques are the ventral slot for ventral herniation in the cervical spine, the hemilaminectomy for access to all lateral portions of the spine and the dorsal laminectomy, primarily used to access the lumbosacral space.

Prognosis is dependent on many factors. The most significant prognostic factor is the patient’s clinical status prior to surgery. Of other consideration is the signalment of the patient, with breed having more of a determining outcome than age or sex. The overall injury to the spine, including the type of herniated disks and the number of herniated disks should also be considered. Finally, the time it took from the initial onset of the disease to the time of surgery is also an important consideration, depending on the clinical status prior to surgery.

Trauma
Animals that are struck by vehicles often have multiple traumas that need to be addressed. Of utmost importance is that the patient is metabolically stabilized. Once an animal is stabilized it should immediately be immobilized to prevent further damage to the spine. While the paraspinal muscles provide excellent internal stabilization of the spine, a fractious, painful or mentally inappropriate animal may hurt themselves further by struggling to move. For this reason, the patient should be immobilized on a rigid board. Proper immobilization is important. Use of sedatives and analgesics is appropriate if they will not compromise the animal’s health. Placing a patient on a radiolucent board also allows radiographs to be taken. While a laterally dislocated or fractured spine will often show up on a single lateral radiograph, a ventral dorsal view is necessary to identify dorso–ventrally displaced fractures. A radiograph may not accurately identify the full extent of the damage to the spine as the paraspinal muscles will pull the spine back into normal or semi normal alignment. For this reason, an MRI is indicated to assess the full damage to the spine. Do not assume that a single injury to the spine has occurred. It is always possible that multiple areas of damage may have occurred. For that reason a complete scan of the localized area should be performed.
Blunt force trauma such as falling off a deck or a cliff is managed similarly to animals that are struck by a vehicle. Proper assessment of vital functions is again very important to make sure that the patient is stable enough for further assessment and anesthesia.

Penetrating foreign bodies refer to gunshot wounds, pointy sticks and arrows (another form of a pointy stick). Knives and spears are usually not too much of a concern as they “bounce” off of the spine. Imaging of the spine when there is a metal fragment can pose a problem in the creation of an artifact know as a magnetic susceptibility. This creates a local distortion of the magnetic field around the tissues that are trying to be imaged. One should assume that these wounds are infected. The penetrating object often pulls in fragments of tissue and hair. These wounds need to be debrided as well as having the spinal cord decompressed.

Radiographs of the spine are essential for evaluating the extent of the injury for prognosis and surgical planning. Radiographs alone are not enough for adequate surgical planning, however, they will often show enough information to allow for decisions of whether or not to euthanize. An MRI is necessary to adequately assess the full gross damage that may have occurred to the spine. An MRI will also tell the surgeon whether or not surgery is necessary or if possible external stabilization is possible.
Surgical decompression and stabilization is often necessary to provide the best chance of recovery. Stabilization surgeries are typically 3 – 6 hours in length depending on the degree of injury. Therefore, it is essential that the patient is stable enough for extended surgery. Often these patients have their MRI performed the day before surgery. While it is important to decompress the spine as soon as possible following the injury, one must take into consideration the risk of anesthesia. Trauma cases usually do not undergo further damage to the spine as long as the patient is immobilized. Even then expanding blood clots, ongoing hemorrhage, edema and movement of bone or foreign body fragments may contribute to ongoing injury.

In cases of trauma, the moment to perform surgery is ideally a balance between as soon as possible and as soon as the patient is stable enough to survive the procedure. Most cases do not continue to deteriorate neurologically following trauma as long as the patient is immobilized.

Prognosis is highly dependent upon the degree of injury, the presence of other injuries to the patient and the skill of the surgeon. Stabilization techniques can be taught, however, on the spot engineering is usually necessary to repair the damaged spine, decompress and do so in as little
time as possible. Postoperative radiographs are necessary to assess reduction of fractures or
positioning of implants if they are used.

Neoplasia

Intramedullary neoplastic lesions of the spinal cord typically present with a history of gradual
weakness in a progressive manner. Since there are no pain receptors inside the spinal cord, these
patients do not often show pain at least early on in the disease. Pain will be present once the
meninges are stretched, as this is the location of pain receptors in the spinal cord. The most
common Intramedullary spinal cord tumors include oligodendrogliomas, neuromas and tumors
that have metastasized to the spinal cord. Spinal cord metastasis is not common. Tumors of the
choroids plexus, in the brain, can spread to lower levels of the central nervous system and “seed”
themselves distal from the primary lesion. These are referred to as “drop mets”.

Since neoplasia in this region of the spine is generally gradually progressive, emergency surgery
is often not warranted. Many patients will present with a sudden worsening of clinical signs and
lose the ability to walk as the functional threshold of the spine is passed. In these cases, surgery
should be performed in a timely manner if not only to lessen the complications associated with a
recumbent patient. Because of the anatomical organization of the descending tracts of the spinal cord, Intramedullary lesions in the C1 – T2 segments of the spine will commonly present with clinical signs that are worse in the forelimbs or began in the forelimbs when compared to the rear limbs. Descending spinal cord tracts destined for the forelimbs are located more centrally within the spinal cord, where those on their way to the pelvic limbs are located more superficially in the spinal cord.

Extradural neoplasia and intradural extramedullary lesions tend to behave in the same manner as intramedullary lesions, with the exceptions that the patient often presents with asymmetrical signs and pain is usually a very prominent clinical finding. As the meninges are either compressed or infiltrated with neoplastic cells, pain receptors in the meninges will begin firing. In cases where the compression is extradural, there is the potential that clinical signs may present very acutely. If you think of the spinal cord as a rope made up of thousands of strands, the damage to the spine from a growing or expanding compression is like cutting one strand of a rope at a time. Eventually the rope will snap and it will no longer function as it is meant to. The spinal cord behaves much in the same way. The central nervous system can compensate for
compression and loss of function only to a point. Eventually the clinical signs manifest and
sometimes the manifestation is very acute.

Tumors either originating from bone or causing boney destruction may weaken the bones of the
spine to the point where a pathological fracture develops. The most common example of these
tumors are osteosarcoma, chondrosarcoma, multiple myeloma, plasmacytoma and
hemangiosarcoma. The spinal column can withstand a large amount of insult and weakening of
its structure due to tumors that destroy bone. A sudden abnormal force directed to the spine can
often lead to acute clinical signs of weakness to paraplegia. As with any other disease that
compresses the spine, the time to perform decompressive surgery will depend on the clinical
status of the patient.

The diagnostic tests that are commonly used in cases of neoplasia of the spine typically start with
survey radiographs and may also include CT scan and MRI. Survey radiographs will show you
foot prints of certain disease process such as collapsed disk spaces and proliferation of articular
facets commonly seen in patients with cervical vertebral malformation / malarticulation. Survey
radiographs are only conclusive in cases of diskospondylitis and tumors that either destroy or
produce bone. A three view metastasis check of the thorax is always recommended in patients
greater than 6 years old and earlier in breeds that have a higher incidence of neoplasia of the nervous system such as golden retrievers and brachycephalic dogs.

As in most cases of disease of the nervous system, an MRI scan is used to rule in or out structural disease of the spine. MRI allows visualization of soft tissues and bone. Additionally, it allows for multiplanar scans so better surgical planning can be performed. As with any compressive disease of the nervous system, the scan should include the entire localized area of interest. In cases where a L4 – S1 localization is made, the scan should include T3 – S1, as not all cases follow the rules completely. Cases of C6 – T2 localization should have C1 – T2 scanned. A recent study in JAVMA looked at the accuracy of localizing a lesion based on reduced withdrawal reflexes in the forelimbs. The study showed that decreased withdrawal reflexes were only associated with C6 – T2 lesions 65.8% of the time (JAVMA, Vol 232, No. 4, page 559 – 563). Additionally, the higher up the lesion in the cervical spine, the less likely the localization was to be accurate.

CT scans are often useful when compared with MRI scans to help with neoplasia that is destroying bone. CT scans can also be helpful when surgical planning may involve the use of implants or bone grafts. Deformities of the spine secondary to benign neoplasia can often create
conditions of scoliosis, kyphosis or lordosis making MRI scans difficult due to poor positioning of the patient. CT scans with advanced 3D software can create a virtual picture of the spine that can be manipulated via computer to give the surgeon the best possible assessment of the disease process and allow for better surgical planning.

Most of the same approaches and techniques for other spinal surgeries are used with neoplastic conditions. Often a myelotomy is necessary in order to remove intramedullary tumors. The client should be well informed that with myelotomy, there is greater chance of a negative outcome following the surgery. Myelotomy of the cervical spine can be associated with death due to the disruption of the descending sympathetic pathways in the lateral tectotegmental tract (Acute Sympathetic Cervical Blockade) of the spine and disruption of the cell bodies that give rise to the phrenic nerve in the C3 spinal cord segment. With myelotomy, the goal is to find a plane of dissection between the tumor and the normal spine. This is sometimes facilitated with ultrasonic surgical tools such as a CUSA ultrasonic aspirator. A durotomy or durectomy is used to approach the intramedullary portion of the spine and to remove tumors that are intradural. With this procedure, the dura is either excised and reflected or removed entirely with the tumor. Oftentimes, both sides of the spine are approached in order to create avenues for implant placement and to provide for more complete dissection of neoplastic tissue. Vertebral body
replacement has been performed and utilizes bone graft tissue that is banked by a local supplier. Because of the natural limitation of surgery involving the nervous system, margins are usually not possible. Because the neoplastic tissue is usually involving the structure itself, the structure often needs to be removed in its entirety. Rhizectomy is the severing of a spinal nerve root and is performed when nerve root tumors are removed. I will try to dissect out as much of the nerve as possible. Occasionally amputation of a limb is combined with surgical removal of nerve root tumors. If performed early prior to involvement of the tumor with the spine, a clinical cure of the cancer is possible.

Prognosis is of course dependent on the location of the lesion, the type of cancer and the treatment options. It is very important to remember that even with surgery alone, survival times for certain types of cancer can be years. Some are curable with surgery and others will benefit from adjunctive therapy such as chemotherapy or radiation therapy. Surgery for spinal tumors has two goals; one goal is to decompress the spine in order to improve clinical status and relieve pain, while a second goal is to identify the neoplastic tissue in order to provide for possible adjunctive therapy and to help in prognostication.

Other cases requiring spinal surgery
Congenital malformation of the spinal cord will often present as a gradually progressive clinical picture. Rarely are these animals acutely affected with the exception of cases where because the spinal column was formed incorrectly, the patient is more prone to problems such as trauma and intervertebral disk disease. The most common of these situations is the hemivertebrae or block vertebrae that are often seen in Pugs and French Bulldogs. Other breeds can be affected as well. Cases of scoliosis, lordosis or kyphosis usually progress very slowly, so that by the time the problem is diagnosed, the damage is usually done. Since the patient is “fully cooked” spinal decompression is usually used to stop the disease process with little hope of reversing the clinical signs. Occasionally a problem is diagnosed prior to the full development of the patient. In these cases, early intervention may allow for a better prognosis while the patient’s skeletal structure is still growing. The spinal cord stops growing before the spine so it is possible that by decompressing the spine, the patient will outgrow further compression. Tethered cord syndrome is a good example of this problem. With a tethered cord, a connection is maintained between the meninges and the skin. As the patient grows the spinal cord (usually at the level of S1 – S3) is dragged up to the skin due to the tether. This puts a gradual compression on the spine and leads to typically incontinence both fecal and urinary and sometimes rear limb weakness. The tether may also create a connection between the dura and the outside environment. A meningocele or
myeloceole may develop. These patients usually have a wet spot right above the base of the tail.

The wet spot is CSF leaking onto the skin. These cases should have surgery performed in a timely manner (within the week of diagnosis) in order to give the patient’s spine the best chance of continued growth and development and to lessen the chance of meningitis forming from the CSF communication. The patients are usually diagnosed at a young age when the owner notices the fecal and urinary incontinence. The skin over the base of the tail may have a “dimple”.

Tethered cord can happen to any breed but is more common in Boston terriers, English Bulldogs and Manx cats.

For the same reason tethered cords are taken care of early, spinal trauma leading to malformation should also be addressed as soon as possible. The goal here is to decompress the spine and stabilize it if needed as early as possible in order to provide the best chance that the patient will outgrow the compression and since the spinal cord is often still growing, this may also allow the spine to develop normally in a situation where it received early damage. If implants are necessary to stabilize the spine, a second surgery may be necessary in the future to remove the implants if they cause complications or possibly to place additional imparts as the patient grows.
Atlantoaxial malformation is not considered a surgical emergency, however stabilizing the patient with a neck brace is considered very important. Surgical stabilization should be performed within a week or two of the diagnosis. Since many of these patients are acutely affected, early decompression and stabilization will give the best long – term results. Surgery may be delayed in very young patients in order to let the bones develop so that the implants will have better purchase. Surgical decompression and stabilization is the treatment of choice for this condition and if no complications are encountered with surgery (complication rate is 25% - 50% depending on technique) the outcome is generally good. Yorkshire terriers represent about 50% of the cases of atlantoaxial instability. It can present at any age and clinical signs are often very acute following a jump or after rough play. A single lateral radiograph of the cervical spine usually provides a definitive diagnosis, however, MRI is still utilized to may sure no secondary lesions such as a spinal cord syrinx are present. Syrinxes complicate the surgery and greatly affect the surgical approach.

Cervical Vertebral Malformation / Malarticulation (CVMM or Wobblers’ syndrome) is typically a gradually progressive disease process. Surgery is usually not an emergency procedure as the goals of surgery and the technique must be taken into consideration. The goal of surgery usually is not to reverse the clinical signs, but rather to prevent the patient from becoming worse. For
this reason surgery should be performed as early in the disease process as possible. As soon as a CVMM case is suspected, diagnostic testing and surgery should be performed. Waiting several months to see if the clinical signs improve will lessen the chance of a positive outcome. Steroids may help with clinical signs but are not doing anything to remedy the primary disease process. Radiographs of the cervical spine will typically show changes to the involved articular facets that give them a “blurry” appearance instead of having crisp definition between the articular surfaces. MRI and multiplanar imaging is necessary to see how severe and at what levels the compression is located. Surgical planning is dependent on whether or not the patient has the “old dog” form of CVMM such as found in Dobermans, or the “young dog” form that is common in Mastiffs and Great Danes. The young dog form tends to have a worse prognosis. This may be due to the size of the patient and limitations to post op physical therapy, or the fact that these young dogs tend to be “fully cooked” once the disease is discovered. Surgery involves both decompression and stabilization using a variety of techniques. The exact engineering of the stabilization apparatus is dependent on the extent of the lesion and the financial means of the owner. Titanium plates with locking screws and bone implants carry the best prognosis, however, older techniques are still considered good choices.

Steroid Use
Much controversy surrounds the use of steroids in dogs and cats suspected to or confirmed to have intervertebral disk disease. Over the years many protocols and therapies have been promoted for the most part based on empirical evidence. Very little scientific evidence is available to support the use of steroids in this manner. It is important to differentiate between the times when steroids may or may not be applicable. We should always remember to “do no harm”. The two main categories that we are concerned with is trauma to the spine due to a sudden compression secondary to a herniated disk and gradual compression of the spine secondary to a slowly or gradually compressive disk. The sudden compressions are usually referred to as Hansen’s Type I intervertebral disk disease. Another classification has been mentioned occasionally in the literature and this refers to “explosive” disks or “Type III disks”. The disks that cause gradual or chronic compression are known as Hansen’s Type II disks.

As clinicians, we have all seen the benefits of steroids and there is no doubt that they can improve clinical status. However, sometimes too much of a good thing can be bad. Side effects secondary to steroids usually preclude their chronic use in patients with intervertebral disk disease.
In 1990 a multi center trial on the use of methylprednisolone sodium succinate (Solu - Medrol®) changed the way that steroids were used in cases of spinal trauma (New England Journal of Medicine, 322: 1405 – 1411, 1990). In this study, it was concluded that a 24-hour infusion of MPSS improved the clinical outcome of those people that received it compared to those that did not receive it or received a placebo. The improvement however, was negligible and essential was the difference between a person being able to lift a finger or slightly lift the wrist. There was, however, a statistical difference and this was received as “ground breaking”. Subsequent studies ended up refuting the previous study and basically saying that in order to be effective the steroid had to be given within 8 hours of the injury. If given after 8 hours a placebo was actually more beneficial. Nonetheless, the data stuck and in veterinary medicine, we modified the protocol to give us the current 30 mg/kg initial dose followed by 15 mg/kg at 2 and 6 hours after the initial traumatic injury.

A recent study (JAVMA, Vol 232, No.3, page 411 – 417) confirmed what many of us have always known; the fact that treatment with dexamethasone resulted in more adverse side effects when compared to dogs treated with other steroids or no steroids at all. Additionally, there was no difference in clinical outcome between the groups that were give steroids (dexamethasone or prednisone) or not given steroids and there was no difference in clinical outcome between the
steroid treated groups. It has been well demonstrated that high doses of methylprednisolone sodium succinate will cause gastric hemorrhage (AJVR, Vol 60, No.8; 977 – 981).

The only steroid I use is prednisone in cases where I think there is a potential for spinal cord inflammation or edema. We all have seen the benefit of dogs that receive steroids and as long as we use appropriate doses, it is unlikely we are going to see negative side effects. In cases of acute suspected or confirmed disk herniation, I recommend using 0.5 mg/kg of prednisone every 12 hours for two weeks, then once a day for two weeks, then every other day for two weeks. This also corresponds to the time we employ conservative management (6 weeks of rest and confinement). Conservative therapy is an option when the patient still has voluntary movement. I do not recommend conservative management in cases where the patient cannot walk on their own. I give it as an “option”, but stress that surgery is the treatment of choice. In cases of chronic suspected or confirmed disk disease, I use the same protocol but try to find the lowest possible dose that still results in a benefit. Remember to tell your clients that the steroids do not remove the external compression; they only help with internal compression secondary to inflammation and edema. The recommended treatment of choice for spinal cord compression is surgical decompression.
Non – steroidal anti – inflammatory medications are usually not beneficial because the lack the lipophillic properties of steroids which are necessary to “get into” the fatty tissue of the nervous system.

Steroids induce ulcers at the anti – mesenteric side of the junction between the transverse and descending colon, so there is little chance that common GI protectants such as acid reducers and sucralfate will be of benefit. Still, we should use them if not only to “cover our bases” in today’s litigious society. Misoprostal, a synthetic prostaglandin probably provides the most benefit by helping to replace that which steroids prevent the body from producing. Using steroids is not wrong, in fact it is a very important factor in managing these cases, just remember to use the appropriate steroid (prednisone) at the appropriate dose (see above) and watch closely for side effects.
Introduction

The approach to intracranial disease should combine an accurate evaluation of the patient via neurological exam, appropriate diagnostic imaging and the possible outcome of therapy (prognosis). An accurate neuroanatomic localization is essential to ensure that the area suspected to have the disease process is evaluated completely. Inaccurate localization usually involves a lack of specific clinical signs that lend evidence to the portion of the nervous system that is affected. While most clinicians are confident in the ability to recognize when an intracranial structure is altered, there are common presentations that can be confusing. Severe cervical pain can often be mistaken for a seizure disorder. Conversely, seizure disorders are sometimes mistaken for cervical pain. Syncope and collapse secondary to cardiac disease may be mistaken for a seizure disorder. Systemic metabolic dysfunction such as renal or hepatic encephalopathy can present with clinical signs associated with lethargy, depression, altered states of mentation and seizures. Seizures secondary to hypoglycemia are difficult to diagnose. An insulinoma may be present in middle aged or older animals, have transient secretion of insulin and therefore sporadic collapse and seizures. When routine blood work is performed, blood glucose levels may be normal. Being able to recognize a specific clinical syndrome is beneficial to clinical practice, however, also being aware of disease entities that might be responsible for the particular clinical signs you are observing is also equally important. The successful outcome of case management begins with a complete
history and a thorough physical and neurological exam. A minimal database consisting of a CBC, biochemical profile and urinalysis is the next step. Additional testing such as fasting and fed bile acids evaluation, serum insulin levels or specific neurotoxin screening should follow. Finally, diagnostic imaging evaluating the thoracic cavity for possible metastatic disease, abdominal ultrasound (if indicated) and advanced imaging such as magnetic resonance imaging (MRI) or computed tomography (CT) is performed.

Treatments for intracranial disease may include conservative therapy, chemotherapy, surgical intervention and/or radiation therapy. Often times, multiple treatment modalities are employed in order to achieve the best outcome. It is important to tailor recommendations to the desires and realistic expectations of the client. It is up to the practitioner to be able to provide the client the best information as far as prognosis so they can make the most informed decision for their pet.

**Common Clinical Entities Associated with Intracranial Disease**

*Seizures:* There are three general categories that seizures can be classified into; primary epilepsy, metabolic disease and structural disease of the brain. Intracranial disease may cause seizures if the disease process involves the forebrain. Any patient that develops seizures secondary to intracranial disease should be treated with anticonvulsants. These may include phenobarbital, potassium bromide, levetiracetam, gabapentin and others. Even if the intracranial disease is removed, seizures may be a persistent clinical sign.
Increased intracranial pressure: The Monro – Kellie doctrine states that within a confined space (the cranial cavity), there is only room for brain, blood and spinal fluid. If anything else is introduced into that space, something has to move or the pressure in that space will increase. For instance, if a brain tumor is present resulting in an addition of mass to the cranial cavity, in order for the space to accept more tissue something has to move. Blood flow to the brain will decrease and normal brain tissue will tend to move down a pressure gradient, which in this case is caudal to the foramen magnum. As the pressure increases brain tissue starts to get pushed under the tentorium cerebelli, the falx cerebri and out the foramen magnum. The Cushing reflex may ensue which results in a decreased heart rate (bradycardia) in order to try to reduce the pressure in the cranial cavity by reducing the amount of blood in the confined space. The most common clinical sign of increased intracranial pressure is altered mentation. As pressure in the cranial cavity increases, metabolic process of the brain slow down due to alterations in blood flow, disruption of neural pathways and alterations and destruction of neurons and their supporting glia. Typically, following an altered state of mentation, pupil changes will be the next indication of a pressure change. As intracranial pressure increases and pressure is placed on the occumotor nucleus pupil size will change. The first pupillary indication of increased intracranial pressure will be anisocoria followed typically by bilateral miosis. As pressure increases and the area around the hypothalamus is compressed, pressure on the origination of the descending sympathetic fibers will result in bilateral mydriasis. Finally, as the pressure continues to increase the brainstem starts to feel the results of compression as the diencephalons starts to move toward the foramen magnum. The ascending reticular activating system (thought to keep us awake) is
affected and quickly followed by cardiac and respiratory pathways. The patient may suddenly die from cardiac or respiratory arrest.

Mannitol is recognized as the most effective diuretic available to decrease intracranial pressure. Most of the beneficial effects of mannitol are thought to arise from its ability to decrease blood viscosity thereby increasing cerebral perfusion pressure. It is also well recognized as a free radical scavenger and osmotic diuretic. By increasing cerebral perfusion pressure to reflect that of systemic blood pressure, vasoconstriction results in a lower intracranial pressure. Mannitol is given as a bolus over 10 minutes at a dose of 0.5–1 gram/kg. The author routinely uses the 1 gram/kg dose. With this dose and a 20% solution of Mannitol, the calculation is simply 5 ml/kg. Although mannitol will not hurt a patient without increased intracranial pressure it is simply known to not be effective in further decreasing intracranial pressure. Contraindications to the use of mannitol include hypovolemia, and current cerebral or intracranial hemorrhage. The latter complication is difficult to be able to assess in veterinary patients. It is safe to assume that when in doubt of the existence of current hemorrhage, give the mannitol regardless. You will save more animals with the use of mannitol that you would hurt if they were concurrently bleeding in the cranial cavity.

Furosemide should be used in combination with mannitol to achieve the maximal effect of reducing intracranial pressure. When to give furosemide in relation to the mannitol is a debatable question. Some feel that furosemide should be given prior to the mannitol in order to decrease the possibility of an initial spike in intracranial pressure that is often
associated with mannitol administration. Others feel that giving furosemide after the mannitol has the greatest effect on decreasing intracranial pressure. The author gives furosemide 15 minutes after administration of mannitol at a dose of 1 mg/kg IV.

In situations where the effects of mannitol and furosemide do not appear to be decreasing intracranial pressure, hypertonic saline may effectively decrease intracranial pressure further. A dose of 0.3 - 0.6 ml/kg of 24% saline may be beneficial. This dose is extrapolated from a human study so it must be emphasized that this is anecdotal evidence for the use of hypertonic saline in this extra label manner. In studies looking at the use of “ultra” hypertonic saline (24% solution) in humans a decrease in intracranial pressure was commonly associated with a concurrent increase in serum sodium concentration by approximately 5 mmol.

There is a direct relationship between cerebral perfusion pressure (CPP), intracranial pressure (ICP) and mean arterial blood pressure (MAP) that can be expressed by the equation:

\[
CPP = MAP - ICP
\]

This relationship basically states that as intracranial pressure increases, cerebral perfusion pressure decreases. Additionally, if mean arterial pressure increases or decreases, there may be a negative effect on cerebral perfusion pressure and intracranial pressure. The maintenance of normal blood pressure through adequate and appropriate fluid
resuscitation is extremely important in managing intracranial pressure. Over hydration, however, may be detrimental by increasing intracranial pressure. Therefore, critical evaluation of systemic blood pressure should be a major tenet to supportive care in patients that are suspected to have or may develop increased intracranial pressure.

Studies have demonstrated that intracranial surgery and specifically a craniectomy (the surgical removal of a portion of the skull without replacement) combined with a durectomy (the removal of the overlying dura) was very effective in decreasing intracranial pressure. Unfortunately, once the craniectomy defect was repaired during closure, intracranial pressure again increased. Subsequently, we assume that removal or biopsy of the disease process, combined with the craniectomy and durectomy may be the most effective way of decreasing intracranial pressure via surgery.

Supportive care: A significant portion of the success associated with treatment of intracranial disease is associated with proper supportive care. As in all neurological patients, thick, clean and dry bedding, maintenance of adequate hydration, elevated head and neck (30 degrees), routine passive range of motion exercises, rotation and massage of down muscles, nutritional support and general stimulation are all essential for a successful outcome. Patients that have received intracranial trauma or had surgery (another form of trauma) are typically some of the most critical patients we see. They demand 24-hour care at a hospital facility, preferably under the watchful eye of multiple specialists such as neurology, internal medicine and critical care. Veterinary technicians
that are trained in a particular specialty are also important components to the operative
and post-operative team.

**Steroids:** The use of steroids in the treatment of head trauma, following intracranial
surgery and in instances where intracranial pressure is thought to be increased is
controversial. It is well recognized that corticosteroids through their anti – inflammatory
actions are beneficial in reducing edema and inflammation. However, corticosteroids
potentiate ischemic injury to damaged neurons. It has also been demonstrated that
humans with elevated blood glucose levels have a poorer prognosis than those with
normoglycemia. This is thought to be due to increased cerebral metabolism at the time of
the head injury. Because corticosteroids result in a transient increase in blood glucose
levels, it is therefore postulated that giving corticosteroids at the time of a head injury
will result in a poorer prognosis. In human medicine it is generally well accepted that the
use of corticosteroids in head trauma is contraindicated. An excellent summary of the
benefits and contraindications of not only corticosteroids but also other treatments for
head trauma as they relate to human medicine is the Brain Trauma Foundation
(www.braintrauma.org).

**Antimicrobials:** The use of antibiotics in instances of intracranial disease is common
practice. However, it is essential to know which antibiotics cross the blood brain barrier
and achieve adequate concentrations in the CNS. Trimethoprim sulfa, clindamycin,
metronidazole and doxycycline are commonly used antimicrobials for treatment of CNS
infections. All attain adequate CNS concentration. Trimethoprim sulfa is effective
against *toxoplasma* and *neospora*. Clindamycin has a broad spectrum of activity against a wide range of bacteria, as well as, protozoal infections. Doxycycline is primarily used to treat rickettsial infections and metronidazole is effective against treating anaerobic infections of the CNS. Metronidazole should be used with caution since one of the possible side effects of this medication is vestibular disease in dogs. Common dosages used to treat giardia, for example can induce toxic side effects. Seizures and central blindness may occur in cats treated with metronidazole. Treatment of the vestibular signs in dogs is through the withdrawal of the medication and it has been shown that oral valium will speed up the recovery.

**Specific Indications for Intracranial Surgery**

*Head trauma*: Surgical treatment of head trauma is indicated in situations where an increased intracranial pressure is not responsive to accepted medical therapies. Primarily this occurs either where there is a mass effect on the brain, such as hemorrhage, or deviation of cerebral parenchyma due to a fractured skull. It should be emphasized that the most important therapy is stabilization of the patient and maintenance of adequate blood volume. Once a patient is stabilized and able to safely undergo anesthesia, imaging can be performed on the brain to assess for ongoing hemorrhage and depressed skull fractures. Some patients will be in a state of coma or stupor whereby, anesthesia is not necessary to perform imaging. Occasionally, less risky anesthesia protocols such as a propofol CRI to maintain sedation are used. Regardless of the form of inhalent anesthesia, it is well recognized that intracranial pressure will be elevated once a patient
is anesthetized. In a patient with an already present increase in intracranial pressure, further increases may precipitate further injury through brain herniation. Therefore, as with any patient with an increased intracranial pressure that may potentially need anesthesia, the risks and benefits of the procedure must be weighed prior to undergoing a potentially risky anesthesia.

In general, if a patient is improving following a head trauma, even if there is demonstrated fractures of the skull, surgery should be delayed until a point where the patient is stable enough to give the best chances of survival following the decompression. Risks associated with depressed skull fractures include the development of a sequestrum of bone, infection (especially if the frontal sinus or inner ear is involved) and sterile abscess or seroma.

Neoplasia: The benefits of surgical treatment of brain tumors in dogs and cats centers around decompression, debulking, biopsy and control of secondary effects of the brain tumor on adjacent brain (seizures and edema). Survival times following surgical removal of brain tumors is highly dependent on the type of brain tumor, its location in the brain, the species involved and the time at which the mass was discovered. Typically, the discovery of tumors in animals occurs at an advanced state of growth of the mass, as compared to humans. We rely on the patient to show us clinical signs of disease rather than tell us when they are feeling the effects of a brain tumor. The advanced state of growth at the time of discovery, therefore often leads to a poorer prognosis. The most important consideration in the possible success of surgical intervention is the location and
accessibility of the tumor. Ideally, all tumors that are superficial and potentially well encapsulated and not infiltrative should be treated with surgery.

The most common brain tumor in dogs and cats is the meningioma. However, the behavior of the tumor is quite different between the species. Meningiomas in dogs tend to be infiltrative and therefore not considered benign. Surgical removal of these tumors is therefore complicated by the poor ability to distinguish tumor tissue from normal brain tissue. Aggressive debulking often leads to the removal of normal brain tissue. Despite this the tumor is rarely removed in its entirety and it would be very misleading for a client to think that a “cure” was possible in the approach to removing a meningioma in a dog. That being said, the author has removed several benign meningiomas in dogs and essentially “cured” the patient of the disease. These successes are more often seen in cats with meningiomas. Feline meningiomas tend to be superficial and well encapsulated. Post-operative complications in cats are reduced when compared to dogs and in general cats tend to tolerate surgical decompression very well. The prognosis for a cat with a superficial meningioma tends to be good. Of course the ultimate determination of a tumor type cannot be made without a biopsy. Surgical biopsy of brain tumors is riskier than CT guided or MR guided biopsy, however, it has the potential to provide a much more likely definitive diagnosis and the added benefits of decompression and debulking at the same time. The ability to approach a brain tumor surgically with the dynamic potential to change the operative goal is essential with any intracranial neoplasia. The surgical plan may change dramatically if once the tumor becomes accessible it is found
that it is either infiltrative or encapsulated. To that end, the surgeon must reasonably
discuss all possibilities that might develop during surgery with the clients.

Mean survival times associated with intracranial surgery are difficult to predict in dogs
and cats. The brain tumor itself is interacting on many levels with systemic function and
possible complications associated either with the primary disease and/or surgery. Still,
many clients like to have a general “feeling” of what the success might be before
committing to brain surgery. It is well recognized that the mean survival time of a patient
with an intracranial mass that has no treatment, whether conservative or surgical is days
to weeks. Conservative treatment alone, primarily consisting of the use of steroids and
anticonvulsants, may be weeks to months. Although, the author has personally treated an
older canine with a suspected meningioma with conservative therapy alone that survived
for nearly two years and had a good quality of life. Surgical treatment will extend
survival times when compared to conservative treatment alone. Surgical treatment
combined with adjunctive therapy such as radiation or chemotherapy has the greatest
potential for long-term survival. The use of steroids in treating patients with
demonstrated intracranial neoplasia can be very beneficial. Often improvement is seen
within hours to days after being treated with moderate doses of prednisone or
prednisolone (0.5 mg/kg BID).

Surgical success is highly dependent on the skill of the surgeon, the location of the mass
and the overall systemic health of the patient. Since intracranial surgery is a relatively
new technique in the management of brain tumors, there is a huge disparity amongst
technical skill in this field. Few residency training programs in neurology have an
adequate caseload and skilled faculty to train future neurosurgeons. The learning curve is
very steep when it comes to intracranial surgery. Therefore, serious consideration should
be given to the ability of the surgeon to perform the proposed operation. Lack of skill
often directs clients away from brain surgery that could potentially have very beneficial
effects for the patient.

Survival times associated with supratentorial masses are significantly longer than those of
infratentorial masses. Adequate exposure to the tumor not only gives the best chance of
removing the greatest abnormal tissue but also reduces damage to adjacent brain and
provides for better decompression. Brain tumors located in the infratentorial space are
naturally more difficult to approach and have a greater risk of damage to surrounding
brain structures. Additionally, since more critical components of the brain are located in
the brainstem, there is a greater risk of intra-operative or post-operative complications
associated with tumors located near the brainstem. It is of course impossible to approach
brain tumors in the lateral or third ventricles without damaging normal brain.

Accessibility to the fourth ventricle is possible through a caudal tentorial approach to the
brainstem and cerebellum. Pituitary tumors and ventral brainstem tumors are best treated
with alternative modalities due to the high risk of complications associated with surgery
at these locations.

The success of brain surgery is also significantly influenced by the general health of the
patient at the time of surgery. Most dogs and cats have had significant systemic
deterioration prior to the diagnosis of the brain tumor due to poor nutrition, stress, pain and the side effects of certain medications that may have been used to treat the patient (such as steroids or NSAIDS). Often following the diagnosis of a brain tumor, a waiting period ensues in order to get the patient to the best systemic health to have a more successful surgical outcome. In a patient that has not been treated with steroids, a few days of steroid therapy at moderate doses, may increase survival due to the decrease in intracranial pressure secondary to the steroids ability to reduce peritumor edema.

**Hydrocephalus:** When medical management of hydrocephalus has failed, surgical shunting of cerebral spinal fluid is indicated. Shunting involves the placement of a ventriculoperitoneal shunt that diverts cerebral spinal fluid from the ventricles to the peritoneal cavity where it is reabsorbed. The shunt is surgically placed in the ventricle cavity and burrowed subcutaneously under the skin to the peritoneal cavity. When the cerebral spinal fluid reaches a pressure that is significant enough to open the diverter valve, fluid will pass into the shunt. Complications from the placement of shunts in animals are most commonly associated with under shunting either from hemorrhage at the surgical site or blockage of the shunt from fibrin deposition or kinking of the shunt tube.

**Caudal Occipital Malformation Syndrome:** Caudal occipital malformation syndrome (COMS) and occipital dysplasia often cause compression of the brainstem and cerebellum to a degree where CSF flow is inhibited. This is thought to result in the formation of a syrinx. A syrinx is a fluid filled cavity within the intramedullary
substance of the spinal cord. COMS is a common clinical entity in Cavalier King Charles Spaniels although other breeds of dogs can also be affected. The most common clinical presentation in Cavaliers is scratching at the shoulder and sudden pain attacks that may look like severe cervical pain. Medical therapy initially involves the use of corticosteroids, which often are able to control clinical signs. The goal is to find the smallest dose of steroids that gives a clinical benefit. If the side effects of steroids prevent their long-term use or they are unable to provide significant improvement, then gabapentin may also have a benefit either combined with the steroids or used by itself. An initial dose of 15 mg/kg every 12 hours is used followed by a 25% increase every two weeks until either the clinical signs resolve or side effects of sedation become too great.

Surgery involves the caudal occipital approach to the brainstem. A craniectomy is performed to expose the cerebellar vermis, portions of each cerebellar hemisphere and a partial dorsal laminectomy of C1. Following this, a fibrous band of tissue is often found compressing the brainstem. There are numerous techniques used to complete the surgery. Some involve the reconstruction of the skull using titanium mesh. The author prefers to cut the fibrous band and reflect the cut edges to the lateral aspect of the laminectomy defect. The tissue is sutured to the adjacent fibrous tissue and muscle with non-absorbable suture material. Clinical improvement is unpredictable. Approximately 30% of the cases will result in reformation of the fibrous band of tissue. Since the band is scar tissue it is not entirely unreasonable that surgery would cause more inflammation and the resulting reformation of the scar tissue. The presentation of clinical signs may occur at an early or late age. It is reasonable to assume that the disease process had been
occurring for some time and that clinical resolution within days to weeks is unreasonable. This is a complicated disease process and surgical correction alone does not appear to be the solution, however, when considered as a last resort for potential improvement it should be considered.

Surgical diseases of the brain have often been thought to be a “lost” cause. As advances in imaging modalities and surgical techniques have occurred over the years, successful management of certain brain diseases via surgery has become more reasonable. Equally important to surgical technique are nursing and supportive care associated with a post-operative patient.