Universidade de Évora - Escola de Ciências e Tecnologia

Mestrado Integrado em Medicina Veterinária

Relatório de Estágio

Equine Medicine and Surgery

Patricia Virginia Alves da Silva

Orientador(es) | Susana Monteiro

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O relatório de estágio foi objeto de apreciação e discussão pública pelo seguinte júri nomeado pelo Diretor da Escola de Ciências e Tecnologia:

Presidente	Rita Payan-Carreira (Universidade de Évora)
Vogais	Paula Tilley (Universidade de Lisboa - Faculdade de Medicina Veterinária) (Arguente) Susana Monteiro (Universidade de Évora) (Orientador)

Évora 2022

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Equine Medicine and Surgery

Abstract

The following work is an externship report, which aims to describe the activities and cases followed during the student's curricular externship in two referral equine Hospitals. The report will be divided into two parts: Firstly, there will be a description of the hospitals, the type of procedures seen and performed, and the skills developed, along with a theoretical introduction and brief mention of the types of cases followed. The second part of the report will be a bibliographic review of Atypical Myopathy disease, which will be supported by a clinical case of a one-year-old stallion.

Keywords: *Equine, surgery, clinics, imagiology, rehabilitation*

Medicina e Cirurgia de Equinos

Resumo

Este trabalho é um relatório de estágio, com o objetivo de descrever as atividades e casuística seguida pelo estudante durante o período de estágio curricular. O relatório está dividido em duas partes: Primeiramente, será feita uma apresentação dos hospitais, dos procedimentos seguidos e das competências desenvolvidas, juntamente com uma introdução teórica e breve menção dos tipos de casos seguidos. Na segunda parte do relatório haverá uma revisão bibliográfica da doença Miopatia atípica, suportada pela apresentação de um caso clínico de um garanhão de um ano.

Palavras-chave: *Equino, cirurgia, clínica, imagiologia, reabilitação*

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List of Abbreviatures

AAEP – American Association of Equine Practitioners

ACTH - Plasmatic adrenocorticotropic hormone

AF – Atrial fibrillation

ALP – Alkaline phosphatase

AM – Atypical myopathy

AST – aspartate aminotransferase

BID – *Bis in die*

CK – Creatinine kinase

CO2 – Carbon dioxide

CT – Computerized tomography

ECG – Echocardiogram

EGUS – Equine gastric ulceration syndrome

EHV – Equine herpesvirus

ELISA - Enzyme linked immunosorbent assay

EOTRH – Equine odontoclastic resorption and hypercementosis

ERU – Equine recurrent uveitis

ETF - Electron transport flavoprotein

FAD - Flavin adenine dinucleotide

FEI – *Fédération Équestre Internationale*

GGT – Gamma glutamyltransferase

GOT - Glutamic-Oxaloacetic transaminase

HGA – Hypoglycin A

IL-5 – Interleukin 5

IM – Intramuscular

IRAP – Interleukin-1 receptor antagonist protein

IV – Intra-venous

JOC – Juvenile osteochondral conditions

Kg – Kilogram

LDH – Lactate dehydrogenase

MADD - Multiple acyl-CoA dehydrogenase deficiency

MCPA - Methylene cyclopropyl acetic acid

MCPF - Methyl enecyl cyclopropyl formyl

MCPG – Methyl enecyclopropylglycine

MRI – Magnetic resonance imaging

OCD – Osteochondrosis dissecans

PCR – Polymerase chain reaction

PO – *per os*

PPE – Pre-purchase examination

PPID – *Pituitary pars* intermedia disfunction

PRP – Platelet-rich plasma

qPCR – Real time quantitative polymerase chain reaction

SAA – Serum amyloid A

SC - Subcutaneous

SDH – Sorbitol dehydrogenase

SID – *Seme in die*

TID – *Ter in die*

TRH - Thyrotropin-releasing hormone

TVEC – Transvenous electrical conversion

Externship report

Introduction

The present work is based on my curricular externship which was divided into two periods. The first one between September 2021 and November 2021 was in Dierenkliniek Wolvega and the second was in Equitom during the month of January 2022, both are referral hospitals of Equine medicine and surgery.

The first period of the externship occurred in Dierenkliniek Wolvega, in the Netherlands, between 6 September and 28 November of 2021. This period was divided into weeklong rotations in the following areas: surgery, ambulatory practice, internal medicine, and appointments/ outpatients. During the surgery rotations the students were able to help prepare the horse for anaesthesia and for the surgical procedure, then helped with the recovery of the horse, and when appropriate, were able to scrub in in exploratory laparotomies and colic surgeries, hernia reductions, castrations, and enucleations. We could observe several standing procedures, such as a nephrosplenic space closure, laser venriculocordectomy, removal of uterine cysts and removal of skin masses, castrations, sinus trepanations, and Caslick sutures. We also helped perform the pre-anaesthetic examinations on the horses for the following day. During this period, we were able to observe several *osteochondrosis dissecans* (OCD) fragment removals, tenoscopies, arthroscopies in articular wounds, tooth removals, castrations, prosthetic laryngoplasties, episcleral cyclosporine implants, sarcoid laser removal, hernial reductions and exploratory laparotomies. The student could also observe several magnetic resonance imaging (MRI) examinations with the horse under general anaesthesia.

In the ambulatory practice we assisted in the appointments that consisted of dental checks, vaccinations, pregnancy checks and some emergency calls. In internal medicine, the externs participated in the patient rounds, performed physical exams, assisted in the medical procedures necessary to the admitted patients, such as ultrasounds, nasogastric intubation, rectal examinations, transfaunation, endoscopies, abdominocentesis, glucose sensitivity tests, over the wire catheter placements, blood analysis, cytologic and faecal examinations, the necessary nursing care, and necropsies. In the outpatient rotation the students aided in the appointments, followed the orthopaedic and outpatient rounds, and assisted in the medical procedures necessary to these patients, as well as in emergency cases. During this rotation we had the opportunity to assist in lameness investigations, pre-purchase examinations (PPE), radiographies, laser therapy, endoscopies, ultrasounds, tracheal washes, nebulisations, electrocardiograms and echocardiographies, dental examinations, ophthalmologic examinations,

neurologic examinations, among others. We were also encouraged to participate in the out of hours emergency calls and surgeries.

In this hospital, most of the horses presented were Friesians, Dutch warmbloods, Standardbreds, and ponies. It was also common to have Icelandic and Fjörd horses.

During the externship, the externs were urged to develop clinical and critical reasoning skills, interpret diagnostic test results, and perform some of the procedures.

The second period occurred in Equine hospital Equitom, in Lummen, Belgium. This period was spent in the surgery and anaesthesia, internal medicine, orthopaedics, rehabilitation, and imaging departments and in hospitalization in variable length rotations.

In the surgery and anaesthesia department the student helped prepare the horse for the surgery and in the recovery, we were also able to assist in several standing procedures, mass removals, observed several arthroscopies for removal of OCD fragments, and a sesamoid fragment removal with debridement of a collateral ligament rupture, a prosthetic laryngoplasty, castrations and exploratory laparotomies. In the internal medicine we followed several procedures done on the patients, such as endoscopies, ultrasounds, radiographs, ophthalmic examinations, thoracocentesis, dental examinations, nasogastric intubations, rectal palpations, and nebulisations. During this period, the externs were responsible for physical examinations, walking and grazing the admitted patients. In the orthopaedics department we followed lameness investigations and pre-purchase examinations and assisted in the treatments, including mesotherapy, interleukin-1 receptor antagonist protein (IRAP) and platelet-rich plasma (PRP). In the rehabilitation department we assisted in the laser therapies, extracorporeal shockwave therapy, cold water spa and Aquatrainer. In the imaging department we observed standing MRI scans, computerized tomography (CT) scans under general anaesthesia, and bone scintigraphy examinations. During the hospitalization rounds we followed the veterinarians in the patient evaluation and treatments and assisted in the administration on drugs, changing bandages and other procedures.

Most of the horses presented were warmbloods and other types of sport horses, but there were also draft horses, ponies and other breeds and species, including donkeys.

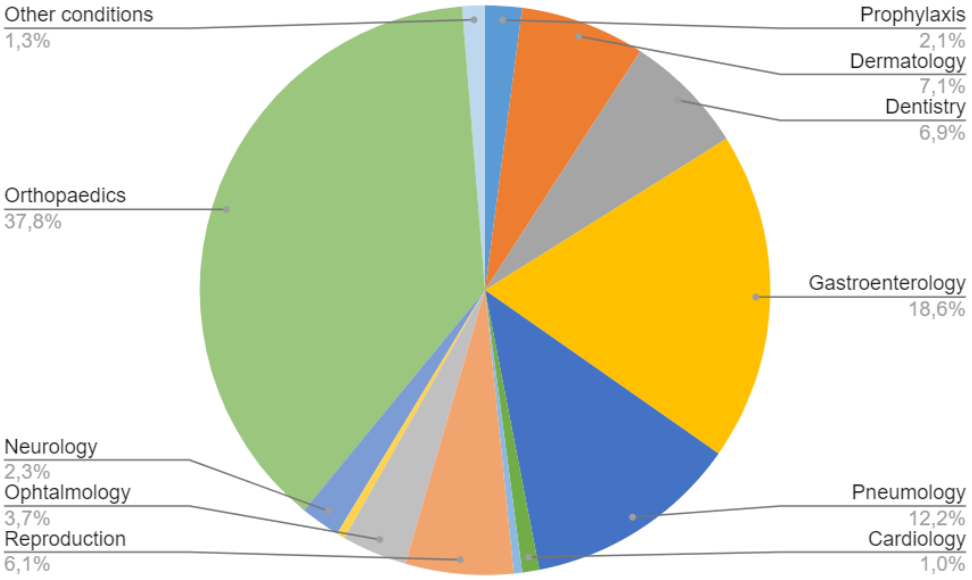
The students were able to participate in various didactic activities in the hospital, such as journal clubs, and were able to develop diverse non-clinical skills concerning husbandry and management of the horses.

Caseload description

During the externship period a total of 623 cases were observed in several medical areas, as shown in Table 1, along with the comparison of the number cases observed in both hospitals. The lesser number of cases observed at Equitom are a result of the fact that the externship was shorter in duration. In Graphic 1 there is a representation of the relative frequency of the cases, divided in the different medical areas, it is seen that most of the cases observed (37,8%) were orthopaedics cases, followed by gastroenterology cases (18,6%). In this section the various medical areas are divided and a brief description of the casuistic in each is provided, followed by a concise theoretical background of the most observed pathologies or most interesting, according to the extern's perspective.

Table 1 - Distribution of cases per body system/medical area.

Cases - System	Dierenkliniek Wolvega	Equitom	Total
Prophylaxis	13	0	13
Dermatology	26	18	44
Dentistry	35	8	43
Gastrointestinal system	92	24	116
Respiratory system	56	20	76
Cardiology	6	0	6
Nephrology and urology	3	0	3
Reproduction	29	9	38
Ophthalmology	10	13	23
Endocrinology	0	3	3
Neurology	8	6	14
Orthopaedics	107	128	235
Other conditions	7	1	8
Total	393	230	623



Graphic 1 - Distribution of cases per body system and relative frequency in percentage.

Prophylaxis

Prophylactic measures consist in procedures taken to prevent diseases. The owner of the horse should keep record of all the treatments the animal has been subjected to, and if the horse is intended for human consumption, then all the information regarding deworming, vaccination and other drug administrations should be on the horse's passport (Gids voor Goede Praktijken, 2019). During this externship, prophylatic procedures were observed during ambulatory practice in Wolvega and are demonstrated in table 2.

Table 2 - Prophylactic procedures

Procedures	Total
Vaccination	11
Deworming	2

In the Netherlands, most of the horses are on pasture for a significant part of the year, which makes them particularly susceptible to internal parasites, the amount and nature of the parasites depend on the grazing strategy. It is advised that the horses do faecal egg counts regularly so an adequate anthelmintic and treatment plan can be advised by the veterinarian (*Gids voor Goede Praktijken*, 2019). In Dierenkliniek Wolvega the faecal egg counts were performed with a McMaster technique by a laboratory technician before the deworming. Faecal egg counts are useful as they estimate strongylid egg shedding in older horses and ascarids in younger horses,

evaluating the efficacy of the current deworming plan, which is particularly important due to increasing resistance to anthelmintics (Nielsen, 2021). Ideally, faecal examinations should be performed throughout the year to every horse on the herd, however it is not always practical, so it is recommended that up to five horses of the same age group are tested to estimate the levels of parasite infection (European Scientific Counsel Companion Animal Parasites (ESCCAP), 2018).

The veterinarian usually advises the vaccination protocol, and it might include immunization against Influenza, Equine Herpes Virus (EHV), Tetanus and Strangles (*Gids voor Goede Praktijken*, 2019). The diseases and vaccination protocols described below correspond to the ones observed during the externship.

Equine Influenza

Equine influenza virus (H3N8) is very contagious, especially in immunologically naïve populations. The global transport of horses is pointed as the main reason for the widespread of the disease. Hence vaccination and strict quarantines, as the most effective ways to prevent outbreaks (Cullinane *et al.*, 2020). Affected horses show high fevers, lethargy, serous nasal discharge, and cough, causing a decrease in performance. To diagnose this infection virus isolation, detection of Influenza A antibodies, hemagglutinin inhibition and real time quantitative polymerase chain reaction (qPCR) may be performed. Most horses recover well from the disease, however, others suffer from serious complications, such as bacterial pneumonia. Ensuring an adequate period of rest is vital for a good recovery, the recommended is one week of rest per each day of fever. Good nursing care, plenty of water and decent quality feed and dust-free bedding should also be available. Non-steroidal inflammatory drugs should be administered in cases of pyrexia, if the horse is well hydrated (Myers and Wilson, 2006; Dilai et al., 2018).

The Fédération Équestre Internationale (FEI) demands that all competition horses are vaccinated for the equine Influenza virus. The recommended vaccination schedule, by FEI is as demonstrated on table 3.

Table 3 - FEI vaccination schedule protocol (Vaccinations, 2019).

Vaccination	Protocol
Primary course	First vaccination – day zero The horses that only have the first vaccination are not allowed on FEI stables
	Second vaccination – day 21 – 92 These horses may compete, if the vaccine was given at least seven days earlier of the day of arrival
First booster	Within seven months after the second vaccination Horses may compete until 6 months and 21 days after the end of the primary course. They may not compete if the last vaccination was within 7 days of arrival
Subsequent boosters	Within one year after the previous booster administration is the minimum requirement. However, to compete the horse has to be in the 6 months and 21 days of the previous booster They may not compete if the last vaccination was within 7 days of arrival

All vaccination history and details must be included in the horse's passport and must have the veterinarian´s clinic stamp (*Vaccinations*, 2019).

Pregnant mares could be revaccinated four to eight weeks before foaling, so they can produce colostrum containing antibodies against Influenza. The maternal antibodies may persist in the foal up to 9 months, therefore foals should only be vaccinated from the sixth to ninth month of life, depending on the titer of antibodies at birth. This first course should include three doses of the vaccine. Foals born from unvaccinated dams, can be vaccinated at three months of age (Myers and Wilson, 2006; Cullinane et al., 2020).

In case of an outbreak all horses demonstrating clinical signs should be isolated even before diagnosis is confirmed, also horses that have been in contact with infected horses. It is particularly important that these horses do not contact with non-exposed horses, especially vaccinated horses that may contact unvaccinated horses, as they may still be subclinical virus shedders. Suspend training in all affected horses and avoid intensive housing and crowding. Afterwards it is important to run diagnostic tests on the horses. Nasopharyngeal swabs can be used for Enzyme linked immunosorbent assay (ELISA), polymerase chain reaction (PCR) or virus isolation may be used to confirm an outbreak. Additionally, an increasing antibody titer in paired serum samples may be of use to differentiate outbreaks from previous contacts with the virus or antibodies from vaccination. Non exposed horses should receive a booster dose of vaccine. Then the premises,

equipment and vehicles should be disinfected with ammonium compounds, formalin, chlorine-based or phenolic disinfectants (Myers e Wilson, 2006).

There are several types of vaccine available, including live attenuated, inactivated, or recombinant vaccines. The latter type is very commonly used, the most common associations are Influenza and tetanus and influenza and Equine herpes virus (EHV), thus providing immunity against both agents. Dilai and associates studied the efficacy of mixed vaccination protocols during primary immunization and found that using different vaccines during prime and boost vaccination is efficient and stimulates a good humoral response. Furthermore, it was noticed that vaccines with influenza/tetanus toxoid association elicited a more pronounced immune response than the EHV/Influenza association (Dilai et al., 2018).

Equine Rhinopneumonitis

Equine viral rhinopneumonitis is a disease caused by EHV types one and four, that is endemic in most countries. Both viruses are associated with respiratory disease and the horses present general signs of viral infection, such as pyrexia, lethargy and inappetence. EHV-4 is thought to replicate mostly on upper respiratory tract; however, EHV-1 is disseminated systemically, inducing vasculitis, and has also been linked with abortion in the final months of gestation, neonatal disease and myeloencephalopathy. Horses infected with these viruses are carriers for life, usually the virus is in its latent state, but in cases of immunosuppression the host becomes a shedder. Horses presenting with herpesvirus-induced myeloencephalopathy present respiratory disease followed by mild ataxia, posterior paresis, and paralysis, which progresses to recumbency. No gross lesions are found *post-mortem*, but histopathology findings are consistent with lymphocytic vasculitis (Pusterla and Hussey, 2014; Davis, 2016; DGAV, 2021).

The diagnosis can be made through serologic testing of neutralizing antibodies of paired samples, virus isolation from nasopharyngeal lavage sampling or PCR from nasopharyngeal swabs. Affected animals should be isolated from other animals, as the virus is spread through respiratory droplets or direct contact with respiratory fluids or abortion materials. The isolation period can go up to a month. The virus persists in the environment from seven to 35 days so fields or stables where there were affected horses should not be used during this period and all the equipment should be disinfected. The American Association of Equine Practitioners (AAEP) suggests that the premises should be isolated for 21- 28 days after the last day in which fever was recorded. (AAEP, 2021). Equine hospitals can also be important places of transmission of EHV, as there are horses from different stables and most of them are immunologically compromised. Both impactful animal and economic losses can occur in result of nosocomial outbreaks (Vandenberghe *et al.*, 2021). Like in most viral infections, there is no specific therapy so the horses should be on rest and supportive care should be provided (Davis, 2016). Vaccination is advised, especially in horses that are in contact with other horses from different origins, as this

disease is highly contagious. However, it is important to note that the vaccine does not prevent the onset of disease (Lunn et al., 2009; DGAV, 2021).

Tetanus

Tetanus is a bacterial zoonosis found in all domestic animals, caused by *Clostridium tetani*. Although it is a disease that occurs worldwide, it is becoming an increasingly uncommon disease (Nout-Loma, 2016). Equines are particularly sensitive to C. *tetani* exotoxins, the bacteria are found ubiquitously in the environment as their spores are very resistant. The clinical presentation of the disease is hypertonia and hyperesthesia of striated muscle, with spasms, and possibly an increased rectal temperature. The muscle spasms are more severe in the area where the infected wound has occurred and become more generalized with time. The animals present a rigid posture, and, when able to walk present a stiff gait, which often degenerates into recumbency. The clinical manifestation of disease can be scored according to Table 4. The clinical score grade can correlate with the prognosis, with most horses dying in the first seven days. Horses with grade one or two, have a reserved prognosis, with grade three have a poor prognosis and horses in grade four should be euthanised (Kay and Knottenbelt, 2007; Gračner et al., 2015; Nout-Loma, 2016). Early veterinary assistance and previous tetanus vaccination are related with better outcomes (Kay and Knottenbelt, 2007; Ribeiro et al., 2018).

Table 4 - Clinical scoring system for tetanus, adapted from (Kay and Knottenbelt, 2007)

Score	Severity	Clinical signs
1	Mild	Slightly stiff gait, but able to walk, nictitans membrane flashing, ears pulled back and able to eat
2	Moderate	Stiff limbs, able to walk, but with difficulty, trismus, and muscle spasm, still able to eat and drink voluntarily
3	Severe	Capable of standing, but not ambulatory, severe trismus, dysphagia, drinking voluntarily
4	terminal	Recumbency, unable to eat or drink

Usually, the diagnosis is based on clinical manifestation and presence of an infected wound. Detection of tetanus toxin is difficult, especially in horses, since they are extremely sensitive to it, a small dose can cause clinical manifestation. Isolation of C. *tetani* from the wound can be attempted or serology can be attempted in unvaccinated horses (Popoff, 2020).

Debriding and cleaning the suspected entry wound, administration of antibiotics, and of tetanus anti-toxin (1500 U) are recommended when there is a risk of infection. Penicillin and metronidazole are usually the antimicrobials of choice used to treat the infection but are ineffective

against clinical disease. Metronidazole has the advantage of presenting a good penetration in necrotic tissues without being inactivated (Nout-Loma, 2016). Supportive treatment consists of sedation and muscle relaxation, which can be achieved with alpha-2-agonists or acepromazine, the administration of benzodiazepines or methocarbamol. The management of the ill horse is also especially important, it is essential to maintain the nutritional and hydration status and place the animals in a dark and isolated box, or putting ear plugs to reduce auditory stimuli (Kay e Knottenbelt, 2007; Nout-Loma, 2016; Popoff, 2020).

Active immunization of the animals is the best preventative measure (Popoff, 2020). Foals have maternal antibodies against the bacteria in the first 10 to 18 weeks of life, so they only should be vaccinated after this time. The first immunization can consist of three doses of the vaccine. The recommended interval between boosters is debatable (Jansen and Knoetze, 1979; Kendall et al., 2015; Ribeiro et al., 2018). The administration of 1500 IU of tetanus anti-toxin in unvaccinated horses can protect horses up to three weeks and should be administered to horses at risk or with an unknow vaccination status (Nout-Loma, 2016).

Dermatology

During the externships, a total of 44 cases were followed, as shown in table 5. Most of the cases observed were wounds (n=15), followed by several types of skin tumours, mostly sarcoids and melanomas.

Table 5 - Dermatology Casuistic

Cases	Total
Urticaria	4
Hypersensitivity to mosquito bite	2
Sarcoidosis	6
Lymphangitis	4
Melanoma	3
Exuberant granulation tissue	1
Cellulitis	9
Wounds	15

Sarcoidosis

Sarcoids are the most common type of skin tumour in horses (Knottenbelt, 2005). Six types of sarcoids are discriminated based on macroscopic appearance and clinical presentation, these are, occult, the less severe and more superficial type, it usually presents with alopecia and have

a more rounded appearance; verrucous, that have a rugged and hyperkeratotic appearance; nodular which appears as a firm subcutaneous nodule with variable size and number. These may be further divided into type A and B, depending on the amount of dermis involvement; fibroblastic, that have a flesh-like appearance, they can also be divided into pedunculate or sessile; mixed; and malignant that are very locally invasive (Knottenbelt, 2005; Taylor and Haldorson, 2012).

There are several known risk factors for the development of sarcoids, as bovine papillomavirus, fly vectors and skin trauma. Histopathologic evaluation of the lesion is necessary to confirm the diagnosis, but it is not recommended unless there is an atypical presentation, as the biopsy may worsen the lesion and result in further proliferation (Taylor and Haldorson, 2012).

Treatment options include conventional excision with wide margins of two or three centimetres, laser or electrosurgical excision, cryotherapy, radiotherapy, chemotherapy, frequently with cisplatin or carboplatin applied topically or intralesionally, topical immune modulation, and antiviral agents, like topical acyclovir and therapeutic vaccinations. Cisplatin electrochemotherapy is a therapy where electrical pulses are delivered on site to increase membrane permeability, potentiating the effect of the therapy, the number of treatments and their regularity depends on tumour type and size. When selecting a treatment, the type of sarcoid and its location, its extension and number, any previous attempts of treatment, the logistics of treatment and owner and animal compliance should be evaluated (Haspeslagh, Vlaminck and Martens, 2016; Knottenbelt, 2019). On the cases observed during the Wolvega externship the horses usually presented more than one lesion, and the selected type of treatment was laser excision, usually these removals were done when the horse was already anesthetized for a different procedure. The skin around the tumour was infiltrated with two percent lidocaine, then surgically prepared with a perimeter of at least one centimetre, and the edges of the tumour were marked with sterile pen, after the excision, the skin was left to heel by secondary intention (Compston *et al.*, 2016). In Equitom a warmblood also presented with several sarcoids along the torso, abdomen, perineum and tail, the chosen treatment modality was cisplatin electrochemotherapy. The horse was placed under general anaesthesia, and the sites were aseptically prepared, then a cisplastin solution was injected into the sarcoids and a two-centimetre margin, the solution had a concentration of 1mg/ml and the dose was 0,3mg/cm^3 of tissue. Within five minutes of the cisplatin injection electrodes were placed on the skin in contact with the tumour surface along with sterile gel. Then electric pulses were applied using an electropulsator, two series of eight electric pulses with a frequency of 500 hertz in two orthogonal directions *per* location. It was projected that the horse should do a total of four treatments with an interval of two weeks, the plan was likely to be adjusted according to the horse's tolerance to the treatment and the lesion's response (Tamzali *et al.*, 2011).

Cellulitis and Lymphangitis

Cellulitis is a spread bacterial infection and inflammation of the deep dermis and subcutaneous tissues. It is a common cause of emergency presentation and clinically it presents as an acute onset of heat, swelling, pain with variable manifestation of lameness and typically associated with pyrexia. The diagnosis is usually made based on clinical signs (Braid and Ireland, 2022). Most often, *Staphylococcus aureus* or other species of *Staphylococcus* or *Streptococcus* are isolated. Lymphangitis refers to the inflammation of lymphatic vessels, and is usually associated with cases of cellulitis, and it presents as a diffuse soft-tissue tumefaction and lymphadenomegaly. Ulcerative lymphangitis in the limbs is commonly associated with infections of *Corynebacterium pseudotuberculosis* and it usually affects the hindlimbs (Chapman, 2014; Rendle, 2017; Braid and Ireland, 2022).

The treatment of cellulitis is very similar to ulcerative lymphangitis and it involves non-steroidal anti-inflammatory drugs, most frequently phenylbutazone (2,2 mg/kg BID), broad spectrum antibiotics, namely an association of penicillin and gentamicin (22 000 IU/kg and 6,6 mg/kg, respectively), trimethoprim sulphadiazine (15 mg/kg BID for five days) or doxycycline (12 mg/kg SID up to five days), compression through bandaging of the limb and hydrotherapy with cold water. Topical medications recommended include silver sulphadiazine creams or chlorhexidine. In some cases, dexamethasone (0,01 mg/kg), may be used to help reduce the risk of chronic swelling, although its use may be controversial, because there is an infectious process present. If the horse is comfortable enough, walking is advised. In cases where the swelling persists despite treatment, ultrasound-guided drainage is advised (Rendle, 2017; Braid and Ireland, 2022).

Insect bite hypersensitivity

Friesian horses, in the Netherlands, are particularly predisposed to develop insect bite hypersensitivity (Grevenhof, van *et al.*, 2007). This disease involves type I hypersensitivity reactions, mediated by Immunoglobulin E, and delayed- type hypersensitivity, type IV, reactions. there may be a genetic component to the disease (Grevenhof, van et al., 2007; Boerma, Back and Sloet Van Oldruitenborgh-Oosterbaan, 2011; Schurink et al., 2011). It may be characterized as a chronic and seasonal allergic dermatitis with pruritus and is usually caused by insects of the *Culicoides* genus. Interleukin 5 (IL-5) seems crucial to the development of insect bite sensitivity, as it promotes the release of leukotrienes and proteins that trigger mast cell degranulation, by the eosinophils (Schaffartzik *et al.*, 2012). A presumptive diagnosis is based on clinical signs, history and elimination of other causes, the definitive diagnosis is achieved through skin biopsy. The prevention of disease is achieved by limiting contact with the *Culicoides* by stabling the horses, using blankets or insect repellents. Corticosteroids, such as prednisone (0,5 – 1 mg/kg PO) or dexamethasone (0,05-0,1 mg/kg PO) may be of help relieving the symptoms but should not be used for long. Fatty acid supplementation and phosphodiesterase inhibitors, such a pentoxyfiline (8-10 mg/kg PO) may be used as adjuvant therapies (Rashmir and Petersen, 2015; Jonsdottir et

al., 2019). Other therapies have been developed but need further testing such as IL-5 vaccines (Schaffartzik et al., 2012; Jonsdottir et al., 2019).

During the externship one four-year-old Friesian horse, that spent most of the time in the field, presented with a complaint of pruritus and focal alopecia. He presented skin lesions on the *ventrum*, similar to the one shown on Figure 1. The horse did not show evidence of external parasites infestation, and the owner had not made any changes to the horse's diet or management, so a presumptive diagnosis of insect bite hypersensitivity based on the signalment and history of the horse was made. A skin biopsy was proposed to confirm the diagnosis, but the owner refused. The horse started fatty acid supplementation and the owner was advised to start applying insect repellents or stabling the horse, he was also instructed to return with the horse in case it persisted, or it got worse.

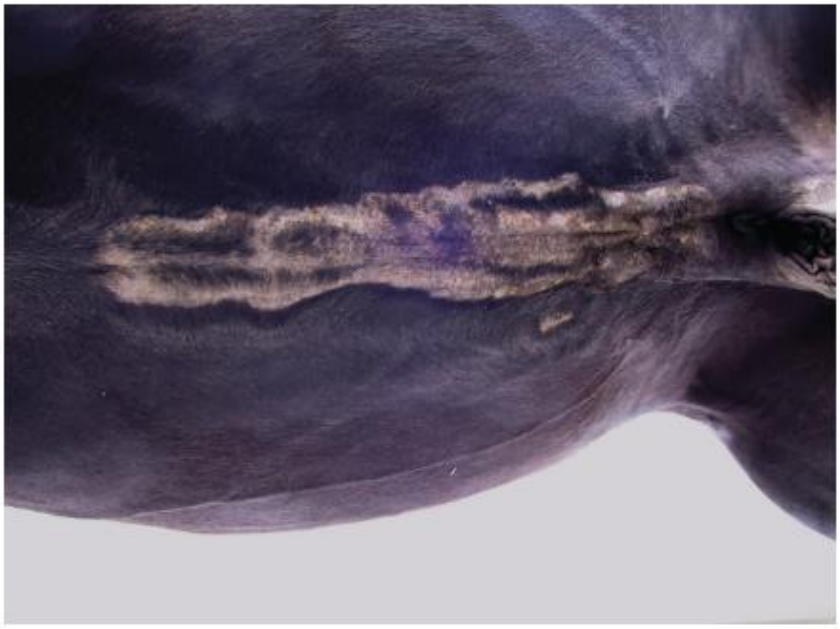


Figure 1 - Pruritic skin lesion in a Friesian gelding with insect bite hypersensitivity (Boerma, Back e Sloet Van Oldruitenborgh-Oosterbaan, 2011)

Dentistry

Regular dental checks should be aimed at the improvement of mastication and digestion of feed, the relief of discomfort associated with sharp enamel points that cause injury to the soft tissues, at reducing stresses on overworn teeth, and at helping the horse be more comfortable with the bit. Hopefully, this will improve mastication, maintain proper alignment and health of the dental arcade. Malocclusion may be associated with weight loss and poor performance (Carmalt, 2007; Tremaine and Casey, 2012).

Horses older than five years, should have their teeth checked at least yearly, if not needed more often. Some upper-level performance horses are recommended to do it every six months (Carmalt, 2007; Tremaine and Casey, 2012). Horses younger than five years might need more regular check-ups, especially when they start training, because as they shed teeth new ones erupt, and may have issues with the bit (Klugh, 2010).

The dentistry caseload is represented on Table 6. Most of the routine dental examinations were observed during ambulatory practice, some of these horses were later referred to the hospital to pursue further investigation of their condition or to perform some treatments, such a tooth removal. In the present report when referring to “dental checks” we are referring to the oral examination itself, the routine treatments to ensure proper occlusion, as well as premolar removals.

Table 6 - Dentistry Casuistic

Cases	Total
Dental checks	33
Equine odontoclastic tooth resorption and hypercementosis	2
Infection	4
Broken teeth	4

Oral examination includes inspection of teeth, gingiva, tongue, salivary systems, and the mucosa. Sometimes these examinations are part of routine health checks or part of a pre-purchase examination, other times they are done to aid the diagnosis of horses with abnormal or biting behaviour or any other equitation issues, weight loss or if there is a suspect of sinusitis. Also, if the horse shows reluctance to prehend food, hypersalivation, oral dysphagia, halitosis, mandibular swelling, unilateral nasal discharge, headshaking or rearing dental disease should be considered, and an oral examination performed (Tremaine and Casey, 2012).

The horse should be well sedated with an alpha-2-agonist like romifidine (0,5-1 mg/kg) or detomidine (10 – 20 µg/kg) in combination with butorphanol (20 µg/kg). The horse should be sedated enough so it does not respond to noxious stimuli, but not enough that it is very ataxic, in this regard romifidine is a good drug because it is less likely to cause ataxia (England, Clarke and Goossens, 1992). Younger horses, or more anxious horses can be given 0,05-1 mg of acepromazine half an hour before the rest of the sedation (Tremaine and Casey, 2012).

A dental speculum and a light source, a head stand or collar, a dental mirror, dental picks, and mouth wash liquid, are essential to perform the examination. Before the speculum is placed, the

mandible should be articulated and displaced in a side-to-side motion to assess lateral occlusion and incisor symmetry (Tremaine and Casey, 2012; Tremaine and Pearce, 2012).

Every tooth should be individually palpated to check for supernumerary teeth, enamel points or hooks, dental caps, diastemas or fractures, and the oral cavity should be checked for ulcers or any other lesions. If the first premolars are present their removal should be discussed with the owner, as their impact is uncertain. If necessary, endoscopy or any other complementary imaging techniques, like radiography or computed tomography scan, should be recommended (Tremaine, 2012; Tremaine and Casey, 2012a, 2012b; Hole and Staszyk, 2016).

Routine treatments may be performed with hand rasps, but ideally should be with motorised or electrically powered tools, as the horses tolerate it better (Tremaine and Pearce, 2012).

In older horses, usually over 20 years-old, the teeth are erupting near the end of the reserve crown, and therefore they are susceptible to periodontal disease, fractures, and other progressive diseases, such as Equine odontoclastic tooth resorption and Hypercementosis (EOTRH). Most malocclusions are no longer amendable, so the goal of treatment in these horses is to relieve discomfort, stop further damage to the arcade and make sure that the horse is able to prehend well (Klugh, 2010; Tremaine e Casey, 2012; Tremaine e Pearce, 2012).

Equine odontoclastic tooth resorption and Hypercementosis

EOTRH is progressive and painful disease primarily affecting the incisor and canines of older horses (older than 14 years-old). The true aetiology of the disease is unknow, but it is thought that a significant strain on the periodontal ligaments of the aged hypsodontal incisor may be in the origin of the lesions. Several factors might be related to the onset of disease, such as, mechanical forces, diet (alfafa), underlying systemic or infectious diseases, genetic predisposition, metabolic disorders affecting blood calcium values or vitamin D, cortisol, or parathyroid hormone, hypervitaminosis A, excessive dentistry, and trauma. Histopathologically, resorption lesions and deposition of reparative cementum are found concomitantly, and the surrounding tissues show signs of inflammation (Pearson et al., 2013; Smedley et al., 2015; Rehrl et al., 2017). The horses may manifest different levels of oral discomfort, periodontitis, gingivitis, gingival hyperplasia, fistulas, tooth mobility or fractures, or may be missing teeth. Radiologically different findings may be observed, such as, different levels of dental resorption and hypercementosis, loss of periodontal space, alveolar bone loss, tooth fractures and osteomyelitis. An example of a horse's radiography is shown on Figure 2 to showcase the most frequent lesions. The radiological findings can relate to clinical significance as shown on table 7. Currently the treatments for EOTRH involve tooth removal, rinsing with 0,12% chlorhexidine and administration of trimethoprim-sulphonamide (15-30 mg/kg PO BID), metronidazole (15-20 mg/kg PO BID) or penicillin procaine (22 000 UI/kg IM BID) and administration of flunixin meglumine, to alleviate the

pain. These horses should be regularly monitored, to ensure the mouth remains in balance with occlusion (Pearson et al., 2013; Lorello et al., 2015).

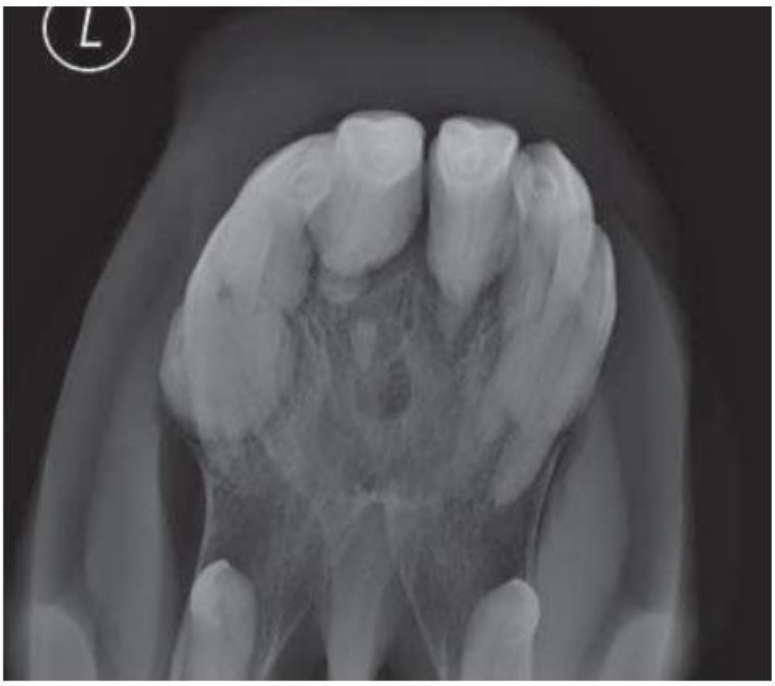


Figure 2 - Radiography in an intraoral projection of remodelling and hypercementosis in the apices of the incisors in a horse suffering from EOTRH, adapted from (Lorello et al.*, 2015)*

Table 7 - Scoring of clinical manifestation of EOTRH according to radiological finding, adapted from (Rehrl *et al.*, 2017).

Score	Severity	Radiological signs
0	Normal	No findings
1	Mild	Tooth shape preserved with a blunted root tip and irregular surface
2	Moderate	Tooth shape mostly preserved, with root tip obviously blunted. The intra-alveolar tooth part is not wider than the clinical crown
3	Severe	Loss of tooth shape, the intra-alveolar tooth part is wider than the clinical crown. The surface markedly rough.

Gastroenterology

Gastrointestinal disease was the most frequent complaint (n=118), after musculoskeletal issues, as previously illustrated on Table 1. Table 8 shows the conditions diagnosed affecting the gastrointestinal system and their frequency. Most of the presented issues were colic or acute abdomen syndrome, followed by Equine gastric ulceration syndrome (EGUS) and colitis. Horses

are particularly at risk for gastrointestinal disorders as they are herbivore hindgut fermenters and have a well-developed caecum and colon. Under natural conditions the horse would spend most of his day foraging, however most horses are confined and have around two meals that consist in concentrates and preserved forages, rich in sugars and starches, which may cause imbalances in the microbial flora, leading to illness (Hesta and Costa, 2021).

Table 8 - Gastroenterology Casuistic

Cases	Total
Megaoesophagus	7
Oesophageal obstruction	8
Equine gastric ulceration syndrome	18
Acute Abdomen/ Colic	61
Colitis	16
Inflammatory bowel disease	4
Peritonitis/ Abscesses	1
Intoxication - endotoxemia	1

Oesophageal obstruction and megaoesophagus

Oesophageal obstruction is usually a consequence of feed impaction in the lumen, it can be caused by concentrated feed, forage, carrots, apples, or other types of foreign bodies. Horses that have issues prehending food, extraluminal masses or oesophageal abnormalities, like megaoesophagus, are particularly at risk. The horses present dysphagia, ptyalism, cough and extension of the head, feed and saliva may also be appearing at the nostrils. As soon as the condition is noticed water and feed should be withdrawed. A full physical examination should be performed, with special care to the patient's hydration status and signs of shock, also the lungs should be carefully auscultated for any indication of inhalation pneumonia, such as increased respiratory sounds. Palpation of the laryngeal and cervical region may also be of help (Chiavaccini and Hassel, 1992; Archer, 2013).

The patient should be sedated with a combination of an alpha-2-agonist and butorphanol, enough so it is less anxious, and lowers the head, to reduce the risk of feed inhalation. Butylscopolamine can be administered at a dose of 0,3 mg/kg intravenously (IV), to reduce smooth muscle tone, as well as anti-inflammatory drugs. A nasogastric tube should be passed, to confirm the diagnosis and, if possible, to break down the impaction with lavages of lukewarm water. It is important to not force the tube as it may cause a tear in the oesophagus. If this approach is not fruitful, then the horse can be placed under general anaesthesia and the oesophagus may be lavaged using a cuffed endotracheal tube. If the obstruction is not resolved withing a few hours then the patient should be placed on IV fluids, to ensure adequate hydration (Chiavaccini and Hassel, 1992;

Bezdekova and Bezdekova, 2012; Archer, 2013; Sanchez, 2016). In cases that the obstruction is not resolved an oesophagostomy can be performed, but there is a high risk of development of strictures and infection (Craig *et al.*, 1989).

After the resolution of the obstruction, an endoscopy should be performed to access the amount of damage, and then repeated after two to four weeks. Sucralfate (20mg/kg every six hours PO) can be administered to reduce the risk of oesophageal ulceration. In the first 24 to 48 hours the horse should be fasted, afterward it can be fed soft food and then transitioned to roughage in the following seven to 21 days (Sanchez, 2016).

The most common complications are oesophagitis, with or without ulceration of the mucosa, and aspiration pneumonia. Oesophageal tears are also possible (Craig et al., 1989; Bezdekova and Bezdekova, 2012).

Megaoesophagus, as mentioned previously, is a risk factor for oesophageal obstructions. It can be defined as a chronic atonia and dilation of the oesophagus (Chiavaccini and Hassel, 2010). It can be diagnosed with endoscopy by its increased size and lack of peristaltic movements. Horses suffering from megaoesophagus may often have history of dysphagia, oesophageal obstruction and/or aspiration pneumonia (Boerma, Back and Sloet Van Oldruitenborgh-Oosterbaan, 2011; Sanchez, 2016). During the externship seven horses were diagnosed with megaesophagus, all these horses were Friesian foals. In fact, in Friesian horses this condition appears to be heritable, and most cases are diagnosed in the first five years of life (Boerma, Back and Sloet Van Oldruitenborgh-Oosterbaan, 2011; Ploeg et al., 2015). The primary cause for this appears to be genetic abnormalities in the collagen of the oesophagus, which can also be found in very young foals not yet demonstrating disease (Ploeg *et al.*, 2015).

Acute abdomen

The most common reason for emergency consults was colic syndrome (n=61). The aetiologic diagnosis were gastric impactions and/or rupture in Friesians, duodenitis- proximal jejunitis, ileal impactions, *Parascaris equorum* impactions in foals, strangulating lipomas, epiploic foramen incarcerations, ileocecal intussusception in a foal, caecal impaction in a donkey, several colon impactions, gas colics, sand enteropathies, nephrosplenic ligament entrapments, colon displacements and colon torsions.

In case of colic the collection of a complete history is very important, such as, when did the clinical signs start and its progression, if there has been any faecal output and if so its consistency, if there were any changes in routine, diet or stabling, if there are any known concomitant diseases or if the horse is taking any medication, routine prophylactic measures or if the horse shows any stereotypic behaviour (Archer, 2013; Cook and Hassel, 2014).

Then, a full clinical examination should be performed, and special attention should be given to the assessment of the horse's cardiovascular state (heart rate, capillary refill time, peripheric pulse), intestinal sounds, the presence of digital pulses and rectal temperature. When assessing the patient, it is important to understand if the lesion is in the small or large intestine, accordingly to the clinical signs. Haematology, blood biochemistry, electrolyte evaluation and lactate measurements should be performed, and a jugular catheter should be placed. Afterward rectal examination should be completed, if there is a large amount of gas N-butyl scopolaminium bromide (0,1 mg/kg IV) should be administered and the palpation should be repeated. Evidence of distention or abnormal location of organs should be noted. A nasogastric probe should be passed, and an abdominal ultrasound should be conducted. It is always advised to pass a nasogastric tube in a horse with a suspect of colic, the amount of reflux should be measured, and if there is only a normal amount, fluids can be administered. A horse should have no more than four litres of gastric reflux. When administering fluids via the nasogastric tube, it is important to keep in mind that a horse's stomach capacity may be estimated in litres as 1/100 of body weight, for example a 500kg horse, would have a stomach capacity of five litres. The ultrasound should be performed with a 3,5-5MHz linear or curvilinear transducer and stomach size, intestinal wall condition, mobility and distention should be evaluated, as well as the presence of excess free fluid, if so an abdominocentesis should be performed. The horse should be fasted, if there are not large amounts of reflux, water should be maintained (Freeman, 2011; Archer, 2013; Rhodes and Madrigal, 2021).

In milder cases an association of butyl scopolamine and metamizole can be administered in a dose of 0,3 mg/kg IV, if the signs of discomfort persist, the horse can be sedated with a combination of an alpha-2-agonist, like xylazine (0,3-0,4 mg/kg IV), and butorphanol (20µg/kg IV or IM), and if necessary, flunixin meglumine (0,25mg/kg IV q8hrs). In the most dramatic cases opioids may ne necessary, such as morphine in a dose of 0,1-0,2 mg/kg IV (Archer, 2013).

The horse should be closely monitored to determine if conservative treatment should be continued or if surgery is indicated. There are several clinical criteria that may aid the decision, such as: presence of tachycardia, moderate to severe pain and little response to analgesia, reduced gastrointestinal sounds, abdominal distension, an increased packed cell volume and total protein, systemic lactate higher than three mmol/L, large amounts of nasogastric reflux, the clinical parameters of peritoneal fluids, distended, immotile small intestine with wall oedema on ultrasound and/or increased colon wall thickness. Horses with uncontrollable pain, severe cardiovascular compromise and gastrointestinal rupture should be euthanized, especially if surgery is not an option (Epstein and Fehr, 2013; Southwood and Fehr, 2013; Cook and Hassel, 2014; Rhodes and Madrigal, 2021).

Regarding surgery, after adequate preparation, the abdomen should be opened and explored through palpation and exteriorization of intestine wherever possible, until the lesion is identified.

The displaced or incarcerated intestine should be corrected, distended viscera should be decompressed and if necessary, severely damaged tissue should be resected (Epstein and Fehr, 2013; Rötting, 2017).

After surgery, the horse's management should be aimed at preventing infection, maintaining the horse comfortable, restore fluid and electrolyte imbalances, re-establish intestinal function, prevent complications such as ileus or endotoxemia and prevent the re-occurrence of colic. The horse should receive intravenous fluid therapy, Ringer's lactate is the fluid of choice, and it can be supplemented with electrolytes as needed. Should receive broad-spectrum antibiotics, and proper analgesia. Enteral nutrition 12 hours after surgery is advised, unless there was extensive intestinal ischemia, ileus or if the horse is still showing colic signs. Parenteral nutrition may be helpful until progressive feeding is possible (Freeman, 2018; Mcgovern, [n.d.]).

Colic surgery complications include ileus, endotoxemia, thrombophlebitis, intestinal adhesions or mesenteric tears, surgical site infection, colitis, laminitis, or pneumonia (Epstein and Fehr, 2013; Freeman, 2018; Mcgovern, [n.d.]).

Pneumology

Respiratory issues are frequent in the horse and usually have significant impact in the animal's performance. These conditions may be divided in the ones that affect the upper respiratory tract, which result in increased resistance to the passage of air, and the ones that affect the lower respiratory tract and, in consequence, the efficiency of gas exchange in the lungs (Art and Warwick, 2014; Ducharme and Cheetham, 2014). The clinical differentiation between these two and some common differential diagnoses are shown on Figure 3. In Table 9 there is a summary of the casuistic observed during the externship and its respective frequency.

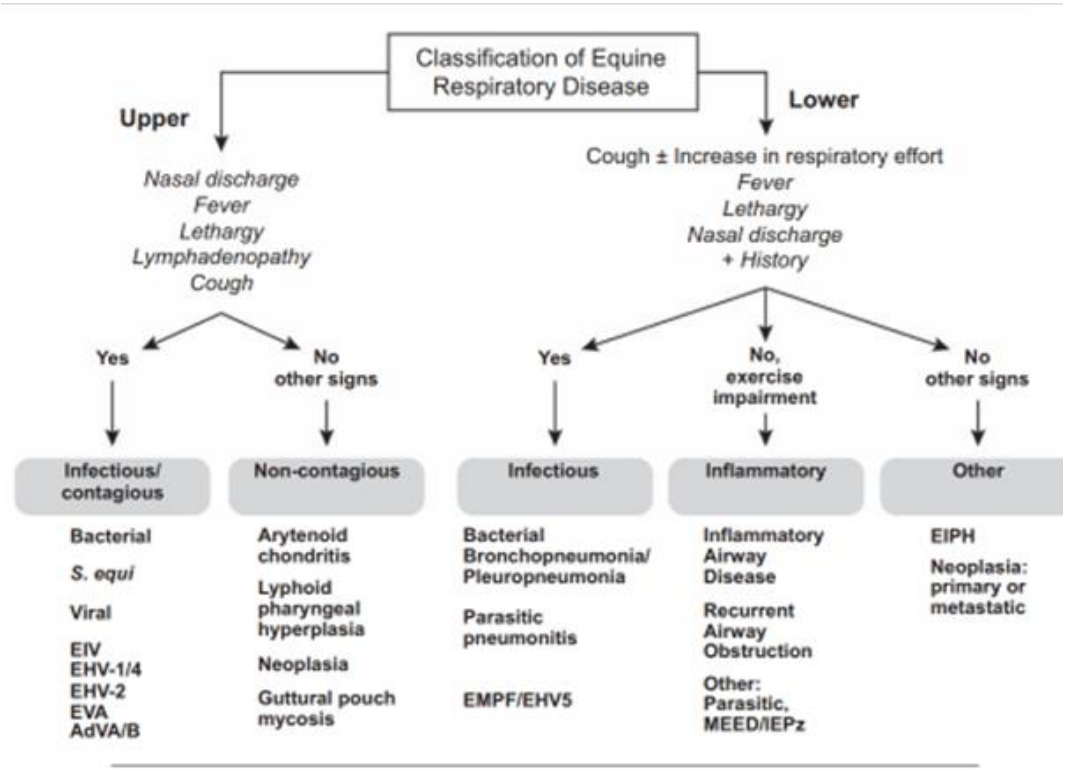


Figure 3 - Diagram differentiating upper and lower airway diseases, adapted from (Davis, 2016).

Table 9 - Pneumology Casuistic

Cases	Total
Sinusitis	8
Sinus cyst	1
Guttural pouch mycosis	1
Strangles	3
Epiglottic entrapment	1
Dorsal displacement of the soft palate	1
Laryngeal hemiplegia	13
Pneumonia	18
Asthma	29
Exercise-induced pulmonary haemorrhage	1

Laryngeal hemiplegia

Laryngeal hemiplegia is an important progressive axonopathy, commonly seen in Warmbloods and Throughbreds (Clercq, Rossignol and Martens, 2018). As the left laryngeal recurrent nerve becomes gradually demyelinised it compromises the enervation of the left cricoarythenoideus dorsalis muscle function, so the arytenoid cartilage abduction is not complete and therefore, not enough air passes through the larynx (McCarrel and Woodie, 2015). Clinically this manifests as decreased performance and respiratory noise. It is usually more severe on the left side, as the nerve is longer and has a more tortuous course. The aetiology of disease may be genetic, but also acquired or iatrogenic, due to the administration of irritating substances or jugular perivascular injections. The definitive diagnosis is achieved through endoscopy, either at rest or dynamic, and then the lesions are graded, based on arytenoid function (McCarrel and Woodie, 2015; Davis, 2016; Clercq, Rossignol and Martens, 2018). The findings of endoscopy at rest may be graded as it is described on Table 10.

Table 10 - Grading of rest endoscopy findings, adapted from (Davis, 2016)

Grade	Description
I	Arytenoid movements are synchronous and symmetrical. There is complete and sustained abduction of the arytenoids
III	Arytenoid movements are asynchronous at some times. There is complete and sustained arytenoid abduction
III	Arytenoid movements are asynchronous, asymmetrical, or both. Complete abduction of the arytenoids is not achieved
IV	Arytenoid cartilage is immobile and vocal fold is complete

If the horse shows exercise intolerance a laryngoplasty surgery is performed, so there is permanent abduction of the arytenoid cartilage, followed by a ventriculocordectomy. If the respiratory noise is the only complaint, then laser ventriculocordectomy might be enough. Laryngeal reinnervation is also an option if some movement is still present (McCarrel e Woodie, 2015). In the laryngoplasty surgery, the horse is placed in lateral recumbency on the table, and non-absorbable suture is placed between the cricoid and arytenoid cartilage. Ventriculocordectomy is the removal of the mucosal lining of the laryngeal ventricle and of a crescent-shape piece of tissue from the vocal cord, this procedure may help with the diminish the respiratory noise and aid with athletic performance, and it can be performed with a diode laser with the horse standing (Ducharme and Rossignol, 2019). After surgery, the horse can be placed in antibiotics for 48-72 hours, and phenylbutazone (2,2 mg/kg PO) or flunixin meglumine (1 mg/kg PO). Horses should be at rest for four to six weeks after the surgery, the only form of exercise should be hand walking. After this period, an endoscopic evaluation should be performed to determine if the horse can be placed in a small paddock, water and feed should be placed on the floor (McCarrel and Woodie, 2015; de Clercq, Rossignol and Martens, 2018; Ducharme and Rossignol, 2019).

Equine asthma

Asthma presents as a chronic and recurrent lower airway syndrome that affects mostly stabled horses. The term encompasses several non-infectious chronic and progressive disorders and may be differentiated into mild/moderate and severe, that represent inflammatory airway disease and represent recurrent airway obstruction, respectively (Couëtil et al., 2016; Bond et al., 2018). This classification only discriminates different clinical presentations, and it does not describe the different diseases regarding to their aetiology and pathogenesis. Different phenotypes of the disease may be identified concerning its immunologic characteristics (Bond *et al.*, 2018). The characteristic clinical factor of this disease is increased respiratory effort at rest, caused by

bronchoconstriction and increased mucus production. In its origin there is a combination of individual and environmental factors, such as previous airway infections, hypersensitivity reactions, presence of allergens and lack of adequate ventilation (Bond *et al.*, 2018; Couetil *et al.*, 2019). The diagnosis is reached based on clinical signs, presence of mucus in the airway, that may be graded during endoscopic evaluation, as suggested on Figure 4 and evaluation of transtracheal or bronchoalveolar wash fluid cytology for the presence of neutrophils and other inflammatory cells.

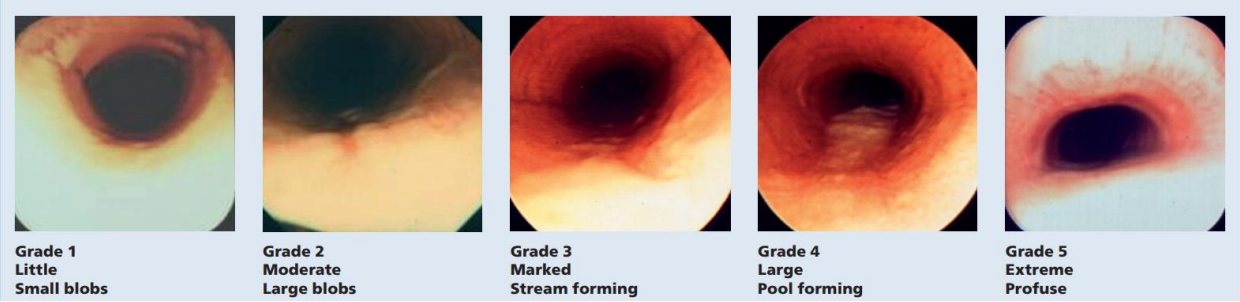


Figure 4 - Grading of mucus accumulation, adapted from (Allen and Franklin, 2007)

In milder cases, alterations in the environment and management of the horse may be sufficient to decrease inflammation. In most cases however, glucocorticoids, like prednisolone (1,1-2,2 mg/kg PO SID) are used as well as bronchodilators, namely clenbuterol (0,8-3,2 µg/kg BID). Omega 3 poly-unsaturated fatty acids are also commonly given. The medication may be delivered via a nebuliser, as shown on Figure 5, if the horse tolerates it, with fewer systemic side effects and good clinical efficiency. Fluticasone (1-6 µg/kg) or beclomethasone (1-8 µg/kg) associated with Albuterol (1-2 µg/kg) are the drugs of choice for this. Medical management of the disease should always be complemented with management and environmental modifications, such as increasing ventilation in the stables or turning out the horses for longer periods of time, replace hay bedding for one that accumulates less dust particles, like wood shavings, immersing feed in water, to further reduce the respiratory burden, and keep feed higher, so the horse does not have to stretch the head while eating, among others (Couëtil *et al.*, 2016) .

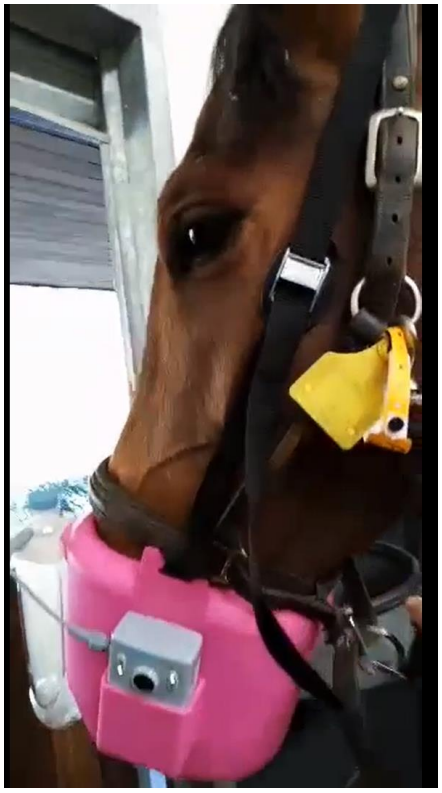


Figure 5 - Nebulisation therapy in a Belgian Warmblood diagnosed with equine asthma

Cardiology

Cardiac abnormalities are frequent in the horse; however, they do not usually produce significant clinical impact. The most frequent complaint associated with heart disease is poor performance (Bonagura, 2019). During the externship, a total of six horses were diagnosed with cardiac issues, as seen in Table 11. The only diagnosed arrythmia was atrial fibrillation.

Table 11 - Cardiology *Casuistic*

Cases	Total
Arrhythmias	4
Valvular disease	2

Atrial Fibrillation

Atrial fibrillation (AF) is a frequent supraventricular arrhythmia in the horse, that usually affects performance (Reef *et al.*, 2014). AF can be characterized as an uncoordinated atrial depolarization resulting from several chaotic re-entry waves that cause ineffective atrial function (Decloedt *et al.*, 2020). Atrial size, structural diseases and ectopic electrophysiological activity are

considered risk factors for the development and perpetuation of AF (Clercq et al., 2014). A presumptive diagnosis can be made during heart auscultation, which should be recognized as an irregularly irregular rhythm, then it can be confirmed with an ECG, in which an irregularly irregular R-R interval with a normal QRS complex and the replacement of P waves by “flutter” or “f” waves, which are oscillations of the isoelectric line, should be present. After the diagnosis is established, a full echocardiographic exam should be performed to identify underlying structural diseases, evaluate valvular function, and measure heart chamber size. Cardioversion can be attempted with transvenous electrical cardioversion (TVEC) or pharmacologically, and is recommended for athletes, if they do not show significant cardiac changes or clinically relevant underlying conditions (Reef et al., 2014; Loon, 2020). TVEC entails a shock delivery via defibrillator through cardioversion catheters during R wave, with the horse under general anaesthesia (Reef *et al.,* 2014). Pharmacological cardioversion is usually done using quinidine sulphate in a dose of 22 mg/kg through a nasogastric tube every two hours, a maximum of five doses a day, until cardioversion or side effects occur. Quinidine sulphate is a sodium channel blocker, which increases the refractory period in the myocardium and prolongs QT and QRS intervals. There is a risk of toxicity so the horse should be closely monitored, and the treatment should be discontinued if it occurs (Reef et al., 2014; Decloedt et al., 2021). During the externship, the presented horses were young Dutch warmbloods in training. Two of the horses had a history of inadequate performance so they were referred to hospital for a ECG evaluation and echochardiograhy, one of the horses was diagnosed during a PPE, and the other was diagnosed when he was anesthetised for an OCD fragment removal. These patients exhibited an irregularly irregular heart rhythm and ECG changes similar to the one shown on Figure 6, but no cardiac alterations in the echocardiography. The recommended treatment was TVEC, as it was not available in the hospital, the horses were referred to Ghent University to perform the treatment. Most horses return to their previous level of work; however, recurrence is a risk, so they should be closely monitored. There are not any known complications specifically associated with TVEC, the identified complications are those associated with the peri-anaesthetic period (Decloedt *et al.*, 2021). Horses that show QRS complexes changes or R-on-T phenomenon or horses that show signs of congestive heart failure are at high risk for collapse, and therefore should not be ridden (Loon, van, 2019).

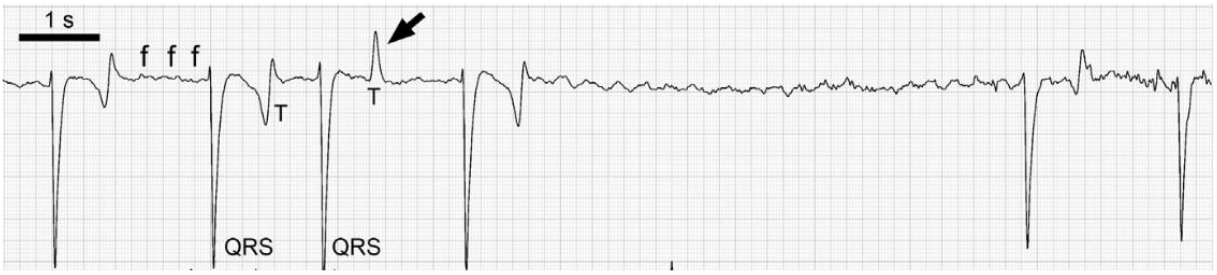


Figure 6 - ECG of a horse with AF from (van Loon, 2019)*. The futter waves are represented with "f".*

Urology

Urologic issues were not a frequent cause of consult or hospitalization (n=3), as seen on table 12. All the patients were male. One Icelandic gelding presented with a urethral stricture, with a complaint of dysuria, one Coldblood stallion showed “colic-like” symptoms and showed bladder distention, and then a foal was referred for oliguria, but showed a normal urine output during his time hospitalized.

Table 12 - Urology Casuistic

Cases	Total
Urethral stricture	1
Oliguria	1
Distention	1

Reproduction

Procedures concerning the reproductive system were followed in several contexts of practice, orchiectomies, uterine cyst removals and Caslick sutures were followed during the surgery rotations, and several mares were followed in both the internal medicine and ambulatory rotations. These procedures are summarized on Table 13.

Table 13 - Reproductive System Casuistic

Procedures	Total
Orchiectomies	22
Pregnancy check	8
Artificial insemination	3
Endoscopic uterine cyst removal	3
Caslick vulvoplasty	1
Abortion	1

Orchiectomy

The most frequently observed procedures were castrations, most of these occurred under general anaesthesia, often also when doing other procedure. Out of the 22 procedures, three were done with the horse standing with an open technique, and the others with the horse under general anaesthesia using a closed technique. Most of the cases were young animals that were castrated before the onset of pronounced stallion behaviour to sterilize them, one had a castration because he presented a mass in the testis, one was a cryptorchid and one other foal had an inguinal hernia.

Before a castration it is important to verify the vaccination status of the horse and administer tetanus anti-toxin, if necessary. A full clinical examination should be performed, and it should be confirmed that both testicles are present in the scrotum. In cryptorchid animals the testis should be located first with ultrasonography, if possible. Antibiotics and non-steroidal inflammatory drugs should be administered perioperatively. Regardless of the technique used, a local anaesthetic block should be performed with an intratesticular lidocaine block, and the emasculator should be placed so the crushing side is proximal do the blade and the winged nut is directed towards the testicle, the emasculator should be left long enough so the spermatic cord is crushed, the time depends on the size of the spermatic cord (Kilcoyne, 2013). The denomination of an open or closed technique concerns the treatment of the parietal tunica vaginalis, in an open castration the tunica is opened before the testicle is exteriorized and before the ligatures are placed, in a closed

castration the tunica is not opened until the spermatic cord is ligated, since in this technique there is less visibility, it should be performed with the horse under general anaesthesia (Searle et al., 1999; Schumacher, 2019; Kilcoyne and Spier, 2021). The wounds are usually left to heal by secondary intention (Kilcoyne and Spier, 2021; Schumacher, 2019).

Various post-surgical complications have been reported, such as, swelling and seroma formation, funiculitis, haemorrhage, eventration, peritonitis, hydrocele formation, penile iatrogenic trauma, or stallion-like behaviour (Kilcoyne and Spier, 2021). The complications observed during the externship were three funiculitis and one scrotal hematoma.

Surgical site infection may occur three to 21 days after surgery, and it is often a result of premature closure of incisions (Kilcoyne and Spier, 2021). Funiculitis is characterized as a chronic infection of the spermatic cord ending after the surgical incisions have healed, with the formation of abscesses. It may manifest with fever, swelling, stiff gait or lameness and discharge from incision wound. Diagnosis can be made with palpation and examination of the scrotal area and a transrectal ultrasound examination. The lesion should be drained and antimicrobial therapy, based on sensitivity tests, should be installed. Usually, *Staphylococcus* sp. or *Streptococcus equi* zooepidemicus can be isolated. If the infection does not resolve with medical therapy, then surgical resection of the infected tissue is necessary for complete resolution. The cord should be emasculated proximally to the infected tissue and then left to heal by second intention (Searle et al., 1999; Kummer et al., 2009; Kilcoyne and Spier, 2021).

Ophthalmology

In Table 14, the caseload of ophthalmology is described. Uveitis and corneal ulcerations were the most frequent cause of complaint. Traumatic eye lesions are frequent in the horse due to its size, the lateral position on the head and the animal's temperament (Lassaline, 2016).

Table 14 - Ophthalmology Casuistic

Cases	Total
Uveitis	14
Corneal ulceration	6
Distichiasis	2
Neoplasia	1

Uveitis

Anterior uveitis is the inflammation of the iris and ciliary body (McMullen and Fischer, 2017). It is frequently secondary to other insults, either ocular or systemic. There is disruption of the blood-ocular barrier caused by the inflammation and spasm in the anterior uvea, which leads to cells and proteins leaking into the anterior chamber causing hypopyon and aqueous flare. In an acute phase there is also swelling and hyperaemia of the conjunctiva, at this stage the cornea might show diffuse oedema and have a decreased intra-ocular pressure. More posterior inflammation of the choroid may also involve the retina, causing chorioretinitis, in which the optic nerve may also show hyperaemia or atrophy and vitreal alterations might also be present. In more chronic cases, there are further changes in the lens and endothelium so secondary cataracts, glaucoma, synechiae, retinal detachment or blindness are possible (Lassaline, 2016). Horses with uveitis usually present with closed eye or photophobia (Dwyer, 2021)

Uveitis might occur secondary to ocular diseases or traumas, such as blunt or penetrating ocular trauma, corneal ulceration, neoplasia, lens rupture, cataracts, or parasitic infections. It might also be caused by other systemic diseases, namely leptospirosis or septicaemia in foals, among others (Lassaline, 2016). It is important to take a thorough history and perform a full clinical examination, assessing if there is any indication of trauma or other concomitant diseases. Then a fluorescein staining should be performed to check if there is ulceration. The horse should be given 1,1 mg/kg of flunixin meglumine IV, and topical atropine for the pain and to reduce the risk of adhesions, respectively. Corticosteroids can be given if there is no sign of ulceration. These horses should be placed in a dark stable and be reassessed frequently (Archer, 2013; Dwyer, 2021).

Equine recurrent uveitis (ERU) is considered the leading cause of blindness in the horse. Usually it is related to the deposition of immune complexes in *Leptospira* spp. infected horses. However, in Appaloosas, a genetic association has been found (Allbaugh, 2017; McMullen and Fischer, 2017).

When episodes occur, they should be treated with topical corticosteroids or non-steroid inflammatory drugs, topical atropine, and systemic anti-inflammatories (Lassaline, 2016; Allbaugh, 2017). The medical treatment should be maintained in the two to four weeks following the episode, and dosage should be slowly tapered (Allbaugh, 2017). There are several surgical options to treat ERU, suprachoroidal cyclosporine implantation, which has been found to decrease the regularity and severity of episodes, or *Pars plana* vitrectomy. In some cases, enucleation is necessary (Townsend, 2019).

Distichiasis

Distichiasis is a condition where aberrant cilia emerge from the eyelid margin. Sometimes the cilia are soft and fine and do not cause discomfort, but other times they are rigid and pointing towards the cornea, and therefore cause corneal irritation or even ulceration. The horses may present with recurrent non-healing corneal ulcerations. This disease is relatively common in Friesian horses, when compared to other breeds, and it is thought to be inheritable (Utter and Wotman, 2011). The cilia may be removed through manual epilation; however, re-incidence is common. Treatments with electrocautery, cryotherapy and partial tarsal plate excision have been described in horses, although the latter usually leaves a significant amount of scar tissue. Electrocautery seems to be a good treatment option as it is inexpensive, and it can be performed in the standing horse. However, there is still a chance of recurrence as cilia from a different follicle, or even the same one, may arise (Hermans and Ensink, 2013; Utter and Wotman, 2011).

Endocrinology

All horses presenting endocrine dysfunction were diagnosed with *Pituitary pars* intermedia dysfunction (PPID) or Cushing disease. And, in fact, it is considered the most prevalent endocrine disease in aged horses (Durham *et al.*, 2014).

Affected horses may develop subtle hair coat changes in the earlier stages of the disease that later turns into hypertrichosis, this may be localized or generalized, as illustrated on Figure 7, they may lose muscle mass, have adipose tissue deposits in certain areas like the necks or the base of the tail and may be lethargic. PPID is more common in older mares, and it can manifest as fertility problems or pseudo lactation (McFarlane, 2011). PPID may cause insulin dysregulation, thus becoming a risk factor for the development of laminitis. Polyuria/polydipsia, excessive sweating, and opportunistic infections due to immunosuppression have also been described. Frequently the owners postpone calling the veterinarian, as they consider part of the clinical signs to be normal in an aged horse (McFarlane, 2011; Mcgowan, Pinchbeck and Mcgowan, 2012; Frank, 2015;).

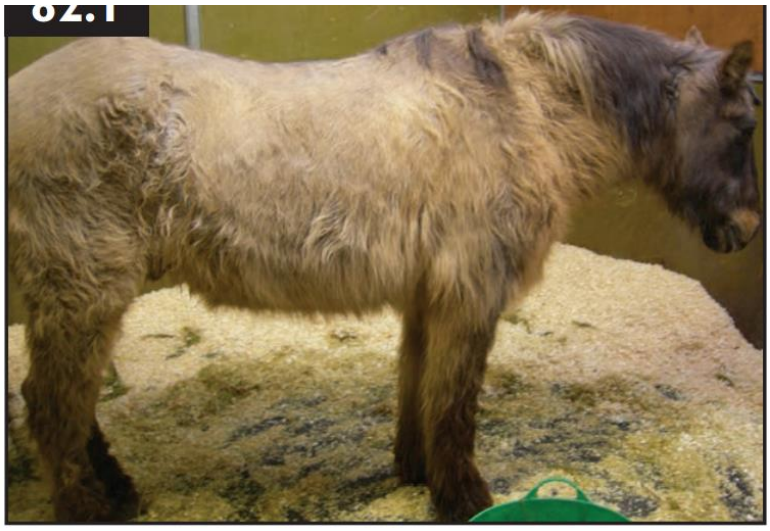
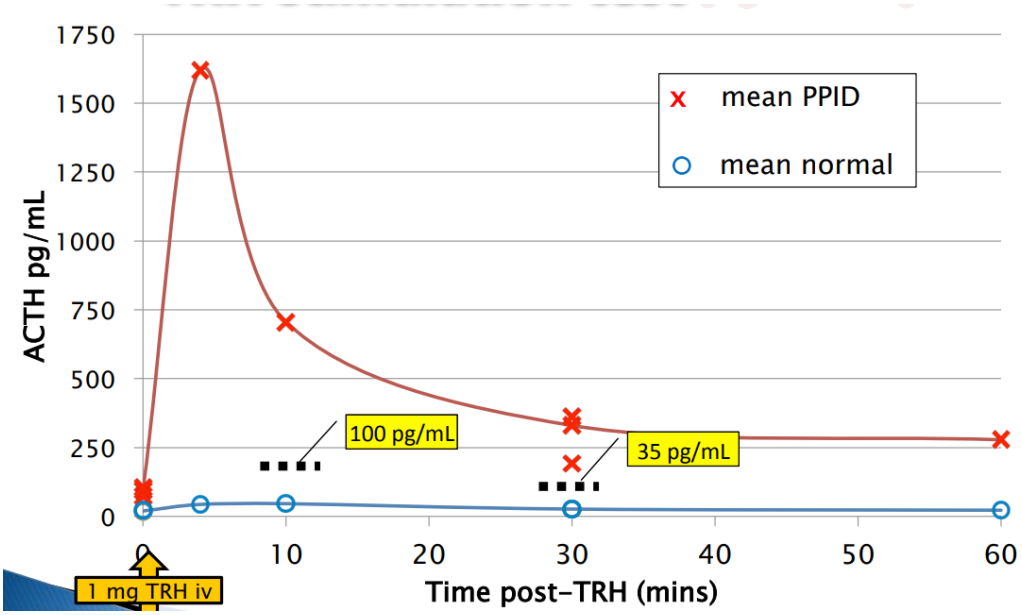


Figure 7 - Twenty-year old pony gelding suffering from PPID showing generalized hypertrichosis, adapted from (Mair and Divers, 2016)

Plasmatic adrenocorticotropic hormone (ACTH) concentration may be measured as a screening test for PPID, or an overnight dexamethasone suppression test of cortisol could be performed. However, these are not very sensitive tests as sometimes horses, especially in earlier stages of disease, have values within range. A thyrotropin-releasing hormone (TRH) stimulation test is more sensitive and should be performed if the ACTH levels are normal. In Graphic 2, the different response between a healthy and a PPID horse to a TRH stimulation test is represented. The range of acceptable ACTH values should be adjusted according to the season, time of the day, if

the horse is in pain or under stress. When stimulated, healthy TRH receptors in the melanotrophs of the *pars intermedia* would release α-melanocyte stimulating hormone, corticotrophin-like intermediate peptide and β-endorphin, in diseased animals they additionally secret large amounts of ACTH (McFarlane, 2011; Durham et al., 2014; Frank, 2015).



Graphic 2 - Comparison of the results of a TRH stimulation test in healthy and diseased horses, adapted from (Durham, 2012)

This condition is primarily managed pharmacologically. Firstly, a dose of 2 µg/kg of pergolide mesylate is tried and the horse is evaluated after 28 days, to see if the dose is appropriate or if it should be increased, until an effective dose is found. Pergolide mesylate is a dopamine receptor agonist, which should restore dopaminergic inhibition of the melanotrophs. The effective dose of Pergolide mesylate is difficult to predict, so therapy should be adjusted considering the animal's clinical evolution. The maximal dosage is at 10 µg/kg daily, when the dosage reaches 6 µg/kg, cyproheptadine (0,25 mg/kg) may be associated. The latter drug is a serotonin antagonist that stimulates the melanotrophs in the *pars intermedia*. In addition to the pharmacologic therapy, the horse will need body clipping, frequent hoof care, regular deworming, dentistry care, and in cases of insulin dysregulation, diet management. It is of paramount importance to document the horse's clinical status, evolution, the treatments performed and to monitor the horse closely (Durham *et al.*, 2014; Frank, 2015).

Neurology

The observed neurology caseload was as demonstrated on table 15. Ten horses presented with ataxia, one presented with a bacterial encephalitis and a horse with infected with West Nile Virus, also two mares presented with a complaint of headshaking.

Table 15 - Neurology Casuistic

Cases	Total
Infectious	2
Spinal ataxia	10
Headshaking	2

Spinal Ataxia

Ataxia can be classified as to its origin as vestibular, cerebellar, or spinal/sensory (Murr and Reed, 2015). Considering the clinical signs, neurologic examination, and history of the horses that presented during the externship with a complaint of ataxia, they were classified as spinal. The diagnosed causes for ataxia were trauma, cervical vertebral stenotic myelopathy, cervical arthritis, and discospondylitis, in one horse a diagnosis was not reached.

In the evaluation of the neurologic system, it is important to first evaluate the level of mentation, head, and body posture, overall behaviour, and interaction pf the animal with the environment. Then, the cranial nerve function should be assessed followed by the evaluation of muscle development, tone, and spinal reflexes, if possible, evaluate and grade the gait abnormalities. An example of grading system is presented in Table 16. Other causes of gait abnormalities should be ruled out, such as lameness or myopathies. for diagnosis it is also very important to consider the relation between the onset of clinical signs and their progression (Archer, 2013; Murr and Reed, 2015).

Table 16 - Grading of gait abnormalities, adapted from (Murr e Reed, 2015).

Grade	Description
0	No abnormalities visible
1	Detectable, but not very evident at normal gate, but worsen when backed, turned, with neck extension and pressure is applied on the hindlimbs.
2	More noticeable at walk, worsens in the same conditions as in grade 1
3	Very obvious at walk, when backed, turned, with neck extension or pressure on hindlimbs falls or buckles
4	Stumbling and falling spontaneously
5	Recumbency

The lesion should be localized, considering the clinical presentation of the horse. First it should be determined if it is a spinal cord/ upper motor neuron injury or if it is a peripheral nerve/ lower motor neuron injury these may be distinguished from lower motor neuron as shown on Table 17. The spinal cord segment or peripheral nerve where the lesion has occurred should be identified (Murr and Reed, 2015). The clinical signs associated with lesions in the different spinal cord segments are presented on Figure 8.

Table 17 - Clinical signs differentiating lower and upper motor neuron injuries adapted from (Murr and Reed, 2015)

LMN weakness	UMN weakness
Flaccid	Spasticity
Decreased tone	Increased tone
Diminished reflex response	Exaggerated reflex response
Profound muscle atrophy	Minimal muscle atrophy
Fasciculations present	Fasciculations absent

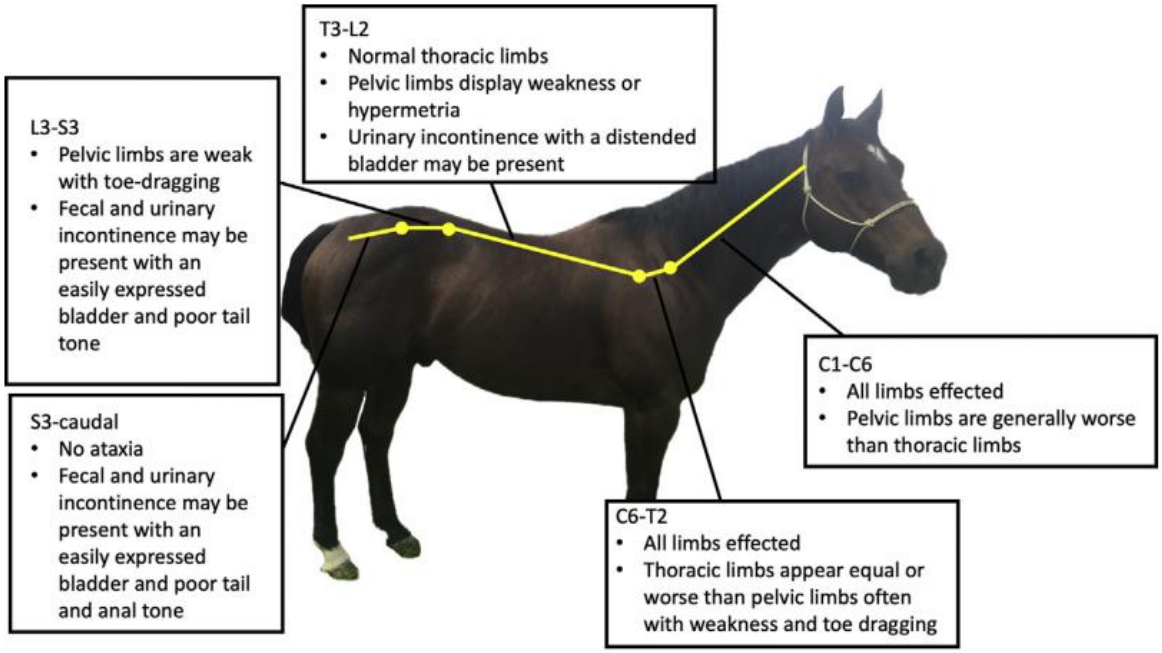


Figure 8 - Clinical manifestation of spinal cord injuries adapted from (Estell, 2021)

Intoxication should also be considered as differential diagnosis, even though it is not a very common cause of spinal ataxia. Several imaging techniques can be helpful in diagnosing the primary cause, such as, radiography for assessing the bone or a myelography if there is a suspect of spinal compression. If it is not diagnostic, then magnetic resonance imaging, computed tomography or bone scintigraphy may be of use (Furr and Reed, 2015).

One- to two-year-old horses presenting neurologic signs should be first tested for EHV. Usually, these horses also present other systemic signs, such as pyrexia, lethargy and anorexia, or there is a history of respiratory disease or abortion in the stables. The horses should be isolated and tested. As previously mentioned, hospitals may be an important source of infection of EHV, thus the need of biosecurity and infection control protocols. The broad aims of such policies are to stop the infection from installing in the population or to contain its spread. Examples of measures that could be implemented are having quarantine areas in separate buildings from the main stables, the limitation of the number of people that may contact the horses, using gloves and sanitizing the equipment between uses, and have written and oral communication of the safety measures to the personnel and visitors (Traub-Dargatz et al., 2004; Weese, 2014; Vandenberghe et al., 2021).

Cervical vertebral stenotic myelopathy

One of the diagnosed causes of ataxia, during the externship, was cervical vertebral stenotic myelopathy or Wobbler syndrome. This disease is known to be the most frequent cause of non-infectious spinal ataxia, especially in Thoroughbreds, Quarter horses and Warmbloods. The breeding of these animals should be thoughtful, as it appears to be a genetic component to this disease (Falco, Whitwell, and Palmer, 1976; Johnson and Reed, 2015).

There are two recognized types of presentation of the disease, both are caused by morphological abnormalities in the vertebrae that induce spinal cord compression through stenosis of the vertebral canal. Type I is usually diagnosed in younger horses, it may be inheritable, but several factors are involved in the development of disease such as history of trauma, growth rate or nutrition. It usually affects a more cranial aspect of the neck (C2–C3 to C3–C4). Type II affects older horses and is related osteoarthritic changes in the articular facets. Clinically the horses present clinical signs of upper motor neuron dysfunction that may vary according to the site of compression. Cerebrospinal fluid analysis may aid the diagnosis of cervical compression and ruling out differential diagnosis (Johnson and Reed, 2015).

Simple radiographies may be performed from a latero-lateral projection. Horses may show bony malformations or articular disease of the cervical vertebrae but the most sensitive and specific way of evaluating compressive lesions is considering the ratio of intervertebral minimal sagittal diameter to sagittal height of the cranial aspect of vertebral body, this should not be smaller than 50%. A schematic representation is shown on Figure 9 (Hudson and Mayhew, 2005).

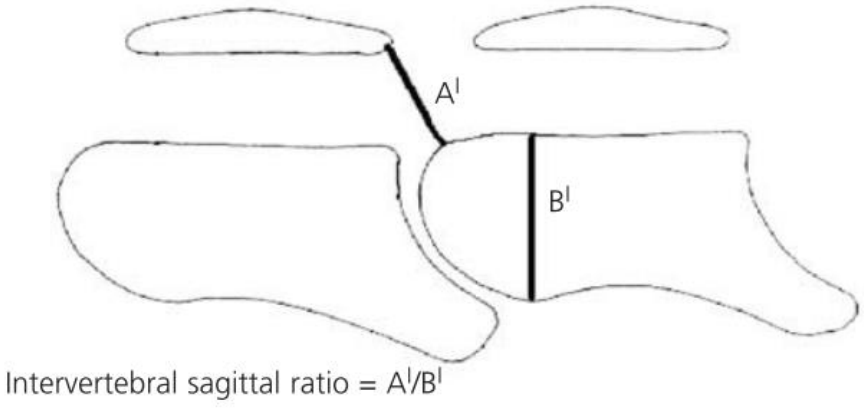


Figure 9 - Intervertebral sagittal ratio anatomic references adapted from (Hudson and Mayhew, 2005)

A myelography is necessary to confirm the diagnosis as usually simple radiographies might not be conclusive. Myelography allows the diagnosis of both static and dynamic compression. Radiographs are taken with the neck in several positions, static compressions are usually found on C5-C6 or C6-C7, and are visible with the neck in neutral positions, while dynamic compressions are more cranial (C3-C4 or C4-C5) and seen with neck flexion. Compressions may be diagnosed with the use of several criteria, one of the most used is the minimal sagittal dural diameter that evaluates the sagittal diameter of the intravertebral sagittal space, a decrease of at least 20% comparatively to the largest diameter is suggestive of compression. Contrast enhanced computer tomography is more accurate than myelography, and therefore may be of use in horses before surgery (Hudson e Mayhew, 2005; Johnson e Reed, 2015; Kondo *et al.*, 2022).

The choice of medical treatment depends on the severity, nature and chronicity of clinical signs, and age of the horse. Mostly, it is aimed at reducing oedema formation, so, non-steroidal inflammatory drugs and dimethyl sulfoxide are frequently used. In young horses, limited exercise and restriction in protein and carbohydrates in the diet may help slow the development of disease. In older horses additional local joint treatment with corticosteroids or hyaluronate sodium may be useful, especially in those who do not show evident neurologic deficiencies (grade zero or two, according to the classification suggested in Table 16) (Johnson and Reed, 2015; Szklarz et al., 2018). Surgical treatment may be recommended in young horses with few myelographic lesions, mild clinical signs, and no concurrent diseases. The success of the intervention may take six months to a year to be determined, but the success rate is estimated at 60% (Fürst, 2019). During the externship, all the horses diagnosed with cervical vertebral stenotic myelopathy (n=3) were young horses (younger than four years) and were euthanized as the owners chose not to pursue treatment.

Orthopaedics

Musculoskeletal problems were the most frequently observed during the externship period (37,8%) as previously mentioned in Graphic 1. The casuistic is summarized in Table 18. The most frequently observed consults were PPE and owner request evaluations of young horses before the initiation of training (n=43).

Table 18 - Orthopaedics Casuistic

Cases	Total
Pre-purchase examination + vetting	43
Back pain	37
Fractures	16
Muscular disease	4
Hoof infections	5
Hoof neoplasia	2
Arthropathy	21
Juvenile osteochondral conditions	35
Laminitis	6
Deep digital flexor tendon lesion	16
Suspensory lesion	17
Superficial digital flexor tendon lesion	2
Flexural deformities	10
Hip luxation	1
Sidebone	3
Bucked shins	2
Navicular disease	7
Ligament injury	8

In both hospitals the orthopaedic consults started with a review of the horse's history and complaint with the owner, then there was a static evaluation of the horse where the animal's attitude, posture, and conformation were observed as well as the presence of tumefactions or wounds, at this time hoof testers may be used as well to detect any pain in this area. Then either an assistant or the owner were asked to walk and then trot the horse several times in a straight line, while the veterinarian observes from different perspectives, afterwards the horse was lunged in both hard and soft surfaces in different gaits. Then flexion tests were performed again to narrow the area where lesion was suspected. In Dierenkliniek Wolvega, at this point imaging studies of the area where lesion was thought to be most probable were performed, usually x-ray evaluation was done and if necessary, ultrasound evaluation of the area was done as well. If a clear primary

lesion was not found or if there were still questions about the source of pain, then perineural or articular blocks were performed to localize the exact anatomic source of lesion and then a MRI evaluation was recommended to reach a final diagnosis. In Equitom, the blocks were performed to localize the painful area, prioritizing the areas where lesion was suspected, and only then imaging studies were performed, if these were not conclusive then more advanced imaging modalities were advised such as bone scintigraphy, MRI or CT. In both hospitals, after a diagnosis was reached a treatment and rehabilitation plan was discussed with the owner.

Juvenile osteochondral conditions

Osteochondrosis (OC) is one of the most important issues affecting sport horses in Europe, as it is a common lesion among young horses. It has a heritable component and so should be taken in consideration when breeding the affected horses (Weeren and Jeffcott, 2013). It affects the growing cartilage of predisposed sites. It may be described as a focal disturbance of endochondral ossification, the abnormal cartilage may present as thickened, collapsed, or develop into osteochondral fragments that separate from the subchondral bone leading to *Osteochondrosis dissecans* (OCD). Although it has a genetic component to it, other factors have been recognized in its aetiology, such as diet, rapid growth rate, conformation, trauma, or diet (Ytrehus, Carlson and Ekman, 2007; Olstad, Ekman and Carlson, 2015; Semevolos, 2017; McIlwraith, 2020).

Subchondral bone cysts or subchondral cystic lesions, in young horses, are also considered clinical manifestations of OC. In the 35 horses presented, seven had endochondral bone lesions, that were found on the first phalange (n=3) and in the medial condyle of the femur (n=4).

The horse may show clinical signs, radiographic evidence of disease, or both. OC is commonly diagnosed in animals with no clinical signs of radiographic examinations as part of pre-purchase examinations (Semevolos, 2017; McIlwraith, 2020).

OC usually affects the articular cartilage and may affect the subchondral bone it is thought that clinical signs develop when the joint surface is dissected by lesions. A failure of cartilage canal blood vessels causes ischemic lesions of the chondrocytes that they supply (*osteochondrosis latens*), that if not spontaneously solved, will persist as an area of necrotic cartilage surrounded by bone tissue (*osteochondrosis manifesta*), when the lesion is surrounded by bone it is turned into fibrous tissue may suffer membranous ossification, if not, it may develop disease (*osteochondrosis dissecans*) (Ytrehus, Carlson and Ekman, 2007). The OCD fragments may then detach or remain loosely attached to its origin, at this moment is when the inflammation of the synovial structures starts to cause the pain and lameness associated with the disease, which may later progress to osteoarthritis. The defects may affect one or more joints, the most injured joints are the femeropatellar, tarsocrural and matacarpophalageal/ metatarsophalangeal joints. The severity of clinical signs and the prognosis following surgery depend on the location of lesions. The lesions of OCD may be diagnosed via radiology or ultrasound, not only the lesions should be

located but the condition of the underlying cartilage should be assessed (Weeren and Jeffcott, 2013; Olstad, Ekman and Carlson, 2015; McIlwraith, 2020; Hoey et al., 2022).

Conservative management of OC lesions involve decreasing the horse's activity level, managing the horse's diet, administration of non-steroidal anti-inflammatory drugs, and intra-articular hyaluronic acid. The OCD fragments should be removed via arthroscopy, and the lesions should be debrided. (McIlwraith, 2013; Ortved, 2017). OCD should not be surgical addressed before eight months of age since the fragments still may be absorbed before this age (Weeren and Olstad, 2015). In some breeds, like the Lusitano horse, osteochondritic lesions may be reabsorbed until the twelve months of age, so surgical intervention should only be performed after that time (Baccarin *et al.*, 2012).

Subchondral bone cysts may be diagnosed on radiographies as well defined, round, radiolucent lesions or as dome-shaped radiolucent areas merging with a flattened joint surface, sometimes computed tomography may be of help in defining these lesions. The lesions may be treated with triamcinolone acetonide intralesionally, systemic anti-inflammatories and rest. Surgical debridement, or lag screw fixation are also possible therapeutic options (McIlwraith, 2013; Weeren and Olstad, 2015; Ortved, 2017; Klein et al., 2022).

Other conditions

There were other conditions observed during the externships that were not included in the medical areas previously described and are summarized in Table 19. There were four horses presented for surgery with complaints of hernias, one was a three-year-old Friesian mare with an abdominal hernia, the other three were foals, older than four months, with umbilical hernias. In the remaining conditions (n=4) a final diagnosis was not achieved.

Table 19 - Other Conditions

Cases	Total
Hernias	4
Idiopathic fever	3
General weakness	1

Atypical myopathy

Cases of Atypical myopathy (AM) have been reported since the 1930s in various parts of the world, but most commonly in Europe (Votion et al., 2004, 2020). Recently, information on the distribution of notified cases of AM from 2006 to 2019 was gathered and the results are shown in Figure 10 (Votion *et al.*, 2020). It appears that the number of outbreaks has been increasing in the last years (Votion et al., 2014; Naylor, 2016). In the United States, a condition has been described, with similar clinical presentation and biochemical profiles, called “Seasonal pasture myopathy”, thus appearing to have a similar underlying pathogenesis (Valberg et al., 2013; Durham, 2014; Votion et al., 2014). In the first reports, the condition was known as Atypical myoglobinuria, however, since myoglobinuria is only one of the present clinical signs, AM is now the preferred designation (Votion et al., 2004).

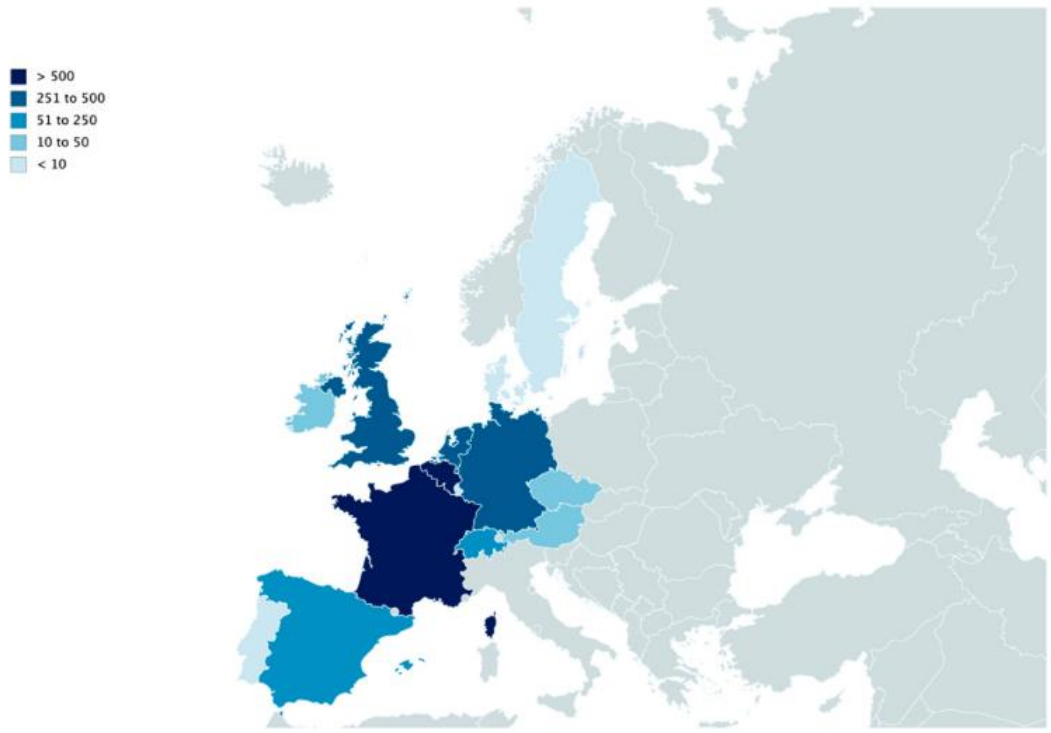


Figure 10 - Distribution of AM cases throughout Europe (Votion et al., 2020).

Aetiology

Equine Atypical Myopathy, in Europe, is believed to be a result of the ingestion of samaras and seedlings of the sycamore maple tree (Acer pseudoplatanus), which is a part of the Sapindaceae family. Hypoglycin A (HGA) was found present in the leaves, seeds, and seedlings of these species (Gillman, Hegeman and Sharp, 2014; Votion et al., 2014). An illustration of the leaves, flowers, and samaras of A. platanus can be seen on Figure 11 (Renaud *et al.*, 2019). Other members of this family are known to cause Hypoglycin intoxications in several species, such as the Ackee fruit (Blighia sapida), that when eaten unripe causes Jamaican vomiting sickness in Humans (Jordan and Burrows, 1937). Furthermore, A. *pseudoplatanus* has been found in or around pastures where the disease has occurred (Votion et al., 2014; Krägeloh et al., 2018).

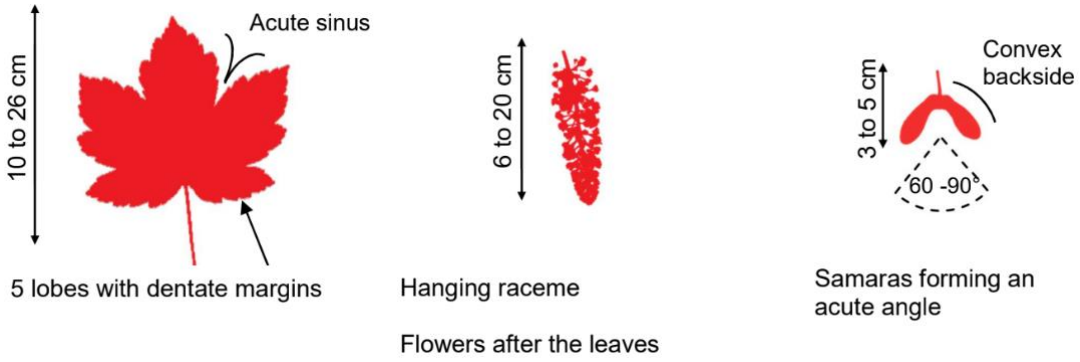


Figure 11 - identification of the leaves, flowers, and seeds of the sycamore maple tree, adapted (Renaud *et al.*, 2019)

HGA is a water-soluble amino acid, its function is mostly unknown but it is thought to be an important part of the plant's defence mechanism. After ingestion and metabolic activation, it becomes an inhibitor of acyl-CoA dehydrogenase, which plays a significant role in fatty acid metabolism (Gillman, Hegeman and Sharp, 2014; Votion *et al.*, 2014; Watson, 2020). HGA is metabolized by transamination and oxidative decarboxylation to the toxic compound methylene cyclopropyl acetic acid (MCPA) in the liver, and then conjugated and excreted in the urine (Bochnia *et al.*, 2015). Moreover, in 2014 Votion *et al.* concluded that the presence of serum MCPA-carnitine, and other MCPA compounds, were a consistent feature in AM diseased animals, and it was not found in diseased controls (Votion *et al.*, 2014). Currently, it is accepted that to develop AM the ingestion and absorption of HGA, followed by metabolism to MCPA-CoA is necessary (Bochnia *et al.*, 2015). In 2019, Bochnia and associates concluded that methyl-enecyclopropylglycine (MCPG), a metabolite analogous to HGA, found in several plants of the Sapindaceae family, and its metabolites could also be responsible for the induction of the disease

when metabolized in methyl enecyl cyclopropyl formyl (MCPF) – coenzyme A (Bochnia *et al.*, 2019).

The large outbreaks frequently happen during the autumn, when there are samaras present in the fields, and then, on the following spring likely due to the ingestion of seedlings. During the spring there are usually fewer cases reported, despite the seedlings still having HGA. This probably occurs because the pastures are of better quality, so the animals ingest less toxic material. Additionally, the availability of feed might lessen the poisonous effects of HGA. Since HGA is water-soluble, contaminated water may also be a source of intoxication (Galen, van et al., 2012; Baise et al., 2016; Votion et al., 2019).

The box elder tree (*Acer negundo*) is another source of HGA intoxication and the main cause of Seasonal pasture myopathy in the United States, a syndrome similar to AM. Even though it is may be present in Europe it is rarely cause of disease. This may be explained by the fact that it is a dioecious tree. Since in Europe it is usually appears as single ornamental trees, they have no seed, hence no HGA content (Sponseller *et al.*, 2012; Votion *et al.*, 2019).

Other maple trees such as the Norway maple (Acer platanoides) and the field maple Acer campestre) were found in sites with A. *platanus*, however, these are considered harmless to horses (Westermann *et al.*, 2016).

Epidemiology

Most reported cases occur in Autumn, and then again, but in fewer numbers, in the following spring. A link between weather conditions and the occurrence of outbreaks has been reported, namely: lack of solar radiation, stormy weather, including winds, rain, and thunderstorms. It has also been reported that the outbreaks stop following heavy frost (van Galen et al., 2012).

Outbreaks occur in sparse pastures, containing or surrounded by trees, most specifically those of the *Acer* genus, and with dead woods or leaves (van Galen et al., 2012). Although the number of sycamore trees has not significantly changed, the number of cases reported has increased. This fact may be explained by an increase in awareness and disease reporting, but it may also be due to a real increase in incidence (Naylor, 2016). It is possible that the climatic changes have affected several characteristics of the aetiologic agent, such as, the *Acer pseudoplatanus* fecundity, the concentration of toxins in the seeds and seedlings of the plant, or the dispersion of toxic materials (Votion *et al.*, 2014). It has also been suggested that the presence of certain funguses or bacteria in association with the plant may increase the morbidity of the disease (Kolk *et al.*, 2013; Naylor, 2016).

It can affect horses in any age group, but it affects mostly young horses (younger than three years old). It has been suggested that the overrepresentation of young horses is due to the lack of selectivity in grazing, and because it is more likely to have younger horses on pastures for longer

periods of time (Galen et al., 2012; Watson, 2020). The grazing behaviour of horses towards A. *pseudoplatanus* was studied, and it showed that the animals tend to avoid materials with a higher phenolic compound's concentration. It is important to note that the horses analysed had a median age of 11 years-old and this might be a learned behaviour (Aboling, Scharmann and Bunzel, 2020). There is also an apparent predisposition in females, which may also be related to the fact that it is more likely for these animals to be turned out. In addition, it has been reported that horses with poor body condition and that have not received regular prophylactic measures, such as deworming, or vaccination have a higher risk of presenting this condition (Durham, 2014). AM results from a muscle energetics imbalance, so it makes sense that horses with less energetic reserves or animals with higher energetic demands, such as growing horses appear to be more sensitive to the toxin (Boemer et al., 2017).

It is important to highlight that the horses affected kept on pastures have not been exercised prior or at the time of the onset of disease (Cassart *et al.*, 2007), ruling it out an exertional myopathy.

The number of horses affected in an outbreak is variable, however, the cases usually occur within a few days of each other and on the same premises or close by. Recurrence in the same area in the following years has also been reported (Durham, 2014). It is important to mention that not all horses grazing in the same fields show a clinical manifestation of the disease, which can be explained by an uneven distribution of toxic materials, resulting in different levels of toxin ingestion. Although it has been shown that the intestinal microbiota has an effect on digestion, metabolism, and immune responses, which could help protect horses from disease. It is not known if there are any intrinsic resistance mechanisms in the horse (Durham, 2014; Wimmer-Scherr *et al.*, 2021; Wouters *et al.*, 2021).

Pathophysiology

AM can be characterized as an acquired multiple acyl-CoA dehydrogenase deficiency (MADD), which affects mainly mitochondrial fatty acid metabolism. Therefore, these horses depend mostly on carbohydrate metabolism for energy (Westermann *et al.*, 2008).

Type I muscle fibres are the most affected. These, opposed to type II muscle fibres are predominantly in slow-twitch muscles, responsible for respiration, stance, and posture, which explains that the horses present mostly symptoms of general weakness and sometimes dyspnoea. Under microscopic examination the injured muscle fibres showed an accumulation of lipids around the myocytes, reflecting the inability to metabolize lipids. (Cassart *et al.*, 2007; Watson, 2020).

As previously mentioned Hypoglycin A is metabolized into MCPA, which then binds to acyl-CoA dehydrogenases, inhibiting long acid chain metabolism. The diversity of clinical presentations in

co-grazing horses can be explained not only by the different doses of ingested toxin but also by the bioavailability. Both HGA and MCPA- conjugate blood levels were lower in the animals that did not show clinical manifestation of disease. It is important to note that this difference was seen more pronouncedly in metabolite conjugates than in the levels of HGA. This suggests that different rates of hepatic metabolism may translate into different severity of clinical signs. The availability of co-factors, like Magnesium, pyridoxal phosphate, thiamine, and co-enzyme A, may also alter the extent of metabolism of HGA, reflecting individual susceptibility to disease. Other dietary factors may limit toxin absorption, as it was seen that animals who were fed supplementary sources of feed had a lower incidence of clinical disease (van Galen et al.,2012; Valberg et al., 2013; Bochnia et al., 2015; Naylor, 2016). HGA and MCPG may be further metabolized through several specific enzymes, like branch-chain oxoacid dehydrogenases and branched-chain amino acid transferases found in some bacteria usually present in the horse's gut microbiome. This suggests that the horse's intestinal flora may have an important role in the development of disease (Wimmer-Scherr *et al.*, 2021).

It is also known that different seeds have different levels of HGA, even if they are from the same tree (Gillman, Hegeman and Sharp, 2014). It has been suggested that HGA levels and seed load may be a response to tree stress. Also, it has been hypothesized that the infection of the tree with the fungus *Rhytisma acerinum*, may have a role in the disease (van der Kolk et al., 2013; Gillman, Hegeman and Sharp, 2014).

The poisoned animals show signs of multiple acyl-coA dehydrogenase deficiency (MADD), and the interruption of these specific metabolic processes shows abnormal biochemical profiles, due to the accumulation of certain metabolites. The acylcarnitine profiles in urine and serum show a distinct increase in free carnitine and in short and medium acylcarnitine chains and mild increases in long-chain acylcarnitines. Urine organic acid profiles show elevated levels of ethylmalonic and 2-methyl succinate along with high glycine conjugates (Westermann et al., 2008; Votion et al., 2014).

When HGA and MCPG are metabolized into MCPA-CoA and MCPF-CoA, respectively, inhibit steps of beta-oxidation of lipids. Short-chain and medium-chain acyl-CoA dehydrogenases are enzymes needed for the first steps of beta-oxidation and are heavily inhibited. There are other inactivated enzymes that are important to lipid metabolism, namely enoyl-CoA hydratases and oxoacyl-coA thiolase, along with the dehydrogenases involved in the degradation of branched-chain amino acids. These selective inhibitions are reflected in the accumulation of acylcarnitines, which may suggest the severity of the disease (Bochnia *et al.*, 2019; Wouters *et al.*, 2021).

AM resembles a genetic disease found in Humans that also causes multiple acyl CoA dehydrogenase deficiency known as Glutaric aciduria type II. This disease causes mutations in the electron transport flavoprotein (ETF) system (Ali *et al.*, 2021). The ETF system serves as an electron acceptor for several flavin adenine dinucleotide (FAD) dehydrogenases, which are vital

for fatty acid beta-oxidation. However, in AM the ETF system appears to be normal and the FAD dehydrogenase activity is reduced. This happens because of the action of MCPA (Karlíková *et al.*, 2016) .

The most important pathophysiologic features of HGA poisoning are a result of the inability of the muscles to use lipids as a primary source of energy, and the increase of glycolysis (Cassart *et al.*, 2007; Karlíková *et al.*, 2016; Lemieux *et al.*, 2016).

There are reports of AM in newborn foals even though they are not consuming pasture or seeds. In one of the cases, the mother was diagnosed in the sixth month of gestation and at the time of delivery, the mother was grazing in a possibly contaminated pasture, even though she did not show clinical signs. It is unsure if the foal was affected by placental transfer of toxins or of these were secreted into the dam's milk (Karlíková *et al.*, 2018). HGA and MCPG may be passed through active transport to the foetus, but afterward, it must be converted into the toxic metabolites to cause disease. During the foetal and neonatal period, the enzymes that metabolize these compounds have low activity levels, which means that there is little risk of these foals becoming intoxicated. It is hypothesized that a congenital defect of any of the enzymes involved in beta-oxidation of fatty acids, can induce a certain synergism with the maple toxins, and is therefore responsible for the development of disease (Sander, Terhardt and Janzen, 2021).

Clinical signs and diagnosis

Horses present with a sudden onset of weakness and stiffness, that may progress to recumbency, myoglobinuria, and very elevated levels of creatinine kinase (CK) activity and aspartate aminotransferase (AST), due to severe rhabdomyolysis. Sometimes, right after the onset of clinical signs, the CK activity levels are high but might increase, so we should measure again after a few hours. The AST and the CK activity levels increase is much higher than what is expected of another myopathy, like an exertional myopathy. Low head position and oedema may be observed as well. Rapid progression of the disease may occur in the first three days, with 80-90% of the horses dying in this period, some other cases are found dead in the field (van Galen et al., 2012; Sponseller et al., 2012; Durham, 2014; Watson, 2020). On Figure 12 a sample of a typical urine of a horse suffering with AM, is shown in comparison to the urine of a healthy horse The dark red/ brown colour caused by myoglobinuria should be noted (Witkowska-Piłaszewicz et al., 2019).

Figure 12 - Urine collected from a healthy horse (left) and urine from a horse suffering from AM (right), adapted (Witkowska-Piłaszewicz et al.*, 2019)*

Other clinical signs may be present, such as congested mucous membranes, tremors, sweating, tachycardia, and, on occasion, arrhythmias, also restless behaviour, mild diarrhoea, and oesophageal obstruction. Gastrointestinal impactions, diminished rectal tone, penal prolapse renal failure, fever and lack of appetite may also be present but are rare. They may also show other signs of hepatopathy. Dyspnoea is a poor prognostic indicator as it indicates damage to the intercostal muscles and the diaphragm (van Galen et al., 2012; Durham, 2014).

Further alterations are also commonly found in the blood of affected horses, such as hyperglycaemia, hypertriglyceridemia, and hypocalcaemia. Metabolic acidosis may also be present, and it is a consequence of the increase of lactate and changes in the serum concentrations of electrolytes. The widespread destruction of cells may cause a combination of hyponatremia, hypochloraemia, and hyperkalaemia, which may also be found in these horses, but may also be the result of renal failure, or excessive loss of fluids, highlighting the importance of close monitoring the of horse's clinical status. Hyperglycaemia and hyperlipidaemia may also cause apparent hyponatremia and hypochloraemia. In patients already showing respiratory distress, it is possible to find respiratory acidosis (van Galen et al., 2012, 2013).

The cardiac muscle may also be affected, in these cases, we might find elevations in cardiac troponin I. The elevations in cardiac troponin and ventricular premature depolarizations cause specific changes in the electrocardiography and echocardiography, namely an increase in QT interval and an abnormal systolic motion in the left ventricle. The QT interval is the time between depolarization and repolarization of the ventricles, if the horse presents with tachycardia, the QT interval is naturally shorter, so in these horses a pathologically increased QT interval may appear normal (Verheyen *et al.*, 2012).

Even though there are no pathognomonic biomarkers in this disease, the most reliable ones are the activity of CK, HGA, and MCPG and the presence of their metabolites, along with the acylcarnitine profiles. The organic acid and acylcarnitine profiles tested in human laboratories may be used to diagnose horses, and it was shown that the increase in metabolites is present several days after the onset of the disease. The most useful biomarkers were: ethylmalonic acid, 2-hydroxyglutaric acid, butyryl glycine, and hex- anoylglycine in urine and acylcarnitines C4-C10 in blood. (Bochnia *et al.*, 2015, 2018; Mathis *et al.*, 2021; Wouters *et al.*, 2021). AM horses show increased levels of acylcarnitines and acylglicines, elevated proteinogenic amino acids, such as alanine and aspartate, decreased serum levels of citrate and purines (adenine, adenosine, guanosine, inosine, hypoxanthine, and xanthine) and reduced levels of riboflavin in serum and in urine (Karlíková *et al.*, 2016). The evaluation of MCPG and the MCPF metabolites in urine is sensitive enough that it is relevant in subclinical cases (Bochnia *et al.*, 2019). A rapid test was developed by Sanders and colleagues that would measure the typical metabolites in a single procedure using a serum or urine sample (Sander *et al.*, 2018).

Post-mortem muscle alterations may be found such as necrosis, muscle pallor, or haemorrhage, especially in the diaphragm, intercostal muscles, neck, and shoulder, and even in the myocardium. On Figure 13, part of the myocardium of a two-year-old cross-bred Fjörd horse suffering from AM is shown where an area of *pallor* can be observed. Also, in histopathologic examinations more severe alterations are found in the diaphragm and postural muscles, which are richer in type I fibres (Cassart *et al.*, 2007).

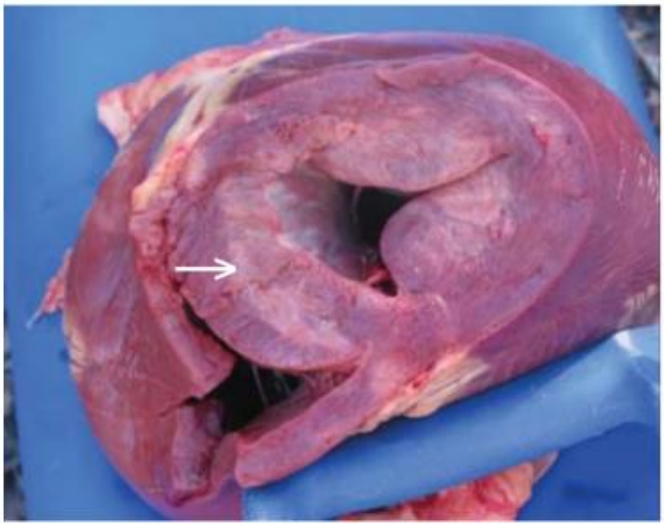


Figure 13 - Myocardium of AM affected horse showing discoloration (white arrow), adapted (Cassart et al., 2007).

The histological characteristic trait of acute *Acer* intoxication is lipid storage myopathy, seen as lipid accumulation in myocytes with Oil red O staining, as shown on Figure 14. The examination may demonstrate lesions compatible with Zenker degeneration, affecting mostly type I muscle

fibres. The histochemical evaluation shows a disorganized NADH tetrazolium reductase staining, the absence of calcium salts precipitates, which is indicative of an intact cytoplasmatic membrane, and a remarkable accumulation of lipid droplets, as shown on Figure 15. It should be noted that despite these alterations the regeneration potential appears to be intact. It is therefore likely that the main lesion is in the mitochondria, suggesting that the disease is the result of a primary mitochondrial failure (Cassart et al., 2007; Van Galen et al., 2012; Bochnia et al., 2019).

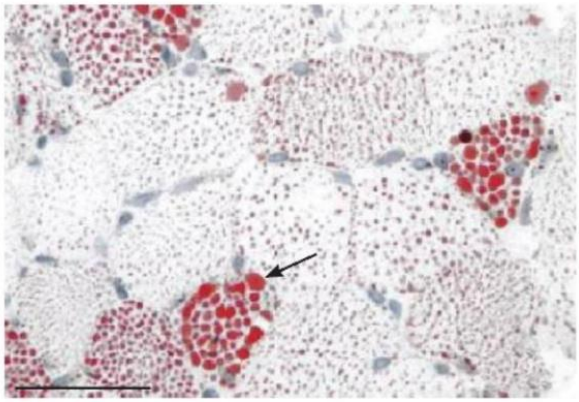


Figure 14 - Oil red O staining of a diaphragm demonstrating lipid accumulation in some fibres (black arrow), (bar=50 µm), adapted from (Haan, 2012)

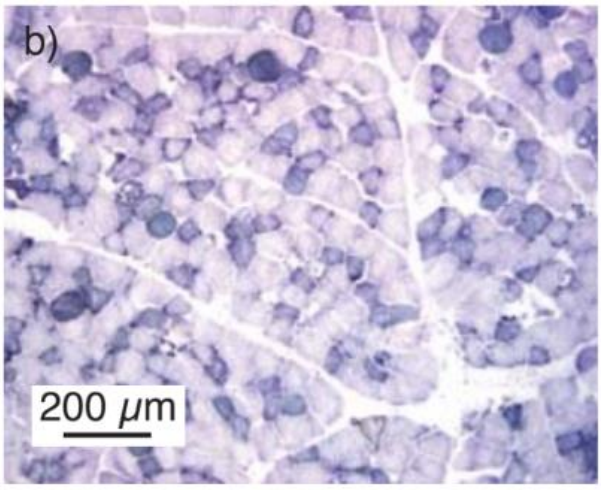


Figure 15 - NADH tetrazolium reductase staining of subscapularis muscle showing an uneven distribution of mitochondrial activity adapted from (Cassart et al., 2007).

Considering the wide variety and inconsistency of clinical signs, the diagnosis may be hard to reach in the field without a detailed history. The most specific clinical signs of this disease are myoglobinuria and the elevations in muscle enzymes. The definitive diagnosis is reached through a muscle biopsy, so that in most cases only a clinical diagnosis is reached, based on history and clinical signs. Although not pathognomonic, the biochemical profiles are highly suggestive (Votion et al., 2004; Van Galen et al., 2012; Votion, 2012; Durham, 2014; Karlíková et al., 2016; Watson, 2020).

Differential diagnosis

AM should be distinguished from other myopathies such as exertional myopathies, nutritional myopathies caused by the deficiency of vitamin E or selenium, polysaccharide storage myopathy in quarter horses, hypokalaemic periodic paresis, post-anaesthetic myopathy or ionophore intoxication. Also, acute clinical presentations should be distinguished from those of colic, endotoxemia or neurologic disorders, like equine grass sickness or tetanus, and other causes of haematuria (Freestone and Carlson, 1991; Votion et al., 2004; van Galen et al., 2012; Valberg, 2018).

Management

Currently, therapy and management of the cases of AM are based on the horse's clinical scenario individually. The treatment is mostly supportive and symptomatic (van Galen et al., 2012; Krägeloh et al., 2018). The main goals of therapy are rehydration and resolution of acid-base and electrolyte imbalances, provision of energy and support of mitochondrial function, removal of toxic metabolites, analgesia, and prevention of further injury and derangements. AM is a very painful disease, where pain management can be very difficult to achieve. This fact allied with the inherent poor prognosis may warrant euthanasia (Durham, 2014; Fabius and Westermann, 2018). If an animal is suspected of acute HGA poisoning, quick veterinarian intervention is of paramount importance. The horse should be moved to a hospital, a stable or another place suitable to receive continued medical attention, be sheltered from the environmental conditions, and be supplied with feed and water (Watson, 2020). If the horse is already recumbent, he should be placed in soft, well-padded bedding and kept warm, by preventing heat loss, instead of actively heating the horse. This is especially important in horses who show cardiovascular compromise as the peripheral vasodilation may further disrupt tissue perfusion. In the early stages, the horse might still be able to get up. Lifting the horse should not be attempted, but he should be turned regularly to prevent pressure wounds (van Galen and Votion, 2013).

The first step of treatment is to rehydrate the animal and maintain a good acid-base and electrolyte balance. The animal's hydration can be assessed through physical examination, packed cell volume, lactate measurement, venous blood gases and electrolytes. Fluid therapy should be provided either intravenously or orally, depending on the horse's dehydration level. Bicarbonate should not be administered, especially if the horse shows dyspnoea, as it will be converted into carbon dioxide (CO_2), which will further increase the partial pressure of carbon dioxide because the horse will not be able to excrete the CO_2 conveniently through the lungs, or if the horse presents hypocalcaemia, because it may worsen the muscle cramping (Corley and

Marr, 1998). A polyionic crystalloid solution, like Ringer's lactate should be administered at a rate of 01-0,2 L/kg in the first 24 hours or enough so the urine clears (Corley and Marr, 1998; van Galen and Votion, 2013).

Myocardium damage is also usually seen in AM horses, so the cardiac function should be closely monitored, through frequent cardiac auscultation, serum troponin I measurements and, if necessary, ECG and echocardiography. If the horse shows persistent tachycardia, pain management should also be reassessed. In cases of myocardial damage and consequent reduced cardiac function, a positive inotrope, such as digoxin (2,2 mg/kg IV) could be administered. If arrhythmias worsen or persist after the electrolyte disturbances are resolved, anti-arrhythmic drugs should be administered (Cassart et al., 2007; Verheyen et al., 2012; van Galen and Votion, 2013).

Nutritional support is important in any critically ill patient but especially in AM where there are deficiencies in energy metabolism. The nutritional status of the patient can be assessed through the levels of triglycerides and glucose in the blood (van Galen and Votion, 2013; Fabius and Westermann, 2018). Fasting should be avoided as it is related to worse clinical outcomes (Dunkel *et al.*, 2020). Since the lipid metabolism is impaired, there is a shift to anaerobic metabolism, so substrate must be provided. Unlike other myopathies, the provided diet should be poor in lipids and rich in carbohydrates. Grass, molasses, carrots, or apples are good options of feed, the horse should still have access to structural fibres, like good quality hay, *ad libitum*. Supplementation of vitamins and antioxidants, such as riboflavin, and carnitine have beneficial effects. Carnitine acts as a detoxifying agent and promotes mitochondrial function, as well as beta-oxidation of fatty acids. Furthermore, it is possible that it enhances the levels of leptin, which activates insulin-sensitive tissues, like the liver, to stimulate glucose uptake. Glycine supplementation may be useful in horses with AM as it helps excrete MCPA (Westermann et al., 2008; van Galen et al., 2012; Fabius and Westermann, 2018). Additionally, an IV solution of glucose/dextrose can be administered if the horse maintains a negative energy balance, it is important to note that these solutions should not be the only source of energy as they may worsen the hyperglycaemia and cause osmotic diuresis. Blood glucose levels should be monitored and, if the horse presents persistent hyperglycaemia, insulin should be administered. In more severe cases the horse the horse can be placed on continuous rate infusion of insulin (0,07 UI/kg/h) as it has a faster onset of action and the plasmatic concentration is more easily controlled, the other option is subcutaneous boluses of protamine zinc insulin (0,1-0,3 IU/Kg BID) that are easier to administer and cheaper, however they take a longer time to take effect and the plasmatic concentrations are more difficult to manage (van Galen and Votion, 2013; Harold McKenzie, 2015; Fabius and Westermann, 2018). The gastrointestinal function should also be closely followed through the horse's appetite, faecal output and its consistency, and by evaluating gut sounds (van Galen and Votion, 2013). Administration of omeprazole or sucralfate may be advised, as pain, the administration of non-steroid inflammatory drugs, anorexia, and stress may increase susceptibility

to the development of gastric ulceration (Fabius and Westermann, 2018). Additionally, horses with AM are more susceptible to haemorrhagic gastroenteritis (Cassart *et al.*, 2007).

The muscular function is monitored through the measurement of CK activity, AST, and lactate dehydrogenase (LDH). CK levels can be hugely elevated without meaning a worse outcome. However, decreasing CK activity levels are a good prognostic sign (Van Galen et al., 2012; van Galen and Votion, 2013). Pain management is one of the main concerns and challenges concerning the management of AM. Therapy with non-steroid anti-inflammatories should not be started until the horse is well hydrated. Meloxicam in a dose of 0,6 mg/kg IV or *PO*, may be the best option. Opioids may be used but are usually not the first choice. Muscle relaxants like acepromazine, benzodiazepines or methocarbamol may be of use since the horses often show stiffness and muscle fasciculations. However, these should be used carefully so the horses do not lay down, as it has been correlated to worse outcomes, also alpha-2-agonists should be avoided because they may cause insulin resistance. Horses present general weakness and may show a lower head position which may cause secondary head oedema, so these horses need to have their head supported. Physiotherapy, if tolerated, may also aid in the horse's recovery. (van Galen et al., 2012; van Galen and Votion, 2013; Fabius and Westermann, 2018)

Urinary issues are also sometimes observed, so urine output, urea levels, signs of dysuria, and hydration status should be frequently monitored. Fluid therapy is of utmost importance, and furosemide or catheterization of the bladder may be necessary, as some horses are not able to urinate.

On admission, some horses present dyspnoea caused by respiratory muscle injury or respiratory alkalosis caused by hyperventilation. Prolonged lateral recumbency, head oedema, and pneumonia aspiration, caused by dysphagia, pain, and stress, may also cause respiratory difficulties. Adequate pain management, proper management of the horse, and adequate symptomatic therapy should resolve the dyspnoea, however intranasal oxygen supplementation or respiratory stimulants, like caffeine or doxapram, may be required. If the dyspnoea is secondary to head oedema an intranasal or intratracheal tube or a tracheostomy may be advantageous. Bilirubin, biliary acids, sorbitol dehydrogenase (SDH), gamma glutamyl-transpeptidase (GGT), and alkaline phosphatase (ALP) are increased in some horses, suggesting liver damage (Westermann et al., 2008). If the liver shows pathological changes, then, the entire pharmacologic regime should be reassessed and adapted. In this case, a solution of dextrose/glucose should be given IV to support metabolism (Cassart et al., 2007; van Galen et al., 2012; Van Galen and Votion, 2013b; Lemieux et al., 2016).

Prognosis

The first 72 hours are the most critical to survival, and horses still alive after five days are likely to recover (Galen et al., 2012). Even though in some horses, recovery can be slow and incomplete, there is not much risk of sequelae (Durham, 2014; Boemer et al., 2017).

Horses that spend more time laying down, regardless of cause, show sweating, anorexia, dyspnoea, and tachycardia usually have a worse chance of surviving, and treatment should be directed firstly at solving these abnormalities (van Galen et al., 2012; Gonzalez-Medina, 2016; Boemer et al., 2017; Fabius and Westermann, 2018;)

Increased CK activity levels, on admission, were not a good survival indicator, because they indicate the amount of muscle damage, and not the type of muscle injured, which appears to be more important. Myocardial damage or damage to the respiratory muscles can mean a worse prognosis, despite less amount of muscle injured (Galen et al., 2012). However, the evolution of CK levels may correlate with the evolution of clinical signs, and therefore with survival (Gonzalez-Medina, 2016; Dunkel et al., 2020). Just like in other myopathies, low levels of calcium may also be related to worse outcomes (Westermann et al., 2008; Boemer et al., 2017). Cardiac troponin I levels are not a good prognosis indicator (Verheyen *et al.*, 2012). Admission levels of glucose are not considered of prognostic value either. Lactate levels are shown to be much higher in horses that do not survive. the hyperlactataemia may be an indicator of anaerobic metabolism (Dunkel *et al.*, 2020; Galen, van *et al.*, 2013). The evolution of the levels of triglycerides is an important predictive factor, as it reflects the success of the nutritional support during hospitalization, and further increases could indicate more severe metabolic (van Galen et al., 2012; Lemieux et al., 2016; Dunkel et al., 2020). A lower body temperature is also found in non-surviving horses, and it may indicate cardiovascular compromise or decreased rectal tone (Westermann et al., 2011; van Galen et al., 2012). Recurrent oesophageal obstruction and the need for sedation were also correlated to worse outcomes (Dunkel *et al.*, 2020).

The variety and relative abundance of specific families of bacteria in the gut microbiome may also be correlated with the worse outcomes, however, it is not clear if these are a consequence of the disease or if they were individual characteristics that may predispose the animal to the disease (Wimmer-Scherr et al., 2021). Certain fatty acid metabolites and acylcarnitine may also have some prognostic potential, namely C2-, C10:2 and C18- carnitines, even though they are not specific diagnostic markers on their own (Boemer et al., 2017; Mathis et al., 2021).

The use of vitamins and antioxidants, such as carnitine, and early hospitalization were correlated with better outcomes (van Galen et al., 2012; Dunkel et al., 2020).

Prevention

To prevent the ingestion of toxic materials, the dead leaves, woods, and sprouts of A. *platanus* should be removed from the fields, and if possible, pastures without many trees around should be used. Trimming the maple trees to avoid the production of flowers and fruits can also be beneficial. During high-risk periods young horses should be stabled or be turned out less time and have supplementary feed. It is also important to ensure that the pastures are not overgrazed (Galen et al., 2012; Krägeloh et al., 2018; Votion et al., 2019). Since the samaras can be spread far from the original tree, it is a good idea to visit pastures in areas close by after stormy weather, and keep these areas fenced off during spring. Also, especially in spring, the horses should not have access to natural courses of water or should not be in fields that favour the accumulation of water, as HGA is water-soluble (Votion *et al.*, 2019). Neither mowing nor herbicide application lowers the concentration of HGA, so the safest option is to burn the contaminated materials, or mechanically remove them. Hay and silage of pastures contaminated with seedlings or leaves of sycamore maples are not safe for horses, as these processes do not lower the concentration of HGA (González-Medina *et al.*, 2019; Votion *et al.*, 2019).

Considering that to develop disease, the HGA and MCPG need to be ingested and absorbed, the administration of activated charcoal might bind the toxins and prevent their absorption, if done right after ingestion. Also, liquid paraffin might diminish the absorption rate of toxin. The administration of these substances could be particularly useful in clinically sound co-grazers, to prevent disease manifestation (Krägeloh *et al.*, 2018).

Clinical case

A one-year-old Frisian stallion was referred to the hospital with a suspected presentation of colic. The owner said that the horse had no previous history of illnesses, and he was fine the day before, when he was placed on the field. This field was surrounded by trees, including maple trees. On arrival, the horse showed severe discomfort, was apathic with a stretched neck posture, but had no evident clinical signs of an acute abdomen. The only alterations found on physical examination were that he showed dark mucous membranes, had a swelling on the scapula and his urine was a dark brown colour. He did not have a fever, a significantly increased heart rhythm or any signs of respiratory distress. Neither did he show specific clinical signs of colic, had evident alterations on rectal palpation, nor had he apparent lesions on abdominal ultrasound.

His bloodwork showed high values for GOT/AST (220 IU/L), LDH (1 290 U/l) and CK, the lactate was two mmol/L and the serum Amyloid A (SAA) value was 450. The CK was around 2 000 000. In the hemogram, there was leucocytosis (15,27x10^9/L) with neutrophilia (12,01X10^9/L).

A strip urine test was made and showed leukocytes, glycosuria, proteinuria, pigmenturia and a pH of 7.

On the following days the horse presented severe hyperglycaemia (117 mmol/L), a slightly decrease in ionized calcium (2,15mmol/L) and sodium (132 mmol/L). Blood gas analysis was performed with a venous blood sample. The blood bicarbonate was slightly decreased (27,6 mmol/L) and there was base excess of 2 mmol/L, which should be between 6,4 to 12,3 in venous samples from a healthy horse, these values suggest that the horse had a slight metabolic acidosis. The blood gas analysis showed a partial saturation of carbon dioxide and oxygen within reference for venous blood (42,5 and 39, respectively) (Soma et al., 1996; Corley and Stephen, 2008).

The most relevant differential diagnosis were: atypical myopathy, white muscle disease and a muscle abscess on the scapular area.

Upon further ultrasonographic evaluation the tumefaction was compatible with a thickening of the longissimus dorsalis muscle, and an abscess was ruled out.

He was placed in a big box, with a thick wood shavings bedding, with food and water available. He was put on IV fluids overnight, was given 15ml of meloxicam IV (60mg/100kg), 5g of ampicillin (1g/100kg BID), vitamin E and selenium. Since he was still painful, he was given a 100 µg fentanyl patch, which also had some muscle relaxant effect, but not enough for the horse to lay down.

Considering, the animal history, clinical signs and CK values of 2 000 000 a presumptive diagnosis of atypical myopathy was made.

The horse started receiving 500ml-1L of a commercial solution that contained B complex vitamins, electrolytes, amino acids, and dextrose, while still receiving around 60L of Ringer's lactate daily, which was then adjusted accordingly, as well as the forementioned medication, except for the ampicillin. Given that the horse did not have a fever and an infectious process was ruled out, the ampicillin was only administered at the time of admission and was then discontinued. He was also supplemented with L-carnitine. After ten days he started to take omeprazole at a dose of 1mg/kg PO to prevent gastric ulceration due to stress, pain and the medication regimen (Sykes *et al.*, 2015). There was always fresh water and roughage available, he was receiving at least 1kg of alfalfa and 2kg of muesli daily. During his hospitalization, the horse maintained good appetite, was able to urinate and passed soft but formed faeces.

As it is common in hospitalized patients, this horse developed thrombophlebitis in his jugular and thoracic veins (Corley and Stephen, 2008). This is a frequent complication associated with prolonged use of IV catheters and the type of material of these catheters, improper placement of such catheters, poor asepsis technique, or administration of irritating substances (Barakzai and Chandler, 2003; Geraghty et al., 2009; Dias and Lacerda Neto, 2013; Schoster and Schoster, 2017). In this case, the horse was on intravenous fluid therapy for several days and receiving intravenous medication, which was then changed to oral administration. Head placement can also be a risk factor to develop jugular thrombophlebitis as it contributes to an abnormal blood flow (Divers, 2003), in this case the horse kept his head lowered most of the time. To decrease the risk of thrombophlebitis an over-the-wire polyurethane catheter with an antibiotic coating should be used (Barakzai and Chandler, 2003). In this horse a 14-gauge over-the-needle polyurethane catheter was first placed on the jugular vein, which was removed after the onset of thrombophlebitis, then a 14-gauge 20 centimetre over-the-wire catheter with an antimicrobial coating was placed on the left thoracic vein, this one also caused thrombophlebitis, so it was replaced by a similar catheter on the other thoracic vein. The horse presented pain on palpation, heat, and tumefaction of the affected veins. The catheter was kept while it was necessary to administer fluids. In AM patients oral fluid therapy should be avoided as nasogastric intubation is contraindicated because it further increases the horse's stress and the rehydration is not as effective as it would be IV (van Galen and Votion, 2013; Fabius and Westermann, 2018). The catheters were flushed at least three times a day with 0,9% saline solution and heparin. The horse was already receiving systemic anti-inflammatory therapy, so he started on a topical cream of heparinoid, to relieve the signs of local inflammation and to help break-down the clot. He had icepacks placed on his front feet twice a day to reduce the risk of developing laminitis as well.

Also, because he had a lower head position, he was attached by the halter, so he did not lie down and had a head support, as shown of Figure 16. He developed skin abrasions in the mandibula, which were addressed with supportive padding between the halter and the skin, daily cleaning of the wounds and application of a topical cream of betamethasone, antibiotics, vitamin A, lidocaine, and chlorhexidine.

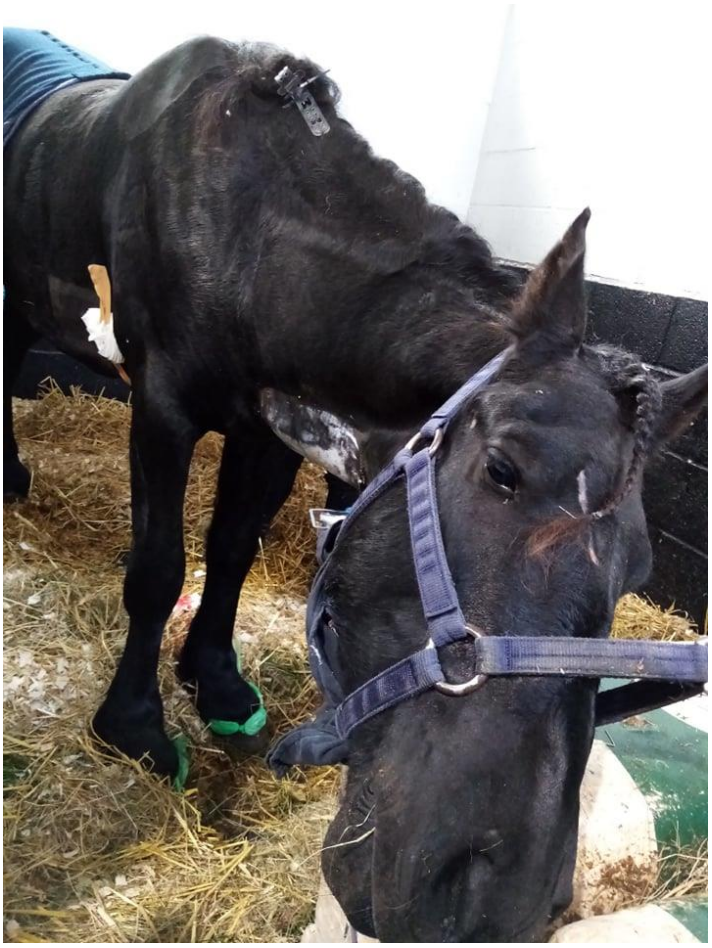
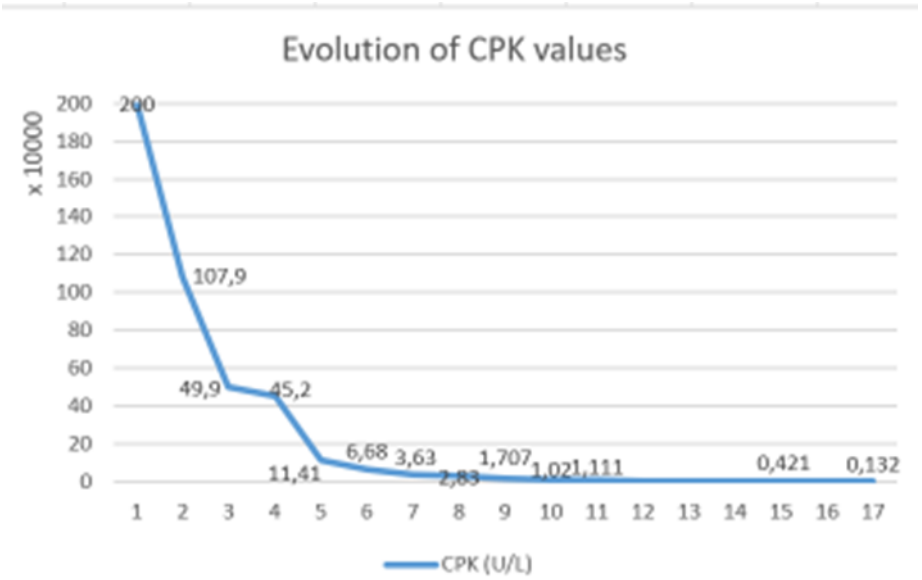


Figure 16 - Horse on the eighth day of hospitalisation with a lowered head position supported by an attachment in the halter and cubes of shavings

On the sixth day the urine was light brown and CK levels were 66 800. The evolution of the CK values is shown on Graphic 3. On the ninth day he was taken off the IV fluids, at this time he was able to move the hindlimbs, still hung his head most of the time but was able to lift it long enough to drink. On day fifteen he started to show more activity and was moved to a different box, he was able to walk stiffly and stay upright. He was discharged on day nineteen and continued to recover at home. The owners were advised to control the horse's rectal temperature daily, he should not have a temperature higher than 38,5ºC. At home, the horse should have been placed in a large box with a tall bedding and be on absolute rest. The horse should also have had access to roughage *ad libitum,* one kg of alfalfa, and one kg of muesli, twice a day, he should also take a L-carnitine and vitamin E feed supplements. After one week, the horse was able to stay upright and maintain a good posture, and improved his overall body condition, as shown on Figure 17.



Graphic 3 - CK values during hospitalisation

Despite presenting with severe discomfort and general weakness, not responding straight away to treatment, and developing some minor complications, this horse did not show signs of respiratory distress or cardiovascular decompensation and spent most of the time standing, which were good prognostic indicatives (Galen et al., 2012).

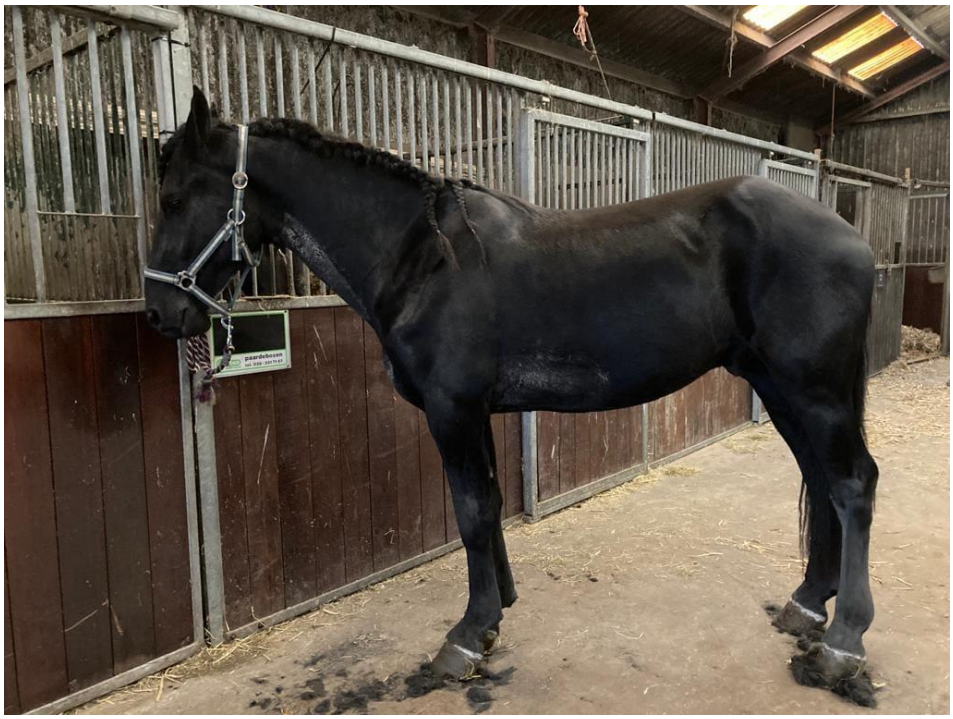


Figure 17 - Horse one week after discharge.

Two other horses of different breeds were also admitted for AM but were euthanized during the first 24-72 hours as they were recumbent and not responding to medical treatment. Additionally, one of the horses already had more severe metabolic derangements, as he presented a blood lactate value of seven mmol/L.

Final conclusions

The curricular externship is a vital part of a veterinarian's education as It allows the students to consolidate knowledge and gain both soft and clinicals skills that are otherwise difficult to obtain. It is the student's first prolonged contact with several paradigms and challenges of the veterinary profession. Performing the externship in two distinct busy referral practices, in different countries, allowed the student to contact with a high volume and variety of cases, reinforcing the knowledge that it was gained during the curricular part of the degree, it also enabled the student to witness issues and clinical scenarios that had not been experienced yet. Due to the number of cases, it sometimes became difficult to properly follow cases individually, however it was very beneficial for the understanding of the different pathophysiologic basis of several diseases and the broad variety of presentations of certain pathologies, as well as the practice of several clinical skills. Frequently the hospitals had different approaches and protocols to similar clinical scenarios which was valuable for the student to further develop critical reasoning skills and its ability to adapt.

This report aimed to describe the caseload followed during the externship, focusing on topics and details that felt most important to the student.

Atypical Myopathy was selected as the topic of the monography because it is an emergent disease which has been the target of extensive investigation in the recent years and also because it was a good example of how important a multidisciplinary approach is when dealing with critical hospitalized patients.

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