Vet Clin Small Anim 35 (2005) 1389–1409
VETERINARY CLINICS
SMALL ANIMAL PRACTICE

Rehabilitation for the Neurologic Patient

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Neurologic disease presents a unique circumstance in which physical therapy has a critical role in maintenance and recovery of function. Dysfunction of the nervous system can cause loss of motor and autonomic function and a range of sensory abnormalities, including loss of sensation (analgesia), abnormal sensations (paresthesia), and heightened sensitivity to stimuli (hyperesthesia). The secondary effects of these problems can be as debilitating and serious as the primary injury. For example, an animal with a peripheral neuropathy may develop muscle contractures that preclude any chance of recovery of function, and the sequelae to recumbency such as decubital ulcers and aspiration pneumonia may be fatal.

A properly designed rehabilitation program should be an important component of the treatment plan of animals with neurologic disease. Such a program should be designed in conjunction with appropriate treatment of the underlying problem and after special consideration of the origin of the neurologic problem (eg, central [CNS] versus peripheral nervous system [PNS], upper or lower motor neuron disease), the severity of the signs, the cause of the signs, their anticipated progression, and the needs of the owner and the pet. This article describes the pathophysiology of injury and recovery in the CNS and PNS, assessment of the neurologic patient, data on the prognosis and expected course of recovery for a variety of different diseases, and rehabilitation exercises appropriate for neurologic patients.

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0195-5616/05/$ – see front matter
doi:10.1016/j.cvsm.2005.08.004

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ACUTE SPINAL CORD INJURY

Pathophysiology

The most common causes of acute spinal cord injury in dogs and cats include acute (Hansen type 1) intervertebral disk herniations, traumatic injuries (causing spinal fractures and luxations or hyperextension injuries), and vascular events such as fibrocartilaginous emboli (FCE). The types of injury caused to the spinal cord include concussion, compression, laceration, and ischemia (Table 1). The primary injury, whether mechanical or vascular in origin, initiates a cascade of events that causes progressive reduction of perfusion and neuronal necrosis [1]. Most of this secondary tissue damage occurs over the 48 hours subsequent to the injury. Most acute spinal cord injuries are self-limiting (eg, fibrocartilaginous embolism) or can be treated surgically (eg, decompression of herniated disk material). The goal is to maximize the functional recovery mediated by spared neural tissue.

Recovery of function in the CNS does not occur by regeneration of neural tissue but rather by the surviving tissue taking on the functions of those axons that have been damaged [2]. So-called "complete" lesions that physically transect the spinal cord tend to cause permanent paralysis, whereas if there is any tissue still crossing the site of a lesion, there is a potential for recovery. This functional plasticity can be enhanced by performing suitable rehabilitative exercises.

One must distinguish between the types of neural tissue that have been injured to predict the expected recovery and to design the most appropriate rehabilitation program. Vascular and pure concussive injuries tend to cause maximum damage to the spinal cord gray matter, killing neuronal cell bodies [3]. If this occurs at a site of functionally important motor neurons (eg, the fourth and fifth lumbar spinal cord segments giving rise to the femoral nerve), the results are devastating. If the injury occurs at the level of the thoracolumbar junction, where the motor neurons innervate the abdominal wall, there is little functional effect. If the vascular or concussive lesion is extensive, the surrounding white matter tracts are also affected, but a subpial rim of axons are often spared [3]. This observation is important when considering the prognosis. Animals with vascular spinal cord injuries often show a sudden and dramatic improvement over the first week. Initially, a zone of edema surrounding the infarcted area of the spinal cord prevents conduction of action potentials. This edema resolves quickly, allowing a return to function to these areas.

Table 1
Tissue trauma associated with common neurologic problems in dogs

Type of injury	IVDD	Fracture/luxation	FCE
Concussion	+	+	−
Compression	+	+	−
Laceration	−	+	−
Ischemia	+	+	+

Abbreviations: FCE, fibrocartilaginous embolism; IVDD, intervertebral disk disease.

By contrast, compressive lesions are more likely to affect the white matter tracts by damaging myelin, deforming ion channels, obstructing blood flow, and ultimately disrupting axons [4]. Surgical decompression of the spinal cord can cause a dramatic reversal of signs if axonal loss and myelin damage are not significant. Damaged myelin takes time to recover, but remyelination of axons in the CNS can occur, leading to recovery of function. If axons have been disrupted, which is common in chronic compressive lesions, the potential for recovery of function is decreased.

Acute intervertebral disk herniations cause compression and concussion of the spinal cord in varying degrees, producing a mixed white and gray matter lesion [5]. The extent of damage can range from minor, with little loss of actual neural tissue and an expectation of full recovery, to extremely severe, effectively causing a complete spinal cord transection.

Lacerations, most commonly seen in traumatic injuries, have more serious implications because the neural tissue is actually disrupted, producing a truly complete injury. The prognosis for recovery from this type of injury tends to be more guarded for animals presenting with a functionally complete spinal cord lesion.

In some cases, surgical treatment of the primary disease may not be completed owing to financial constraints of the owner or other health issues. For example, following a traumatic injury that causes a spinal fracture, the animal may have severe cardiac arrhythmias that preclude prolonged anesthesia, or the owner may not be able to afford surgical stabilization. In such cases, recovery may be possible with rehabilitation as long as further injury does not occur. The main mechanisms for further injury include instability causing repeated spinal cord concussion and compression and severe persistent compression of the spinal cord. Of these, spinal instability can be addressed by suitable external splinting and management of the animal, but the physical therapist should always be aware of the potential for causing further damage in such cases. One must also consider the effect of ongoing compression of nerve roots as they exit intervertebral foraminae. Nerve root compression can cause severe pain and may be a limiting factor in the management of such cases.

Assessment

Several important questions must be answered by the patient assessment.

- All systems should be reviewed and all health problems identified, including coexisting orthopedic problems.
- The neurologic lesion must be localized accurately to one (or more in the case of trauma) of four different regions of the spinal cord: the first to fifth cervical spinal cord, the sixth cervical to second thoracic spinal cord, the third thoracic to third lumbar spinal cord, and the fourth lumbar to the third sacral spinal cord (Table 2).
- The severity of the lesion must be assessed. The particular parameters to evaluate for the different localizations to generate the necessary information are listed in Table 3.
- The degree of hyperesthesia should be assessed and the potential source of pain identified (eg, postoperative pain, muscle spasticity, nerve root entrapment).

recumbency or holding it off the ground in a position in which it is comfortable. Pressure is applied to the bone of the digits using hemostatic forceps gently at first to stimulate a withdrawal reflex, and then the pressure is increased (the aim is to stimulate the periosteum) until a conscious response is elicited. Deep pain perception is believed to be mediated by small diameter, polysynaptic, diffuse pathways in the spinothalamic and priopriospinal tracts that lie deep within the white matter. As such, a serious injury must occur to interrupt conscious perception of pain. At the time of an acute injury, loss of deep pain perception indicates a functional spinal cord transection. Nevertheless, it does not mean that there is anatomic transection, and, in the long term, loss of deep pain perception does not necessarily imply a complete spinal cord lesion [9].

Tetraplegia with loss of deep pain perception is an unusual presentation because cervical spinal cord injuries severe enough to cause loss of deep pain perception will also cause paralysis of the respiratory muscles and loss of sympathetic tone to the heart, with most patients dying before they reach the veterinarian. The exceptions to this are severe gray matter lesions of the brachial intumescence (usually the result of FCE) that can cause loss of deep pain perception in one or both thoracic limbs while deep pain perception is preserved in the pelvic limbs.

Respiratory function

The most severe and potentially life-threatening grade of cervical injury causes tetraplegia with compromise of respiratory function. It is vital that respiratory function is evaluated in any tetraplegic animal and that hypoventilation or other respiratory compromise (such as aspiration pneumonia) is identified before embarking on exercises that may exacerbate the problem. For example, the weight of water in a hydrotherapy bath may cause decompensation of an animal that is hypoventilating.

Prognosis and Recovery

If the underlying spinal cord disease has been addressed and is not ongoing, any animal that has intact deep pain perception in its affected limbs has the potential to recover useful function.

Paraparesis

For the paraplegic animal, the best prognostic guide is the presence of deep pain perception. Extensive information exists about the prognosis for and rate of recovery of animals that have suffered an acute intervertebral thoracolumbar disk herniation. One study showed a direct relationship between the rate of recovery and body weight and age [9]. A relatively high percentage of dogs that recovered from paraplegia with loss of deep pain perception had persistent mild urinary (32%) or fecal continence (41%). The same study looked at the long-term recovery of dogs with disk herniations that did not regain deep pain perception. Approximately 40% of these dogs recovered apparently voluntary motor function and tail wag, although they did not recover deep pain perception or continence. The mean time to recovery of motor function was just over

9 months, and one dog took 18 months. The recovery of a voluntary tail wag preceded the recovery of pelvic limb function and in most cases was present within a month of injury, serving as a useful prognostic indicator. This motor function is hypothesized to be mediated by surviving subpial axons [9].

Less information is available on the exact course of recovery of dogs that have sustained FCE or traumatic injuries. The most accurate prognostic indicator for both types of injury is the presence of deep pain perception. All dogs with intact deep pain perception have the ability to recover from these injuries unless there is ongoing damage. For example, if a dog with a spinal fracture remains unstable, it could deteriorate to a paraplegic condition with loss of deep pain perception. Some work has been done on the prognosis of dogs with spinal fractures and loss of deep pain perception. If there is displacement of the vertebrae at the time of injury, it is extremely unlikely that there will be recovery of function. If the vertebrae are not displaced, the odds of a recovery of function are improved, although they do not reach the 50% chance of recovery noted with disk herniations [9]. The recovery from FCE is notable in that there can be a rapid improvement in the first 7 to 10 days after injury. This observation probably reflects the fact that the lesion often centers on gray matter, with a zone of surrounding edema affecting the white matter.

There has been much discussion of the phenomenon of "spinal walking." This behavior develops in rodents and cats following surgical transection of the spinal cord and has been postulated to occur in dogs. Nevertheless, in one of the author's (NJO) experience, dogs with traumatic spinal cord injuries in which there is significant displacement of vertebrae and loss of deep pain perception (ie, suggesting an anatomic transection of the spinal cord) do not recover useful motor function despite prolonged efforts at rehabilitation, although they develop pronounced reflex movements in their pelvic limbs. A group of dogs will recover motor function (albeit, disconnected and crude) without recovery of deep pain. These dogs invariably have sustained a disk herniation and have a voluntary tail wag (ie, it occurs when they see their owner). It is likely that these dogs have some intact axons running across their lesion, and that the dogs are more similar to humans in that they do not develop useful spinal walking.

Tetraparesis

There is far less objective information on the rate of recovery of tetraparetic dogs from different types of injury. In general, the involvement of all four legs can make rehabilitation more difficult; therefore, the course of recovery may be more protracted. As noted previously, it is extremely unusual to encounter an acutely injured tetraplegic patient with loss of deep pain perception in all four legs. If one did encounter such a case, the animal would be unlikely to survive. Any animal with hypoventilation as a result of its injury carries a poor prognosis unless it can be mechanically ventilated.

Rehabilitation

The goals of a rehabilitation program for acute spinal cord disease include reducing postoperative and muscular pain, maintaining joint range of motion,

reducing the development of muscle atrophy, and restoring neuromuscular function. These goals can be achieved through a rehabilitation program that incorporates exercise, functional activities, and therapeutic modalities (Table 4) [10,11].

Passive and Reflexive Exercises

Passive exercises should be performed in neurologic patients who lack voluntary movement or strength or whose proprioceptive deficits preclude a normal gait.

Passive range of motion

Placing each joint through a normal range of motion will help maintain joint health in patients who have deficits in voluntary movement [12]. Passive range

Table 4
Guidelines for rehabilitation activities for patients with cervical or thoracolumbar spinal cord disease

Step 1: Immediately postoperatively (neurologic stages 1 and 2)
- Cold-packing the incision
- Range of motion exercises
- Massage of limb muscles
- Nursing care
  - Provide soft, padded, and dry bedding
  - Turn patient at least every 4 hours to prevent decubital ulcers, every 2 hours in ideal situations
  - Keep patient clean and dry
  - Water and food easily accessible
  - Bladder and bowel care
- Assess feet and bony prominences for ulcers or abrasions; protective boots may be used if needed

Step 2: Able to support weight (no limb movements) (neurologic stage 3)
- Passive range of motion exercises
- Standing exercises
- Standing in water
- Neuromuscular stimulation

Step 3: Initial limb movements (neurologic stage 4)
- Passive range of motion exercises
- Standing exercises
- Pregait and weight-shifting activities
- Walking (treadmill, dry land), depending on level of assistance required
- Swimming (with support)
- Neuromuscular stimulation

Step 4: Good limb movements (neurologic stage 4)
- Passive range of motion exercises
- Sit-to-stand exercises
- Balance and coordination exercises
- Walking (treadmill, dry land, sand, snow)
- Swimming (with support)

Step 5: Near-normal gait (neurologic stage 5)
- Balance and coordination exercises
- Walking (longer duration, up inclines or stairs)
- Swimming

of motion (PROM) exercises will not improve strength or muscle mass; active range of motion is necessary to stimulate muscle tissue. PROM should be performed with the patient lying in lateral recumbency on a well-padded surface. The uppermost limbs should be put through gentle flexion and extension of each joint within the patent's comfort zone. In patients with spinal cord injury, there is usually increased muscle tone or spasticity. To overcome this tone, one should avoid placing his or her hands on the bottom of the patient's foot (which may elicit an extensor reflex). Placing graded pressure behind the stifle or in front of the elbow can relax the tone. In severe cases of increased tone, gently flexing the digits may decrease extensor tone. Once each joint has been put through 15 to 20 cycles, each limb may be put through bicycling movements for another 15 to 20 repetitions. The patient is then flipped and the exercise repeated on the contralateral limbs. This exercise should be performed three to four times per day until the patient can ambulate.

Flexor reflex stimulation

In patients with upper motor neuron deficits, elicitation of a withdrawal reflex in the forelimb or hind limb causes active flexion of the elbow and carpal joints or stifle and tarsal joints, respectively, thereby improving muscle tone. This exercise is performed by placing the patient in lateral recumbency and pinching the interdigital skin of the upper limb. As the reflex causes the limb to retract actively, resistance is achieved by the therapist holding the foot, creating a gentle "tug-of-war" in which the patient is pulling more forcefully to withdraw the limb from the therapist's grip. This exercise should be performed for three to five repetitions per limb, three to four times per day.

Patellar (extensor) reflex stimulation

Similar to the flexor reflex, stimulation of the patellar reflex will enhance muscle tone and strength in patients with weak or intact femoral nerves. This exercise should be performed in patients with upper motor neuron deficits to take advantage of their normal to hyperactive extensor reflex. To stimulate contraction of the quadriceps muscles, the patient is placed in a standing position with the hind feet placed squarely on the ground. The animal may require assistance to maintain this position. The patient's hind end is then gently raised (enough to lift their toes off the ground) and lowered, such that the animal is required to support their body weight as their hind end is lowered to the ground. The patient may be kept in a standing position until they start to collapse; at this point, the animal is supported and returned to a standing position. Alternatively, the extensor reflex can be evoked by the therapist placing his or her hand on the bottom of the patient's foot and pressing toward the body. This should be repeated 15 to 20 times, and the exercise performed two to three times per day.

Active Exercises

These activities are designed to improve muscle strength, neuromuscular balance, and coordination in patients who have at least some voluntary movement of their limbs. In patients with acute disease, loss of neuromuscular function

will be of greater importance than muscle atrophy, and the choice of rehabilitation activities will reflect this. In humans with traumatic spinal cord injury, early (within 2 weeks of injury) intervention with resistance training has been shown to improve motor activities and function [13,14].

Sit-to-stand exercises

The sit-to-stand exercise strengthens stifle and hip extensor muscles and is indicated in patients with enough motor activity and strength to stand up with minimal to no assistance. The patient is placed in a sitting position and prompted to stand up on all four limbs. This activity should be repeated three to five times and performed two to three times per day until near-normal movements and gait have been restored. It may be performed before other active exercises; however, if the patient appears too fatigued, the activities should staggered.

Assisted walking

When some voluntary movement is present, having the patient perform several short walks per day will improve muscle strength and neuromuscular coordination. A padded sling (commercially available or one home-made from a stockinette or Vetrap bandaging material) should be used to support the hindquarters as necessary. If recovery is anticipated to be prolonged, a cart or counterbalance wheelchair can be used to facilitate ambulation. Nonslip flooring is ideal to encourage proprioceptive recognition and appropriate limb placement. Commercially available booties may provide additional traction. A land or underwater treadmill may also be used. Treadmill walking has been shown to encourage a consistent and symmetric gait in humans with hemiplegia [15], and buoyancy from an underwater treadmill or pool will help to support the patient's body weight [16]. The patient should be walked slowly for 2 to 5 minutes depending on their ability. It is best to stop before the patient has fatigued, performing multiple short walks per day rather than one or two longer ones.

Ambulation activities

Once the patient is able to walk, even with residual proprioceptive deficits, some resistance may be added to improve muscle condition. This resistance may involve walking up a sturdy incline, briskly in an underwater treadmill, with resistive exercise bands, on sand, or through snow. The depth of water, sand, or snow will influence the amount of resistance against which the patient must work. For underwater activities in postoperative patients, one of the authors (KBH) recommends waiting 7 to 14 days following surgery and confirming that the surgical wound has healed. As is true for assisted walking, resistive walking should be limited to 2 to 5 minutes as dictated by the patient's fatigue level. It may be performed daily to every other day until a normal gait has been restored.

Swimming

Aquatic therapy can be beneficial by minimizing weight-bearing forces [16] and allowing the patient to improve joint range of motion [17] and muscle strength.

placed in a moist towel can be applied to the surgical incision for 10 to 15 minutes. This application may be repeated every 4 hours during the inflammatory period. One should closely monitor patients if moist towels are used during the recovery from anesthesia or sedation (use dry towels in sedated animals). Continued inflammation (pain, redness, and swelling) of the surgical site beyond 48 hours may be indicative of an infection and should be assessed appropriately and managed.

Therapeutic ultrasound

The application of therapeutic ultrasound to soft tissues helps alleviate pain while improving tissue blood supply and healing (speed). Ultrasound may be beneficial for epaxial muscles that are experiencing muscle spasms. Its use is contraindicated over an exposed spinal cord, and continuous mode ultrasound is not recommended in postoperative neurosurgical patients. In nonsurgical patients with acute spinal cord disease and neuromuscular spasm, ultrasound may be applied to the epaxial muscles to help manage pain and muscle spasm.

Neuromuscular stimulation

The application of neuromuscular electrical stimulation (NMES) in patients with acute spinal cord disease may be beneficial to increase tissue perfusion, decrease pain, and delay the onset of disuse muscle atrophy [20,21]. In patients with lower motor neuron disease, stimulation of the affected muscle groups will delay the onset and severity of neurogenic muscle atrophy.

The use of electrical stimulation is preferred for muscle groups that are not already experiencing spasms. It is contraindicated over surgical sites following a laminectomy or pediculectomy until adequate healing has taken place. NMES should be applied to the muscle groups of affected limbs once a day for 15 minutes each until the patient is ambulating with mild-to-moderate ataxia.

CHRONIC SPINAL CORD INJURY

Pathophysiology

Chronic spinal cord diseases are a common and insidious problem in older dogs of large and small breeds. They usually result from degenerative changes of the vertebrae and their associated soft-tissue structures. Examples include cervical spondylomyelopathy ("wobbler" syndrome in all of its forms), Hansen type II intervertebral disk disease of the thoracolumbar and cervical spine, spinal malformations such as atlantoaxial subluxation and spinal stenosis, and cystic diseases such as subarachnoid cysts and syringohydromyelia. Degenerative lumbosacral disease primarily affects peripheral nerves of the cauda equina and is discussed in the section on peripheral neuropathies. Neoplastic disorders also cause chronic compression, and, if the underlying cancer is slow growing or has been treated definitively, rehabilitation should have an important role in the treatment plan.

In general, chronic compressive diseases produce neurologic damage by compressing neural tissue, causing demyelination, deforming axonal membranes, and eventually killing axons [4,22,23]. Recovery will be enhanced by

decompression of the spinal cord if this is viable without causing a dramatic deterioration in signs. Nevertheless, histopathology of chronic compressive diseases such as caudal cervical spondylomyelopathy shows that there is significant gray matter damage [3,23]. This damage may reflect compression of the blood supply to the spinal cord and may also be the result of small concussive injuries to the spinal cord as the spine moves caused by the hypertrophied soft tissue, such as annulus fibrosus, or the hypertrophied bone, such as articular facets. There is potential benefit from strengthening the spinal musculature to minimize any sudden movements and to maintain a normal range of motion in the spine.

Assessment

The approach for assessing the chronically paretic animal is identical to that for the acutely paretic animal. Identification of other chronic conditions such as degenerative joint disease of the stifle joints is extremely important, and long-term secondary effects of the neurologic disease should be noted (eg, chronic urinary tract infections owing to impaired urination). Hyperesthesia may be a significant problem in these patients, in particular in animals with cervical disease. The severity and possible causes of that hyperesthesia should be determined. In addition, owing to the chronicity of the signs, any significant muscle atrophy should be documented and taken into account when designing the rehabilitation program.

Prognosis and Recovery

The expectations and therapeutic goals for recovery are different when dealing with chronic versus acute spinal cord injuries. First, the spinal cord lesion usually results from some underlying often poorly understood structural abnormality of the spinal cord or vertebral column. For example, although wobbler syndrome is postulated to result from underlying instability of the cervical spine, it is difficult to demonstrate instability in radiographic or biomechanical studies. Although the spinal cord may be decompressed surgically and stabilized, this may not correct the abnormality that triggered the problem, or it may change the dynamics of the adjacent spine. A complete cure is unusual, and recurrence of signs is relatively common. As noted in the section on pathophysiology, the role of physical therapy in addressing the actual underlying spinal abnormality may be critical and is a field that needs to be developed. A second problem is that, with chronic spinal cord diseases, the gradual accumulation of damage allows the animal to compensate functionally; therefore, signs become evident once a large amount of irreversible damage is present. The anticipated recovery is not as rapid and complete when compared with that in acute spinal cord injuries. It is preferable to begin conservative or surgical treatment and rehabilitation while the animal is still ambulatory.

The outcome of the surgical management of caudal cervical spondylomyelopathy using a variety of procedures has been reported. In general, even if the animal is nonambulatory, approximately 80% of dogs will recover the ability to walk in the long term, although at least 20% of these recovered dogs will have a recurrence.

Rehabilitation

The goals of a rehabilitation program for chronic spinal cord disease include reducing postoperative and muscular pain, improving joint range of motion, correcting muscular atrophy, and restoring neuromuscular function. These goals can be achieved through a rehabilitation program that incorporates therapeutic modalities and exercise (see Table 4).

Passive and Reflexive Exercises

Passive exercises should be performed in neurologic patients who lack voluntary movement or strength, or whose proprioceptive deficits preclude a normal gait. In patients with chronic disease, joint range of motion will be determined by the chronicity and magnitude of the neurologic deficits. In these patients, baseline values for joint ranges of motion should be determined to establish which joints are the most compromised and will require preferential attention.

Passive range of motion

Placing each joint through a normal range of motion will help maintain joint health in patients who have deficits in voluntary movement and will help restore lost range of motion [12]. The methods for PROM have been described previously. Passive exercises will not improve strength or muscle mass. PROM in chronic patients should be performed three to four times per day until the patient is able to ambulate or has reached a recovery plateau.

Stretching

In joints that have lost range of motion, PROM activity should be combined with stretching exercises to help restore function in the affected joint. The affected joint and adjacent muscles should be prewarmed with a warm pack or massage. PROM should be performed to the joint. Upon reaching the respective endpoint of flexion and extension, the therapist should exert gentle traction to maintain the joint at the upper limit of flexion or extension, respectively. A gentle "bouncing" motion may be applied to assist in the breakdown of periarticular fibrous tissue. Following stretching, a cold pack can be applied to the joint if the patient experiences discomfort.

Flexor and patellar (extensor) reflex stimulation

Flexor and extensor reflex stimulation for patients with chronic neurologic disease is similar to stimulation in patients with acute neurologic disease. The stimulation should be performed 20 times, with two to three sessions per day.

Active Exercises

Active exercises are designed to improve muscle strength, neuromuscular balance, and coordination in patients who have at least some voluntary movement of their limbs. In patients with chronic disease, muscle atrophy may be almost as important as loss of neuromuscular function, and the rehabilitation protocol should address both of these conditions.

Sit-to-stand exercises

As described earlier, sit-to-stand exercises strengthen stifle and hip extensor muscles and are indicated in patients with enough motor activity and strength to stand up.

Assisted and resistive walking, swimming, balance, and coordination exercises

Assisted and resistive walking activities, swimming, balance, and coordination exercises are similar to the walking activities described for patients with acute neurologic problems. These activities are of particular importance in patients with chronic disease because of their potentially protracted recovery.

Therapeutic Modalities

Cold-packing, therapeutic ultrasound, and neuromuscular stimulation

Therapeutic modalities can be used in patients with chronic neurologic problems as described previously for the management of patients with acute neurologic problems. NMES helps recondition muscles atrophied from chronic disuse [21].

PERIPHERAL NERVE INJURY

Pathophysiology

Common causes of peripheral nerve injury include fractures (eg, the femoral fracture that damages the sciatic nerve), intramuscular injection (usually affecting the sciatic nerve), traumatic brachial plexus avulsion, and poor surgical technique. Vascular injuries can also occur, the most common of which is iliac thrombosis in cats causing a distal sciatic neuropathy, but thrombosis of the brachial artery can also cause thoracic limb monoparesis. Peripheral nerves differ from their CNS counterparts in that they regenerate at rates as fast as 1 mm a day [24]. Nerves must be in a Schwann cell environment for this regeneration to occur. Peripheral nerve injuries have three levels of severity [25] as follows:

- In neurapraxia, axonal conduction is lost without disruption of the axon. This injury usually results from compression, transient ischemia, or blunt trauma. Loss of conduction may be a result of myelin damage or insufficient energy to maintain axonal resting potential.
- In axonotmesis, the axon integrity is lost, but the endoneurium and Schwann cell sheath it lies within are still intact, providing the opportunity for regeneration back to the correct target. Successful regeneration may occur, particularly if the axon is damaged close to its target.
- In neurotmesis, the entire structure of the nerve is disrupted. The axon has the ability to regenerate but needs to find a Schwann cell sheath to do so, making it much more difficult. The prognosis for recovery from such injuries is guarded, even with surgical intervention.

In peripheral nerve injuries, one must consider sensation and muscle atrophy. Regenerating peripheral nerves, and indeed any disease causing a peripheral neuropathy, can cause unpleasant abnormal sensations (paresthesia) and hyperesthesia, both of which can result in self-mutilation. A sequela to denervation of a muscle is severe muscle atrophy, which over time may lead to muscle contracture and, in growing animals, to skeletal deformities.

Assessment

In the same manner as for spinal cord injuries, the exact location of the lesion and its severity must be determined by the neurologic examination. The muscles innervated by each nerve should be known [26]. It is also useful to refer to references depicting the cutaneous sensory zones of peripheral nerves [27,28]. Severity of the lesion is determined by assessing the level of motor function and assessing for deep pain sensation. Electrophysiologic evaluation of the muscles and nerves using electromyography (EMG) and nerve conduction velocity studies allows a more detailed description of the severity and course of the injury [29,30]. Muscles that are completely denervated develop spontaneous electrical activity when at rest, although such changes do not appear for at least a week after denervation of a muscle. Nerve conduction studies should be interpreted with care. Immediately after an injury, conduction may be lost across the site of injury, whereas the distal portion of the disrupted nerve can continue conducting for a period of hours to days. As nerves regenerate and sprout to innervate denervated muscles, the size of motor units increases; therefore, the size of motor unit potentials on EMG increases [30].

Prognosis and Recovery

As a rule, neurotmesis carries a poor prognosis unless immediate surgical intervention to reconnect the severed nerve occurs. Animals with axonotmesis or neurapraxia carry a better prognosis. Neurapraxia usually reverses within 2 weeks of injury, although damage to myelin slows recovery to 4 to 6 weeks. The recovery from axonotmesis is governed by the proximity of the injury from the target muscle, the severity of muscle atrophy, and the development of contractures. If the damage has occurred far from the target muscle (eg, at the brachial plexus), by the time the axon has regrown, severe muscle contractures could limit recovery.

Brachial plexus injuries tend to involve the caudal two thirds of the plexus (radial, median, ulnar, and lateral thoracic nerves and the sympathetic innervation of the head) or the complete plexus, although cranial plexus injuries have been reported [27]. It is easy to be misled when evaluating animals with caudal plexus injuries, because there is preservation of musculocutaneous function and elbow flexion. This function is not useful for recovery of the ability to bear weight and should not be used to determine the prognosis. Instead, it is important to test deep pain perception, particularly in the lateral digit [31]. The absence of deep pain in this digit implies severe radial nerve injury. If it does not reappear within 2 weeks of injury, the prognosis for recovery of useful motor function in that limb is guarded.

Rehabilitation

The goals of a rehabilitation program for patients with lower motor neuron injury include restoring and maintaining joint range of motion, improving muscle strength, restoring neuromuscular function, and preventing self-mutilation and trauma to the affected limb. The lack of spinal reflexes and corresponding

muscle tone in these patients poses a unique challenge to their rehabilitation, and emphasis must be placed on restoration of muscle and joint function.

Passive and Reflexive Exercises

Because of the dysfunction of the spinal reflex arc in patients with lower motor neuron deficits, passive exercises should be performed in these patients until a near-normal gait is established.

Passive range of motion, stretching

These options are used in the same manner as in patients with acute and chronic neurologic problems. Patients with lower motor neuron deficits may benefit from stretching of affected and antagonist muscles. Loss of tone to antagonist muscle groups predisposes patients to joint contractures. Massage of a mildly contracted muscle group may also be beneficial in restoring its function and should be performed two to three times per day after prewarming the region.

Flexor and patellar (extensor) reflex stimulation

In patients with a sciatic nerve deficit, elicitation of a withdrawal reflex may not be possible. Nevertheless, progress should be monitored by serial evaluations of the spinal reflex arcs. In patients with weak or intact withdrawal reflexes, stimulation of the flexor reflex will improve muscle tone and neuromuscular coordination. Patients with femoral nerve injury require a lot of assistance to maintain this position. A balance ball (Swiss ball) may be used to support the trunk while slowly lowering the hind limbs to the ground. The patient's hind end is then gently raised (enough to lift their toes off the ground) and lowered, such that the animal is required to support their body weight as the hind end is lowered to the ground.

Radial nerve stimulation

Patients with mild radial nerve deficits will benefit from being challenged to bear weight on their forelimbs. Patients who lack any elbow or carpal extension (eg, brachial plexus avulsion) should not perform this activity until some extensor muscle tone is present. The exercise is performed by placing the patient in a standing position while supporting the trunk and forelimbs. With the animal's forefeet placed squarely on the ground, the amount of weight-bearing support is gradually reduced. When the patient starts to collapse in the forelimbs, the therapist supports the animal and returns the patient to a standing position. A balance ball or custom orthotics may similarly be used to support the patient. The exercise is repeated five times, two to three times per day.

Active Exercises

These activities are designed to improve muscle strength, neuromuscular balance, and coordination in patients who have at least some voluntary movement of their limbs. In certain patients with peripheral nerve disease affecting more than one limb, loss of neuromuscular function may preclude some of these activities.

Sit-to-stand exercises, assisted and resistive walking, and swimming

Patients with sciatic nerve deficits can often perform sit-to-stand exercises because they require active stifle extension but only passive stifle and tarsal flexion.

Balance and coordination exercises

Balance and coordination exercises benefit patients with peripheral nerve injuries. They are performed as described previously.

Therapeutic Modalities

Neuromuscular stimulation

The application of NMES in patients with peripheral nerve disease may delay the onset of neurogenic muscle atrophy and recondition the affected muscles [21,32,33]. When an affected muscle is completely denervated, electrical muscle stimulation is the modality of choice. Affected muscle groups should be stimulated once a day for 15 minutes each.

NEUROMUSCULAR DISEASE

Pathophysiology

Neuromuscular diseases include neuropathies, junctionopathies, and myopathies. The most common neuropathies that require rehabilitation include immune-mediated polyradiculoneuritis (also known as Coon Hound paralysis in dogs), infectious neuritis (eg, *Neospora caninum*), degenerative or toxic neuropathies (eg, either breed related or secondary to diabetes or insulinoma), and compressive neuropathies (eg, degenerative lumbosacral disease). Botulism is the most important junctional disorder that requires rehabilitation. There are many different myopathies, including infectious/inflammatory (immune-mediated polymyositis and protozoal myositis), degenerative (muscular dystrophy), and metabolic myopathies. A wide variety of pathologic processes occurs and needs to be considered carefully before designing a rehabilitation program. For example, an animal with X-linked muscular dystrophy may develop dramatic myonecrosis or myocardial failure after excessive exercise.

In general, diseases of the lower motor neuron cause dramatic and rapid muscle atrophy, and, over time, contractures may develop and restrict joint motion. In addition, there may be involvement of the esophagus, laryngeal, and pharyngeal muscles, causing potentially fatal dysphagia and aspiration pneumonia. These changes can be complicated by hypoventilation, particularly in a recumbent animal. In myopathies and botulism, the heart may be involved, causing yet another potentially fatal complication.

Assessment

Following the standard assessment, specific points that must be assessed in a patient with generalized lower motor neuron disease include the following:

- The severity and distribution of lower motor neuron signs should be recognized by making a distinction between an ambulatory and nonambulatory status and between nonambulatory tetraparesis and tetraplegia.

- Respiratory function should be assessed for evidence of hypoventilation (arterial partial pressure of carbon dioxide by arterial blood gas measurement) or aspiration pneumonia.
- Esophageal, pharyngeal, and laryngeal function should be assessed by careful questioning of the owner about voice changes, coughing after eating or drinking, and regurgitation. Thoracic radiographs should be taken to identify megaesophagus.
- Cardiac function should be assessed. Ideally, echocardiography should be performed in the presence of generalized myopathies.
- The presence and severity of muscle atrophy and joint range of motion should be evaluated to establish a baseline.

Prognosis and Recovery

Although the prognosis and course of recovery are closely linked to the underlying disease, the following general statements can be made:

- Esophageal, pharyngeal, and laryngeal dysfunction worsen the prognosis, particularly if the animal has aspiration pneumonia. This potential complication needs to be remembered by the physical therapist when performing exercises with the animal.
- Hypoventilation to the extent that the animal needs to be mechanically ventilated significantly worsens the prognosis.
- The more severe the muscle atrophy, the more protracted the recovery. The development of muscle contractures can preclude a recovery even when the underlying disease has been resolved.
- If the underlying disease process cannot be cured (eg, X-linked muscular dystrophy, inherited neuropathy such as the laryngeal paralysis polyneuropathy complex), the role of the physical therapist is to palliate the animal's signs. It is very important not to precipitate a crisis by causing aspiration pneumonia or an episode of myonecrosis. Physical therapists can also recommend appropriate protective and assistive devices and prevention and positioning techniques and can provide caregiver instruction for home care and safe transfer techniques in the event of hospice situations.

Some guidelines are available for the expected course of recovery for some of the common self-limiting diseases. The recovery from botulism requires the production of new proteins to replace those bound by the botulinum toxin and usually takes approximately 3 weeks [34]. If the animal can be supported through this period successfully, it should recover. Most dogs with polyradiculoneuritis take 3 to 6 weeks to recover from this immune-mediated disease [35]. In both of these diseases, the animals require intensive physical therapy and supportive care during the recovery period to survive.

Rehabilitation

The goals of a rehabilitation program for generalized neuromuscular disease are determined by the particular disease pathophysiology and specific neurologic deficits. Because generalized weakness and lower motor neuron dysfunction are common clinical signs of most neuromuscular disorders, rehabilitation

of these patients includes attention to housing, maintaining joint range of motion, preventing neurogenic muscular atrophy, and restoring neuromuscular function. These goals can be achieved through a rehabilitation program that incorporates exercise and therapeutic modalities.

Passive and Reflexive Exercises

These exercises are performed as described previously.

Active Exercises

Sit-to-stand exercises, assisted and resistive walking

Active exercises are used in dogs with neuromuscular diseases as described earlier. Walking in an underwater treadmill is particularly useful in patients with generalized neuromuscular disorders because the buoyancy will help compensate for their weakened state [16]. Owing to the muscle weakness and risk of drowning, it is important to maintain control of the patient's head at all times while in the water.

Swimming

When swimming a patient with generalized neurologic disease, it is important to support them at all times using manual assistance or a life preserver. As is true for underwater treadmill use, the therapist must maintain control of the patient's head at all times to prevent drowning or aspiration. These patients fatigue easily; therefore, swimming should be limited to 1 to 3 minutes every 2 to 3 days.

Therapeutic Modalities

Neuromuscular stimulation

The application of NMES in patients with generalized neuromuscular dysfunction may be beneficial to increase tissue perfusion and minimize the onset of neurogenic muscle atrophy. NMES should be applied to muscle groups of affected limbs once a day for 15 minutes each.

SUMMARY

The rehabilitation of dogs with neurologic disease involves a combination of active and passive exercise, functional activities, and therapeutic modalities. The key to maximizing the patient's functional recovery is cooperation and participation of the patient, the owner, and the therapist.

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