

Cerebellar Diseases in Companion Animals

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Cerebellar diseases are relatively uncommon presentations in companion animals. However, these disorders are one of the easiest to localize based on clinical signs due to the unique role the cerebellum plays. The cerebellum functions primarily to coordinate (harmonize) muscle activity of the head, trunk and limbs. The cerebellum controls the rate, and range of movements but does not actually initiate the motor activity. Initiation is controlled from motor neurons in the cerebral cortex or brain stem motor nuclei.

Clinical signs in animals with cerebellar disease are characterized by ataxia with minimal to no weakness, a wide based stance, dysmetria, intention tremors and, rarely, vestibular abnormalities and opisthotonos. The ataxia often involves the trunk with a swaying gait when the animal moves. At rest, animals with cerebellar disease tend to assume a wide based stance because they are so incoordinated they fall if they fail to assume this posture. The dysmetria is characterized by exaggerated movements of the head and limbs, generally being hypermetric, rather than hypometric in character. Animals may have delayed initiation of motor activity an exaggerated (hypermetric) response and replace the limb to the ground forcefully rather than gently. Gaits are often described as goose-stepping, jerky or choppy. This type of abnormality can be noted during gaiting or when evaluating postural reactions (hopping and placing). Tremors are common in animals with cerebellar disease and mostly involve the head but the rest of the skeleton may develop tremors, particularly when initiating movement, i.e. intention tremors. This may be particularly obvious when the patient is attempting to eat or drink. They often submerge the muzzle under water and then when removing the head go too far up. Signs often are absent or difficult to detect when the animal is at rest or asleep. The involvement of the head in the problem separates spinocerebellar tract disease from cerebellar disease. With cerebellar disease if the animals head is elevated and quickly released they often have a rapid downward fall of the muzzle and it may strike the floor forcefully since they can't coordinate the dropping of the head to slow it appropriately. Fine tremors of the eyes (nystagmus) can also be seen, especially when the animal can be induced to move its eyes in the orbit. In diffuse cerebellar diseases animals may have bilateral absent menace reactions. If lesions involve the caudal areas of the cerebellum, the flocculonodular node, vestibular signs can also be observed. Mild anisocoria may also be seen with unilateral cerebellar lesions in which the contralateral (unaffected) side has a mildly dilated pupil. If cranial aspects of the cerebellum are affected, opisthotonos may be observed as well.

Causes for cerebellar signs are many, but neonatal infections (panleukopenia), degenerative conditions (abiotrophies), inflammatory diseases (fungal, rikettsial, bacterial, protozoal) and neoplasia account for the majority of cases seen in clinical practice. Occasional toxic exposure may lead to tremors and incriminated toxins include mycotoxins, hexachlorophene, lead, organophosphates, carbamates, pyrethrins, pyrethroids, ivermectin, bomethaline, and theobromine. Toxins generally have additional neurological and systemic signs of illness in addition to the tremors.

In cats, the most important cause is in-utero or early postal natal infection with feline panleukopenia (parvovirus). Vaccination of pregnant queens or kittens under two to

three weeks of age with modified live virus vaccines can also lead to cerebellar hypoplasia. Infection must occur prior to the first two to three weeks of life when the cerebellum is undergoing final development for signs to develop postnatally. Viral infection prevents normal maturation of the cerebellum and leads to cerebellar hypoplasia. This is often obvious when the size of the cerebellum of affected kittens is compared to a normal brain. Signs are evident when the kittens first begin to ambulate as symmetrical, non-progressive tremor, ataxia, and dysmetria. Since the problem is non-progressive, if signs are mild to moderate, these kittens can live normal lives but will always have problems with coordination. They tend to adapt to the problem with time so may appear to improve clinically and may make acceptable house pets.

In dogs, degenerative causes are the most common group seen in animals under one year of age. A number of abiotrophies have been described in the literature and lists of affected breeds can be found in your reference texts. Abiotrophy refers to premature degeneration of neurons secondary to abnormal metabolism. This leads to progressive worsening of cerebellar signs and death or euthanasia due to the severity of disease. Definitive diagnosis requires brain biopsy to confirm, usually done following a necropsy.

An uncommon, but important cause in young adult dogs (9 mo. to 4 yr.) is “idiopathic cerebellitis”, also referred to as “little white shaker disease” and “steroid responsive tremor syndrome-SRTS”. Affected dogs tend to be young, generally miniature (<15 kg), white dogs (50%). Most commonly reported breeds include, Maltese, Poodles, West Highland White Terriers, Samoyeds, and Spitz, although occasional non-white animals are also seen. Signs are acute in onset, symmetrical and progress rapidly to peak over two to three days. Severe intention tremors, and ataxia that disappear during rest or sleeping are characteristic. No other neurological deficits are generally seen. Some animals resolve spontaneously, although most benefit from steroid and/or muscle relaxant therapy. Lab work is generally unremarkable except for mild lymphocytic-plasmacytic inflammation present in CSF (< 40 WBC, primarily lymphocytes). It has been hypothesized that the cause for this syndrome is viral or immune mediated. Immune mediated disruption of neurotransmitter metabolism resulting in reduced conversion of tyrosine to dopamine is proposed by some authors. Dopamine is an important neurotransmitter for the regulation of movement. Other explanations include altered function in cells of similar embryologic origin that produce melanin and neurotransmitters and possibly genetic factors. Since this disease occurs in non-white and non-purebred dogs genetic influences seem to be less likely. Therapy involves immunosuppressive dosages of prednisone that are tapered over 8 to 12 weeks. Initial dosages are 2.2 mg/kg/day in divided dosages q 12 hours. This is given for four weeks tapering by 50% every month until alternate day dosages are achieved (0.25 mg/kg/day on alternate days). Diazepam may also provide relief when given at 0.5 mg/kg every 8 hours for one month, then decrease to twice daily for 4 weeks then given once daily for 4 weeks. I have generally used only prednisone with a high degree of success. Some animals will relapse when the steroids or diazepam are discontinued and will need longer therapy, for several months to keep clinical signs in remission. Most animals have a dramatic clinical response in 48 to 72 hours following the beginning of steroid therapy.

Usually returning to normal. Occasionally animals will relapse weeks to months after the initial episode and need another course of therapy.

Suggested Reading

Wagner, SO, Podell, M, and Fenner, WR: Generalized tremors in dogs: 24 cases (1984-1995). J Am Vet Med Assoc, 1997; 211, 731-735.

Bagley, R, Kornegay J, Wheeler S, et al. Generalized tremors in Maltese: Clinical findings in 7 cases. J Am Anim Hosp Assoc. 1993;29:141-145.