

Ataxia and ataxic lameness in a horse practice

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Regardless of the cause, lameness represents the most frequent condition preventing a mount from being ridden. Whenever constantly shifting lameness occurs, the head and spinal area should be considered as the possible cause.

1. Definition

What is ataxia: ataxia or lack of muscular coordination is a loss of ability to coordinate movement.

It is not a form of lameness. A distinction is made between cerebral, cerebellar and spinal ataxia, depending on where in the central nervous system the injury is located.

2. Causes

In the case of cerebral ataxia, the cause of the lack of muscular coordination lies in the area of the cerebrum, diencephalon or midbrain. It occurs in horses in connection with traumatic or infectious diseases of the central nervous system.

With cerebellar ataxia, the dysfunction originates in the cerebellum. The cerebellum is the coordination centre enabling all movements such as controlling muscle tone, balance, motor performance, etc. to be executed correctly. Clinical relevance exists with Oldenburg and Arab horses and Gotland ponies. Cerebellar ataxia consists of a congenital or hereditary degenerative change in the cerebellum.

Spinal ataxia, which is the subject of this paper, has long been known in horses. It consists of a constriction of the cervical, thoracic and lumbar region of the spinal canal.

In foals the cervical form of spinal ataxia

predominates. This is caused by rollover injuries resulting in extreme ventral flexion of the cervical spine. The dorsal ligaments of the cervical spine are overstretched as a result, leading to inflammation and subsequent circulatory disturbance in the apophysis. This ultimately leads to apophysial necrosis, impaired linear growth of the cervical vertebrae and consequently to the head of the vertebra sliding dorsally out of the corresponding vertebral socket on ventral flexion of the cervical vertebra in question. As a result, the cervical spinal cord is constricted and crushed in places. This leads to disturbed sensomotor function patterns.

Spinal ataxia in older horses occurs principally as the result of slipped discs, inflammation, bruises, fissures, prolapsed articular hydrops and restricting processes.

3. Symptoms

The gait of foals and young horses becomes increasingly unsteady especially in the hindquarters. Colts are affected more frequently than fillies. When the animal turns, its hindquarters collapse outwards. When it brakes suddenly, the hindquarters remain unsteady and tend to overtake the front of the body. The same happens when the animal travels downhill.

In less severe cases, constantly shifting lameness, stiff back and neck, short strides and pinched tail are observed.

4. Diagnosis

Table Diagnosis

- Visualisation
- Ataxia tests such as turning, sudden braking, reining back, tail test
- Sensitivity palpation
- X-ray

5. Prognosis

Always very cautious with medical treatment "lege artis".

6. Therapy

Drug treatment administered:

- glucocorticoids
- anabolic agents
- selenium
- vitamin E
- antiphlogistics
- mannitol solutions

Surgical treatment:

As the horse is a food source, orthodox medical treatment brings the vet into conflict with the law or increases the administrative burden.

7. Bioresonance therapy

In my practice I have found bioresonance therapy to be the most successful form of treatment.

Table First treatment session

- Basic therapy
- Geopathy compensation
- Eliminate scar interference
- Stimulate eliminating organs

After basic therapy (in most cases program no. 130) the main blocks such as radiation stress (program no. 700) and scars (program no. 910) are treated. The

input electrode is placed on the back of the horse's neck for this.

The eliminating organs are stimulated with programs 430 (liver), 930 (lymph system) and 480 (kidneys). The input electrode is placed on the appropriate organs.

The modulation mat is placed on the animal's back or fastened around the base of the neck.

In the case of generalised joint problems, allergens, foreign matter and bacteria (mainly Borrelia) should also be tested out and eliminated if the result is positive.

Only then are the affected segments of the spine treated according to the cause of the problem.

Table Causes of the complaint

- Bruising
- Strain
- Stretched ligaments
- Formation of oedemas
- Haematoma
- Nerve crushing
- Nerve degeneration
- Slipped disk
- Vertebral fracture
- Arthrosis
- Vertebral fissure
- Vertebral fracture

It is not possible to define all the causes of a complaint with certainty as the precise time the problem began is not always known and the diagnostic aids which provide an image cannot always be analysed with precision.

Determining the cause therefore depends upon what the attending vet learns of the animal's medical history and his experience and understanding of the development of the disease.

CASE STUDIES

Case 1: Gelding, 2 years old

Previous history:

Quarter horse, treated several times by vets, "set" by osteopath, various diagnoses of lameness.

Own examination:

When walking free, I noticed the horse was very stiff in the back, its tail was pinched, neck and head held straight out stretched upwards. Trot and gallop were hard and staccato-like.

Ataxia tests were negative. The gelding would not allow its ears to be touched.

When palpated the horse reacted very sensitively by thrusting its head upwards when the region of the occipital bone and the first two cervical vertebrae were touched. The remainder of the back was pain-free.

On questioning the owner extensively, it emerged that the gelding was often tied up. It resisted this, pulling violently on its halter, in so doing bruising the back of its neck.

Own diagnosis:

Relatively acute bruising on the back of the neck with oedematization and inflammation of all the surrounding connective tissue structures.

Own treatment on 1st day:

Basic therapy with no. 130
Geopathy compensation no. 700
Elimination of scar interference no. 910
Stimulation of eliminating organs:

- no. 430
- no. 930
- no. 480

Main therapy:

Oedema no. 610
Connective tissue process no. 923
Connective tissue disease no. 433

The next day the animal only reacted moderately to palpation.

Own treatment on 2nd day:

Main therapy:

Oedema no. 610
Connective tissue process no. 923
Damaged intervertebral disc no. 440, 341

Own treatment one week later:

Main therapy:

Connective tissue process no. 923
Damaged intervertebral disc no. 440, 341
Arthrosis no. 821, 633

After this treatment the animal was completely pain-free and would allow its ears to be touched. The pain spots were completely insensitive.

When walking free the animal's head and neck were rounded and held tilted downwards, its back swung freely and it carried its tail.

Its strides were light and long.

The gelding has not so far had any relapse.

Case 2: Stallion, 3 years old

Previous history:

Oldenburg breed, had been in various clinics, had been treated by osteopaths. Diagnosis: spinal ataxia.

X-rays from clinic not abnormal.

Own examination:

Animal had huge coordination problems when walking free. When braking its hind quarters "overtook" the front of the animal's body.

Tail test positive. Impossible to rein back. The stallion was very aggressive.

Sensitivity palpation in the region of 2nd, 3rd, 4th cervical vertebra extremely positive. The remainder of the back was hypersensitive.

As a foal the horse had often tumbled.

Own diagnosis:

Spinal ataxia, cervical spinal cord crushed damaging the posterior column system.

Probably also arthrosis on the intervertebral joints.

Own treatment on 1st day:

Basic therapy with no. 130

Geopathy compensation no. 700

Elimination of scar interference no. 910

Stimulation of eliminating organs:

- no. 430
- no. 930
- no. 480

Main therapy:

Damaged vertebral disc no. 440, 341

Arthrosis no. 821, 633

Oedema no. 610

Connective tissue disease no. 923

Own treatment on 2nd day:

Main therapy:

Damaged vertebral disc no. 440, 341

Arthrosis no. 821, 633

Oedema no. 610

Connective tissue disease no. 923

The stallion still displayed slight sensitivity to palpation. He was less aggressive and took to the treatment well.

Own treatment 1 week later:

Main therapy:

Damaged vertebral disc no. 440, 341

Arthrosis no. 821, 633

Connective tissue disease no. 923

Nerve degeneration no. 271, 230

During this treatment the animal struck the loosebox wall with its left hind leg in a reflex action.

Neuralgic pain no. 911, 423

Sensitivity palpation was negative. The stallion seemed to be able to control itself better when walking.

Own treatment 1 week later:

Main therapy:

Connective tissue disease no. 923

Nerve degeneration no. 271, 230

Neuralgic pain no. 911, 423

Weekly therapy over a six month period alternating between:

Damaged vertebral disc no. 440, 341

Arthrosis no. 821, 633

Connective tissue disease no. 923

Nerve degeneration no. 271, 230

At the end of the six months, the stallion walked normally. The ataxia tests were negative.

Now, eighteen months later, the animal (now a gelding) can be ridden successfully. He now has a perfect temperament.

Case 3: Mare, 1 year old

Previous history:

Warmblood. When in a herd of yearlings run over by the rest of the herd. Could only stand up if assisted. Diagnosis by a colleague: Fracture of the 3rd lumbar vertebra. Recommended slaughtering the animal.

Own examination:

The mare staggered when standing. Sensitivity palpation in C2, C3 region extremely positive, in L3 region extremely positive. Rapid movement caused the horse to fall over. Active walking was not possible. X-rays were not taken due to the cost and general anaesthetic. It was essential to treat the horse as it came from a valuable blood line and, if successful, the animal could be used for breeding.

Own diagnosis:

Trauma of C2, C3, L3 with crushing of the bone marrow, conductivity disorders, possible fracture or fissure of aforementioned vertebrae.

Own treatment on 1st day:

Basic therapy with no. 130

Geopathy compensation no. 700

Elimination of scar interference no. 910

Stimulation of eliminating organs:

- no. 430
- no. 930
- no. 480

Main therapy:

Damaged intervertebral disc no. 440, 341

Oedema no. 610

Connective tissue disease no. 923

During treatment the mare was very nervous and struck out in an uncontrolled manner with her forelegs. She kept bending her rear legs.

Own treatment on 2nd day:

Main therapy:

Damaged intervertebral disc no. 440, 341

Oedema no. 610

Connective tissue disease no. 923

The animal was able to stand more steadily and remained calm during the treatment.

Own treatment on 3rd day:

Main therapy:

Oedema no. 610

Connective tissue disease no. 923

Mesenchymal therapy no. 433 (Pain intensification, immunological response)

Nerve degeneration no. 271, 230

Own treatment 1 week later:

Main therapy:

Oedema no. 610

Connective tissue disease no. 923

Damaged intervertebral disc no. 440, 341

Arthrosis no. 821, 633

The animal was pain-free when palpated. Its forelegs were taking its weight normally. The mare could stand and walk in a specific direction using its hind legs. The following week the animal fell while running with the panic-stricken herd and slid under a fence.

The mare was separated (but still allowing contact with the other horses). The problem with its hindquarters had deteriorated again.

The aforementioned treatment was performed each week over a six month period.

Now, about 1 year later, the horse walks normally. When walking quickly, agitated and trotting, the hind legs, especially the right leg, land irregularly. The mare has grown to a normal size, has no visible pain and can definitely be used for breeding.

Bibliography

Handbuch Pferdepraxis

[Equine practice manual], Dietz/Huskamp