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ATAXIA – RECOGNITION AND APPROACH

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Ataxia is the same as incoordination. It is one of the most important neurological signs to recognize due to its importance in localizing lesions within the nervous system. Ataxia is an inability for the patient to coordinate the position of its head, trunk and limbs into space. Ataxia is a sensory, not motor dysfunction that can only be identified when the patient moves. The type of the ataxia is characterized through a complete neurological examination (mental status, gait and posture, postural reactions, evaluation of cranial nerves and spinal reflexes and pain perception). Special attention should be given to the gait and posture.
Ataxia and weakness (paresis) are often confused with each other. The main difference between ataxia and paresis is that ataxia affects coordination without affecting strength, while paresis affects only strength.

Diagnostic approach

A detailed history should be taken to assist in the identification of the cause of the ataxia. While most patients with ataxia have a primary neurological disease, it is important to know that metabolic diseases (e.g. hypoglycemia, hypocalcemia), toxins (e.g. lead, organophosphates), and drugs (e.g. Phenobarbital, metronidazol) can cause ataxia. Once a detailed history is obtained, physical and neurological examinations should be performed. The neurological examination enables the clinician to identify the type of ataxia. Once the type of ataxia is identified, further diagnostic tests should be performed according to the type of ataxia and the lesion localization.

Types of Ataxia

There are 3 types of ataxia, namely proprioceptive, cerebellar and vestibular.

Vestibular ataxia is the easiest to recognize. Vestibular ataxia is characterized predominantly by a head tilt, the side of the head tilt usually indicating the side of the lesion. Other common signs of vestibular ataxia are leaning, falling, rolling, occasionally circling, strabismus and nystagmus. The severity of vestibular signs depends on a number of factors, but it is usually worse in the acute phase of the disease. It is important to differentiate between central and peripheral central vestibular disease because the differential diagnoses and prognosis differ greatly. Patients with central vestibular disease have changes in mental status (most commonly somnolence) and deficits in proprioceptive positioning and/or hopping. Vertical nystagmus or positional nystagmus (one that changes direction when altering the head position) may also be seen. The proprioceptive positioning deficits are ipsilateral to the head tilt, except in cases of paradoxical vestibular syndrome, where proprioceptive deficits are contralateral to the head tilt. Central vestibular signs are associated with rostral medullary lesions (brainstem) or with lesions in the flocculonodular lobe of the cerebellum, and are commonly caused by encephalitis or tumors. In peripheral vestibular disease, as the lesion involves the inner ear receptors located outside of the brain (petrosal part of temporal bone), the patient does not display changes in mental status or proprioceptive positioning deficits. The nystagmus is always in the same direction, either horizontal or rotatory, but not vertical.

Cerebellar ataxia is characterized by dysmetria (inability to control the rate and range of stepping movements), which is usually manifested by hypermetria (exaggerated step). It is normally easier to recognize a hypermetric gait in the thoracic limbs. It is important to differentiate this sign from thoracic limb spasticity or hypertonicity, which often accompanies proprioceptive ataxia secondary to cervical myelopathies. Hypermetria is

manifested by a prolonged flexion of the step (protraction), while spasticity causes the thoracic limbs to appear rigid or spastic. Other signs characteristic of cerebellar ataxia are head and whole body tremors, intentional tremors, and wide pelvic limb stance and gait. Patients with pure cerebellar ataxia do not display weakness (paresis) or proprioceptive positioning deficits, as they have no involvement of the upper motor neurons, or conscious proprioceptive tracts, respectively. This can be very useful in distinguishing cerebellar from proprioceptive ataxia.

Proprioceptive ataxia is the type primarily related to spinal cord diseases. This ataxia can be differentiated from vestibular and cerebellar ataxias by the absence of head involvement (tremor or tilt). Proprioceptive ataxia may be seen with brain lesions (brainstem, thalamus, basal nuclei, or cortex), but is much milder, and other brain signs are usually more obvious than the ataxia (somnolence, behavior changes, cranial nerve involvement, circling, seizures). As proprioceptive ataxia is commonly associated with spinal cord diseases, this discussion will focus on this aspect. Proprioceptive ataxia is a phenomenon of the spinal cord's white matter, reflecting a dysfunction of the sensory tracts carrying unconscious proprioception (dorsal, ventral, and cranial spinocerebellar tracts, as well as the cuneocerebellar tract). Clinical signs seen with proprioceptive ataxia are truncal sway (wobbliness) and abnormal limb stance and gait such as circumduction, abduction or adduction with the limbs crossing with each other as the animal walks. Proprioceptive ataxia is the very first sign observed with spinal cord compression, and may or may not be accompanied by proprioceptive positioning deficits (conscious proprioception = CP deficits or knuckling). Usually, patients with spinal cord disease have ataxia associated with proprioceptive deficits, however, many dogs with chronic spinal cord disease display ataxia, without proprioceptive (CP) deficits. This can be explained by the fact that the tracts carrying conscious proprioception (fasciculus gracilis and cuneatus) are different from those involved in unconscious proprioception and responsible for ataxia. It is therefore the gait examination (presence or absence of proprioceptive ataxia), and not the evaluation of proprioception (knuckling) that conclusively defines the involvement of the spinal cord.

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