Neurology

PERIPHERAL NERVE INJURY

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Traumatic peripheral nerve and nerve root injuries are common in companion animals and may be caused by motor vehicle accidents, fractures (humeral, pelvic, proximal femoral), fracture/luxations (sacroiliac, sacrocaudal), bite wounds, gunshot wounds, and iatrogenic lesions (misplaced intramuscular injections, surgical “misadventures”). The nerve injury may result from compression, crushing, stretching, laceration or complete transection. Clinical signs associated with peripheral nerve injury include: pain, proprioceptive deficits, lower motor neuron type motor dysfunction (paresis/plegia, muscle hypotonia, hypo/areflexia, neurogenic muscle atrophy), and hypo/anaesthesia. Peripheral nerve injuries have been classified based on the degree of functional and structural integrity of the nerve trunk.

Neurapraxia – refers to a transient interruption in nerve function (impulse conduction) due to ischemia and/or mild paranodal demyelination. There is no structural damage to the axons and their supportive connective tissue. Neurapraxia is the mildest form of nerve injury and it is commonly caused by blunt trauma or compression. The degree of proprioceptive and motor dysfunction can be variable, nociception is usually preserved. Neurogenic muscle atrophy is unlikely to occur. Recovery is usually spontaneous, complete and occurs within one to two weeks once the compression and edema resolve. Local demyelination may take 4 to 6 weeks to resolve.

Axonotmesis – few to several axons and surrounding myelin are disrupted (structural damage), but the Schwann cells, their basal lamina and the endoneurium remain intact. Wallerian degeneration occurs. Distally to the point of injury the axons and their myelin sheaths degenerate and undergo phagocytosis. Degenerative changes also occur in the axons proximal to the injury site but usually involve only one to three nodes of Ranvier. Axonotmesis may result from severe stretching or crush injury of the nerve. The degree of proprioceptive, motor and nociceptive deficit is proportional to the number of axons that are damaged. In general, significant neurological dysfunction and neurogenic muscle atrophy are expected. Axonal regrowth occurs spontaneously (1 mm/day) along the connective tissue scaffold, but the time until return to function depends on the extent of injury and the distance from the denervated end-organs.

Neurotmesis – is the most severe type of injury and is characterised by complete severance of the nerve trunk (axons, Schwann cells, supportive connective tissue). It is associated with complete proprioceptive, motor and nociceptive dysfunction (i.e. no deep pain perception). Neurogenic muscle atrophy is severe. As in axonotmesis the distal segment undergoes Wallerian degeneration, but the proximal axons will not regrow to their end-organ since there is no guiding scaffold (Schwann cell basal lamina and the endoneurium have been disrupted). Scar tissue tends to interfere with sprouting axons and may result in neuroma formation. Consequently, surgical intervention is necessary to assist regenerating axons to reach and reinnervate their appropriate end-organs.

The neurological examination, especially if repeated overtime, may help distinguishing between neurapraxia and axonotmesis or neurotmesis, however it might be difficult or even impossible to differentiate severe axonotmesis from neurotmesis on the basis of clinical assessment. Our ability to estimate clinically the severity of a nerve injury is mostly based on the relationship between the diameter of a nerve fibre and its susceptibility to compression. Large myelinated fibres, (supplying mainly proprioceptive function) are the most sensitive to injury. The slightly smaller myelinated fibres controlling motor function are the next most susceptible. Small non-myelinated fibres supplying nociception are the most resistant to compression. Therefore, with increasing compression of a nerve, proprioceptive function will be impaired at first, followed by motor function and finally by nociception. The presence of deep pain sensation in the dermatome of a certain

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nerve implies a much better prognosis than its absence. A thorough knowledge of the dermatomes and autonomous zones of the principal nerves of the limbs is essential to perform an accurate assessment of sensory function. It is important to remember that with brachial plexus nerve root avulsion there may be inconsistency in the pattern of sensory versus motor deficits as the ventral nerve roots appear more susceptible to damage than the dorsal nerve roots. Clinical signs associated with dysfunction of most commonly affected spinal peripheral nerves have been thoroughly described.

The diagnostic investigation for patients with peripheral nerve injury will include:

- a minimum data base consisting of complete blood count, serum biochemistry profile and urinalysis to assess general health condition prior to anaesthesia for electrodiagnostics and/or diagnostic imaging. In certain cases it might be indicated to perform also thoracic radiographs, abdominal ultrasound and a coagulation profile.

- Electrodagnostic tests are useful in assessing nerve integrity, functionality, severity of damage, distribution of nerve injury and in monitoring reinnervation.

Immediately after a brachial plexus or a peripheral nerve injury electromyography (EMG) can be used in the awaken animal to determine if some axons are still functional in the injured nerve and therefore if the muscle is still innervated by that nerve. By eliciting a withdrawal reflex (stimulating an area that the animal can definitely feel) and simultaneously recording EMG activity from a flexor muscle, motor unit action potentials will be recorded if any muscle function remains. For example, the presence of motor unit activity in the biceps brachii muscle of a dog or a cat with brachial plexus injury implies that some functional axons must have survived through the musculocutaneous nerve. However, the absence of compound muscle action potentials does not indicate the severity or permanency of the nerve damage because transient conduction block due to edema may also prevent conduction down a nerve.

Motor nerve conduction velocity (MNCV) and the recorded amplitudes of muscle evoked action potentials provide an accurate evaluation of the severity of the damage to the LMN. F-wave studies allow assessment of ventral nerve root function and may be useful in cases of proximal motor nerve injuries such as brachial plexus avulsion.

Sensory function can be assessed by means of sensory nerve conduction velocity (SNCV) study and cord dorsum potential (CDP). For example, in a dog with sensory dysfunction following brachial plexus injury, the absence of the CDP with a normal SNCV indicates a complete nerve root injury above the dorsal root ganglion.

Intraoperative nerve action potential are used in human medicine in order to identify functional regeneration of an injured nerve and accurately trace the length of regenerating axons and the length of nonviable nerve. These information are extremely helpful to decide whether and exactly where segmental nerve resection and reanastomosis should be performed.

- Survey radiographs are indicated when the nerve injury is likely to be associated with fractures (i.e. humeral, femoral, pelvic, sacrocaudal) or with intramedullary pinning. Ultrasound may be helpful in assessing nerve anatomy and further characterise traumatic nerve lesions in companion animals. We have successfully used ultrasound to visualise sciatic nerve entrapment following penetrating wound in the caudal thigh of dogs previously attacked by a boar. A recent study in human medicine has shown that ultrasound can be a useful diagnostic aid in the determination of the precise localisation of the injured site along the involved peripheral nerve, the type of injury and the diagnosis of neuroma.

Computerized tomographic (CT)-myelography is indicated in the diagnosis of brachial plexus nerve root avulsion since it allows visualization of meningeal diverticula and abnormalities of the spinal nerve roots. In a recent prospective study of 40 human patients suffering severe brachial plexus injuries, CT-myelography was found to be 85% accurate in predicting the intraoperative findings.

MRI provides fine anatomic detail of soft tissue and can be used to visualise nerve structures. In veterinary medicine MRI has been successfully used in the diagnosis of peripheral nerve sheath tumors and will certainly have a role in the diagnosis of peripheral nerve injuries. In human medicine, it has been demonstrated that conventional MRI can detect signal changes in injured nerves and in denervated muscles. In addition, in human medicine, the use of...
custom designed phase array coils has led to the development of MR neurography (MRN). MRN produces images with higher resolution improving the ability to visualize both normal and abnormal peripheral nerves in various regions of the body. These advanced diagnostic imaging techniques may have an important role in the assessment of peripheral nerve and brachial plexus injuries in companion animals in the near future.

Treatment of peripheral nerve injury depends on the cause, the severity and the site of the lesion. In general, anti-inflammatories and analgesics are indicated to relieve inflammation and pain. Physical therapy should be started in the early stages following nerve injury in order to maintain range of motion and minimise muscle atrophy. If the nerve injury produces monoparesis and knuckling such as with radial or sciatic nerve lesions, the paw should be protected by means of commercially available boots, splints or special bandages.

When the primary cause of nerve injury can be identified (trauma by fractured/dislocated bone, excessively long femoral intramedullary pin, entrapment by inflammatory/fibrous tissue) it should be addressed surgically as soon as possible (fracture repair, intramedullary pin removal/shortening, neurolysis).

If the nerve has been severed sharply (e.g. by a piece of glass or a knife), and the wound is not contaminated, immediate nerve repair is recommended. Special microsurgical instrumentation and magnification (ocular loupes or operating microscope) are required to perform a meticulous neurorrhaphy. The surgical repair must be accomplished with the least possible trauma to minimise inflammation and fibrosis. The sutures have to be positioned with no longitudinal and circumferential tension at the repair site. Excessive scar formation at the suture line will markedly decrease the progression of regenerating axons. An essential part of neurorrhaphy is accurate anatomical alignment of the nerve fascicles. Failure to match the fascicles in the two segments prevents regenerating axons from reaching their appropriate end-organs. The surgical techniques (epineural, perineural, nerve grafts) to perform nerve repair have been described.

When the nerve lesion is caused by stretch and/or compressive forces associated with a closed traumatic injury, it is not possible to determine immediately whether the lesion is neurapraxic, axonotmetic or neurotmetic. Therefore medical management (analgesics, anti-inflammatories) and frequent re-evaluation (neurological examination, electrodiagnostics, and if indicated also diagnostic imaging) are advised. It is interesting to note that these guidelines are quite similar to the ones used in human medicine even though patient cooperation during clinical and electrodagnostic assessment is much higher than in veterinary medicine and more sophisticated facilities are usually available. Hence, we can state that frequently the greatest diagnostic aid in patients with closed traumatic nerve injuries is the passage of time. Unfortunately, while awaiting for a possible improvement some complications may occur, these include: muscle atrophy and joint contracture (that can be prevented/ addressed with physiotherapy), abrasion of the dorsal surface of the paw (preventable with adequate foot protection), and paraesthesias with self mutilation. This latter complication results from abnormal sensation in an affected area due to pathological changes in the peripheral and/or central nervous system and may be difficult to control pharmacologically.

In those instances where limb dysfunction is profound and nerve damage is chronic (no improvement after 3 to 6 months post-injury), severe and irreversible, treatment options are quite limited. Muscle-tendon transfer alone or in association with carpal/tarsal arthrodesis may be indicated as a salvage procedure in selected cases. When these techniques have failed or are not indicated and delayed nerve repair is not an option, limb amputation may be the only possibility.

Future developments in the treatment of peripheral nerve injury include the use of neurotrophic and neurotropic factors to stimulate axonal survival and regeneration (trophic effect) and to direct growing axons to their proper target organ (tropic effect).

REFERENCES


