THE WOBBLER SYNDROME IN HORSES

by

Clara PRADIER

Promotors: Prof. Dr. Paul Simoens
Dr. Sofie Muylle

Literatuurstudie in het kader van de Masterproef

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I. Summary and keywords

The Wobbler syndrome is a neurological disease characterized by typical symptoms: incoordination, spasticity, hypermetria and ataxia. It is most often seen in young horses with a rapid growth and extensive training. The symptoms are the consequence of a lesion of the cervical spinal cord due to compression. The compression is created by static or dynamic stenosis of the spinal canal or, in older horses, a degenerative joint disease. These symptoms are also seen in non-wobblerian disease such as Equine Protozoal Myelitis or Equine Herpesvirus Myelopathy.

The anatomical pathways involved in the Wobbler syndrome are the general proprioception tracts i.e. the spinocerebellar tracts and the deeper upper motor neurons. Symptoms occur when they are no longer functional.

The etiology of the disease is unknown. A multifactorial base is suspected with factors such as excess and imbalance in the nutrition, external trauma and rapid growth. A genetic base has been long suspected and is still controversial.

The diagnosis is based on the horse history, radiography and myelography. More recently, MRI and CT have been used to confirm the diagnosis or in case the classical diagnosis technics were not successful.

Keywords: Horse – Wobbler syndrome – Neuroanatomy – Cervical Vertebral Malformation – Cervical Stenotic Myelopathy
II. Introduction

The definition of the name Wobbler syndrome is not easy since its signification has been evolving over the years without a general consensus and represents two different things. According to some authors, the syndrome is an ataxia with typical clinical signs, such as the hind limbs crossing over each other while circling, difficulty to step back and more (3,4,5). Any disease which produces this pattern of symptoms is part of the differential diagnosis of the Wobbler syndrome. This is the clinical point of view. Any clinician or veterinarian who observes the typical clinical pattern of the wobbler horse, he will classify it as such before even trying to find the cause. In practice, for many years the veterinarian gave the diagnosis Wobbler syndrome freely to any horse that moved unsteadily or unsurely back and forth or from side to side as if the horse were drunk (1). This uncoordinated walk is called wobbling and the horse is called a wobbler (16). Any lesion in the cervical region will cause a horse to wobble. The differential diagnosis for the clinical ataxia includes a long list of diseases, such as Equine Protozoal Myelitis, Equine Herpesvirus Myelopathy and many others, all causing the same symptoms due to lesions of the spinal cord.

This generic use of the name caused a real confusion but it is now considered out-dated. More recently the definition and even the name of the syndrome have evolved. The name Wobbler syndrome is no more representative of an association of clinical symptoms of a clinical ataxia but of a disease being part of the diagnosis for the gait deficiency. As such the name Wobbler Syndrome is now considered a miscalling (7,16) and is now generally named Cervical Vertebral Malformation (1,5,6,11,12,13). It is also known as Cervical Stenotic Myelopathy (2,3,4,5,9,11,13,15), Cervical Vertebral Compressive Myelopathy (8), Acquired Vertebral Malformation-Malarticulation (7,16), Cervical Vertebral instability (1,13), Cervical Vertebral Stenosis (1) or Equine sensory ataxia (11,13).

Even if the name is cause for confusion, there is a general agreement that the Wobbler Syndrome is caused by anything that creates a compression on the spinal cord leading to the wobbler’s typical signs, and that stenosis is the cause of the signs without revealing the origin of the stenosis or compression. Cervical Vertebral Malformation and Cervical Stenotic Myelopathy are, in the more recent literature, the most used naming for the Wobbler syndrome.

Another complication is the difference of signification of the name between authors. For example, for some authors Cervical Vertebral Instability is a synonym of Wobbler syndrome or Cervical Vertebral Malformation (1), whereas for others it is a subdivision of Cervical Vertebral Malformation and serves as a synonym for dynamic cervical stenosis (11,16).

In other words, the name “Wobbler syndrome” has two signification that differ. First it is, from the point of view of the clinician, a pattern of typical symptoms whether these signs are caused by Vertebral Cervical Malformation or any other disease affecting the spinal cord in the cervical region. A horse that comes to a clinic and present the symptoms of ataxia, spasticity, hypermetria, incoordination will be considered a wobbler horse therefore associating the non-wobblerian disease to the Wobbler syndrome (Cervical Vertebral Malformation). Second, the “Wobbler syndrome” name is a disease by
itself, on the same level as Equine Protozoal Myelitis or Equine Herpesvirus Myelopathy and therefore part of the differential diagnosis for ataxia or gait deficiency.

A way to differentiate Cervical Vertebral Malformation from the non-wobbler diseases is the typical evolution of a “true” wobbler horse. The Wobbler syndrome caused by Cervical Vertebral Malformation will slowly evolve and then stabilise (13). It will neither worsen nor improve, in contrast to cases caused by an Equine Protozoal Myelitis or Equine Herpesvirus Myelopathy.

The Wobbler syndrome is thus now considered as a form of clinical ataxia, characterized by incoordination, spasticity, hypermetria and ataxia (2). The name Wobbler comes from the first obvious symptom that is the “wobbling” of the horse.

Cervical Vertebral Malformation or Cervical Stenotic Myelopathy is a very common disease in horses that leads to a compressive myelopathy. It is characterized by malformation of the vertebrae, stenosis of the spinal canal and compression of the spinal cord which causes the symptoms (12).

There are two forms of Cervical Vertebral Malformation: a Vertebral Foramen Stenosis, which can be either static or dynamic, and a Degenerative Joint Disease of the (cervical) vertebral joints.

The Vertebral Foramen Stenosis is a disorder frequently seen in young athletic horses from 4 to 24 months of age, whereas Degenerative Joint Disease can occur at any age but is more common in older horses.
III. Anatomical notions and modifications

The Wobbler syndrome is a focal compressive myelopathy that can occur in any of the cervical vertebrae. In younger horses, it is more common at the cervical vertebra 3 to the cervical vertebra 4 or at the cervical vertebra 4 to the cervical vertebra 5 due to a static or a dynamic stenosis. In older horses, the cervical vertebra 5 to the cervical vertebra 7 regions presents a degenerative osteoarthropathy of the synovial articulations also known as Degenerative Joint Disease.

A. Review of the anatomical structures involved

1. The brain message and the reflex arc

   a) Hind limbs

Voluntary movements start with the brain emitting a message along the descending upper motor neuron pathways indicating to the muscle that it should contract. The message slides down the spinal cord, reaches the lower motor neuron or effector neuron, which will communicate the message to the motor neuron endplate. The muscle contracts.

Second, the sensitive receptors positioned in the muscle, the tendons and the articulation, will send a neural message to the brain informing it of the muscle contraction and the spatial position of the limb. This information goes up the proprioceptive nerve in the limb and reaches the spinal cord where it ascends to the brain and informs it. The nerve impulse will also directly inform the limb itself of the situation via the reflex arc. The nerve impulse will follow the dorsal spinocerebellar tract when it originates from the muscle receptor and the ventral spinocerebellar tract when it comes from the sinew.

The limb is informed via a reflex arc, as the proprioceptive nerve transfers the nerve impulse to a connector neuron, which is a short neuron situated in the spinal cord that communicates the nerve impulse to the effector neuron. The later is the same effector neuron that had received the UMN message.

The brain will integrate the message via the ascending pathways constituted by the dorsal and ventral spinocerebellar tracts. Their primary neurons are situated in the spinal ganglia. Their afferent axons will reach the spinal cord via the dorsal nerve root and form a synapse with the secondary neurons in the dorsal horn of the grey matter. Their axons form the proprioceptive pathways in the spinal cord to the brain: the spinocerebellar tracts. They are located superficially in the lateral funicule of the spinal cord (10).
**Reflex arc and high integration**

b) Forelimbs

The situation in the forelimbs is similar to that in the hind limbs, but the proprioceptive tract from the limb to the brain is the rostral spinocerebellar tract, which is situated more ventrally in the spinal cord than the dorsal and ventral spinocerebellar tracts (10).

The main sensory pathways


Fig.3: (from Simoens P. 2014)

B. Anatomical pathways involved in the Wobbler syndrome

Lesions in the white matter of the cervical spinal cord cause the typical symptoms of the Wobbler syndrome. The anatomical pathways involved in the sensory ataxia in the Wobbler syndrome are the general proprioception tracts i.e. the spinocerebellar tracts. Their superficial position in the spinal cord render them more fragile and exposed to compression. Spasticity and paresis occur when the deeper upper motor neurons are no longer functional. Prolonged compression will initially damage the superficial structures and progressively spread to the deeper areas (9,10,13).
C. Anatomical modification

1. Bone and soft tissue

Several pathological changes can be observed in case of Cervical Vertebral Malformation caused by abnormal biomechanical forces.

In the first type, the stenotic-malformation type, there is a disproportionately shorter vertebral body compared with a caudally extended vertebral arch and a narrower vertebral canal, especially at the caudal vertebral orifice. The vertebrae present a funnel shape that will lead to instability in the intervertebral articulation. This instability in adjacent vertebrae will cause angular deviations, a dorsal “flare” of the caudal epiphysis of the vertebral body (osteophyte formation on the articular processes), degenerative osteoarthritis and osteosclerosis of the dorsal lamina. There is a decreased cartilage differentiation and a higher bone density sometimes associated with necrosis and osteoclasia. Furthermore, fibrovascular and fibrocartilaginous proliferation of the soft tissues, such as the ligamentum flavum, joint capsule thickening and extradural synovial cysts, can also play a role in the spinal cord’s compression.

In cases of abnormal articulation of the vertebrae, the cranial orifice of the vertebral foramen of the more caudal vertebra is usually narrowed dorsoventrally (7,8,13).

Medial section of the C3 vertebra. Note the narrow caudal orifice of the vertebral foramen and the elevation of the vertebral body at the level of the caudal epiphysis.

Fig.4. (from de Lahunta et al. 2008)
In the second type, Degenerative Joint Disease, the Cervical Vertebral Malformation results from a chronic malarticulation in the more caudocervical vertebral articulations due to osteoarthritis at a young age or an injury followed by malarticulation. Spinal cord compression will begin when the proliferation of the articular processes and joint capsules extend in the vertebral foramen. Changes in ligaments and a synovial cyst formation associated with degenerative joint capsule will also contribute in the compression. Bone proliferation and joint capsule thickening lead to a dorsolateral compression of the spinal cord. (7,8,13)

Caudal aspect of the C3 vertebra after disarticulation at necropsy. Note the compression of the spinal cord in the caudal orifice of the vertebral foramen.

Fig.5. (from de Lahunta et al. 2008).

2. Spinal cord
Cervical Vertebral Malformation will eventually create a compression of the spinal cord leading to a focal lesion (9,13,14).

Macroscopically, a characteristic cervical spinal cord focal pressure-induced lesion, with damages mostly to the white matter and also to the grey matter, can be observed. Also a flattening or a decreased height of the spinal cord compared to adjacent segments can occur. This lesion may be 1 to 2 cm long (9,13).

Microscopically, the lesion site presents a myelin loss in all funiculi with evidence of axonal degeneration such as neuronal fibre swelling and astrocytic gliosis. A fibrosis process is indicated by an increased amount of perivascular collagen and macrophages. The ventral and lateral funiculi are extremely sensitive and will easily degenerate under compression or pressure. Sometimes the dorsal
funiculi can be spared. In severe lesions cavitation in the white mater, i.e. vacuolated spongy degeneration, can be replaced by hypertrophied astrocytes (8,11,13).

A secondary fibre degeneration or Wallerian-type degeneration occurs in the descending tracts caudal to the lesion and in the ascending tracts cranially. The degeneration occurs secondary to the lesion’s degeneration and is not caused by the compression. The Wallerian-type degeneration pattern can be used in order to find undetected primary lesions. It is characterized by myelin and axonal loss, perivascular fibrosis in the white matter and astrocytic gliosis distant from the lesion. Sometimes a complete absence of neurons in the dorsal and ventral horn of the grey matter can be noted with myelin loss only cranial to the lesion and not caudally (8,11,13)

Fig.6. (from Nout, Reed 2003)

Haematoxylin and eosin stained crossed section of the cervical spinal cord viewed at x40 magnification. This is the image of the ventral funicular at the level of C3-C4. In the top right corner the ventral median fissure is present. This cross-section demonstrates axonal swelling (large arrow head), axonophagia (small arrow heads) and axonal dropout (arrows).

Fig.7. (from Nout, Reed (2003)

Cross-section of the cervical spinal cord showing the affected proprioceptive tracts during spinal cord compression.
The pattern of neuropathologic lesions that occur with compressive myelopathy associated with Cervical Vertebral Malformation. The portions of the lesions in the transverse sections cranial and caudal to the focal region reflect the Wallerian degeneration of the cranial and caudal projecting pathways, respectively. (N.B. In an animal above is cranial and below is caudal)

Fig. 8. (from de Lahunta et al. 2008)

IV. Clinical signs and neurologic examination

Cervical Vertebral Malformation is characterized by the dysfunction of the upper motor neurons (UMN) and general proprioceptive tracts that are located in the spinal cord. The typical symptoms are spasticity, paresis, hypermetria and ataxia (1,2,5,9,11).

Clinical signs are usually symmetrical and located in all four limbs. The pelvic limbs tend to have more obvious signs due to the more superficial location of the pertaining neurons in the spinal cord (1,13). Another explanation is a bigger distance of the pelvic limbs from the gravity centre of the horse and more upper motor neurons synapses in the grey matter of the cervical intumescence (13).
However, it can occur that forelimb ataxia is more severe in horses with stenosis of the cervical vertebra 6 to the cervical vertebra 7 due to a compression of the cervical intumescence (5). In older horses, neck pain and lameness of the forelimbs can sometimes be observed in case of Degenerative Joint Disease without clinical signs of spinal compression. Occasionally, in Degenerative Joint Disease, a synovial cyst can develop causing a dorsolateral compression resulting in an asymmetrical ataxia and paresis (6,7).

More rarely, nerve root compression due to proliferative articular processes at the nerve's vertebral canal exit can occur with possible muscular atrophy of the neck muscles as well as prominent articular processes of the fifth and sixth cervical vertebrae, cutaneous hypoalgesia and hyporeflexia of the cervical reflexes adjacent to the place of spinal cord compression. These symptoms are the result of peripheral nerve compression and are more commonly seen in horses older than four years (13).

At rest, the affected horse can have a basewide stand, abnormal limb placement and a delayed proprioceptive response to repositioning (5,11,13,15). Horses may present chipped, worn or squared hooves due to toe-dragging (5,11,13,15). This is a sign that the neurological syndrome has been present for quite a time.

The neurological examination must be done on a nonslippery surface. Its purpose is to evaluate the patient’s gait. Normal postural reaction evaluations are too dangerous in neurological patients. Therefore it will only consist in walking in a straight line, circling, walking off and on step, backing, and pulling the tail. Attempting the more typical neurological test of “hopping” would be too dangerous. These positions require normal Upper Motor Neuron en General Proprioception tracts functions and will exacerbate the clinical signs in an affected horse. (8)

The Wobbler syndrome is graded in order to classify the severity of the symptoms. Some authors discern four neurological grades going from 0 to 4; 0 is a perfectly healthy horse with no symptoms and 4 is a falling horse at normal gaits (1). For others it can go to a fifth grade indicating recumbency (12,15). The difference of grade between the front and hind limbs can give an indication about the place and the type of lesion. If the hind limbs are two degrees or more higher than the front limbs it indicates that there is usually more than one lesion. If the hind limbs are only one grade above the front limbs it is a focal lesion. If the grades are similar in front and back then the lesion is located between the cervical vertebra 6 and the cervical vertebra 7 (1).

A. Walking in a straight line

The Wobbler horse demonstrates gait deficit such as varying stride lengths with a typical short forward phase of the stride and a low foot arc of his forelimbs, floating or dragging of the hooves, outward or inward swaying of the limbs (circumduction), unsteady movements of the pelvis and the trunk, and extension of the head and neck (6,8).

Head elevation can worsen the floating of the thoracic limbs and/or buckling of the pelvic limbs. (8)
B. The step
In conditions where the Wobbler horse is supposed to avoid an object lying on the floor, walking on and off a step, or in a slope, or simply passing over a ditch, the absence of information brought to the brain about the position of the horse’s limbs by the proprioceptive tracts will make the horse look at his feet and exacerbate the limbs movements in order to pass the “obstacle”. Most horses will hit the step or stumble. Elevation of the head will exacerbate these signs. (8,9)

C. Circling
Walking in tight circles is the most sensitive clinical test. The wobbler horse has a slow protraction of the limbs, will pivot on the inner limb while the spastic, rigid outer limb circumducts. Sometimes the horse will scuff its hooves, step on itself, or strike the inside of its own limb. Some horses may present the typical signs of laceration of the heel bulbs or the medial side of its limbs, typically called the “wobbler’s heel” from overreaching and interference (6,8,9).

D. Backing
The horse prompted to back will take a basewide stand or lean backward. While backing, the limbs drag and protract very slowly. Sometimes the horse will step with his front hooves on his hind hooves. In some cases, the horse can fall. (6,8,9)
E. Pulling the tail

To evaluate the paresis, the tail should be pulled ipsilaterally to the limb in, or just before, the weight-bearing phase of the movement. The diseased horse will sometimes stumble and try to correct its posture. (6,8,9)

Fig.11. (from Wobbler Syndrome a Disappointing Diagnosis, DCKEquineHospital.com 2014)

V. Etiology

The specific cause of the Wobbler syndrome is unknown. It appears to come from developmental orthopaedic disease of the appendicular skeleton such as physitis, joint effusion, osteochondrosis and flexural limb deformities (5). The high incidence in Thoroughbreds suggests a genetic predisposition. Some studies disclaimed a genetic basis (14,17,18,19) whereas others claim a genetic influence on Cervical Vertebral Malformation in the length of the neck, cervical vertebral mechanics and the horse’s size (14,20) or a genetic increased sensitivity of the cartilage growth to environmental factors (21). It is presumed to be a multifactorial disorder, factors going from genetic predispositions, to overnutrition (high plane nutrition and micronutrient imbalance), environmental and iatrogenic. (13)

The syndrome is often seen in male, young, large horses that are rapidly growing (1,5,13,14). Genetic selection for a rapid growth will create an imbalance between muscle and skeletal structures (1,9,13). A disparity between the vertebral column and the spinal cord will occur, causing a focal stretching, associated with a high plane nutrition and micronutrient imbalance: high in calcium/phosphorus, zinc and carbohydrate to encourage even more this rapid growth. Furthermore copper deficiency seems to be one of the factors leading to Cervical Vertebral Malformation by causing a defective lysyl oxidase which is necessary for connective tissue formation. An excess of zinc is the cause of a secondary copper deficiency. Carbohydrate excess creates an endocrine imbalance with increased insulin and decreased thyroxin concentrations creating the impossibility for the cartilage to adequately mature (1,8,13,14).
Sport horses are often subject at a young age to a vigorous training program that can lead to trauma or abnormal biomechanical forces that will contribute in the formation of the syndrome. All these factors will lead to the static or dynamic narrowing of the spinal canal and instability between vertebrae causing a compression of the spinal cord (5,13).

A second type of Wobbler syndrome is generally seen in older horses due to osteoarthritic enlargements of articular processes, bony and soft tissue proliferation. Nevertheless many horses suffering from osteoarthritic enlargements of the articular processes do not show any neurological signs; therefore only predisposed individuals with a relatively narrow vertebral canal are more likely to develop the syndrome. The pathogenesis of the disease in older horses is uncertain but external trauma and exercise in a predisposed animal is considered the best explanation (13).

VI. Diagnosis

A clinical diagnosis is important and will be based on the detailed horse history given by the owner or the rider and the recognition of the typical neurological symptoms. Breed and age of the horse are also good indicators. The definitive diagnosis of the Wobbler syndrome is made with radiography and myelography. Most likely, no diagnostic technique is sufficient on its own. Therefore complementary diagnostic techniques exist but are no often used:

1) Magnetic Resonance Imaging
2) CT scanner
3) Cerebrospinal fluid analysis
4) High resolution Cerebrospinal fluid electrophoresis

A. Radiography

To diagnose a suspicion of Wobbler Syndrome, strictly lateral radiographs of the occiput, all the cervical vertebrae and the thoracic vertebra 1 on a standing horse with the neck neither extended nor flexed are necessary. A light sedation can be of use since the long exposure time requires the horse to stand still (2,5,8).

Several radiographic features related to the pathological changes and anatomical modification of the bone structures and soft tissues can be observed on the radiographs. (2,8,11,13,15)

1) A mild subluxation of the vertebrae equivalent to the degree of dorsal angulation or misalignment between two vertebrae.
2) Vertebral body physeal enlargement and dorsal direction of the caudal physis.
3) Osteoarthritis and bony proliferation of the articular processes.
4) Osteochondrotic changes, associated with Degenerative Joint disease, including incomplete or delayed postnatal ossification of the articular processes.
5) Caudal extension of the dorsal aspect or dorsal laminae of the vertebra over the cranial physis of the vertebra that follows.
6) “Flare” of the caudal vertebral epiphysis of the vertebral body
7) Sclerosis of the dorsal laminae.
8) Soft tissue calcification dorsal to the intervertebral space and of the laminae of the caudal cervical vertebra.
Schematic drawing of the cervical vertebrae in neutral position illustrating the survey radiographic changes in Cervical Stenotic Myelopathy: subluxation, apparent extension of the vertebral arch, dorsal projection of the caudal epiphysis, degenerative changes of the articular processes. Note that in most cases C6 can easily be recognized by its broad lateral process.

Fig.12. (from van Biervliet et al. 2006)

Following the neutral position radiographs, three different radiographs should be taken in order to show the site of stenosis: a normally extended neck, a flexed neck and a “as much as possible” extended neck (2,5,9,13). All cervical vertebrae must be evaluated; assessing all vertebral bodies, vertebral foramina and articular processes. The measurement of the vertebral foramina and the flexion between the vertebrae will indicate the stenosis more precisely. In case of Degenerative Joint Disease in a wobbler horse the osteoarthropathy should be obvious on the radiographs. Ventro-dorsal radiographs may be necessary to diagnose a unilateral compression occasioned by an enlarged articular process in the case DJD.

Two measurements are made: the minimum sagittal diameter and the minimum flexion diameter of the vertebral canal. They are then used for the Radiographic Cervical Stenotic Myelopathy scoring (2,5,9,13).

1. **The minimum sagittal diameter**

   The minimum sagittal diameter is the measurement of each vertebral foramen made between the line that represents the roof of the vertebral foramen to the line representing its floor. This is the narrowest dorsoventral measurement and should be measured at the narrowest part of the vertebral foramen (2,9).

2. **The minimum flexion diameter**

   The minimum flexion diameter is taken on a radiograph of the neck undergoing as much flexion as possible in an anesthetized horse. This measurement is made between two vertebrae, from the most cranio-dorsal point of the caudal vertebral body to the closest portion of the dorsal aspect of the cranial vertebral foramen. It measures the vertebral canal’s size at the narrowest point (9).
3. **Radiographic Cervical Stenotic Myelopathy scoring**

The minimum sagittal diameter and the minimum flexion diameter are considered impossible to interpret due to magnification. Therefore a third system was created: radiographic Cervical Stenotic Myelopathy scoring, also called corrected minimum sagittal diameter. The stenosis is assessed by two ratios: the intravertebral ratio and the intervertebral ratio.

**Intravertebral and intervertebral ratio method**

The intravertebral vertebral ratio method is the ratio of the minimum sagittal diameter and helps calculate the size of the vertebral foramen. The sagittal ratio is obtained by dividing the minimum sagittal diameter of the vertebral foramen by the maximum sagittal diameter of the vertebral body. Both measurements are taken at the cranial aspect of the vertebra and perpendicular to the vertebral canal. The magnification problem of the minimum sagittal diameter alone is eliminated since the two measurements are taken in the same plane. A ratio below 50% (13) or 52% (2,5,7,8) for the cervical vertebral articulations 3 to 4, 4 to 5 and 5 to 6 and below 52% (13) or 56% (2,5,7,8) for the cervical vertebral articulation 6 to 7 is indicative for a foramen narrowing and a high chance of Cervical Stenotic Myelopathy. The test accuracy is up to 89-90% (8,13). Although the test is sensitive and specific in order to find a vertebral foramen narrowing, it does not indicate the place of spinal cord compression. Therefore, the results should not be used to determine the diagnosis alone and must be combined with other results or it will lead to false-positive results. Myelography is the most accurate diagnostic ante-mortem tool to diagnose Cervical Stenotic Myelopathy.

The intervertebral sagittal ratio has been proposed to obtain an even more precise notion of the occurrence of the spinal compression than with the intravertebral ratio. It is the ratio between the minimal distance from the most craniodorsal point of the vertebral body to the most caudal point of the vertebral arch of the more cranial vertebra and the maximum sagittal diameter of the cranial region of the caudal vertebra. The overlap between the results of the affected horses and the nonaffected horses creates the need for more research on the intervertebral sagittal ratios. (8,13)

![Fig.13.](from van Biervliet et al. 2006)

**Schematic drawing of the cervical vertebrae illustrating the sagittal ratios**: the intravertebral sagittal ratio is calculated as the ratio of the minimum sagittal diameter of the spinal canal (green line) to the maximum sagittal diameter of the vertebral body, taken at the cranial aspect of the vertebra and perpendicular to the spinal canal (red line). The « intervertebral » sagittal ratio is the ratio of the minimum distance taken from the most cranial aspect of the vertebral body to the most caudal aspect of the vertebral arch of the more cranial vertebra (blue line) and the maximal sagittal diameter of the vertebral body (red line).
Myelography is performed under general anaesthesia and in lateral recumbency. It should be used when the radiographs could not determine any lesion or to confirm the radiographic observations (1,13). It is also the diagnostic technique used to differentiate a static stenosis from a dynamic stenosis. A needle is placed in the subarachnoid space of the cerebellomedullary cistern at the foramen magnum in order to inject a radiopaque agent, generally iohexol (8), iopamidol (13) or meromazide (9), that is often wrongly (1) called dye. The head is elevated to an angle of 30° for 5 to 10 minutes in order to make the radiopaque agent flow caudally (fig15). Adequate positioning of the neck is extremely important. Flexion is considered achieved when the nose is positioned between the carpi. It is important to know that, on a flexed myelogram, narrowing of the dorsal dye line, also called the dorsal myelographic column, at the intervertebral junction indicates a compression of the spinal cord. For diagnostic accuracy it is now considered that the reduction should be 70% in order to avoid false-positive diagnoses. For more accuracy, the dural diameter reduction should be used. It represents the total reduction of the dural sac. Its value is obtained by a ratio between the minimum dural diameter at the intervertebral junction and the maximal dural diameter at mid-vertebral body (fig17). A reduction greater than 20% is deemed diagnostically worthy. A definitive diagnosis of the site of compression is rendered difficult in many cases by the presence of narrowing at several sites of the vertebral canal in many cases. (8,9,13)
1. **Cervical Static stenosis**
The stenosis is considered static if compression is observable regardless of the neck position and apparent on the normally extended and the flexed views. The articulations between the cervical vertebra 5 and the cervical vertebra 6 and from the cervical vertebra 6 to the cervical vertebra 7 are the predisposition places for a static stenosis. Cervical static stenosis occurs most often in older horses (13,8).

2. **Cervical Dynamic stenosis**
The stenosis is considered dynamic if a stenosis is apparent only on the flexed view. In a neutral position the cervical vertebral canal appears to be wide enough and only the movement will show the site of compression (fig16). Predisposition places are the articulations between the cervical vertebra 3 and cervical vertebra 4, and between cervical vertebra 5 to cervical vertebra 6. It is most common in younger horses (13,8).

![Photos showing the head positioning during myelography. (A) Head elevation is used to provoke a caudal flow of the contrast medium. (B) Adequate flexion can be achieved with a neck collar as illustrated.](image)

Fig.15. (from van Biervliet et al. 2006)
a) Cervical myelogram demonstrating dynamic compression of the cervical spinal cord at C3-C4. The neutral view demonstrates subluxation of the vertebral bodies at C3-C4 (arrow), strongly suggestive of spinal cord compression. b) Spinal cord compression is obvious when the head and neck are flexed. At C3-C4 the sagittal ratio is 31%. The dorsal column reduction is 83% and sagittal dural diameter reduction is 53%. Cervical Vertebral Stenotic Myelopathy at C3-C4 was confirmed by histopathology.
C. Magnetic Resonance Imaging (MRI) and Contrast-enhanced Computed Tomography (CECT)

False-positive and false-negative results have been reported using only radiographs and myelograms (13). A recent study has compared the diagnostic efficiency between radiographs, myelogram and MRI (12).

CECT can be used for presurgical evaluations at the suspected site of spinal cord compression or for detection of lateral compressive lesions that can’t be detected using radiography or myelography. The use of CECT allows an accurate detection and gives significant information over the location and severity of the lesions.

The difficulty in the use of a MRI or a CT scanner is the size of the horse sometimes not permitting an evaluation of the entire cervical vertebral column. (12,13,15)

Transverse magnetic resonance image showing the absence and presence of spinal cord compression. The image on the left is at the level of C4-5. The spinal cord is round, with circumferential spinal fluid (green arrows). From the same horse, the image on the right is at the level of C5-6 and demonstrates a narrowed canal area with ventral compression of the spinal cord, attenuated cerebral fluid and encroachment of the articular processes joints (yellow arrows). Compression of the spinal cord at C5-6 was confirmed on histological examination.

Fig.18. (from Janes et al. 2013)

D. Cerebrospinal fluid (CSF) analysis and electrophoresis

Cerebrospinal analysis is used to differentiate non-infectious ataxia (Wobbler syndrome) from infectious ataxia if the radiographs and myelogram findings are not conclusive.

High Cerebrospinal fluid electrophoresis can be used to help diagnose Cervical Stenotic Myelography (13).
VII. Discussion

The term “Wobbler syndrome” as it stands now has two meanings. In strict sense, Wobbler syndrome is a clinical condition characterised by ataxia, whereas in a large sense it is used for describing the association of pertaining symptoms including ataxia, incoordination, hypermetria and spasticity. Based on recent pathologic research about the etiology of the disease, the term “Wobbler syndrome” has been considered to be “nonspecific and it should be avoided” (7) or “superseded by more descriptive categorization” (16). More specific terms such as Cervical Vertebral Instability, Cervical Vertebral Malformation or Cervical Stenotic Myelopathy have been proposed as synonyms or as alternative terms. Still, further studies are needed to obtain consensus about the precise definition and a uniform nomenclature of the various aspects associated with “Wobbler syndrome”.

In contrast to the confusing terminology, the diagnosis for the disease is well-established, as much recent research using the newest technology has improved the detection of the disease. The pathological and post-mortem aspects of the disease are also well defined and well studied.

The etiology and exact pathogenesis of the disease are still unknown and controversial. There is a general consensus about the multifactorial origin of the disease, as a combination of genetic propensities, overnutrition, and environmental factors such as extensive training or external trauma, but more research on the exact etiology and on the different aspects of the multifactorial development of the disease seem necessary.
The Wobbler Syndrome in horses

VIII. References

