ROCKY MOUNTAIN VETERINARY NEUROLOGY

Neurology/NeuroSurgery
Stephen B. Lane, DVM
Diplomate ACVIM-Specialty of Neurology
Veterinary Neurology/Neurosurgery

Optic Neuritis

The optic nerves are the second paired set of cranial nerves (nerves of the brain) in companion animals. They are made up of millions of individual nerve processes (axons) with their fatty covering (myelin) that allows them to transmit electrical impulses at incredibly fast speeds of 50-100 meters per second. Each eye gives rise to one optic nerve, made up of the individual nerve processes of the nerves making up the retina, enclosed in a protective covering, the epineureum, exiting the back of the eye and entering into the cranial vault through a bony hole called the *optic foramen*. Once inside the skull, the optic nerves run along the floor of the cranial vault, beneath the brain. Each nerve crosses over the other (optic chiasm) to enter the rostral brainstem on the opposite side and provide visual input to the opposite visual cortex. By doing so, vision is represented on the opposite side from where it was initiated. Said another way, vision on the right is represented in the left visual cortex. It is important to remember that not all the never fibers cross over at the chiasm. This means that visual input from both visual fields is represented in both visual cortices. This is why binocular vision is present in animals and humans. Differences in the degree of crossover of these visual nerve fibers and the degree of lateral (to the side) set of the eyes, allow certain species greater binocular vision. The greater the degree of crossover and the farther the eyes are set to the side of the head results in greater binocular vision.

While the optic nerves transmit visual information in the form of electrical impulses to the visual cortex of the brain, they also carry information relative to the amounts of light in the environment controlling the pupil size and light emitted to the retina of each eye. Change in the optic nerve function can be represented by visual dysfunction, pupil size change or irregularity, and loss of pupil size response to light (papillary light reflex). ***Optic neuritis is an inflammation of the optic nerve(s).*** Optic neuritis appears to occur in all breeds and age groups with a predilection for middle aged, female dogs of 3-6 years of age. An acute onset of visual dysfunction, dilated pupils, lack of constriction of the pupils in bright environments, and difficultly in negotiation of surroundings is common. A sudden onset of reluctance to walk in dark or new environments is often the first evidence that something is wrong. Bumping into objects, reluctance to perform normal activities or tricks and difficulty in finding objects requiring visual capabilities is compromised or lost. This may be manifest by difficulty finding a thrown ball, catching or finding a treat and reluctance to perform activities requiring vision.

The diagnosis of optic neuritis is often based upon systemic assessment, appropriate laboratory testing and specialized imaging. Ophthalmologic examination is important to exclude diseases of the retina that can also cause similar signs as optic neuritis. This examination can also provide information regarding the appearance of the optic nerve as it begins at the retina, the *optic disc*. Swelling and discoloration of the optic cup (where the nerve fibers exit the retina) can often be noted with inflammation of the optic nerve behind the eye. Further ancillary testing utilizing a full blood profile to identify internal conditions capable of causing visual dysfunction such as hepatic disease (hepatic encephalopathy), over active adrenal glands (hyperadrenocorticism) and leukemia should be performed if indicated.

Definitive testing utilizing magnetic resonance imaging (MRI) and cerebrospinal fluid analysis (CSF) is often necessary to accurately diagnose the cause of optic neuritis. Inflammatory diseases capable of causing inflammation of the visual nerves include immune-mediated (idiopathic and paraneoplastic optic neuritis), granulomatous meningoencephalitis (GME)) as well as infectious etiologies (Canine Distemper Virus, Toxoplasmosis/Neosporum spp. and Rickettsial spp). In addition, infiltrative nerve diseases, as seen with lymphoma or leukemia of the lymphatic system, lymphoplasmacytic neuritis, and nerve-sheathe-related neoplasia, have also been appreciated.

Definitive diagnosis of optic neuritis is important in definitive therapy and long-term prognosis. Antibiotic therapy using Clindamycin or Doxycycline is used to treat infections caused by Protozoal spp. and Rickettsial spp. causes. Immune mediated inflammation of the visual nerves is the most common cause for optic neuritis. This means that infectious causes are uncommon. The use of aggressive immunomodulant treatment is often necessary to regain visual nerve function and maintain a remission state and long-term visual function. Two categories of immunomodulants (agents that modulate the immune system) exist for routine use in veterinary medicine. **Glucocorticoids** such as *Dexamethasone* or *Prednisone* are employed early in the coarse of treatment. Attempts to reduce the dosage or gradually withdraw glucocorticoid therapy are the goal. Oral **chemotherapeutics**- *Lomustine (CeeNu), Cytosine, and Azathioprine (Imuran)* are used alone, or in conjunction with glucocorticoid therapy. **Cyclosporine** is the latest immunomodulant to be used with nervous system disease, including optic neuritis. While in it's infancy with this syndrome, it appears to be an effective drug in the control optic neuritis.

Long-term prognosis with optic neuritis is dependent upon the underlying cause and response to treatment. Compliance ensures as successful an outcome as possible. With greater diagnostic ability and aggressive therapy, the long-term outlook for vision is considered good. Your neurologist will direct you as to appropriate testing and follow up necessary.