

ATAXIA – CLINICAL APPROACH

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Abstract

Ataxia is defined by the loss of movement coordination and it represents one of the most important clinical signs in localizing the neurological lesion. The ataxic patient finds itself in the impossibility to coordinate head, trunk, limbs and tail position. Ataxia is a sensorial dysfunction that can only be observed when the patient moves.

Ataxia is often mistaken with paresis (weakness of the limbs). Unlike paresis, ataxia only affects coordination and not muscle strength. A detailed patient history should be provided in order to identify 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, hypocalcaemia), 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 localization of the lesion. There are three types of ataxia, namely proprioceptive, cerebellar and vestibular.

Keywords: ataxia, incoordination, neurological examination, issue, localization.

INTRODUCTION:

Ataxia is the same thing as incoordination. It is one of the most important neurological signs that must be recognized due to its importance in localizing lesions within the nervous system. Ataxia is an inability to coordinate the position of the head, trunk and limbs into space. It is a sensory, not motor dysfunction that can only be identified when the patient moves.

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.

Locomotion is thought to be controlled at the level of brain stem; however, an exact anatomic gait center (nucleus) has not been identified. Supratentorial (forebrain) structures are important for voluntary initiation of movement. The cerebellum, while not necessary for the initiation of movement, is important for coordination of movement. Cerebellar influences coordinate and smooth body movements by controlling rate, range and force of limb motion.

MATERIALS AND METHODS:

All animals were investigated according to the same plan.

The patients were examined following the neurological examination form, which contains:

- Status;
- Proprioception;
- Posture;
- Cranial nerves;
- Spinal reflexes;
- Panniculus;
- Perianal reflex.

The general examination plan involves:

- Anamnesis;
- Clinical exam;
- Neurological examination;
- Hematological and biochemical exam;
- Rx and/or ultrasonographic;
- Urine tests (summary, sediment, bacteriological exam);
- Specific tests (Toxoplasmosis, Carre's disease, FIP, Rabies, Neosporosis);
- Hormonal tests (Hypothyroidism);
- RMN/CT;
- CSF exam;
- Cardiological exam;
- Ophthalmological exam.

A detailed anamnesis should be taken to help identify the cause of ataxia.

While most patients with ataxia have a primary neurological disease, it's important to know that ataxia may also be caused by metabolic diseases (e.g. hypoglycemia, hypocalcemia), toxins (e.g. lead, organophosphates) and drugs (e.g. Phenobarbital, Metronidazol). Once a detailed anamnesis is obtained, physical and neurological examination should be performed. The neurological

examination enables the clinician to identify the type of ataxia. Once the type of ataxia is identified, further tests should be performed according to the type of ataxia and the localization of the lesion. Ataxia literally means „lack of order” and is sometimes described as incoordination. Ataxia can result from a variety of anatomical lesions within the nervous system, most commonly of the cerebellum, vestibular system and the spinal cord sensory pathways.

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

Proprioceptive ataxia (Figure 1):

The anatomical diagnosis includes the spinal cord (T3-L3). The neurological signs are: abnormal postural reaction, UMN (upper motor neuron) paresis in limbs and normal to increased spinal reflexes. Eyes and head posture are most affected.



Figure 1. German Shepherd, 9 years old, Male, T3-L3 lesion

Vestibular ataxia (Figure 2):

The anatomical diagnosis includes the vestibular nuclei, the vesicular portion of CN VIII or the vestibular receptors. Neurological lesions can be unilateral or bilateral. Unilateral lesions determine head tilt, leaning, falling or rolling to one side, abnormal nystagmus, strabismus. Postural reactions are normal in peripheral lesions and abnormal in central lesions. Bilateral lesions determine a crouched posture, refuse to move and wide head excursions.

Cerebellar ataxia (Figure 3):

The lesion is localized in the cerebellar cortex. Neurological signs are: broad based stance, symmetrical ataxia, truncal ataxia, intention tremor of the head, vestibular deficits, hypermetria, delayed and exaggerated response to postural reaction testing, menace deficit with normal vision, without paresis or abnormal mentation.



Figure 2. Caniche, 12 years old, Male, Haemorrhagic CVA

Dysmetria is an improper estimation of distance during muscular activity, manifested as a loss of synchronous limb movements. Dysmetria includes both hypo- and hypermetria. Voluntary muscular movement overreaches the intended goal in hypermetria.



Figure 3. Westie, 5 months old, Male, Cerebellar atrophy

Hypermetria is more commonly recognized than hypometria. Both abnormalities are most often associated with lesions of the cerebellum or cerebellar pathways. For example, in case of hypermetria, the loss of cerebellar input that normally stops the flexion phase of gait induces exaggerated movement.

Spasticity is a state of increased muscle tone and commonly results from upper motor neuron (UMN) lesions.

Spasticity is observed in gait as lack of normal flexion or as floating (failure to adequately flex the limbs during gait). Stiffness associated with decreased step length is commonly seen in peripheral neuromuscular apparatus diseases (LMN cell body, nerve roots, peripheral nerve, neuromuscular junction and muscle). Dogs with neuromuscular diseases may also have a stiff, stilted, choppy gait primarily due to muscle weakness. These abnormalities can appear episodic or as the level of exercise increases in myasthenia

gravis. A similar appearance may occur in dogs with pain, primarily from musculoskeletal diseases. Paresis can be traduced as neurological weakness without complete paralysis or although implies some voluntary motion. The degrees of paresis can occur in some animals as result of retaining the ability to walk and in other cases in animals that are

unable to stand and support their own weight. Paresis may be observed in walking as dragging of the paws. Abnormal toe posture may suggest underlying paresis. Paresis first occurs with lesions in the midbrain caudal to the red nucleus. The severity of gait impairment increases progressive towards the caudal central nervous system.

Table 1. Differential diagnosis of ataxia using neuroanatomical localization (most common causes).

Disease mechanism	Spinal cord	Brainstem Central vestibular	Cerebellum	Vestibular
Vascular	Fibrocartilaginous embolism	Brain infarct Brain hemorrhage	Brain infarct Brain hemorrhage	-
Inflammatory	Toxoplasma, Neospora, Rickettsial, Fungal, Canine Distemper, Rabies, Meningoencephalitis	Toxoplasma, Neospora, FIP, Rickettsial, Fungal, Bacterial, Canine Distemper, Rabies, Meningoencephalitis	Infectious encephalitis (Distemper, Toxoplasma, Bacterial, Neospora, Fungal, FIP, Rabies, Rickettsial)	Otitis media/interna Nasopharyngeal polyp
Trauma	Spinal fracture Traumatic disc hernia	Head trauma	Head trauma	Head trauma
Toxic	N/A	Metronidazole toxicity	Marijuana 5-fluorouracil	Aminoglycosides Topical iodophors Loop diuretics Topical clorhexidine
Anomalous	Atlantoaxial Subluxation (C1-C5) Syringomyelia Subarachnoid cyst	Chiari-like syndrome Hydrocephalus	Chiari-like malformation Cerebellar hypoplasia	Congenital vestibular disease
Metabolic	N/A	Hypothyroidism	N/A	Hypothyroidism
Idiopathic	N/A	N/A	N/A	Acute idiopathic perihel vestibular disease
Neoplastic	Primary or metastatic spinal column or spinal cord tumor	Primary or metastatic brain tumor	Primary or metastatic brain tumor	Middle and inner ear tumor
Nutritional	-	Thiamine deficiency	N/A	N/A
Degenerative	IVDD Cervical spondylo-myelopathy.	Storage diseases Other neurodegenerative diseases	Storage diseases Neurodegenerative diseases	N/A

Lameness (decreased or non-weight bearing on a limb(s)) is usually associated with pain of the limbs from musculoskeletal diseases. A similar clinical abnormality (and possibly pain) can also occur in nervous system dysfunction, referred to a nerve root signature. This abnormality often occurs in a single thoracic limb due to cervical spinal compressive disorders (intervertebral disk extrusion). The same phenomenon may be seen at the pelvic limb. Often

the affected limb may appear painful at manipulation, mimicking an orthopedic problem.

RESULTS AND DISCUSSIONS:

At the Medical Clinic of Faculty of Veterinary Medicine 96 cases were examined: 23 of them were cats and 73 were dogs that presented different types of ataxia.

The most common type is vestibular ataxia, followed by spinal and cerebellar ataxia. We observed that vestibular ataxia is more frequent in dogs than cats, especially the older ones.

In few cases the etiology was an infectious/inflammatory affection of the internal ear and some of them had neoplastic formations (otoscopic exam, Rx, RMN)

We observed that in German Shepherd and its mixed breeds, usually over 7 years old, the most common is spinal ataxia (disc hernia or traumatic injuries).

At young and small sized dogs cerebellar ataxia is the most frequent due to congenital anomalies (cerebellar hernia or aplasia diagnosed clinically, neurologically and using RMN).

CONCLUSIONS:

Is it a neurological or orthopedical problem?

Clinical/neurological investigations are mandatory in case of abnormal gait (the animal should be observed in large spaces to be able to see the way it walks: in a straight line, zig-zag or circles).

To be able to establish a neuroanatomical diagnosis we should first determine the type of ataxia.

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