DEGENERATIVE MYELOPATHY

Adapted by Leslie Manis, Health/Genetics Chairman, ASTC

Note: This article is for informational purposes only. DM has not been identified in the Sealyham Terrier as a health problem. This article is an adaptation of Degenerative Myelopathy--Disease Basics, published at http://www.caninegeneticdiseases.net/DM/basicDM.htm

[Every effort has been made to ensure accuracy of information. However, this is not a substitute for prompt veterinary care. Any similarity to other publications is unintentional. Published online at Sealyhealthguard.org, 7/20/11]

Degenerative myelopathy is a progressive disease of the spinal cord in older dogs, usually beginning between 8 and 14 years of age. The first sign is a loss of coordination in the hind legs; it can begin in one leg and then the other. Within 6 months to a year, the dog will become paraplegic. If signs continue to progress, incontinence may occur and eventually weakness will develop in the front legs.

The only way to confirm diagnosis is to examine the spinal cord under the microscope after death. Any disease that affects the spinal cord can cause similar signs of loss of coordination and weakness. Many of these other diseases can be treated effectively. The most common one is herniated intervertebral disks. This can often affect short-legged, long-backed dogs. It can usually be seen with X-rays of the spine and myelogram. Myelography involves using a contrast medium and X-rays to examine the spinal cord. More advanced imaging techniques like CT scan or MRI may be used. A neurologist can aid in diagnosis, if necessary. Other causes of weakness and loss of coordination include tumors, cysts, infections, injuries and stroke. Degenerative myelopathy is a diagnosis of elimination of these other possibilities.

DM begins in the white matter in the chest (thoracic) region of the spinal cord. This white matter contains fibers that transmit commands from the brain to the limbs, and feelings from the limbs to the brain. There is loss of the fibers themselves as well as the stripping away (demyelination) of the insulation of these fibers. This interferes with communication between the brain and limbs.

There are no treatments that have been clearly shown to stop or slow the progression of DM. The dog's quality of life can be improved by good care, physical rehabilitation, pressure sore prevention, monitoring for urinary infections, and use of harnesses or carts.

There is a DNA test available which clearly identifies dogs that are clear (two normal copies of the gene, G-G), those that are carriers (one normal copy of the gene, one mutated copy, G-A), and those dogs at a much higher risk for developing degenerative myelopathy (two mutated copies, A-A). Not all dogs with two mutated copies develop DM. Researchers now seek to understand what triggers the development of clinical symptoms in some, but not all of these A-A dogs.

As of July 2008, researchers have found the mutation present in 43 breeds. It is possible that the genetic background that predominates in some breeds prevents the development of symptoms even in A-A dogs. At this time, they have the required evidence that there's

an association between DM symptoms and the mutation in the following breeds: Boxer, Cardigan & Pembroke Corgis, Chesapeakes, German Shepherds, Rhodesian Ridgebacks and Standard Poodles. Of these breeds, G-G and A-G dogs have never been confirmed to have degenerative myelopathy. All dogs with DM have A-A (two mutated copies of the gene), but as stated above, the reverse is not necessarily always true.

Now that the DNA test has been developed to identify dogs at risk for developing degenerative myelopathy, researchers can work to discover what triggers its development, and find ways to prevent it. Until then, breeders can add this as a valuable tool for informed, balanced breeding decisions.

For more info on the DNA test:

http://www.caninegeneticdiseases.net/DM/ancmntDM.htm

END