The term “wobbler” is sometimes used as a general designation for dogs that are incoordinated due to a variety of spinal cord diseases. However, the term “wobbler syndrome” is usually reserved for a combination of cervical spinal cord and nerve root compression that occurs in large-breed dogs as a result of developmental malformations of the vertebrae, instability, or degenerative changes (arthritis) secondary to instability. These changes and instability can also lead to intervertebral disk failure and development of type I or type II intervertebral disk disease. Type II disk disease is more common, and is characterized by age-associated degeneration of the disk, with bulging into the spinal canal and slowly progressive spinal cord compression. A more technical term for “wobbler syndrome” is cervical spondylomyelopathy. Other terms for this disease that have been used include cervical spondylopathy, cervical spondylotic myelopathy, cervical spinal stenosis, cervical malformation/malarticulation syndrome, cervical vertebral instability, and cervical vertebral instability-malformation syndrome. They are all referring to the same problem.

Wobbler syndrome is primarily a disease of large and giant breed dogs. Dobermans and Great Danes are the breeds most commonly affected (roughly 5.5% of Dobermans and 4.2% of Danes). Other affected breeds include Weimaraners, although any large or giant breed dog can have the disease. I am unaware of any study that reports the prevalence of wobbler syndrome in Weimaraners, but I have observed neurologic deficits in many elderly Weims and know of a few younger and middle-aged Weims that have been diagnosed with this problem. In the 2012 online health survey conducted by the WCA, representing 482 Weims, wobbler syndrome was reported in 1.7% of respondents, ataxia reported in 1.2% of respondents, and degenerative myelopathy reported in 1% of respondents. Considering that some of these diagnoses might or might not be misreported, the prevalence of wobblers in that survey was anywhere from 1.7% to 3.9%.

The cause of cervical spondylomyelopathy is unknown. While age-related degenerative joint disease, disk fibrosis, etc. likely play a role, it is also likely that genetics and nutrition influence vertebral development and growth, which can result in differences in spinal canal size (stenosis) or stability (with instability resulting in increased arthritis of the cervical facets). A genetic influence is suggested by the disease being more prevalent in certain breeds, but also the observance that it appears to occur more commonly among related dogs within a breed. Dietary factors that have been thought to be associated with this disease include overfeeding (especially during the puppy/developmental period) and excessive calcium. I think most likely the syndrome is caused by instability of the cervical articulations, which over time leads to arthritis and enlargement of the articular facets. Eventually these enlargements compress the spinal cord, resulting in ataxia and other neurologic
signs. The enlargements are also more likely to compress the cord, or to compress it at an earlier age, if the spinal canal is also narrow. Instability and stenosis can be due to both genetic and nutritional factors, most likely a combination of the two. Overfeeding can result in rapid growth, especially in a genetically predisposed dog, which can lead to abnormal stresses in the vertebral bodies as they mature. The age of onset of clinical signs is probably inversely proportional to the initial degree of instability - that is, the more unstable the cervical articulations to begin with, the faster the arthritis accumulates and dogs show neurologic signs at an earlier age. For these reasons, dogs that start to show clinical signs at an early age (for example age 6-8) are likely more problematic for a breeding program than dogs that begin to show clinical signs at a more advanced age (for example 12-14).

Clinical signs of cervical spondylomyelopathy consist of slowly progressive neurologic deficits in all four limbs, with the hind legs generally worse than the front limbs. Problems localized to the cranial cervical spine usually are associated with floating forelimb movement, whereas problems in the caudal cervical spine (more common) are associated with short, choppy strides in front. Both locations result in ataxia, knuckling, loss of conscious proprioception, and delayed postural reactions in the rear limbs. Neck pain is usually not a feature of this syndrome, being more commonly associated with type 1 intervertebral disk protrusion. However, the dog may resist dorsal extension of the neck. As severity increases, the dog may start to suffer falls and/or cannot walk unassisted.

Useful diagnostic tests to determine the cause of the neurologic signs include radiographs +/- myelogram, CT (computed tomography), and/or MRI (magnetic resonance imaging), which help differentiate cervical spondylomyelopathy from trauma/fracture, neoplasia, intervertebral disk disease, discospondylitis, vertebral osteomyelitis, meningomyelitis, or fibrocartilagenous embolism.

Treatment depends on the severity of disease and, to some extent, the age of the dog at the time of onset. Prognosis varies, depending on how quickly clinical signs progress, the age of the dog, and whether or not surgery is performed. Medical therapy, consisting of exercise restriction, use of a harness instead of a collar, anti-inflammatory drugs (NSAIDs or steroids) and muscle relaxants will temporarily relieve clinical signs, but will also allow progression of spinal cord compression. One study found that 54% of dogs treated medically improved, whereas 27% were unchanged at long-term follow-up.

More than 20 different surgical techniques have been described for treating cervical spondylomyelopathy in dogs. Surgical decompression more consistently leads to clinical improvement than medical treatment (80% of dogs improve after surgery), but is not without risk. In one study of 771 dogs, mortality rate with decompressive surgery was 3% on average. Other possible complications of surgical treatment include postoperative worsening of clinical signs, penetration of the vertebral canal or transverse foramina with implants, implant failure, and eventual development of a compressive lesion next to the stabilized one in approximately 20% of dogs. Despite these complications, surgery is generally recommended as definitive treatment, especially in younger dogs. Surgery is not cheap and can require prolonged aftercare.