A collection of information about Wobblers Disease

SPONDYLOLITHESIS

WOBBLERS SYNDROME

Description

Wobblers syndrome is caused by a narrowing or malformation of the spinal cervical (neck) vertebrae which causes pressure on the spinal cord by the lower cervical (neck) vertebrae due to either a malformation of the vertebra or a malocclusion (when the vertebrae do not come together properly). This causes anywhere from a mild, to a severe affect in the dogs gait. Other conditions can mimic the symptoms. The only definitive diagnosis of Wobblers Syndrome or Spondololithesis, is a mylogram where dye is injected into the spinal column and then the neck is flexed and x-rayed.

Breeds affected: - Dobermans and Great Danes primarily - young Danes more commonly affected. Dobermans - young and old, can grow through the problem as youngsters, more commonly seen in middle aged to older Dobermans (3 to 9 years of age) Other breeds who have a similar if not identical syndrome described include the Boxer, Basset, Bull Mastiff, St. Bernard, Weimeraner, Labrador Retriever, German Shepherd, Rhodesian Ridgeback, Dalmatian, Samoyed, Old English Sheepdog, Irish Setter, and the Borzoi. Males are affected more often, in a ratio of 2:1

Cause

The cause of Wobblers Syndrome is unknown, although a link to fast growth and genetics is suspected. According to the Merck Veterinary Manual, "The cause is unknown, although rapid growth rates and nutrition, mechanical factors, and genetics may be implicated." Some breeders say that there has been a marked decrease in the incidence of not only Wobblers Syndrome, but other diseases that occur during the early, fast growth stages of Great Danes, when weight is kept down and growth rate has been slowed nutritionally.

Symptoms

Symptoms usually appear first in the rear legs as a mild uncoordination in gait (ataxia) and can escalate to involvement of the forelegs as well. The severely affected dog moves like a drunk and the uncoordination shows up most when the dog is walked and then moved sharply into a turn. An unsuspecting owner might simply conclude that his older puppy was just clumsy. Overly clumsy young Great Danes should be Wobbler suspects.

In Great Danes, Wobblers Syndrome most commonly appears from 10 months to a year and a half of age although it can manifest as old as 4 or 5 years, and as young as 5 weeks. In Doberman Pinschers it usually doesn't appear until the dog is 4 or 5 years old.
Diagnosis

A veterinarian will do a neurological work up on the dog and this often includes not only cervical spine x-rays, but a mylogram x-ray. A mylogram is not only dangerous to the dog, but is expensive. The owner should thoroughly investigate the advisability of this procedure, especially since if it is Wobblers Syndrome, surgery may not be the best option.

Treatment

Treatment of Wobblers Syndrome can include the use of corticosteroids, a neck brace and surgery. The surgery fuses the 2 unstable vertebrae which relieves the pressure on the spinal cord. Unfortunately this also puts further stress on adjoining vertebrae which can cause instability to recur in them. Many Wobblers can live a long and pain free life with little or no treatment. Others deteriorate quickly and euthanasia then becomes the only kind choice.

Wobbler's Syndrome

by Bruce R. Wittels, D.V.M.

This is the first in a series of articles that will discuss specific disease entities of bone caused, in part, by improper or over nutrition. Some of the information to be presented in this and future articles will have been discussed previously but will be put into specific consideration to the disease entity covered. It is my desire to provide an understanding of the total picture of proper nutrition and a well balanced and strong skeleton upon which these modern day giants are to support their massive bodies. If the foundation of any structure is weak, then the weight which is born by it will cause this foundation to break and fall.

The principal breed affected with Wobbler's Syndrome is the Great Dane. Cervical Spondylopathy or Canine Wobblers Syndrome as the lay person knows it, consists of any uncoordination or lameness caused by pressure on the spinal cord as it travels through the neck (at any age for any reason).

In Great Danes, a true wobbler is usually seen between the ages of 3-12 months of age. The outstanding symptom is a slowly developing, progressive, uncoordination and paralysis of the hind limbs. This paresis and/or uncoordination is caused by pressure on the spinal cord as it passes through the vertebrae of the neck, the last three being the most prominent sites. The animal may appear clumsy, "wobbling" or display swaying of the rear legs. On slick surfaces it may be difficult or impossible to walk. The dog may fall when attempting to turn and the toes of the hind feet may drag when walking. The dog usually has its legs spread out as far as possible trying to better balance itself. Initially front leg involvement is minimal or undetectable. As the disease progresses, a characteristic short choppy rolling stride is seen on what appears to be somewhat rigid forelimbs. As the
compression of the spinal cord becomes more severe, the front legs can have the same signs as the rear legs. There is usually little or no neck pain but the dog may stand with the neck flexed which usually alleviates some of the spinal cord pinching. Many dogs will object to neck manipulation and may even collapse when the movement is forced.

On occasion, there can be an acute onset of signs. They are usually more severe than with the progressive type. Forelimb involvement is usually present. There is also a higher incidence of neck pain with the acute onset. This type of wobblers is seen most frequently in five to seven year old Doberman Pinchers and is rarely seen in young Danes. This must be differentiated from similar signs caused by cervical disc protrusion or a fractured vertebrae.

The pressure of the spinal cord is due to improper formation of the anatomical parts surrounding the spinal cord during growth. The pressure can be due to one or a combination of the following:

1. weak ligaments which hold the vertebral bodies in place - this allows irregular movement of the vertebral bodies as the neck is placed in various positions (subluxations) and causes a pinching of the spinal cord.

2. hyperplasia of the yellow ligaments - normally these are thin loose elastic sheets located between the arches of adjacent vertebrae. Just beneath them is the small epidermal space which is immediately above the spinal cord. With enlargement of these ligaments, the epidermal space is obliterated and the yellow ligaments push directly on the spinal cord.

3. malformation of the vertebrae - this can happen in various ways i.e.:
   a. Ventribal or spinal canal that is too small for the diameter of the spinal cord
   b. Assymetric cartilage defects which cause vertebral subluxations
   c. Osteochondrosis of the articular facets which also lead to subluxations
   d. Stenosis of the cranial orifices of vertebral foramina which causes a pinching of the nervous system

**DIAGNOSIS**

This is based on the breed, age, history and results of both a physical and neurological examination. Diagnosis is confirmed by radiographic examination. A minimum of three views should be taken: a side view with the neck in normal position, a side view with the neck in a flexed position (head pointed towards the feet), and a side view with the head in a skyward position. A subluxation of one or more vertebrae may be the only causative agent and is often only seen when the neck is in and extended or flexed position. At times, a view of the neck from top to bottom must be taken. A mylogram might be needed to properly determine the exact compression sites. Other disease entities which can cause similar symptoms that must be differentiated from are: disc protrusions, infectious, neoplastic, vascular, or traumatic disorders of the spinal cord. Other bone disorders
common to the giant breeds can cause gait abnormalities but are usually presented with a lot of pain and do not show any nervous system involvement.

CAUSES
The exact reasons why the aforementioned vertebral abnormalities occur are unknown. There is probably a genetic factor that comes into play. This is determined by a high incidence of Canine Wobblers seen in certain families of Danes. It is difficult to separate the genetic from the environmental factor (nutrition, management and activity) as most of these families are kept in the same environments because many breeders keep the pups for three to four months thus keeping the family under a similar environment. Deformity and displacement of the cervical vertebrae secondary to a long neck, large head and rapid growth has been proposed. (Weight et al 1973).

A vertebral canal that is too small for the diameter of the spinal cord could easily be caused by nutritional hypercalcitonism. As discussed in my second article, Nutrition of the Newly Born and Growing Individuals (GDR Jan/Feb '85). Calcitonin is released from the thyroid gland when the animal is fed a diet high in calcium. Its function is to retard bone re-absorption and thus decrease blood calcium levels. Remember, calcium is kept within strict limits in the blood stream. In normal growth, expansion of the spinal canal must be perfectly synchronized with growth of the spinal cord. Expansion is achieved by resorption of bone inside the vertebral canal. With hypercalcitonism, the retarded rate of bone resorption prevents proper expansion of the canal - therefore the diameter of the spinal canal is decreased and has an irregular contour. The spinal cord grows independently of the surrounding bone and is thus pinched by the nonresorbed bone therefore causing Cervical Spondylopathy.

Nerves leave the spinal cord, to go to other parts of the body, through spaces formed by notches is the vertebrae called foramina. A narrowness in these spaces will cause a pinching of the nerves leaving the spinal cord which in turn causes inflammation and swelling of the spinal cord itself thus causing wobblers syndrome. Hypercalcitonism causes narrowness in the vertebral canal can also yield a stenosis of the cranial orifices of the vertebral foramina due to its effect of slowing bone resorption. Hyper or over nutrition can also give the same results as an increased growth rate without proper mineralization and body controls can cause enlarged or asymmetric bone formation of the vertebrae in the neck as well as in any other bone in the body. This could easily result in pinching of the nerves as an overabundance of bone can yield improper notch formation.

Although the vertebrae may not be as flexible as other joints in the body - they do move and have joint surfaces as do other bones. The joint surfaces called "articulaion facets" are lined with a cartilage the same as those of other joints. Improper formation of these surfaces can cause the vertebrae to move improperly and cause luxation or subluxation which can cause pinching of the spinal cord. A mechanism that can cause such a situation is Osteocondritis Dessecans (OCD). For now, let's describe OCD as a disturbance of endochondral ossification (one type of bone formation) which
leaves the joint cartilage thicker than normal. It is therefore further away from the blood supply leaving it more susceptible to injury and cell death which will cause poor rotation of the joint and hence subluxation. This can happen at all or only one of the facets in the cervical spine. Again the last three vertebrae are most commonly affected. Osteochondrosis will be more thoroughly discussed in the next issue of the Reporter. The main cause of OCD is overnutrition or ad-lib feeding. Hedhammer et all (1974) studied the relation between overnutrition and skeletal disease in growing Great Danes. In general - dogs fed ad libitum had smaller than cervical vertebral foramina (notches) with subsequent spinal cord compression as well as asymmetric vertebral articular facets. There was also OCD in the facets displayed as defects in the cartilage covering the facets. They concluded that "excessive intake of food rich in protein, energy, calcium and phosphorous accelerates growth and can induce various skeletal changes including changes in the cervical vertebrae that result in the wobbler syndrome."

TREATMENT
The treatment of cervical spondylopathy depends on the severity of the neurological involvement and the longevity of the disease process. Treatment is directed at stopping further cord trauma and at decreasing the current neurological deficits.

The acute form of canine wobbler syndrome is truly a medical emergency. The dog is very often quadriplegic and immediate veterinary attention must be sought in order for the spinal cord to be saved. Generally the veterinarian will treat the animal with diuretics (i.e. mannitol) and high dose steroids to decrease the swelling and inflammation in the cord as well as ascorbic acid to protect the myelin sheath that surrounds the nerves. This treatment may last as long as 2 to 3 days. Depending on the degree of improvement, decompressive surgery is usually required to prevent further trauma to the cord. If via rigorous medical treatment, the animal regains full neurological function, surgery may be delayed from one to three weeks with the animal probably kept in a neck brace. If, however, little or no improvement is rendered via medical care - surgery must be done within a few days in order to preserve the integrity of the spinal cord. The more time that passes with the spinal cord compressed and therefore without nutrients and oxygen, the higher the likelihood of some degree of permanent paralysis. The two most used surgical techniques are Dorsal Laminectomy (where the top of the spinal canal is removed thus allowing the spinal cord to rise out of the confining environment) and Ventral Decompression which removes ulcerated disc material as well as certain ligaments under the spinal cord and some of the vertebral body that may be compressing the cord. Choice of surgical technique is made by the orthopedic surgeon or by someone that has had much experience with spinal surgery as well as the special instrumentation needed for this complicated procedure. Even with the correct facilities and techniques there are always possibilities of post operative complications.

Dogs with slowly progressive signs of Wobblers Syndrome often respond well to a combination of cage rest and corticosteroid therapy. I usually apply a neck brace as well to provide some stabilization of the neck and at the same
time some traction as well. This is only if the syndrome is caught in the early stages. Remember that the above will only alleviate the effects of compression of the cord and does not and will not correct the vertebral and ligamentous changes. The IMMEDIATE thing to do is to reduce the protein level of the diet. A protein level not to exceed 22-24% should be fed. Any and all mineral supplements should be discontinued. All food and water must be elevated to further reduce neck tension. Ball playing or any action where the head is dipped down to snatch up an object is to be eliminated. A very concerted effort to restrain this dog from exercise is to be instituted as this can easily yield an increase in the likelihood of vertebral subluxation or further damage to the spinal cord. It is a known fact that signs of joint instabilities are less severe in animals kept in a sedentary environment than those allowed unrestricted exercise. Hopefully if this is discovered early and the diet is changed while the bones are still forming and little recurrence of cord trauma occurs, the remaining formation of bone will be normal and the condition can stabilize itself to the point that surgery will not be needed. Surgery for the slowly progressive form of cervical spondylopathy may not be as rewarding as with the acute form, if it is discovered late since the damage to the spinal cord may be permanent. It may however, prevent further damage to the cord.

What can we do to prevent canine wobblers syndrome? My own observations and present studies indicate a probable genetic factor that may predispose certain lines or individuals to producing wobbler offspring. You as breeders must strongly consider the use of these lines or individuals for breeding purposes. You must answer to your own conscience and morals. The goal of breeders and the purpose of breeding is to strive to ever better the breed and those of you in breeding solely for the monetary gain must seriously reconsider this motive!!!

One thing that we all must do is to refrain from that archaic urge to supplement the excellent commercial foods available as the main cause of growing bone disparities in the giant breeds is hypernutrition. For those of you who have found that one or two pups out of every litter or every other litter have shown signs or have developed into wobblers, I recommend that you do your very best to eliminate the environmental elements that might encourage or enhance the pups’ chances of developing into a wobbler. This would entail feeding the entire litter the adult form of the high quality food (22%-24% protein) starting at weaning time. Don’t use Iams puppy food or A.N.F. 30 for instance. The extra calcium and protein in these foods could easily cause signs of the above. I would feed a multiple vitamin and absolutely no additives. Perhaps a little canned food to increase the palatability. Each litter must be treated individually. Don’t equivocate your litter to your friend’s. Don’t ignore the possibility that your dogs might be more efficient in digesting foods and utilizing a higher percentage of available nutrients than someone else’s. This could give the same effect of overfeeding without the addition of any supplements. Periodic radiographs may be advantageous to early detection of wobbler development. Again it is very important to put as little stress on the necks of these pups - directions to be followed as previously described.
SUMMARY
There is no cut and dry answer or definition as to why or where this disease entity originates - this is a fairly new area of veterinary science. We are finding strong evidence suggestive to a genetic entity in this syndrome and have found definite proof that nutrition plays a major role in the development of wobblers and other bone disorders in the giant breeds. Again this is due to our wanting only the best for our dogs and knowing that the giant breeds need more nutrition than average dogs. Where we tend to go wrong is in the belief that if a little more is good, than a lot more must be better. Take an honest look at your own feeding program and look for areas where you might be able to improve the lives of your Danes.

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Wobbler Syndrome - in large breeds of dogs. (Caudal Cervical Vertebral Malformation and Malarticulation) (Caudal Cervical Spondylopathy and Myelopathy)

(Nota #1)

This syndrome has also been called the "wobbler" syndrome, cervical vertebral instability, and cervical spondyloolisthesis. The term "Wobbler" describes a nonspecific clinical picture, and the terms instability and spondyloolisthesis do not accurately reflect the complexity of the syndrome nor the fact that instability is often not demonstrable. The name cervical spondylopathy more accurately reflects the complexity of the syndrome and therefore has become widely accepted.

The outstanding clinical feature is a slowly progressive upper motor neuron paraparesis and incoordination. Cervical pain may or may not be present. The neurologic deficit in the thoracic limbs is usually minimal and occasionally nondetectable. The reason the pelvic limbs seem more affected than the thoracic limbs is unknown, but deLahunta (1977) suggests that the more superficial position of the pelvic limb spinocerebellar tracts in the spinal cord at the site of the compression may be the reason. He also suggests that the further distance of the pelvic limbs from the center of gravity of the animal may be important.

Although the spinal cord compression is usually in the caudal cervical area (area of lower motor neuron supply to the forelegs), upper motor neuron signs predominate in the forelegs. This finding reflects the fact that chronic spinal cord compression affects the white matter more severely than the gray matter. In some cases there is a lower motor neuron involvement, usually expressed as bilateral atrophy of the scapular muscles. The foreleg gait observed in many cases is quite characteristic. The forelegs appear somewhat rigid and the stride is short, choppy and rolling. With greater degrees of compression the thoracic
limb gait resembles the pelvic limb gait, that is, paretic and incoordinated. Occasionally the onset of signs is acute, and the dog is quadriplegic when presented to the clinician.

(Only #2)

Diagnosis...Breed, age and clinical signs suggest the diagnosis of cervical spondylopathy, which is confirmed radiographically. Changes seen on noncontrast radiographs may consist of (1) change in shape of the vertebral body with apparent loss of the cranioventral corner of the vertebral body. (2) abnormal width and shape of the disc space cranial to the vertebral body defect. (3) calcification of the affected disc, (4) reactive osteophyte formation and end-plate sclerosis resulting from the instability of affected disc, (5) malformation of the articular processes. Myelography is required to identify the nature and precise location of extra-dural soft-tissue masses: disc hernation, thickening of the ligamentum flavum and joint capsule, or thickening of the dorsal longitudinal ligament. The use of a hyperextended lateral projection is of great value in demonstrating the maximum degree of cord compression.

Vertebral instability, either alone or in combination with vertebral malformation and/or soft tissue stenosis has been suggested as an initiating cause of spinal cord compression and associated neurologic abnormalities (VanGundy 1988 - Dobes).

Etiology (causative factors) - still obscure and include - genetics, rapid growth, nutrition. The high incidence in certain breeds suggests heredity is a contributing factor.

Structural Aspects - combined bone and soft tissue lesions (spinal cord compression) at C6-C7 and C5-C6 are most commonly seen, however C4-C5, and C3-C4 can be affected.

Malarticulation - allowing ventral displacement. This can be stable or unstable - the unstable cases are often only visible on flexed lateral views (Spondylolisthesis).

Malformation - changes can be secondary to malarticulation; the cranial edge of the vertebral foramen may be stenotic with or without deformities of the vertebral body. Arthritic changes, intervertebral disc degeneration and collapse, disc protrusion etc. can occur in various combinations in older dogs. Most dogs have a combination of both malarticulation and malformation.

Destruction of Neurons - in severe cases, neurons are destroyed at the site of injury, usually at C6 and C7 spinal cord segments. Milder cases myelin may be lost at the site of injury, causing some loss of function that can be repaired over time if the lesion is stabilised.
Clinical Signs - vary widely. Seen as early as 2-4 months of age and as late as 8-9 years of age.

Neurologic dysfunction (signs visible) are due to the spinal cord compression exhibited in that dog. Most commonly seen in the younger dogs is hindquarter incoordination, wide based exaggerated movement and proprio-receptive deficits (stumbling, scrapping of toes). Frequent turning will often cause the dog to fall over in the hindquarters. Front legs are usually less affected than the hind-limbs, but affected dogs may have a restricted action, the limbs appearing rigid.

Rate of progression is variable according to the severity of spinal cord compression and the extent of instability and damage.

Young Dogs - especially Great Danes (less than 2 years of age), frequently have dorsal spinal cord compression due to elongation of the cranial aspect of the dorsal arch of the affected vertebrae. Dobes, Danes may be 6 months or younger when initial signs are noticed. Bull Mastiffs are generally less than 1 year of age. Affected Bassets are male, generally less than 6 months of age and have a malformation of C3.

These dogs frequently have severe ventral spinal cord compression secondary to collapse of the intervertebral disc and proliferation of fibrous tissue around the unstable area.

Older dogs often exhibit pain on movement of the neck, particularly during flexion. Signs can be as mild as neck pain to tetraplegia (paralysis of all 4 limbs).

Diagnosis - When studying this disorder it must be determined that a neurological disorder exists and not one of several skeletal diseases that occur in young dogs including OCD, HD, HOD. Most of these disorders are asymetrical, the gait appears stiff and pain can be palpated in affected joints or limbs. Neurological cases with cervical lesions have poor control over the position of the limbs, hence the wide based stance, stumbling and proprio-receptive deficits (righting relaxes of the feet).

Disorders such as congestive cardiomyopathy or hypothyroidism are frequently diagnosed in the Great Dane and the Doberman. TSH testing is recommended in any Dobermann Pinscher with clinical signs consistent with cervical spondylyoeyelopathy. Supplementation of hypothyroid dogs with T4 products is recommended, and in some dogs may result in dramatic clinical improvement. (Ettinger 1989) Hypothyroid neuromuscular dysfunction symptoms include - weakness, stiffness, reluctance to move knuckling or dragging of the feet with excessive wear of the dorsal surface of the toenails and muscle wasting.

OCD of the cervical vertebral articular surfaces of young Great Danes may be a
causative factor of cervical vertebral instability in this breed. Lesions seen were similar to those seen in Cervical Osteochondrosis in swine (Olssen 1980).

Plain radiographs are useful for a rough diagnosis if there is vertebral malformation, however a myelogram is necessary for positive confirmation of the areas of spinal cord affected by compression. Apparent "tipping of vertebrae", or coning of the vertebral canal can be enormously misleading in demonstrating the involved vertebral interspaces. Without myelograms, accurate and complete diagnosis cannot be made. If surgery is needed, myelograms are essential in the selection of the most appropriate surgical techniques that will stabilise the neck and allow the surgeon to assess the long term prospects of the affected dog.

Treatment - depends on several factors - severity of the symptoms, age, suddenness of onset, long term prospects.

Conservative - rest and corticosteroids are most effective in many cases. Surgery is advocated where response to corticosteroids is poor and clinical signs and/or the radiographic signs are severe. Younger dogs are often treated this way initially, however if signs persist, surgical intervention is necessary if long term improvement is to be gained. Occasionally a young dog may "grow out" of their problem by enlarging their vertebra canal sufficiently to accommodate the spinal cord. Older dogs, where the pain is readily controlled with rest and medication, can often be managed on long term cortico-steroid therap.

Surgical - rule of thumb on this type of treatment is based on the age of the dog i.e.. if 6-8 years- surgery is probably of benefit. A 10 year dog may be better managed on tablets. Numerous methods are described and can vary depending on the cause and site of compression - plates, dorsal and ventral laminectomy, vertabral body screws etc. Treatment is aimed at stabilisation and decompression of the effected section of the spinal cord. Neurological deterioration can occur subsequent to surgery (months or years later) due to invovement of an adjacent disc space. Prognosis - of affected dogs is as varied as their vertabral column lesions and neurological deficits. In general the more severe the neurological dysfunction, the less favourable the prognosis.
Post surgical improvement depends on the elimination of further injury to the spinal cord and remyelination of the damaged nerve tissue.

Genetics - A simple recessive mode has been suggested in the Great Dane and Doberman. Higher incidents are seen in males. An autosomal recessive mode for the Borzoi (generally affects older adults) has also been suggested. However there appears to
be a gender influence as well (females are primarily affected in the Borzoi).

References: Veterinary Neuroanatomy and Clinical Neurology, De Lahunta 1983

Wobbly Doberman goes chiropractic

I just read about the Doberman who was diagnosed with wobblers syndrome, and I felt I had to write to you.

Major, our Doberman, had the same diagnosis by both the emergency-room vet and our regular vet. He was staggering, with little feeling in his back legs and much pain when he moved. Both vets suggested euthanasia, but I wasn't ready for that.

When the vet said a vertebrae was pressing on one of Major's nerves, I remembered that I'd been successfully treated by a chiropractor for the same thing. It took me three days to find a chiropractor who would treat a dog, but I finally did. By that evening, Major was no longer howling in pain when he moved. By the end of the second week he was running and playing happily. He never suffered the problem again and died five years later in his sleep.

Below is a link to Dr. Fox - I have never used him for reference but you may choose to do so.

Send your questions to Dr. Michael Fox in care of United Feature Syndicate, 200 Madison Ave., New York, NY 10016.