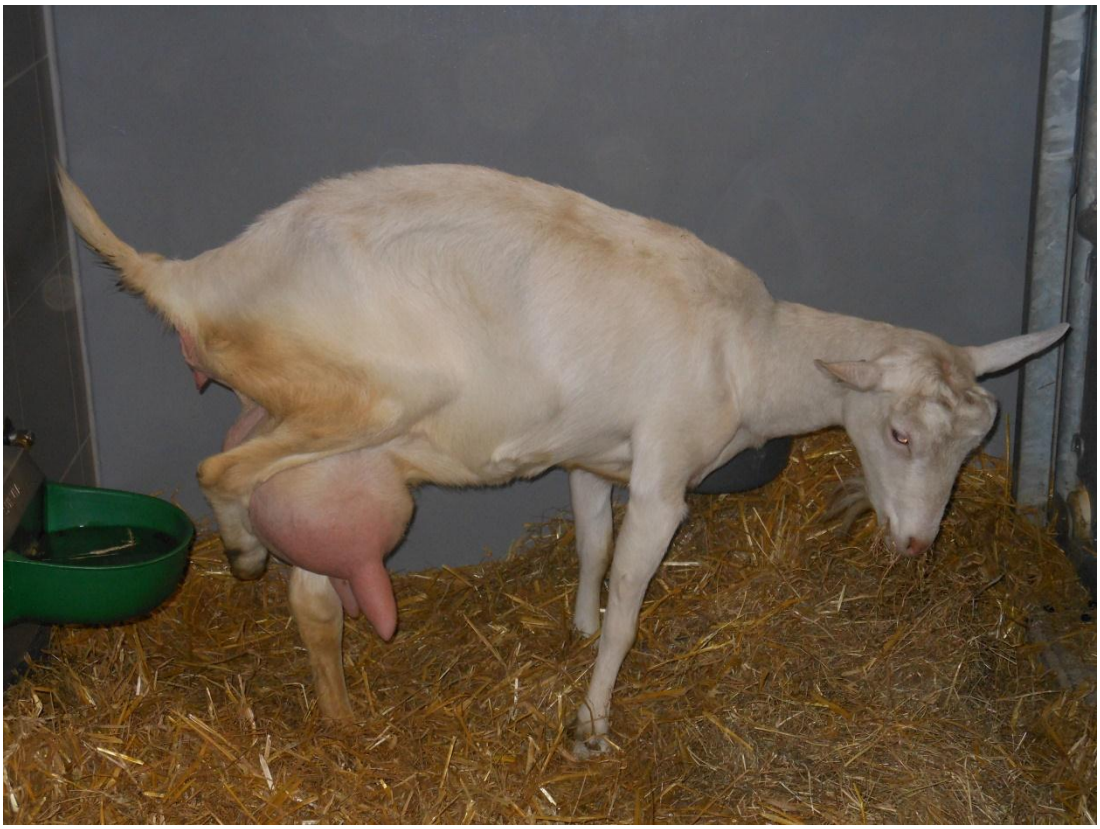
De Gezondheidsdienst voor Dieren

Progressive bilateral (spastic) paresis, ataxia posterior and a decreased perineum tone in Dutch dairy goats:

Pathophysiology



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Abstract

Progressive bilateral (spastic) paresis, ataxia posterior and a decreased perineum tone in Dutch white dairy goats was an uncommon syndrome in dairy goat farming in The Netherlands. The aim of this study was to study the pathophysiological background of progressive bilateral (spastic) paresis, ataxia posterior and a decreased perineum tone in dairy goats. Therefore several methods have been used for data collection: farm visits, blood clinical chemistry, clinical and neurological examination under clinic conditions, quantitative electromyography (QEMG) and pathological examination. No significant differences were observed between cases and controls in blood clinical chemistry. Clinical-neurological observations were: kyphosis of the lumbosacral region, pelvic tilt, moderate musculature of the hind quarters, hypotension of the perirectal and perivaginal area and an atonic tail. The locomotion was characterized by paresis and ataxia posterior with extended tibio-tarsal joints. The perineal reflex was absent and the withdrawal reflex and patellar reflex were decreased. The QEMG measured spastic muscle contractions in the lateral vastus muscle of the quadriceps muscle of the thigh and the biceps femoris muscle. Pathological examination found no abnormalities at macroscopic level. The most likely diagnosis on the basis of clinical-neurological examination was a lesion in the thoracolumbar and lumbosacral segments of the spinal cord. Further research was necessary to determine a more accurate localization of the abnormality.

Keywords: goat, spastic, prolapse, paresis, neuropathology, spinal cord

Introduction

The syndrome *progressive bilateral (spastic) paresis, ataxia posterior and a decreased perineum tone in Dutch white dairy goats* was formerly known as progressive (spastic) paresis posterior in combination with vaginal and/or rectal prolapse. This syndrome was discovered by the “Veekijker”, a monitoring system of The GD-Animal Health Service in Deventer, The Netherlands in 2012. This monitoring system is financially supported by the Dutch Ministry of Economic Affairs (EZ) and the Product Board for Livestock and Meat (PVV). This syndrome was most common in pregnant yearlings, during the last trimester of gestation, but it was also observed in yearling bucks. The animals develop bilateral (spastic) paresis posterior and showed the following symptoms: a steep position of both hind legs, (spastic) stretch of the hind legs, pelvic tilt, kyphosis, abnormal crossing of the hind legs in standstill and moderate musculature. Some cases exhibited hypotension of the perirectal and/or perivaginal area in combination with the other clinical signs. In severe cases, a vaginal and/or rectal prolapse occurred. These prolapses presented as a passive process, there was no straining observed (Koekkoek, 2013).

In prior research (Koekkoek, 2013) the incidence, risk factors and the clinical observation of this syndrome investigated by means of surveys sent out to dairy goat farmers and farm visitors, farm visits, blood clinical chemistry and pathological examination. The response from commercial dairy goat farmers (famers keeping more than 100 animals) to the survey was 28% (97/342), 46 farmers (47%) of the responders indicated they had observed symptoms of bilateral (spastic) paresis posterior and/or prolapse (rectal and/or vaginal). One hundred and seventy one (171) goats were observed with clinical signs of bilateral spastic paresis posterior on 33 different dairy goat farms and 407 goats with rectal and/or vaginal prolapse on 47 different dairy goat farms. Goats with both symptoms of bilateral (spastic) paresis posterior and prolapse (rectal and/or vaginal), were detected in 37 animals on 11 farms. In total, 615 dairy goats observed with symptoms of bilateral (spastic) paresis posterior and/or a prolapse (rectal and/or vaginal). Sixteen point two (16.2) percent of the farmers observed a progression, 16.5% a regression whereby in some cases complete recovery. A steady condition was reported by 32.5% farmers and 34.8% reported different courses in different animals. Farm visitors (veterinarians and nutrition advisors) were sent out a similar survey, the response was 31% (50/160). Forty- two point nine (42.9) percent of the responders observed symptoms of bilateral (spastic) paresis posterior, 69.4% observed symptoms of prolapse (rectal and/or vaginal) and

15.2% observed goats with both symptoms. Percentage affected animals in a herd according to farm visitors vary from 0.5 to 15.5%.
There was no apparent difference in incidence of clinical signs between conventional and organic dairy goat farms, and none of the risk factors studied in this research turned out to be related to this syndrome (Koekkoek, 2013) . This means that the etiology of this syndrome was so far totally unclear.

There probably were several risk factors or causes correlated with bilateral (spastic) paresis, ataxia posterior and a decreased perineum tone. Therefore, in this research, the syndrome was approached from a different angle: the pathophysiology of the clinical signs.

The aim of this study was to study the pathophysiological background of progressive bilateral (spastic) paresis and ataxia posterior and a decreased perineum tone by Dutch white dairy goats. This has been achieved objective by clinical and neurological examination of goats with these specific clinical signs. The clinical signs and findings on a repeated neurologic examination did result in an accurate anatomical diagnosis. It was important to localize the lesion for specific pathological examination.

This research consisted of different methods to collect data: farm visits, blood clinical chemistry, clinical and neurological examination under clinic conditions, QEMG on case animals and pathological examination.

Materials and Methods

Farm visits and animals

Four farms were selected on the basis of the response to the surveys made by another researcher previously (Koekkoek, 2013). These four farmers reported signs of spastic paresis and wanted to come in contact with GD-Animal Health Service to discuss this subject. Only commercial dairy goat farms with over 100 dairy goats and almost exclusively Dutch white dairy goats were included to this study. The objective of the farm visits was to select animals with clinical signs of spastic paresis and ataxia posterior and decreased perineum tone (appendix 1) for further research in the clinic of the Department of Farm Animal Health at the Faculty of Veterinary Medicine at the University of Utrecht. Two farms with case animals were visit, one with a history of this syndrome and one farm with problems of kyphosis. The first farm was an organic farm with 200 Dutch white dairy goats. The case animal on this farm was a 22 months old goat and the only animal with symptoms of spastic paresis and ataxia posterior and decreased perineum tone. The symptoms had arisen when the goat was pregnant (± 9 months old). The second farm was a conventional farm with 1400 goats. In the past, 20% of the goats on this farm had symptoms of spastic paresis and vaginal/rectal prolapse. The case animal on this farm was an 11 months old goat with symptoms of spastic paresis since a few weeks. Both case animals were selected for further research. Farm inspection was carried out on all four farms. Blood sampling was done on the two case farms, on each farm one case and two controls. The case animals were the same animals selected for further research. The third farm was a farm with a history of this syndrome, but without clinical cases at this moment. The farmer of the fourth farm described problems resembled the spastic paresis, but upon inspection the goats had only kyphosis without symptoms of spastic paresis. The two case animals were placed in research in the clinic of the Department of Farm Animal Health at the Faculty of Veterinary Medicine at the University of Utrecht. The goats were housed together on straw and were fed a goat ration containing of grass silage, straw and hay, and tap water was available at libitum. Stress was prevented as much as possible. This was accomplished by the goats housed in a department only for small ruminants, thermo neutral conditions: temperature of 18˚C, no draught, daylight, no loud noises and good veterinary animal handling.

Human endpoint was defined as: constant vaginal or rectal prolapse and/or not eating and drinking longer than 24 hours and/or not being able to stand independently.

Blood clinical chemistry

Blood samples for clinical chemistry were collected during the farm visits from one (1) case and (2) control animals. There were also clinical chemistry results from previously visited included. Blood sampling was done lege artis by jugular vene puncture (BD Vacutainer®, Flashback Blood Collection Needle, BD Vacutainer®, One Use Holder, and BD Vacutainer®). Three tubes were taken from each goat, one heparin tube and two

serum tubes. The following blood parameters in serum were determined: haemolysis index, creatine phosphokinase (CPK), anorganic phosphorus, calcium all by Ultra violet UV)/Visible light (VIS)-spectroscopy, potassium (Ion-Selective Electrode module (ISE)), magnesium (Inductively Coupled Plasma (ICP) Atomic Emission Spectroscopy (AES)), copper (ICP-Mass Spectrometry (MS)), osteocalcin , C-terminal cross-linked telopeptide (CTX) both by ELISA. Glutathione peroxidase (GSH-px) was determined in heparin (UV/VIS). The chosen analysis for clinical chemistry were accredited by the Council of Accreditation and performed by the GD-Animal Health Service, Deventer, The Netherlands.

There were two ELISA's available for osteocalcin: IDS® (Immunodiagnostic Systems Limited, United Kingdom) and Quidel® (Quidel Cooperation, United States of America). Both tests were used to investigate which test was most useful in goats (appendix 2).

Clinical examination

The goats were observed and examined clinically and neurologically for seven consecutive days. Upon arrival at the clinic, the study started with a general impression of the goats, based on Kuiper and van Nieuwstadt (2008) (appendix 3). The goats were examined once a day according a protocol based on clinical examination of Kuiper and van Nieuwstadt (2008) (appendix 4) and neurological examination based on Kuiper and van Nieuwstadt (2008) and Constable (2004) (appendix 5). Tools for clinical and neurological examination were: stethoscope, clinical thermometer, Taylor reflex hammer, artery forceps and a pen light.

Quantitative needle electromyography (QEMG)

A quantitative needle electromyography (QEMG), including motor unit action potential (MUAP) analysis was performed on the lateral vastus muscle of the quadriceps muscle of the thigh, the biceps muscle of the thigh and the ventral serrate muscle in both case goats. The material and method have been described by Wijnberg *et al*. (2002). The examination was conducted using a portable EMG apparatus (Nicolet Meridian EMG apparatus, Nicolet Biomedical Inc, Madison, Wis.), connected to a portable computer (Topline 8000,Topline, Hoevelaken, The Netherlands) for recording and saving the signals, and a disposable 23 G concentric EMG needle (EMG concentric needle electrode, Nicolet Biomedical Inc, Madison, Wis. diameter 0.60mm, length 75mm, recording area 0.068mm^2). A surgical pad was used as the ground electrode and was attached to the goat using a belt and connected to the preamplifier. MUAPs were generated by voluntary contractions of the measured muscles. The EMG needle was inserted in different locations in each muscle. Sweep speed was 20-100ms/division, band pass was 20-10k,50Hz, and amplifier gain was 100-200µV/division (Wijnberg *et al.* 2004).

Pathological examination

Pathological examination of the two case animals was performed according to a special pathology protocol based on findings of clinical examination (appendix 6).

Data analysis

Case and control animals were compared statistically with a two sample T-test.

Results and Discussion

Clinical chemistry blood

Results of clinical chemistry collected from the selected case animals and control animals on the farms of origin and in addition, there were also devious results included (table 1). Reference value of The GD-Animal Health Service in Deventer, The Netherlands (GD) were compared with reference value found in the literature.

Table 1: Clinical chemistry blood parameters, case and control: mean, (range), standard deviation (SD), P value, reference value

Parameter	Cases mean, (range), SD (N = 14)	Controls mean, (range), SD (N = 12)	P	Reference value
CPK (IU/l)	304.71 (121 – 600) 151.71	268.92 (105 – 525) 137.74	0.54	49.1± 2 (adult)(Stevens *et al.* 1994) 24.0 ± 7.0 (Saanen adult) 48.0 ± 22.0 Saanen kids 40-59 days old) 22.0 ± 8.0 (Saanen kids 240-259 days old) (Boss, 1977) 13 – 210 (GD)
Calcium (mmol/l)	3.34 (2.17 – 2.52) 0.12	2.41 (1.98 – 2.68) 0.22	0.31	2.23 – 2.93 (Feldman *et al.* 2000) 2.2 – 3.6 (Smolders *et al.* 2010) 2.52 (Mellado *et al.* 2006) 1.57 (young) 2.23 (old) (Ahmed *et al.* 2000) 2.53 (1.65 – 3.03) (Mellado *et al.* 2006) 2.43 ± 0.18 (Stevens *et al.* 1994) 2.2-3.6 (GD)
Phosphor (mmol/l)	2.33 (1.7 – 3.3) 0.49	2.23 (1.1 – 3.6) 0.67	0.52	1.36 – 3.17 (adults) 2.68 – 3.33 (juvenile) (Sherman, 1983) 1.712 (1.03 – 4.13) (Mellado *et al.* 2006) 2.26 ± 0.7 (Stevens *et al.* 1994) 1.1 – 2.4 (GD)
Potassium (mmol/l)	6.68[1] (5.3 – 8.2) 1.13	6.60 (4.6 – 8.7) 1.21	0.88	3.5 – 6.7 (Feldman *et al.* 2000) 3.5 – 5.2 (Ahmed *et al.* 2000) 4.0 – 7.0 (Smolders *et al.* 2010) 4.0 – 7.0 (GD)
Copper (µmol/l)	16.56 (11.8 – 23.9) 4.02	15.34 (11.8 – 22.1) 3.02	0.40	9.13 – 25.2 (sheep) (Kaneko *et al.* 1997) 14 – 24 (Smolders *et al.* 2010) 43 – 82 (Ahmed *et al.* 2001) 12.60 (3.15 – 42.50) (Mellado *et al.* 2006) 14 – 24 (GD)
Magnesium (mmol/l)	1.16[2] (0.95 – 1.9) 0.30	1.07[2] (0.96 – 1.23) 0.09	0.44	1.23 – 1.59 (Feldman *et al.* 2000) 1.10 – 1.93 (Ahmed *et al.* 2000) 0.79 (0.31-1.32) (Mellado *et al.* 2006) 0.9 – 1.7 (Smolders *et al.* 2010) 0.8 – 13.0 (GD)
Vitamin B12 (pmol/l)	377.29 (92 – 592) 165.22	431.50[3] (107-647) 184.54	0.42	77.49 – 1476 (mean: 533.65) (Al-Zadjali *et al.* 2004) >221 (GD)
GSH-Px (IU/g HB)	770.93 (595 – 1132) 157.07	769.67 (541 – 1112) 182.45	0.99	200 – 1000 (Smolders *et al.* 2010) 120 – 600 (GD)
Osteocalcin (µg/l)	56.55[4] (49.1 – 64.0) 10.54	74.58[4] (42.5 – 126.1) 36.29	0.55	9.2 (cattle) (Larsen *et al.* 2001) 0.24-0.28(cattle) (Kamiya *et al.* 2010) 15 – 25 (GD)
CTX (µg/l)	0.85[5] (0.39 – 1.42) 0.41	0.82[5] (0.37 – 2.13) 0.66	0.92	0.2 – 0.8(GD)

[1] Potassium: 9 case animals, [2] magnesium: 8 case animals, 10 control animals, [3] Vitamin B12: 14 control animals, [4] Osteocalcin: 2 case animals, 4 control animals, [5] CTx: 6 case animals, 8 control animals

There are few studies on reference of clinical chemistry parameters of the dairy goats, in particular references of Dutch dairy goats. It was therefore questionable whether the right references for Dutch dairy goats were used. Research of Smolders *et al.* (2010) investigated the blood samples of different Dutch goat herds and compared the results with available literature.

The difference between case and control animals was not significant ($P > 0.05$). It was unlikely to demonstrate an effect, because the numbers of the goats were small (14 case, 12 control), and for some parameters very small (osteocalcin: 4 case, 2 control). In other words, the probability of a type II error was present.

The CPK levels in blood were increased for both case and control animals. Eight (8) of the fourteen (14) case animals and 7 of the 12 control animals were above the reference value. This was an expected result for case animals, because clinical-neurological examination and pathological examination have shown a moderate musculature. On the other hand, this syndrome was probably a chronic process without severe muscle breakdown. There was a wide range between mild muscle and severe muscle damage. Small increase in serum CPK may be resulted from moderate muscle damage after intramuscular injections, transport or fighting. Acute muscle damage was associated with hundredfold increase in CPK (Smith & Sherman, 2009).

The calcium blood results of both the case and the control animals were within the normal reference value except one case and one control animal. These two results were slightly lower than the reference value of both GD and others. Hypocalcaemia was associated with a decreased consciousness, decreased in appetite, progressive paralysis of striated and smooth muscles resulted in obstipation and a decreased rumen motility associated with development of bloat (Cockcroft, 1999). Norwegian study found that forty goats with clinically hypocalcaemia had an average plasma calcium concentration of 0.93 mmol/l (range 0.73 – 1.28), compared thirty-six normal goats with an average of 2.48 mmol/l (Overby, 1980). The two results were slightly lower, therefore hypocalcaemia was unlikely.

The phosphor levels in blood for both case and control animals were increased compared with the reference of GD. It was also questionable of the reference used for phosphate in goats was correct. Mellano *et al.* (2006) found the reference 1.03-4.13 mmol/l for goat.

The potassium levels for both case and control animals were increased compared with the reference of the GD, but also other references (Feldman *et al.* 2000, Smolders *et al.* 2010, Ahmed *et al.* 2000). High potassium levels in ration have a negative effect on magnesium and sodium utilization (Smolders *et al.* 2010). But the magnesium levels in the blood of these were within the reference value.

The copper levels in the blood were in six (6) of the fourteen (14) case animals and five (5) of the twelve (12) slightly lower than the reference value. The goats with lower copper levels came from different farms. Copper deficiency can be divided in primary copper deficiency as result of low intake, or secondary due to high concentrations of sulphur and iron or molybdenum. Copper, sulphur and molybdenum formed in the rumen thiomolybdates, which reduced the copper availability (Pugh, 2002). Congenital copper deficiency in young kids was associated with sway back and enzootic ataxia. Copper deficiency in adult animals was often not associated with signs, but may be associated with depigmentation of the hair coat, diarrhoea and anaemia (Smith & Sherman, 2009). There were no symptoms of copper deficiency observed on the farms.

The blood test results for vitamin B12 were in two (2) of the fourteen (14) case animals and three (3) of the twelve (12) control animals slightly lower than the reference value. The goats with lower copper levels came from different farms. Johnson *et al.* (2004) demonstrated that goats with a vitamin B12 deficiency was associated with a decline in erythrocytes, reduced weight gains, dry scruffy hair coat and hepatic lipidosis in Omani goat.

Selenium was analysed by determination of the enzyme GSH-px. GSH-px was increased compared with the reference value. The reference value of the GD was derived from cattle. Dercksen *et al.* (2007) investigated the requirement of selenium by dairy goats and compared the GSH-px values of goats with other animal species. It showed that a similar amount of selenium in the ration of goats, GSH-px values were higher than other animal species. Probably goats used selenium more efficient.

The IDS® osteocalcin test gave undetectable osteocalcin values for goats, however, the Quidel® gave detectable values. The values of the Quidel® were as expected for young adult animals (Counotte, 2013). The reference range for osteocalcin was the reference range for pigs. Compared with studies on bone metabolism in

goats, this reference range seemed to be useful for goats (Liesegang *et al.* 2003, Liesegang *et al.* 2006, Liesegang, 2008). The number of goats were small (2 case, 4 control), and different in age, stage of lactation and pregnancy. The first group of three goats (1 case, 2 controls) were goats from 18 months old, the first lactation. The other group of three goats (1 case, 2 controls), were young animals of 10 months old, not in lactation.

The CTX levels in the blood were in four (4) of the eight (8) case animals and one (1) of the six (6) control animals slightly higher than the reference value of GD. The reference value of CTX was also the reference value of pigs. The reference range of CTX is used in pigs and seemed to be useful in goats (Liesegang *et al.* 2003, Liesegang 2008). There was no significant difference found in bone formation and bone absorption between case and control goats. This was an unexpected result, because these goats had a decreased mechanical loading of the hind legs (Iolascon *et al.* 2013). It may be explained by that the disease was a chronic process and the bone metabolism was stable: no bone formation or absorption.

Previous researcher had the clinical chemistry discussed in detail (Koekkoek, 2013).

Clinical examination

Behaviour and level of consciousness

General impression was not abnormal except the behaviour and level of consciousness, because the goats were less alert than expected in dairy goats. General examination showed abnormalities in posture and gait.

Posture and gait

Clinical observations of the posture of the goats showed the following symptoms: kyphosis of the lumbosacral region, pelvic tilt (figure 1), moderate musculature of the hindquarters (semitendinosus muscle, semimembranosus muscle and biceps muscle of the thigh) (figure 2), abnormal crossing of the hind legs, wherein in these goats the right hind leg was spastic crossed in front or behind the other hind leg (figure 3 and 4) and an exhibited hypotension of the perirectal and perivaginal area (figure 5). The tail had an atonic posture. An abnormal position of the limbs was may be a result from upper motor neuron (UMN) or lower motor neuron (LMN) disease (Constable, 2004). An arched back was associated with lame cows. The arched back in these goats was probably a sign of pain (Sprecher *et al.* 1997).

Figure 1: Kyphosis and pelvic tilt.

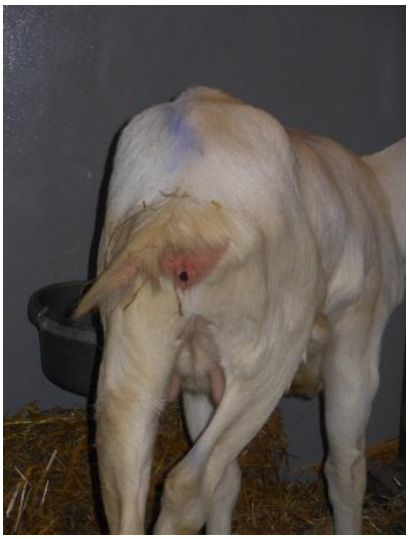

Figure 2: Moderate musculature of the hindquarters.

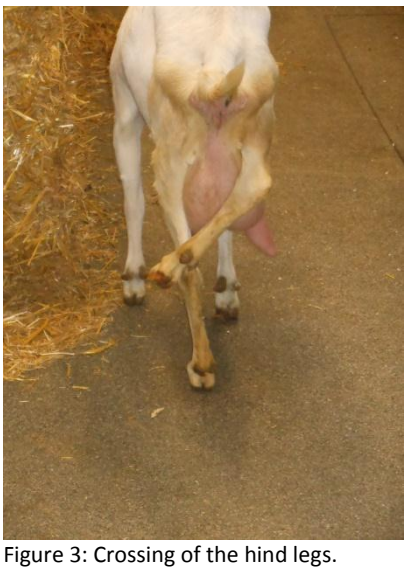

Figure 3: Crossing of the hind legs.


Figure 4: Crossing of the hind legs.


Figure 6: Lying position.

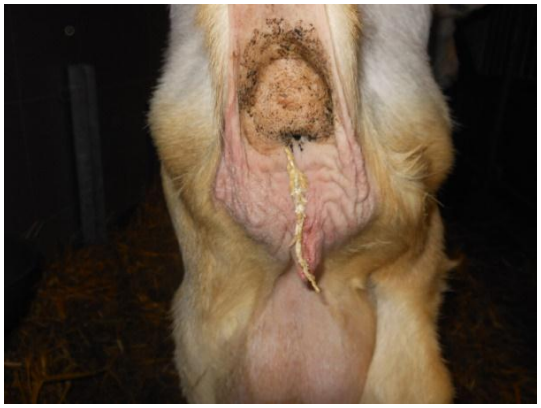
Figure 5: Exhibited hypotension of the perirectal and perivaginal area.

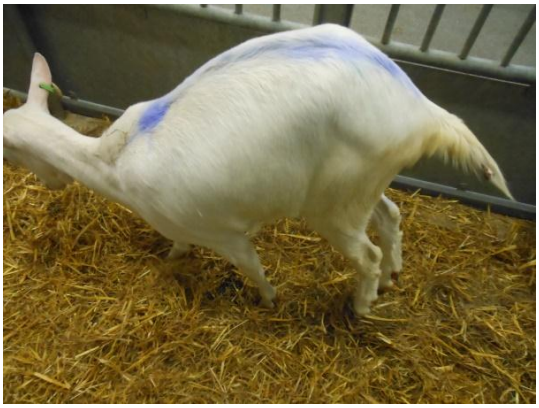
Figure 7: Walking on two fore legs.

It was questionable of their lying behaviour was abnormal, because the goats often lay with extended tibio-tarsal joints (figure 6). The goats were reluctant to stand up and one of the goats walked after standing up on two fore legs with the rump in the air (figure 7). Standing was difficult and was accompanied by stiffness. Probably these symptoms were sign of pain. The locomotion after standing up was characterized by ataxia posterior and paresis posterior with extended tibio-tarsal joints (figure 9). The symptoms of the young seemed more severe than the symptoms of the older goat.

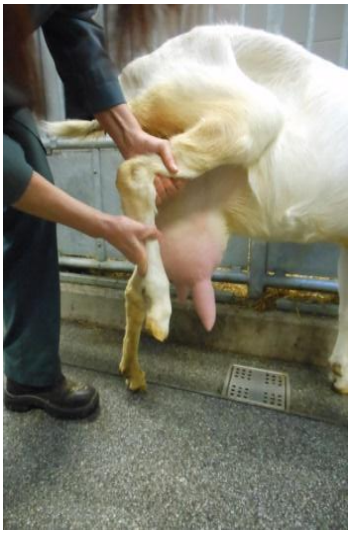
Figure 8: Extension tibio-tarsal joint.

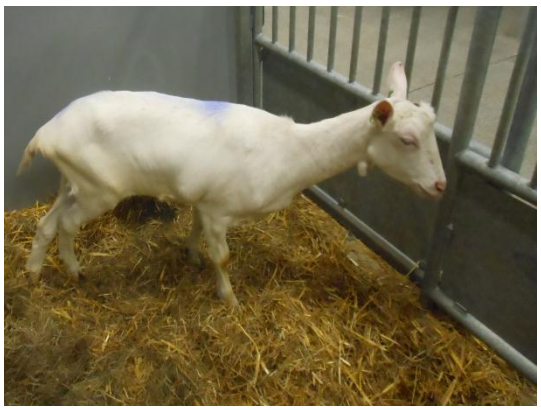

Figure 9: Walking with straight tarsi.

Symptoms of spastic paresis in goats have been previously described. Kral & Hlousek (1973) examined a three-year-old Saanen buck with symptoms of spastic paresis. The buck was reluctant to stand up, walked after standing up on two forelegs with the rump in the air and straight tarsi. Spastic paresis has been also observed in pygmy goats (Baker *et al.* 1989). The affected animals showed spastic contractions of the gastrocnemius muscles in the hind limbs. The contractions resulted in extension of the tibio-tarsal joint and kyphosis of the back. The most likely diagnosis on the basis of clinical signs was a defect in the myotactic reflex that resulted in a reduced inhibition or an overstimulation of the efferent motor neurons (Baker *et al.* 1989). These symptoms correspond with those of the goats in this research. Symptoms of spastic paresis were a well-known in cattle. Bovine spastic paresis (BSP) was a neuromuscular disorder characterized by an overextension and a spastic contraction of the gastrocnemius muscle of one of both hind legs and a limited increase in body condition (Ledoux, 2001). Pariset *et al.* (2013) suggested that BSP was correlated to a defective glycinergic synaptic transmission and a modification of calcium signalling proteins. The symptoms of spastic paresis in Saanen buck, the pygmy goats and the BSP looked like the symptoms of the two Dutch dairy goats in this study. However, these studies do not described symptoms ataxia and exhibited hypotension of the perirectal and perivaginal area.

Prolonged movement or trotting resulted in more flexible locomotion. The head of the goat was moved down to a lower position when the most affected hind leg was in stance. This finding was consistent with hind leg lameness in horses. The head movement is used to reduce load on the lame hind leg (Kelmer *et al.* 2005).

Reflexes

The reflexes in the protocol (appendix 5) were divided into postural reactions (stell reflexes, correction reflexes), cerebral reflexes, spinal reflexes, pathological reflexes and pain perception. The postural reactions were normal in the forehand, but abnormal in the backhand. Hopping (lifting three of the four legs and hopping on one leg forward and sideways) was abnormal in the backhand, because the hind legs were uncoordinated with spastic movements positioned. Hopping on the right hind leg was impossible in both goats. Hemi- walking (lifting ipsilateral foreleg and hind leg and walking forward) was difficult to perform because the hind legs were not moved forward. The cross reflex, one of the correction reflexes, was difficult to perform in the hind legs and not consistently positive or negative. It was questionable of this reflex was usable in goats. The conscious limb placement was tested by the obstacle test and was uncoordinatedly performed by the hind legs. The proprioception was tested by foot displacing by turning it onto its dorsum and pushing at the level of the withers and the pelvis. Foot displacing was difficult to perform on forelimbs and hind limbs, but pushing out balance was well corrected by the goats. It was questionable of the foot displacing was properly implement in goats. According to Constable, this reflex was useful in goats (Constable, 2004).

The cerebral reflexes were not abnormal in contrast to the spinal reflexes. The reflex of the withers and the panniculus reflex were absent. The sensory part of the panniculus reflex was accomplished by the peripheral nerves of the related dermatome. The motor arc was accomplished by the lateral thoracic nerve and spinal cord segments C8 and T1 and stimulates the muscle contraction of the cutaneous trunci muscle. This reflex was normally absent in dermatomes caudal of L3 in ruminants (Getty, 1975). It was doubtful whether these reflexes were normally present in goats. According to Constable, this reflex was useful in goats (Constable, 2004). The perineal reflex was absent and the tail was atonic. This was an expected result, because the exhibited hypotension of the perirectal and perivaginal area observed during the examination of the posture. The perineal reflex was integrated by the spinal cord segments S2 trough S4 and caudal rectal and pudendal nerves. The patellar reflex was decreased, but not absent. The patellar reflex needs an intact femoral nerve and intact spinal cord segments L4 and L5 (Getty, 1975). Only damage of the femoral nerve resulted in decreased

or absent patellar reflex. Severe damage of the femoral nerve resulted in muscle atrophy of the quadriceps muscle and an inability to support weight on the affected hind limb (Kirk *et al.* 1987). In standstill, the affected hind limb was slightly flexed and the hip of the affected limb was kept slightly lower (Paulsen, 1978). Symptoms of only femoral nerve injury did not correspond with the symptoms of the goats with bilateral spastic paresis and ataxia posterior and a decreased perineum tone. Therefore, an abnormality in the spinal cord was more likely. The withdrawal reflex was present in the forelegs and not present in the hind legs by a normal stimulus, however, a strong stimulus (pain perception) resulted in a withdrawal reaction. This reflex in the hind legs needed intact spinal cord segments L5 through S1 and an intact sciatic nerve (Getty, 1975). Only damage of the sciatic nerve led to weakness in the hind limbs and knuckling of the fetlocks. The patellar reflex with sciatic nerve damage was increased or normal as the result of reflex contraction of the quadriceps muscle of the thigh by the femoral nerve. The patellar reflex was not inhibited by muscles of the hind legs that were innervated by the sciatic nerve. Symptoms of only sciatic nerve damage did not correspond with the symptoms of goats with bilateral spastic paresis and ataxia posterior and a decreased perineum tone. Therefore, an abnormality in the spinal cord was more likely. The pain perception was normal.

There were no control animals used and the neurological exam was not a standard examination in goats, this could potentially affect the interpretation of the examination. Because there were no control animals and the small group of case animals (N = 2), the clinical examination were not compared statistically.

Pain

It was questionable whether these clinical symptoms were painful. The physiological parameters like heart rate, respiration rate and temperature were normal. Also the behaviour of the goats was quite normal, but the goats seem to be less active than normal healthy goats. The goats were not sensitive when touched and during performed passive movements. Ruminants species were flight animals and do not show overt abnormal behaviour or pain symptoms, to avoid attracting attention from predators as survival strategy (Livingston, 2010).

Qualitative needle electromyography

The QEMG recordings showed increased (spastic) muscle contractions in the lateral vastus muscle of the quadriceps muscle of the thigh, left had higher values than right (figure 10 and 11). The left biceps femoris muscle of the thigh also had higher values than the left (figure 12 and 13). This was not consistent with the findings of the neurological examination, because the right hind leg of both goats was more severe affected. The young goat had severe abnormal MUAPs than the older goat. This was an expected result, because the clinical abnormalities in the young goat also seemed more severe. The lateral vastus of the quadriceps were innervated by the femoral nerve. This was the same nerve, which was necessary to generate the patella reflex (Constable, 2004). There was possibly a link between these findings. The QEMG recordings showed no obvious myotonic or neurogenic pattern, but the recordings seemed more like a neuropathy. Not all the muscle groups were measured, therefore the QEMG had to be interpreted as a coarse indication.

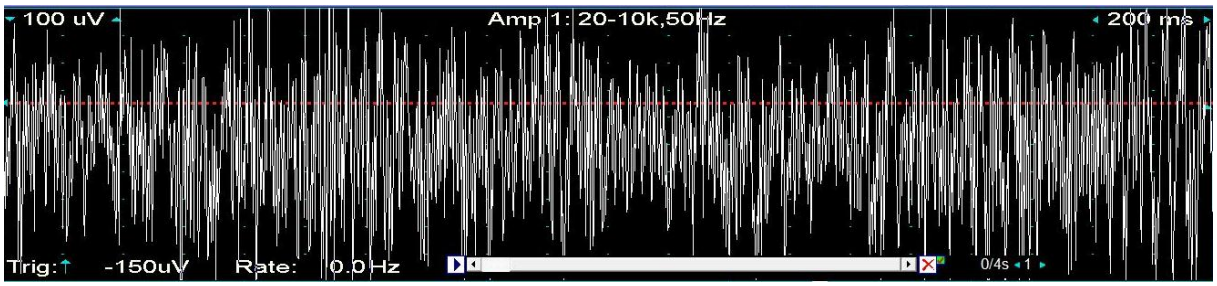

Figure 11: Quantitative electromyography of the left lateral vastus muscle of the quadriceps muscle of the thigh in the 11 months old goat with progressive bilateral (spastic) paresis, ataxia posterior and a decreased perineum tone, displayed increased muscle contractions. The electromyography was performed, using 100µV and 200ms/division.

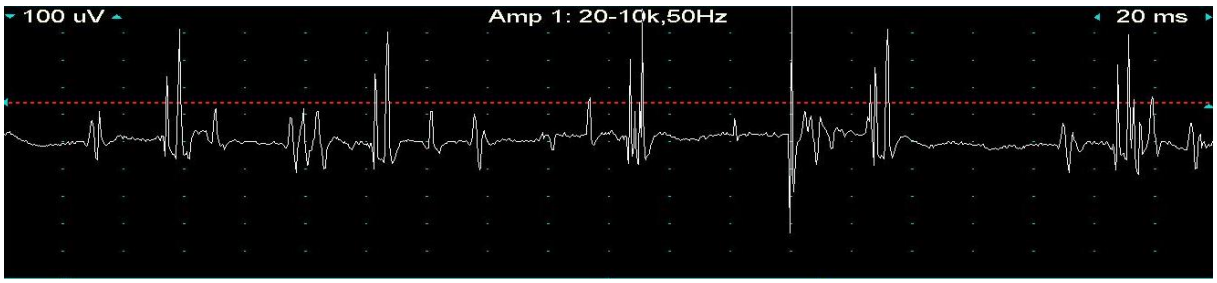

Figure 12: Quantitative electromyography of the right lateral vastus muscle of the quadriceps muscle of the thigh in the 11 months old goat with progressive bilateral (spastic) paresis, ataxia posterior and a decreased perineum tone, displayed normal muscle contractions. The electromyography was performed, using 100µV and 20ms/division.

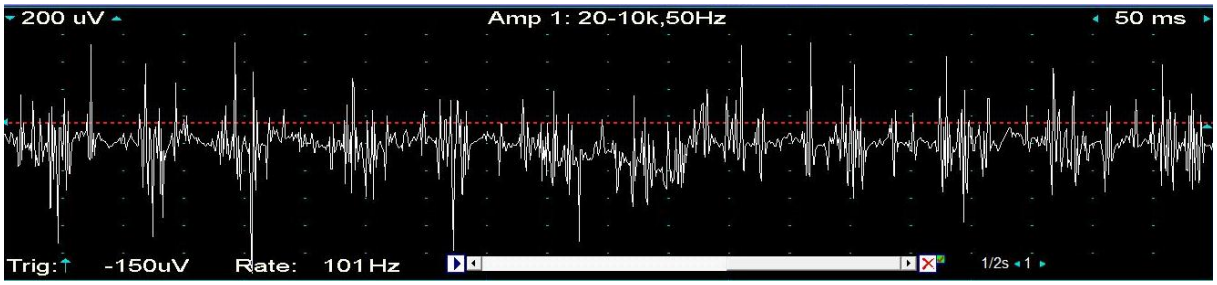

Figure 13: Quantitative electromyography of the left biceps muscle of the thigh in the 22 months old goat with progressive bilateral (spastic) paresis, ataxia posterior and a decreased perineum tone, displayed increased muscle contractions. The electromyography was performed, using 200µV and 50ms.

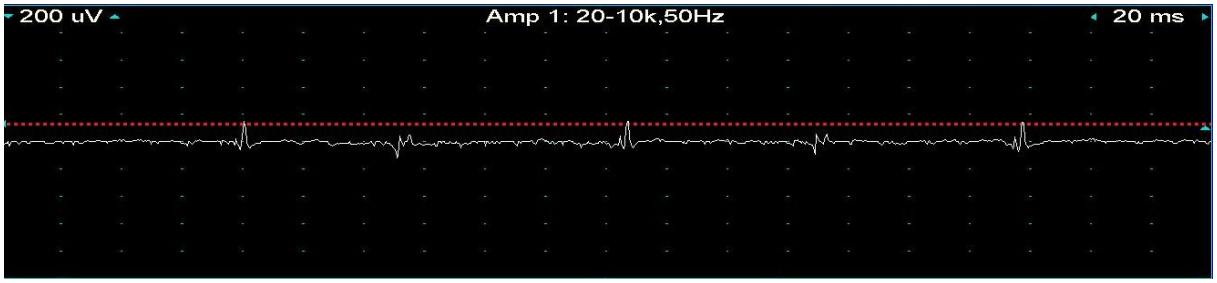

Figure 14: Quantitative electromyography of the right biceps muscle of the thigh in the 22 months old goat with progressive bilateral (spastic) paresis, ataxia posterior and a decreased perineum tone, displayed normal muscle contractions. The electromyography was performed, using 200µV and 20ms.

Pathological examination

No abnormalities were found at macroscopic level. Probably there were abnormalities found at histology. Pathological examination of the pygmy goats with spastic paresis were also no abnormalities found in the spinal cord, the gastrocnemius muscle or peroneal or tibial nerves (Baker *et al.* 1989).

Localisation of the lesion

Localisations of the different neurological findings were shown in figure 14. The symptoms of the goats could be caused by lesions in the spinal cord and/or lesions in the peripheral nerves. Lesions in the spinal cord were differentiated in Upper Motor Neuron (UMN) disease and Lower Motor Neuron (LNM) disease. Lesions of the UMN resulted in reduction of the arbitrary motor function and reduced inhibition of the LMN caudal of the lesion. Symptoms of UMN injury are: (spastic) paresis, increased muscle tone and exaggerated reflexes which resulted in symptoms of ataxia. Lesion of the LMN resulted paresis, decreased muscle tone and decreased spinal reflexes in muscles which are innervated by related nerves. Lesions in the peripheral nerves resulted in clinical symptoms of LMN injuries such as weakness, muscle atrophy, paresis, paralysis and altered reflexes. The goats in this research showed symptoms of UMN disease (extended tibio-tarsal joints) and LMN disease and/or peripheral nerve injury (decreased spinal reflexes)(Constable,2004).

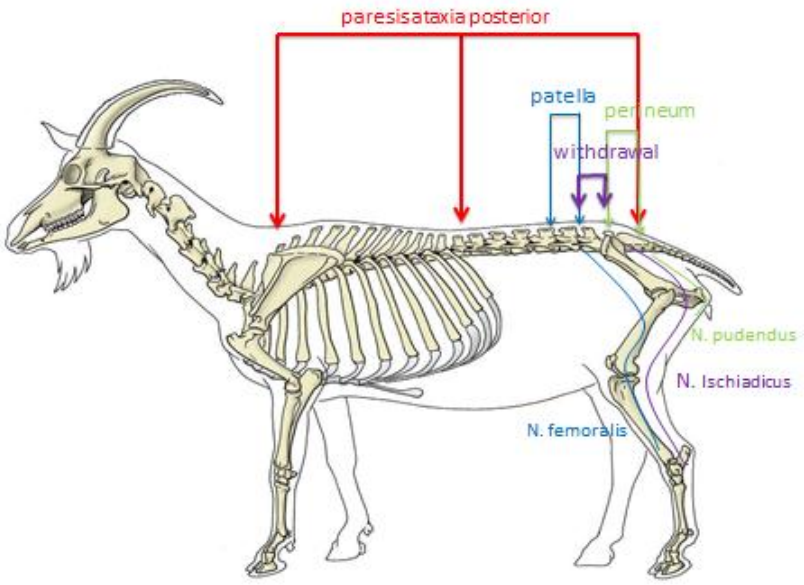

Figure 14: Localisations of neurological findings (Dyce *et al.* 2010).

Possible aetiology

The aetiology of this syndrome was unknown. Several causes or combinations of causes were possibly. Some farmers and farm visitors may have suggested that mycotoxins in the feed have been associated with these symptoms. Karki *et al.* (2008) described the Dhakeri-Bange syndrome in goats due to *Penicillium* and *Aspergillus* spp in Banke District of Nepal. Goats exhibited symptoms of knuckling of the fetlocks and pelvic limbs, without flexor weakness or ataxia, apathy and digestion disturbances as ruminal stasis and diarrhea. The symptoms were associated with feeding of moldy forage; this syndrome was called Endemic Mycotic Polyneuropathy. Van der Lugt *et al.* (1993) described an acute *Aspergillus clavatus* intoxication by twenty-three cattle caused. Grain sorghum meal was contaminated with *A. clavatus*. The animals developed nervous symptoms such as paresis of hind limbs, knuckling over the fetlocks, hypersensitivity and muscle tremors. Affected animals were euthanized 2-7 days after displaying clinical signs due to intoxication. *A. clavatus* neuromycotoxicosis was associated with toxic neuronopathy/axonopathy with primary damage to the neuron characterised by chromatolysis and with secondary or concurrent axonal degeneration. The above described symptoms of mycotoxin intoxication do not resembled the symptoms of the goats with bilateral spastic paresis and ataxia posterior and a decreased perineum tone. The likelihood that these problems can be associated with mycotoxins in the feed was small.

Conclusion

None of the blood-parameters measured significantly different between cases and controls. The most likely diagnosis on the basis of clinical-neurological examination was a lesion in the thoracolumbar and lumbosacral segments of the spinal cord. These goats showed symptoms of both UMN and LMN problems; probably it was a UMN and LMN disease. The QEMG could not differentiate between myopathy and neuropathy. The pathological examination did not result in finding abnormalities correlated to the clinical symptoms. The clinical examination resulted in a better definition of the disease:
"Symptoms of progressive bilateral (spastic) paresis, ataxia posterior and a decreased perineum tone in Dutch white dairy goats."

Further research was necessary to determine a more accurate localization of the abnormality, probably with MRI of the thoracolumbar, lumbar and lumbosacral part of the spinal column to localize a possible abnormality in the spinal cord. For the interpretation of neurological examination, it was important to examine not only case animals, but also use control animals. For a better

differentiation between myopathy and neuropathy, an extensive QEMG was needed.

There was also more research needed to determine the risk factors or causes of this syndrome.

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Appendix

1. Protocol selectie klinische representanten

Criteria klinische representant:

- Nederlandse witte melkgeit/bok van een commercieel melkgeitenbedrijf in Nederland
- bilaterale parese/paralyse/ataxie posterior
- kruisen van de achterpoten in stilstand
- steile stand van de achterpoten
- (kanteling van het bekken)
- (slappe spiegel)
- (prolaps van vagina en/of rectum)

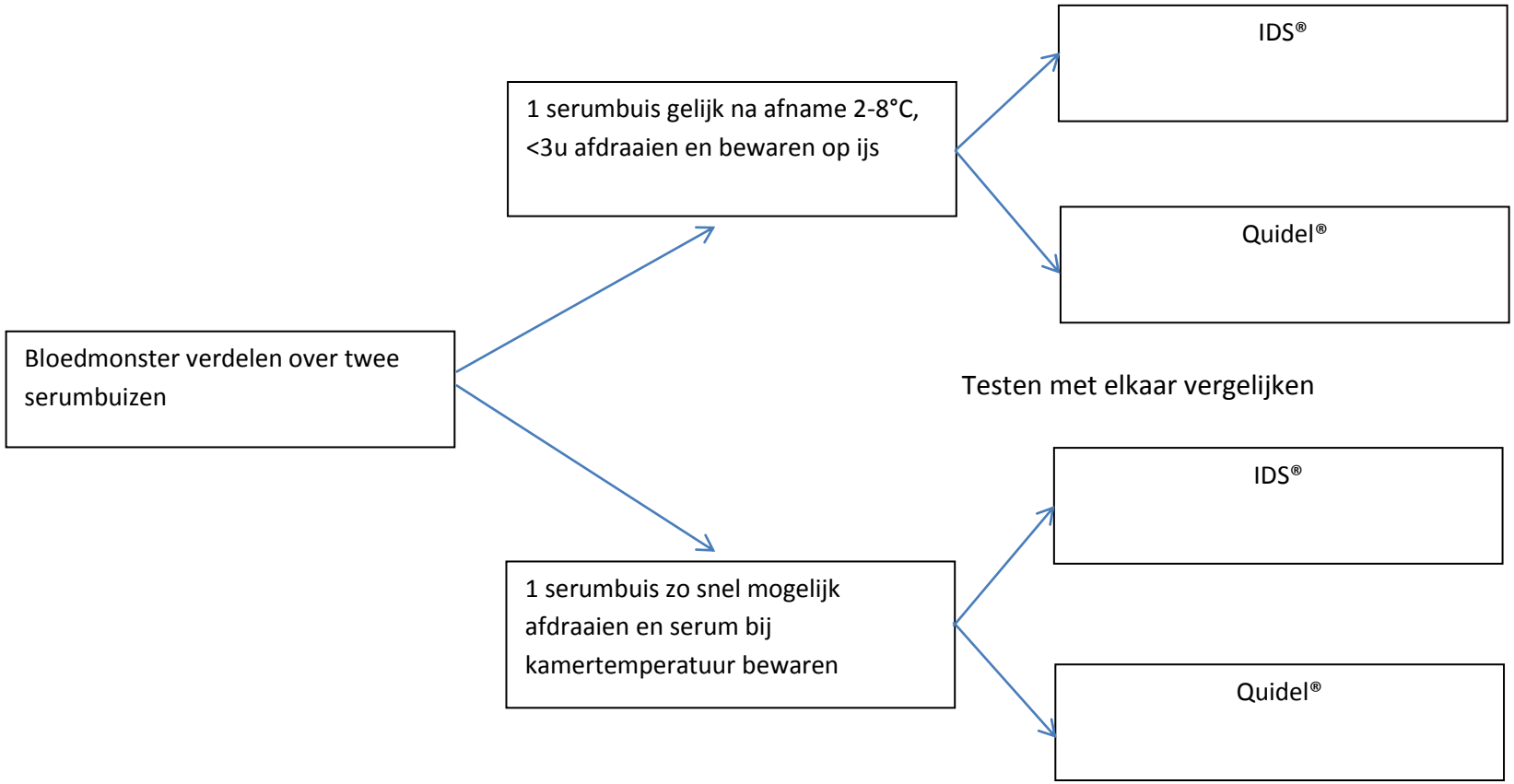
Dieren moeten worden uitgesloten als:

- gebroken ledematen
- mechanische beschadiging waardoor kreupelheid ontstaat (verwondingen, kapotte mediale/laterale banden, klauwontstekingen)
- geen beiderzijdse problemen aan achterbenen
- enkel prolaps van vagina en/of rectum zonder parese/paralyse/ataxie van achterbenen
- andere lichamelijke afwijkingen of ziektebeelden vertoont

2. Protocol test osteocalcine

Plan test osteocalcine IDS® vs. Quidel® onder kliniekomstandigheden

1. Twee bloedjes nemen in serumbuis
2. 1 bloedje gelijk na afname koel bewaren (2-8graden Celsius) en binnen 4 uur afdraaien en bewaren bij <-20°
3. 1 bloedje zo snel mogelijk afdraaien en het serum bij kamertemperatuur bewaren
4. Beide bloedjes testen in beide ELISA's (IDS® en Quidel®)
5. Uitslagen met elkaar vergelijken



3. Protocol algemene indruk

Invulformulier klinisch onderzoek (omcirkel de juiste score) houding en gang: zie neurologisch onderzoek

Datum: Tijd:	**Geit**					**Geit**					**Geit**				
Gedrag	Rustig	Evenwichtig	Angstig	Agressie		Rustig	Evenwichtig	Angstig	Agressie		Rustig	Evenwichtig	Angstig	Agressie	
Pensvulling	Slecht	Matig	Goed	Overmatig		Slecht	Matig	Goed	Overmatig		Slecht	Matig	Goed	Overmatig	
Conditiescore	1	2 2.5	3 3.5	4	5	1	2 2.5	3 3.5	4	5	1	2 2.5	3 3.5	4	5
Vacht	Schoon	Vuil				Schoon	Vuil				Schoon	Vuil			
Hoornige structuren	gb		Te lange klauwen	Ingegroeide hoornen		gb		Te lange klauwen	Ingegroeide hoornen		gb		Te lange klauwen	Ingegroeide hoornen	
IHOSKA's	gb	Anders				gb	Anders				gb	Anders			
Datum: Tijd:															
Gedrag	Rustig	Evenwichtig	Angstig	Agressie		Rustig	Evenwichtig	Angstig	Agressie		Rustig	Evenwichtig	Angstig	Agressie	
Pensvulling	Slecht	Matig	Goed	Overmatig		Slecht	Matig	Goed	Overmatig		Slecht	Matig	Goed	Overmatig	
Conditiescore	1	2 2.5	3 3.5	4	5	1	2 2.5	3 3.5	4	5	1	2 2.5	3 3.5	4	5
Vacht	Schoon	Vuil				Schoon	Vuil				Schoon	Vuil			
Hoornige structuren	gb		Te lange klauwen	Ingegroeide hoornen		gb		Te lange klauwen	Ingegroeide hoornen		gb		Te lange klauwen	Ingegroeide hoornen	
IHOSKA's	gb	Anders				gb	Anders				gb	Anders			

4. Protocol algemeen onderzoek

Algemeen onderzoek **Geit:**

1. **Ademhaling:**
   a. Frequentie: ……ademteugen/min (10-30/min)
   b. Diepte: ◯ oppervlakkig ◯ vold. diep ◯ te diep
   c. Type: ◯ te costaal ◯ costo-abdo ◯ te abdominaal ◯ anders: ……
   d. Ritme: ◯ regelmatig ◯ onregelmatig

2. **Pols:**
   a. Frequentie: ……slagen/min (70-90/min)
   b. Ritme: ◯ regelmatig ◯ onregelmatig
   c. Kwaliteit: ◯ equaal ◯ inequaal ◯ krachtig ◯ zwak

3. **Temperatuur**:…….. °C (38.6-40.0°C)

4. **Huid, beharing en hoornige structuren**
   a. Huid: ◯ gb ◯ geur ◯ kleur ◯ temperatuur ◯ dikte ◯ oplichtbaarheid ◯ turgor ◯ laesies ◯ sensibiliteit ◯ bloedingen ◯ oedeem
   b. Beharing: ◯ gb ◯ glad ◯ aangesloten ◯ vuil ◯ glanzend ◯ kale plekken ◯ ectoparasieten
   c. Hoornige str: ◯ gb ◯ grootte ◯ vorm ◯ aard opp. ◯ kleur ◯ temperatuur ◯ pijnlijkheid

5. **Slijmvliezen** (conjunctiva, sclera, bek)
   a. Kleur: ◯ papierwit ◯ bleek ◯ roze ◯ rood ◯ afwijkend: ……
   b. Vochtigheid: ◯ vochtig ◯ droog ◯bloedingen ◯ laesies

6. **Lymfeknopen** (grootte, vorm, consistentie, pijnlijkheid, verschuifbaarheid)
   a. Mandibularis: ◯ gb ◯ afwijkend……
   b. Retrofaryngealis: ◯ gb ◯ afwijkend……
   c. Boeg: ◯ gb ◯ afwijkend……
   d. Vang: ◯ gb ◯ afwijkend……
   e. Mammari: ◯ gb ◯ afwijkend……

5. Protocol neurologisch onderzoek

Neurologisch onderzoek en houding & gang

1. Gedrag en bewustzijnsniveau:

◯ verhoogd ◯ normaal ◯ verlaagd ◯ dwangbewegingen
◯ stereotypieën

2. Houding & locomotie

Houding

a. Kop: ◯ gb ◯ torsie lengte as ◯ ventraal

b. Hals: ◯ gb ◯ scheve stand ◯ extensie hals ◯ flexie hals

c. Rug: ◯ gb ◯ kyfose ◯ lordose ◯ scoliose

d. Staart: ◯ atonie ◯ normotonie ◯ hypermetrie

e. ◯ onwillekeurige bewegingen ◯ spasmen ◯ tonische krampen ◯ clonische krampen ◯ tremoren

Locomotie

a. Rechte lijn:……
……

b. Bochten maken:……..

c. Draf: ……
……

d. Coördinatie:
   i. Locomotie: ◯ gb ◯ eigen poten aantikken ◯ struikelen ◯ ataxie ◯ dysmetrie ◯ hypermetrie

e. Kracht:
   i. Parese: ◯ gb ◯ mono ◯ para ◯ tetra ◯ posterior
   ii. Paralyse: ◯ gb ◯ mono ◯ para ◯ tetra ◯ posterior

f. Onwillekeurige bewegingen
   i. ◯ gb ◯ tremor ◯ tic ◯ myoclonieën

3. Schedel en wervelkolom

- Inspectie en palpatie

	Vorm	Positie	Symmetrie	Laesies	Deformiteiten	Pijn	Crepitatie
Kop							
Wervelkolom							

Beschrijving afwijkingen:

- Percussie
  - Hals: ◯ gb ◯ afwijkend: .....
  - Rug: ◯ gb ◯ afwijkend:.....
- Passieve bewegingen
  - Beweeglijkheid kop: ◯ gb ◯ afwijkend:.....
  - Beweeglijkheid hals: ◯ gb ◯ afwijkend:.....
  - Beweeglijkheid rug: ◯ gb ◯ afwijkend:.....

4. Spieren

Lichaamsdeel	Spierafwijking	Spiertonus	Spierspasmen
Hals	Vorm – symmetrie – consistentie (hyper-/atrofie) – temperatuur pijnlijkheid Beschrijving:	atonie hypotonie normotonie hypertonie	
Rug	Vorm – symmetrie – consistentie (hyper-/atrofie) – temperatuur pijnlijkheid Beschrijving:	atonie hypotonie normotonie hypertonie	
RV	Vorm – symmetrie – consistentie (hyper-/atrofie) – temperatuur pijnlijkheid Beschrijving:	atonie hypotonie normotonie hypertonie	
LV	Vorm – symmetrie – consistentie (hyper-/atrofie) – temperatuur pijnlijkheid Beschrijving:	atonie hypotonie normotonie hypertonie	
RA	Vorm – symmetrie – consistentie (hyper-/atrofie) – temperatuur pijnlijkheid Beschrijving:	atonie hypotonie normotonie hypertonie	
LA	Vorm – symmetrie – consistentie (hyper-/atrofie) – temperatuur pijnlijkheid Beschrijving:	atonie hypotonie normotonie hypertonie	
		knipmes – Shiff-Scherrington	

5. Reflexen (houdingsreacties, cerebrale en spinale reflexen)

Houdingsreactie

- Stellreflexen:
  - Optisch: ◯ aanwezig ◯ afwezig
  - Labyrintaire en proprioceptieve: ◯ aanwezig ◯ afwezig
- Correctiereflexen
  - Dubbeltreden:

- RV: ◯ aanwezig ◯ afwezig
- LV: ◯ aanwezig ◯ afwezig
- RA: ◯ aanwezig ◯ afwezig
- LA: ◯ aanwezig ◯ afwezig

- Kruisreflex
  - Voor: ◯ aanwezig ◯ afwezig
  - Achter: ◯ aanwezig ◯ afwezig
- Huppelreacties
  - Voor: ◯ aanwezig ◯ afwezig
  - Achter: ◯ aanwezig ◯ afwezig
- Plaatsingsreacties
  - Optische plaatsing: ◯ aanwezig ◯ afwezig

Cerebrale reflexen

- (II) N. opticus
  - Dreigreflex: ◯ aanwezig ◯ afwezig
  - Pupilreflex: ◯ aanwezig ◯ afwezig
- (III) N. oculomotorius
  - Ptosis: ◯ aanwezig ◯ afwezig
  - Dilatatie pupil: ◯ aanwezig ◯ afwezig
  - Divergerende strabismus: ◯ aanwezig ◯ afwezig
- (IV) N. trochlearis
  - Rotatie bulbus naar buiten + beneden: ◯ aanwezig ◯ afwezig
  - Scheve pupilspleet: ◯ aanwezig ◯ afwezig
- (V) N. tricheminus
  - Afhangende onderkaak: ◯ aanwezig ◯ afwezig
  - Kauwstoornissen: ◯ aanwezig ◯ afwezig
  - Palatum durum reflex: ◯ aanwezig ◯ afwezig
  - Ooglidreflex: ◯ aanwezig ◯ afwezig
  - (cornea reflex) ◯ aanwezig ◯ afwezig
  - Zuig- en kauwreflex: ◯ aanwezig ◯ afwezig
- (VI) N. abducens:
  - Convergerende strabismus: ◯ aanwezig ◯ afwezig
- (VII) N. fascialis
  - Eenzijdige paralyse: ◯ aanwezig ◯ afwezig
  - Partiele paralyse (neus + liphelft): ◯ aanwezig ◯ afwezig
- (VIII) N. vestibulocochlearis
  - Gehoor: ◯ aanwezig ◯ afwezig
  - Scheve hoofdhouding: ◯ aanwezig ◯ afwezig
  - Cirkels lopen: ◯ aanwezig ◯ afwezig
- (IX) N. glossopharyngeus:
  - Slikproblemen: ◯ aanwezig ◯ afwezig
- (X) N. vagus
  - Slikreflex: ◯ aanwezig ◯ afwezig
  - Hoestreflex: ◯ aanwezig ◯ afwezig
- (XI) N. accessorius
  - Neurogene spieratrofie aangetaste zijde: ◯ aanwezig ◯ afwezig
- (XII) N. hypoglossus
  - Paralyse tong: ◯ aanwezig ◯ afwezig

Spinale reflexen

Staand dier

- Schoftreflex: ◯ aanwezig ◯ afwezig ◯ versterkt ◯ pathologisch
- Huidrimpelreflex: ◯ aanwezig ◯ afwezig ◯ versterkt ◯ pathologisch
- Staartreflex: ◯ aanwezig ◯ afwezig ◯ versterkt ◯ pathologisch
- Anusreflex: ◯ aanwezig ◯ afwezig ◯ versterkt ◯ pathologisch
- Scrotumreflex: ◯ aanwezig ◯ afwezig ◯ versterkt ◯ pathologisch

Liggend dier

- Patellareflex: ◯ aanwezig ◯ afwezig ◯ versterkt ◯ pathologisch
- Radialisreflex: ◯ aanwezig ◯ afwezig ◯ versterkt ◯ pathologisch
- Buigreflex: ◯ aanwezig ◯ afwezig ◯ versterkt ◯ pathologisch

Pathologische reflexen

- Gekruiste strekreflex: ◯ aanwezig ◯ afwezig
- Massale respons: ◯ aanwezig ◯ afwezig
- Postreflectoire clonus: ◯ aanwezig ◯ afwezig

6. Pijnperceptie

- Diepe pijnperceptie: ◯ aanwezig ◯ afwezig

6. Protocol pathologisch onderzoek

Sectie protocol specifiek voor spastische parese en prolaps recti/vaginae

Naast algemene sectie dient aandacht te worden besteed aan de volgende punten:

- **Body Condition Score**
  ◯0 ◯1 ◯2 ◯3 ◯4 ◯5
- **Maag-darmkanaal**
  - Beoordeling van de mest**, indien afwijkend**:
    - Natief preparaat maken van duodenum, jejunum, ileum, colon en rectum; deze preparaten worden semi kwantitatief beoordeeld door afd. parasitologie op aanwezigheid van coccidiën; potje mest bewaren.
    - Onderzoek clostridium infectie en/of andere oorzaken voor aantasting van de darmen **die (heftig) persen als gevolg kunnen hebben.**
    - Histologisch onderzoek aangetaste delen darm
- **Lever, nieren, hart, milt, longen**
  Sample nemen voor microscopisch onderzoek (ook indien macroscopisch niet afwijkend.)
  M.n. nieren belangrijk: hoog fosfaat in bloed
    - Urinemonster nemen
- **Urogenitaal tractus**
  - Beoordeling van rectum en vagina op verschijnselen van prolaps:
    - Zwelling
    - Verslapping van de wand van vagina/rectum
    - Afwijkingen

- **Centraal Zenuwstelsel** (fixatie in 10% gebufferde formaline)
  - Hersenen in toto veiligstellen
  - Ruggenmerg in toto veiligstellen + uittredende zenuwen uit het lumbale gebied
  - Bij verkrijgen materiaal centrale zenuwstelsel dient de wervelkolom te worden beoordeeld op abnormaliteiten
  - Van hersenen worden van één helft cerebrum, cerebellum, hersenstam en verlengde merg microscopisch beoordeeld en het ruggenmerg t.h.v. plexus brachialis en lumbalis; rest bewaren in pot.
  - **Zenuwen**
    - **Zenuwen voorpoten**
      - **N. radialis**
      - **N. axillaris**
        - Sample nemen voor histologisch onderzoek
        - (zenuw opspannen langs een stokje)
    - **Zenuwen achterpoten links en rechts beoordelen**
      - **N. femoralis**
      - **N. sciaticus met N. tibialis en N. peroneus**

        - **N. pudendus**
            - Sample nemen voor histologisch onderzoek
            - (zenuw opspannen langs een stokje)
    - **Cauda equina**
        - Sample nemen voor histologisch onderzoek
    - **Ruggenmerg** (o.b.v. neurologisch onderzoek)
        - **L4 en L5**
        - **L5 tot en met S1**
        - **S2 tot en met S4**
            - Sample nemen voor histologisch onderzoek

- **Spieren**
    - **Spieren van de achterhand (beiderzijds) beoordelen**
        - Sample nemen voor histologisch onderzoek
            - M. biceps
            - M. semitendinosis
            - M. semimembranosis
            - M. vastus lateralis van de quadriceps
    - **Spieren van de lendenen beoordelen**
        - Sample nemen voor histologisch onderzoek

- **Botten: achterpoten**
    - Indien macroscopische afwijking: sample nemen voor histologisch onderzoek
- **Gewrichten:**
    - Beoordeling van snijvlak op abnormaliteiten
    - Indien macroscopische afwijking: sample nemen voor histologisch onderzoek