What happens during breakdown and repair of the suspensory ligament and what are different treatments aimed at?

Izabella Granswed

Sveriges lantbruksuniversitet
Fakulteten för veterinärmedicin och husdjursvetenskap

Vad händer vid skada och läkning av gaffelbandet och vad är målet med olika behandlingar?

What happens during breakdown and repair of the suspensory ligament and what are different treatments aimed at?

Izabella Granswed

TABLE OF CONTENTS

SAMMANFATTNING

Skador på gaffelbandet är ett stort problem inom hästvärlden och framförallt inom ridsporten. Eftersom många tävlingshästar drabbas finns idag höga krav på veterinärer inom både diagnostik och behandling för att göra de drabbade hästarna friska igen. Då gaffelbandet är en svårläkt struktur så finns ännu ingen revolutionerande behandling att tillgå för att optimera läkning. Detta har medfört att flera olika behandlingar testas och används för att komma närmare en konsensus för skadan. Då man som veterinär rekommenderar behandling och förebyggande åtgärder är det viktigt att man är medveten om varför gaffelbandet går sönder och även vad som händer under läkningsprocessen. Behandlingen går ut på att minska den akuta inflammation som sker direkt efter skada, för att senare underlätta för återuppbyggnad av gaffelbandets struktur. Man vill dessutom hindra bildning av ärrvävnad som predisponerar för ytterligare skador då den saknar samma styrka och elasticitet som frisk vävnad.

SUMMARY

Injuries to the suspensory ligament represent a huge problem in equine veterinary medicine. As many of the affected horses are elite-competition athletes, there are high expectations on veterinarians to make these horses fully functional again.

The suspensory ligament has several features which makes the healing process complicated. So far no treatment has shown to optimize the healing process. This has led to the testing of many new treatment methods, in order to find a consensus for the injury. When recommending treatments and preventing factors to hinder injuries to the suspensory ligament, it is important to know *why* the particular ligament has been ruptured. It is also important to study exactly what happens during the healing process.

Existing treatments aim at minimizing the acute inflammation that occurs directly after the injury and then ease the process of regaining the structure of the ligament tissue. The aim is also to prevent scar tissue from forming and becoming abundant. The reason for this is that scar tissue lacks the strength and elasticity of the healthy ligament tissue.

INTRODUCTION

Suspensory ligament injuries are common reasons for why sport horses become lame and put off training. A study from 2009 in Great Britain shows that it was the second most common injury for dressage horses (Murray *et al*., 2009). Injuries to the suspensory ligament are of special interest since many of the affected horses are elite-competition athletes and expected to reach the same level of performance as before the injury. Therefore, there are high expectations on veterinarians to make horses with ligament injuries fully recovered. Moreover, owners expect that their injured horse will be treated, even if there is little research and documented evidence of the treatment methods at hand.

Different methods of how to treat suspensory ligament injuries are a hot topic in equine veterinary medicine. Several new methods have been described in recent years. The question of how these methods work in relation to what actually happens when the suspensory ligament is broken down and rebuilt, is however not clear. It is important that veterinarians understand the pathobiology of the injury and also what different treatments are aimed at to optimize healing.

This essay will illustrate the pathogenesis and the healing process of a damaged suspensory ligament. It will also present how some of the most common treatments could improve healing.

MATERIALS AND METHODS

The study is based on scientific papers, studies and publications relevant to the topic. The following web sites were used for finding the reports:

1. http://www.pubmed.com
2. http://www.sciencedirect.com
3. scholar.google.com

The phrases used for search were "suspensory ligament injuries", "treatment of suspensory ligament injuries" and "diagnostic methods". These phrases were combined with "equine AND + OR horse". The library at the Swedish University of Agricultural Sciences was also used; to find publications and books describing the anatomy of the suspensory ligament and to acquire useful information about different diagnostic techniques.

LITTERATURE REVIEW

Anatomy, function and composition of the suspensory ligament

The suspensory ligament, or the interosseous muscle, is a strong tendinous band. It arises from the proximal end of the third metacarpal bone (MCIII) and the distal row of the carpal bones in the front limbs. In the hind limbs it arises from the proximal end of the third metatarsal bone (MTIII) and the distal row of the tarsal bones.

The ligament lies deep to the flexor tendons between the second and fourth metacarpal bones. It has one surface towards the MCIII/MTIII and the other surface towards the flexor tendons and divides into two branches that attach to the proximal sesamoid bones. The branches give rise to a medial and a lateral band, both extending to the common digital extensor tendon on the dorsal end of the foot.

The suspensory ligament is usually divided into three parts: the *proximal part* where the ligament originates, the *body of the ligament* which lies along the third metacarpal bone and the *lower branches of the ligament*, that insert into the sesamoid bones.
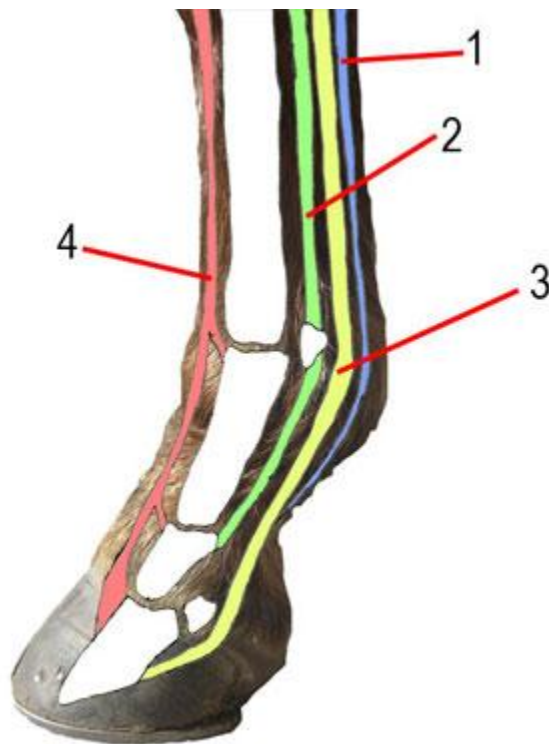
Figure illustrated by Stina Qvarnström (Copyright © Equiart) shows a simplified version of how the tendons in the horse are located. Note that the extension of the two branches of the suspensory ligament to the common digital extensor tendon is not visible. 1. Superficial flexor tendon 2. Suspensory ligament 3. Deep flexor tendon 4. Common digital extensor tendon.

The function of the ligament is to provide support for the proximal fetlock joint. It prevents the fetlock joint from excessive dorsal flexion and also limits the palmar flexion of the joint

because of the branches stretching to the common digital extensor muscle. Furthermore, it helps to minimize concussion (König & Liebich, 2007).

The suspensory ligament has tendinous features even though it differs a bit in composition, compared to the other flexor tendons (Zousa *et al*., 2010). It is referred to as neither a tendon nor a muscle, but a complex ligamentous structure that originates from a muscle through evolution (Soffler & Hermansson, 2006).

The suspensory ligament is composed of nearly 70% water and 30% dry mass (Dowling et al., 2000). The dry mass contains fibroblasts and tendon specific tenocytes which are arranged along the length of the tendon (Soffler& Hermansson, 2006). The tenocytes produce the extracellular matrix (ECM), which is characterized by connective tissue with collagen, proteoglycans, glycoproteins, glycosaminoglycanes and vascularized zones (Dowling et al., 2000). The dry mass also contains about 10% muscle fibers (Soffler& Hermansson, 2006), and between those adipose tissue can be found (Cunningham, 1883).

About 85% of collagen is collagen I and the remainder collagen III. Collagen I fibrils are strictly organized in fibers that form a parallel pattern and makes it possible for the tendon to attach to the bone. Collagen III is mainly present in scar tissue and lacks the strength and elasticity of collagen I (Soffler& Hermansson, 2006). The ligament seems to have more collagen III than other tendinous structures and also a higher frequency of glycosaminoglycanes. The fibroblasts have shown to have a more narrow appearance than the tenocytes, which makes it a complex ligamentous structure (Zouza *et al*., 2010).

Injuries to the suspensory ligament

Injuries to the suspensory ligament can differ in severity depending on where the injury is located, and also how severe the damage is. Predominantly the injury is located on the first proximal third of the ligament, which gives rise to proximal suspensory desmitis (PSD). This is an inflammation of the proximal aspect of the suspensory ligament. However, desmitis can also arise in the body or branches of the suspensory ligament. When an injury has occurred, horses are usually more lame on soft ground than on hard ground since the suspensory ligament then is more loaded (Dyson, 2007).

Acute injury

A common reason for an injury is overstretching the ligament, usually through hyperflexion of the metacarpal/metatarsal joint (Murray *et al*., 2009), leading to a single overload of the ligament and a rupture of the collagen fibers. There is however a contradiction to whether these injuries have undergone a gradual breakdown of ECM, which predisposes for a single overload, or if it is a single overstretch that is responsible for the injury.

Degenerative injury

If the ligament is gradually broken down, the injury is referred to as degenerative suspensory ligament disease (DSLD) or "overuse injury". This happens when the repetitive nature of loads on the tendon creates damages to ECM at a higher rate than the cells within the tendon can repair. Overstimulation has shown to increase inflammatory cytokines and destructive enzymes, which contribute to damage that accumulates and finally reach a level that gives clinical signs. However, also to less stimulation has indicated catabolic gene expressions that contribute to degradation of ECM and loss of function (Dahlgren, 2007).

In degenerative injuries, fibrils are reformed into larger bundles with fibroblasts in the middle. This hinders the blood supply of the fibroblasts that either die or become chondrocytes who try to heal the damage by producing more cartilage-like scar tissue (weak and inelastic) instead of collagen I (strong and elastic). This is likely to predispose for new injuries because of lost elasticity and strength. It also explains why the risk of repeated injuries is high. If the injury reaches a chronic form, oedema is often present and characterized by fibrous tissue, which thickens the ligament and gives rise to enlargements (Gibson & Steel, 2002).

Bone-related traumas and other reasons for injury

Injuries to the suspensory ligament can also arise through percussion, cutting or a bone-related trauma, e.g. avulsion fractures. These are small fractures in the bones which create uneven areas and bone fragments that disturb the suspensory apparatus (Gibson & Steel, 2002). Injuries have also shown to occur due to stress fractures, exostoses and uneven areas on MCIII/MTIII, which are rubbing against the ligament and create a secondary desmitis (Dyson, 2007).

Pathobiology of injury and repair

The pathobiology can be divided into three phases; first an acute inflammation phase, second a proliferation phase and last the remodeling phase.

1. *The inflammation phase* usually last 1-2 weeks and is characterized by heat, swelling of the area, pain on palpation and immigration of inflammatory cells (Dahlgren, 2007). Phagocytes clean up the area, fibroblast produces scar tissue and broken collagen fibrils and blood vessels give rise to small hematomas within the ligament (Soffler & Hermansson, 2006). A release of vasoactive and chemotactic factors initiate angiogenesis, stimulate cell-proliferation and also contributes to recruitment of cells involved in wound reparation. There is also an increase of growth-factors and inflammatory cytokines (Dahlgren, 2007).

2. *The proliferation phase* usually starts a few days after the injury and can last up to a few months. The endotenon (the space between collagen fibrils) is hypertrophied and a massive cell invasion can be seen. Many of the cells are low differentiated, presumably progenitor cells, but there are also fibroblasts which change their morphology and becomes bigger. Tenocytes get an increased gene-expression of collagen I and collagen III, and collagen III becomes more abundant in the tissue than collagen I. New ECM starts to form and the area becomes hypercellular. After about 4 weeks, fibrous tissue and granulation-tissue are starting to form and ECM is organized into a random pattern. At this level of healing the ligament can only withstand minimal exercise (Dahlgren, 2007).

3. *The remodeling phase* starts about six weeks after the injury and can continue for six months or sometimes even 12 months. It is the most critical phase if the horse should be able to return to full work again (Dahlgren, 2007). The scar tissue formed in the earlier phases of the injury is not comparable to the collagen I fibers that were present before the injury and seems to consist of mostly collagen III fibers, which are not fully organized. In this phase, the fibers seem to adapt and get a more linear structure along the tendon, even though the strength and elasticity will not reach the same level as before the injury (Gibson & Steel. 2002).

The healing process was long thought to be similar to that of the other tendons but ultrasonographic pictures show that the suspensory ligament does not heal as well as the other tendons. It also shows more persistent hypoechoic areas, periligamentous soft tissue fibrosis, hyperechoic scar tissue and calcification. These findings can be seen both during and after the healing process (Soffler & Hermansson, 2006).

Differential diagnoses for suspensory ligament injuries could be desmitis of the accessory ligament of the deep digital flexor tendon, carpometacarpal/tarsometatarsal joint pain, and also palmar/plantar cortical fatigue fractures of MCIII/MTIII (Dyson, 2007).

The prognosis is said to be better on the front limbs than on the hind limbs and several factors have to be considered when judging the severity and prognosis of the injury. Acute injuries have also shown to have better prognosis than chronic injuries with degeneration of the ligament (Dyson, 2007).

In a clinical study by Dyson (2007), almost 90% of horses went back to full work within three months. However, chronic disease and injury of the hindlimbs seem to be more challenging. In the same study, horses with proximal injury of the hindlimbs, only 14% (6 of 42) went back to full work within one year, all of which had been lame for less than five weeks.

Treatments

There are several methods in use for treating suspensory ligament injuries, some of which will be mentioned in this study. However, none of the methods have alone shown any remarkable results in healing blinded and double-blinded studies (Herthel, 2001).

The purpose of a chosen treatment is however to minimize muscle and skeletal pain, provide support for the suspensory apparatus (Halper *et al*., 2010) and maximize collagen I formation and organization. It is also desirable to minimize scar tissue (Sutter, 2007) and stimulate new ECM formation and organization (Dahlgren, 2007).

Conservative treatment
By a so called conservative treatment the horse gets box-rest and controlled walking exercise, usually for several months. This treatment is the most used and is often combined with clinical, ultrasonographical and radiographical controls, for monitoring the healing process (Dyson, 2007). Dahlgren (2007) also emphasizes that the rehabilitation program must match the healing phase of the ligament.

NSAID and Glucocorticoids /Antiinflammatory drugs
NSAID and Glucocorticoids can be used in the acute phase of an injury to reduce inflammation (Gibson & Steel, 2002) and swelling - to minimize the risk of compartment syndrome (Dyson, 2007). Compartment syndrome is the compression of blood vessels, muscles and nerves inside a closed compartment.

Cell therapy
The idea of cell therapy is to make cells differentiate into mature fibroblasts capable of building new ECM, as well as producing growth-factors and cytokines (Dahlgren, 2007).

Mesenchymal stem cells (MSC), and other bone marrow components for injection, seem promising for the future, according to Sutter (2007). The idea of this technique is that the cells can differentiate into tendon-specific fibroblasts at the injured site, and create the appropriate

wound matrix. Sutter claims that horses have more connective tissue progenitors in their bone marrow aspirate than humans, which would make them even better candidates for surgery.

In a retrospective study by Herthel (2001) on 100 horses, 92% of the candidates returned to full work after surgery, compared to 15,6% of 66 horses that were not treated. Sutter (2007) thus concludes that more research must be done and that the expectations are speculative at best. Herthel also points out that there can be a risk of damaging the ligament because of the large volume of fat cells and bone spicules commonly found in bone aspirate. Bone marrow contamination of blood has also been discussed as a risk factor.

Platelet rich plasma (PRP)
The idea of injecting PRP to injured tendons and ligaments is to produce a scaffold in the form of a fibrin-clot that forms and infuses growth factors into the lesion (Dahlgren, 2007). This method has become popular in recent years, especially in the USA (Sutter, 2007).

It is the high concentration of growth factors PDGF, TGF-b, FGF, VEGF, IGF-I and EGF, released from platelet a-granules that make PRP especially interesting for tendon and ligament healing (McCarrel & Fortier, 2008).

PRP has shown to provide a provisional matrix which increase collagen I production, tenocyteproliferation, neovascularization, and also increase early strength, organization and fiber pattern alignment (Sutter, 2007).

Mc Carrel & Fortier (2008) compared the temporal release of growth-factors from PRP, Bone marrow aspirate (BMA) and lyophilized platelet product (PP) in vitro. Their results support further in vivo investigation of PRP for treatment of tendonitis and desmitis.

Waselau *et al*, (2008) investigated the outcome of 9 standard breed race-horses with moderate to severe mid-body suspensory ligament desmitis (MSD). The horses were treated with one intralesional injection of PRP and a gradually increased exercise program. All 9 horses returned to race and the authors conclude that the treatment have an excellent prognosis for that.

Acellular urinary bladder matrix (UBM)
UBM is a lyophilized (freeze-dried) powder, derived from the extracellular matrix of the basement membrane of swine urinary bladders. It is non-cellular, but believed to recruit growth factors and regenerative cells from the circulatory system and other tissues - and differentiate into new healthy tissue (Wallis *et al*., 2010).

UBM has shown to increase angiogenesis in other species (not specified by the author) 5-7 days after injection. Other benefits could be providing a scaffold for collagen deposition, recruiting new growth-factors to the site and minimizing excessive fibrous tissue formation (Wallis *et al*., 2010).

According to Dahlgren (2007) it could theoretically provide a scaffold for cellular migration into the lesion, and also help organize ECM. Accompanying bioactive molecules could also stimulate cell recruitment.

A study by Wallis *et al*. (2010) on collagenase induced suspensory ligament injuries thus showed that there were no histological differences between the group treated with UBM and

the control group. However, there was a trend towards milder lameness for the treated group, compared to the control group.

Shock-wave therapy

Shock-wave therapy involves pulse acoustic pressure waves sent to a specific area. The pressure waves generate high stress forces that act upon cellular interfaces. The treatment seems to relieve pain and stimulate healing. However its mechanisms are poorly understood (Gibson & Steel, 2002). According to Dyson (2007) shock-wave therapy, or radial pressure wave therapy, is useful in some cases, although the results are not guaranteed.

In a study of 16 horses with proximal suspensory desmitis or a similar pain by Imobdem et al., (2007), the authors concluded that one session with shock-wave treatment did not ease lameness, even though it could not be excluded in certain individuals.

Several authors refer to a study by Chen *et al*. (2003) which shows that shock-wave treatments promoted healing of collagenase induced achilles-tendinitis of rats by inducing growth factors TGF-b1 and IGF-1. These growth-factors showed to play an important role in cell-proliferation and tissue regeneration in the treated tendons.

Neuroectomy

Removal of the deep branch of the lateral plantar nerve has become a routinely used method of treating lameness caused by PSD (Pauwels *et al*., 2009). In a study series of two, made by Dyson and Genovese (2003), 5/6 and 8/9 horses went back to 100% performance.

In a study by Kelly (2003), 78/84 (91%) horses went back to full performance. The study by Pauwels *et al*. (2009) showed that neuroectomy of the deep branch of the lateral plantar nerve could cause neurogenic atrophy of the muscle fibers in proximal parts of the suspensory ligament. Dyson (2007) mentions anecdotal reports of ulnar neurectomy or neurectomy of the lateral palmar nerve for management of PSD, but states that she has no personal experience of the results.

DISCUSSION

When looking at the anatomy of the suspensory ligament it is understandable that the area is subject to great loads of physical pressure in a horse. The suspensory ligament has to provide support for the proximal fetlock joint and prevent it from excessive dorsal flexion. At the same time it has to hinder palmar flexion.

Since healthy tissue mainly consists of collagen I fibers, elastic and strictly organized, this composition is desirable. As the goal with treatment is to regain a healthy tissue, the treatment must aim at getting back the strength and elasticity found in collagen I fibers. Therefore, scar tissue containing the weaker and more inelastic collagen III must be hindered.

As the repetitive nature of loads on the tendon creates damage to the ECM, the cells in the tendon must be able to repair the tissue with the same speed as it is broken down (Dahlgren, 2007). If not, injuries will arise and scar tissue, containing more collagen III than healthy tissue, is created. Since Collagen III lacks the same elasticity and strength as Collagen I, the maximum load on the tendon is more rapidly reached.

When discussing degenerative injuries it should be considered that a micro damage, without clinical signs, could develop into a larger injury. As the micro damage tries to heal, minimal scar tissue forms which weakens the ligament at the injured site. This predispose for new injuries.

Since there are three phases of injury and repair, which all are characterized by different happenings, it should be considered that the ligament is in favor of different treatments during different phases of healing. Alternatively, some treatments could be more successful than others, depending on in what phase of healing the ligament is. However, Dyson (2007) concludes that most studies made on treatments have lacked control groups.

NSAID and Glucocorticoids can be used in the acute phase of an injury, to reduce inflammation and swelling - and minimize the risk of compartment syndrome (Dyson, 2007). As the first phase of healing is the inflammation phase with heat, pain and swelling (Dahlgren, 2007), one could understand that the risk of compartment syndrome increase, and also that this phase of injury should be controlled.

During the proliferation phase a massive cell invasion can be seen. Collagen I and III get an increased gene expression and new ECM starts to form. This phase goes together with the remodeling phase, which is the most critical phase according to Dahlberg (2007). This is due to the fact that the remodeling of scar tissue, to a more organized and elastic tissue, becomes evident and that this step often fails. It is therefore in this phase of healing that most treatments are designed to improve effective healing.

Cell therapy is built on the idea that mesenchymal stem-cells differentiate into mature fibroblasts, capable of producing new ECM. Growth-factors and cytokines should also be produced to – so far in theory - boost healing. If applied during the healing phase, the idea seems very promising, also concluded by Sutter (2007). However, just like many other techniques used for tendon and ligament healing, this method lacks placebo-controlled studies, even though there are some interesting in-vitro studies made (Sutter, 2007).

In the retrospective study by Herthel (2001), 92% of the candidates returned to full work. However, some important information was missing. For example, lameness duration,

location, lesion size and hind limb versus front limb were not fully declared. This lack of input makes it hard to evaluate the study. It seems that Sutters (2007) conclusion of more research, instead of speculations, is relevant in this case, even if the method seems promising. It is also important to consider the risks that are associated with collecting bone-marrow aspirate and injecting into tendons. Sutter (2007) mentions secondary injuries by bone spicules as a possible negative consequence. Even if stem cells are favorable for healing ligament injuries, the risk of creating a secondary damage should always be considered when choosing a treatment. If the risk of secondary damage is big there is also a risk of misleading information when the results are to be evaluated. Possibly the aspirate should be cleaned from bone spicules before injected in the tendon, to get better results. In short, there are several questions that need to be answered before more accurate conclusions can be drawn.

Platelet rich plasma (PRP) seems to work similar to cell therapy as both aim to increase growth-factors. The high concentration of growth factors, especially in platelet a-granules, would in theory contribute to a better healing (McCarrel & Fortier, 2008). Sutter (2007) has investigated cell-based theories and states that PRP have shown to provide a provisional matrix which increase collagen I production, tenocytproliferation, neovascularization and also increase early strength, organization and fiber alignment. However, the PRP method lacks placebo-controlled studies in vivo even if the author mentions positive in vitro studies. The author discusses how the technique works and also how tendons in rats and humans have shown to heal better. But, he does not provide any actual results on healing suspensory ligament injuries with PRP on horses.

Mc Carrel and Fortier (2008) compared the temporal release of growth-factors for PRP, PP and BMA and also measured their effect on ligament and tendon gene expression. They found that platelet concentration was highest in PRP and PP, and also that growth-factor concentration reflected platelet concentration. Hence – PRP and PP had the highest amount of growth factors. They also concluded that some growth factors in PRP even increased after contact with the tendon/ligament structures which would be optimal for ligament healing. However, the study is made in vitro and might not reflect the result of a suspensory ligament injury of a real case. Therefore, the authors now support in vivo studies with PRP and PP.

Waselau et al. (2008) supports the injection of PRP together with controlled walking exercise. However, his study only contained nine horses and the low number of horses in the study makes it hard to evaluate whether PRP alone had any remarkable effect on the healing. If more horses had been used, including a control group with only conservative treated horses, the study would be more reliable.

Dahlgren (2007) has summarized the pathobiology of tendon and ligament injuries in a review article. She states that injections with UBM are promising, at least in theory, and that it could provide a scaffold for cellular migration into the lesion, as well as organizing ECM and provide cell recruitment. She presents facts but has not made any studies herself. Wallis et al. (2010) seems to agree in theory, and as UBM has shown to increase angiogenesis in other species it would be relevant for healing ligaments. However, the study by Wallis showed no difference between the treated group and the control group on collagenase induced suspensory ligament injuries. The author concluded that UBM did not have a positive effect on the healing process. However, there is always a question of whether collagenase induced injuries is comparable to real cases or not.

Since Shock-wave therapy generates high stress forces and act upon cellular interfaces (Gibson & Steel, 2002), it is on one hand believed to stimulate healing. On the other hand, it

"only" seems useful in some cases according to Dyson (2007). As the mechanisms are poorly understood, there is little fact to evaluate whether they could affect healing in a positive way. Both these articles discuss shock-wave therapy but have not made any studies on the method themselves.

In the study by Imobden *et al*. (2007) it was concluded that one session of shock-wave therapy did not improve lameness. The study thus aimed at measuring lameness and did not take healing into consideration. It could thus be seen that shock-wave did not have any analgesic effect after only one session. It would, however, be interesting to see whether lameness improved after several treatments and also if healing was affected.

The report that gives most support of the shock-wave therapy method, is the study by Chen *et al*. (2003). It shows that shock-wave treatment can promote healing of collagenase induced achilles-tendinitis in rats. The treatment induced growth factors TGF-b1 and IGF-1, both which have important roles in cell-proliferation and tissue regeneration in tendons. The results explain the mechanisms of shock-wave, but unfortunately the study is not performed on horses. The treated tendon (rats) does not have the same composition as the suspensory ligament and the injuries are collagenase induced.

When discussing neuroectomy, it is important to note that the treatment only acts upon the symptom and not on the actual injury. The nerve innervating the area is removed and pain is lost. This is quite evident in the studies by Dyson & Genovese (2003) and Kelly (2007) as both have high scores in treating lameness (86% and 91% returning to full work). Unfortunately some information that could be used to evaluate the studies are missing. Dyson & Genovese (2003) base their studies on "unpublished data" and "personal communication" from their own work. Whereas the study by Kelly (2007) have been demonstrated on an EVCS (European College of Veterinary Surgeons) meeting and information is limited.

Furthermore, one shall not forget the possible negative consequences associated with this treatment such as neurogenic atrophy of the muscle fibers in the proximal parts of the suspensory ligament (Pauwels *et al*., 2009). If an area is not innervated, the surrounding muscles seem to get a gradual breakdown. This gives less support to the suspensory ligament and might predispose for new injuries. It should thus be noted that Pauwels *et al*, (2009) only had one horse in their study, which decrease the study's credibility.

In summary, we can conclude that more research within the area of injury, healing and treatment of suspensory ligament injuries is necessary. It is needed for an adequate understanding of the mechanisms of the disease and for the development of effective treatment methods.

There is a general agreement of why the suspensory ligament in horses breaks. Healing mechanisms are also generally agreed on, judging from studies and reports on the subject. There are however contradictory findings of how to best treat the injuries. Researchers in the area agree on the fact that inflammation in the acute phase should be controlled. Also, the main purpose of later treatments is to promote new ECM, collagen I formation and alignment and prevent scar tissue from being abundant. Since no treatment alone has given any remarkable results in achieving this, this important area of study and research must be further explored.

LITTERATURE

Cunningham, D.J. (1883). The Development of the Suspensory Ligament of the Fetlock in the Foetal Horse, Ox, Roe-Deer, and Sambre-Deer. *J Anat Physiol* 18(Pt 1), 1-12.

Chen, Y. J.,Wang, C. J.,Yang, K. D., Kuo, Y. R., Huang, H. C., Huang, Y. T., Sun. Y. C & Wang, F. S. (2004). Extracorporeal shock waves promote healingof collagenase-induced Achilles tendinitis andincrease TGF-b1 and IGF-I expression. *Journal of orthopedic research* 22: 854-861.

Dahlgren, L. A. (2007). Pathobiology of Tendon and Ligament injuries. *Clin Tech Equine Pract* 6: 168-173.

Dowling, B. A., Dart, A.J., Hodgson, D.R and Smith, R.K.V. (2000). Superficial digital flexor tendonitis in the horse. *Equine Vet J*, 32: 369-378.

Dyson, S. (2007). Diagnosis and Management of Common Suspensory Lesions in the Forelimbs and Hindlimbs of Sport horses.*Clin Tech Equine Pract* 6: 179-188.

Dyson, S & Genovese, R.L. (2003). The suspensory apparatus. In: *Diagnosis and Management of Lameness in the Horse*, 1st edn., Eds: M.W. Ross and S.J. Dyson,W.B. Saunders, St Louis. pp 658-662.

Gibson, K.T. & Steel, C.M. (2002). Tutorial article - Conditions of the suspensory ligament causing lameness in horses. *Equine Veterinary Education* 14(1), 39-50.

Halper, J., Khan, A. & Mueller, P.O.E. (2011). Degenerative Suspensory Ligament Desmitis - A New Reality. *Pakistan Veterinary Journal* 31(1), 1-8.

Herthel, D. J. (2001). Enhanced Suspensory Ligament Healing in 100 horses by Stem Cells and Other Bone Marrow Components. *AAEP Proceedings* vol 47: 319-321.

Imobden, I., Waldern, N. M., Wiestner, T., Lischer, C. J., Ueltschi, G &Weishaupt, M. A., (2009). Short term analgesic effect of extracorporeal shock wave therapy in horses with proximal palmar metacarpal/plantar metatarsal pain. *The Veterinary journal* 179: 50-59.

Kelly, G. (2007) Results of neurectomy of the deep branch of the lateral plantar nervefor treatment of proximal suspensory desmitis. In: *16th Annual Scientific Meetingof the European College of Veterinary Surgeons*, Ed: M. Schramme, ECVS,Dublin. pp 130-134.

König, H.E & Liebich, H. G. (2007). *Veterinary Anatomy of Domestic Mammals, textbookand colour atlas*. 3rd edition. Stuttgart: Schattauer GmbH.

McCarrel, T & Fortier, L. (2009). Temporal Growth Factor Release from Platelet-Rich Plasma, TrehaloseLyophilized Platelets, and Bone Marrow Aspirate and Their Effect onTendon and Ligament Gene Expression. *www.interscience.wiley.com*. DOI 10.1002/jor.20853.

Murray, R. C., Walters, J.M., Snart, H., Dyson, S. J & Parkin, T. D. H. (2009). Identification of risk factors for lameness in dressage horses. *The veterinary journal* 184, 27-36.

Pauwels, F. E., Schumacher, J., Mayhew, I. G & Van Sickle, D. C. (2009). Neurectomy of the deep branch of the lateral plantar nervecan cause neurogenic atrophy of the muscle fibres in theproximal part of the suspensory ligament (*M. interosseous III*). *Equine Vet . J.* 41 (5) 508-510.

Soffler, C. & Hermanson, J.W. (2006). Muscular design in the equine interosseus muscle. *Journal of Morphology* 267(6), 696-704.

Souza, M.V., Weeren, P.R.v., Schie, H.T.M.v. & Lest, C.H.A.v.d. (2010). Regional differences in biochemical, biomechanical and histomorphological characteristics of the equine suspensory ligament. *Equine Veterinary Journal* 42(7), 611-620.

Sutter, W. W. (2007). Autologous Cell-BasedTherapy for Tendon and Ligament Injuries. *Clin Tech Equine Pract* 6:198-208.

Wallis, T. W., Baxter, T. M., Werpy, N. M., Mason, G. L., Frisbie, D. D & Jarloev, N. (2010).Acellular Urinary Bladder Matrix in a CollagenaseModel of Superficial Digital Flexor Tendonitisin Horses. *TW Wallis et Al.* Vol 30, No 7.

Waselau, M., Sutter, W. W., Genovese, R. L & Bertone, A. L. (2008). Intralesional injection of platelet-rich plasma followed by controlled exercise for treatment of midbody suspensory ligament desmitis in Standardbred racehorses. *J Am Vet Med Assoc* 232: 1515-1520.