UNIVERSIDADE DE LISBOA

Faculdade de Medicina Veterinária

TENOSCOPIC EXAMINATION OF THE DIGITAL FLEXOR TENDON SHEATH: A RETROSPECTIVE ANALYSIS OF 86 HORSES (2016)

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DISSERTAÇÃO DE MESTRADO INTEGRADO EM MEDICINA VETERINÁRIA

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To my family and friends, for all the love, dedication and support during these years, and especially to my beautiful mother, who raised me to be strong, passionate and to always follow my dreams. After six years of adventures, hard work and determination, this one is for you Mum.

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Abstract

TENOSCOPIC EXAMINATION OF THE DIGITAL FLEXOR TENDON SHEATH: A RETROSPECTIVE ANALYSIS OF 86 HORSES (2016)

The digital flexor tendon sheath (DFTS) enfolds the digital flexor tendons and their associated structures, protecting and facilitating its movements. Tenoscopic examination of the DFTS has become a routine procedure in equine surgical practice since it is a minimal invasive technique that allows the exploration of the sheath structures with direct observation of lesions and therefore, confirmation of a diagnosis and, in some cases, early treatment.

In this work, medical records of horses that underwent tenoscopic examination of the DFTS in 2016 at four different equine hospitals in the United Kingdom had their medical records reviewed. Eighty-six cases (93 DFTSs) were included in this study. There were 31% Thoroughbreds, 28% Warmbloods, 20% Coldbloods, 12% Ponies and 9% unknown. Ninety-eight percent of horses were lame at the time of clinical examination, effusion was present in 94% of the DFTSs and the hindlimbs were more frequently intervened (61%). Palmar/plantar annular ligament (PAL) constriction (43%) was the most common pathology, followed by sheath penetration (27%), and tears of the flexor tendons (24%) and manica flexoria (MF) (19%). Other diagnosis included tendonitis, infectious tenosynovitis, fibrosis, diseases of sesamoidean ligaments and ganglion cysts.

Results show that PAL constriction, flexor tendon and MF tears were diagnosed more frequently in the hindlimbs (72.5%, 64% and 90%, respectively), while traumatic injuries affected more forelimbs (60%). Coldbloods and Ponies were predisposed to MF tears and PAL constriction, Warmbloods to PAL constriction and flexor tendon tears, and Thoroughbreds to traumatic injuries. Young horses (<10 years) had a higher incidence of traumatic injuries, whereas older horses (>10 years) were commonly diagnosed with MF tears and PAL constriction.

Tenoscopy was of extreme importance as a diagnostic method, considering that MF tears were only identified by other diagnostic methods in 61% of cases, flexor tendon tears in 67% and PAL constriction in 53%. In conclusion, tenoscopy was proven to be a simple and useful diagnose and treatment method of DFTS pathology, even though the long term follow-up of the analysed horses was not studied.

Keywords: horse; tendon; digital flexor tendon sheath; tenoscopy.

Resumo

TENOSCOPIA DA BAÍNHA DIGITAL DOS TENDÕES:
ANÁLISE RETROSPETIVA DE 86 CAVALOS (2016)

A bainha digital dos tendões (BDT) envolve os tendões flexores digitais e as estruturas associadas aos mesmos, protegendo-os e facilitando os seus movimentos. A examinação tenoscópica da BDT tornou-se um procedimento de rotina em cirurgia de equinos, visto ser uma técnica cirúrgica minimamente invasiva que permite a observação direta de lesões, confirmação de diagnósticos e, em alguns casos, tratamento precoce.

Neste trabalho foram revistos os registos médicos dos cavalos que foram submetidos a tenoscopia da BDT em 2016 em quatro hospitais de equinos no Reino Unido. Oitenta e seis casos (93 BDTs) foram incluídos no presente estudo sendo que 31% eram de raça puro-sangue inglês, 28% de sangue quente, 20% de sangue frio, 12% póneis e 9% de raça desconhecida. Noventa e oito porcento dos cavalos exibiram claudicação e 94% dos membros tinham distensão da BDT. Os membros posteriores foram os mais intervencionados (61%). Constrição pelo ligamento anular palmar/plantar (LAP) (46%) foi a doença mais comum, seguida de lesão percutânea da bainha (27%), e lesões marginais dos tendões flexores (24%) e roturas da manica flexoria (MF) (19%). Outros diagnósticos incluíram tendinite, tenosinovite infeciosa, fibrose, doenças de ligamentos sesamoideus e quistos.

Os resultados indicam que a constrição pelo LAP, lesões marginais dos tendões flexores e roturas da MF são mais frequentes nos membros posteriores (72.5%, 64% e 90%, respetivamente), enquanto lesões percutâneas afectam mais os membros anteriores (60%). Cavalos de sangue frio e póneis foram mais propensos a roturas da MF e constrição pelo LAP, cavalos de sangue quente a constrição pelo LAP e lesões marginais dos tendões flexores, e puro-sangue inglês a lesões traumáticas da BDT. Cavalos jovens (<10 anos) tiveram maior incidência de lesões traumáticas mas cavalos mais velhos (>10 anos) foram mais afetados por roturas da MF e constrição pelo LAP.

A tenoscopia é de extrema importância como método de diagnóstico, considerando que roturas da MF apenas foram identificadas por outros métodos em 61% dos casos, lesões marginais dos tendões flexores em 67% e constrição pelo PAL em 53%. Em conclusão, foi provado que a tenoscopia é uma técnica simples e útil de diagnóstico e tratamento de doenças da BDT, embora o acompanhamento da recuperação do cavalo a longo prazo não tenha sido estudado.

Palavras-chave: cavalo, tendão, bainha digital dos tendões, tenoscopia.

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List of Abbreviations and Symbols

% - Percent
ºC - Degree Celsius
BEVC - Bell Equine Veterinary Clinic
CDET - Common digital extensor tendon
CI - Confidence interval
cm - Centimetres
CSA - Cross-sectional area
CSL - Cruciate sesamoidean ligament
DDAL - Distal digital annular ligament
DDFT - Deep digital flexor tendon
DFTS - Digital flexor tendon sheath
DGVG - Donnington Grove Veterinary Group
dL - Decilitres
DM - Digital manica
EDTA - Ethylenediamine tetraacetic acid
g - Grams
hr - Hour
IPJ - Interphalangeal joint
ISL - Intersesamoidean ligament
kN - Kilonewton
L - Litres
LDET - Lateral digital extensor tendon
LF - Left forelimb
LH - Left hindlimb
LT - Longitudinal tear
Mc2 - Metacarpal bone 2
Mc3 - Metacarpal bone 3
Mc4 - Metacarpal bone 4
MCP joint - Metacarpophalangeal joint
MF - Manica flexoria
MHz - Megahertz
min - Minute
mL - Millilitres
mm - Millimetres
mm^2 - Square millimetres
MPa - Megapascal
Mt2 - Metatarsal bone 2
Mt3 - Metatarsal bone 3
Mt4 - Metatarsal bone 4
MTP joint - Metatarsophalangeal joint
N - Newton
NEH - Newmarket Equine Hospital
No. / N / n - Number
OSL - Oblique sesamoidean ligament
P1 - Proximal phalanx
P2 - Middle phalanx
P3 - Distal phalanx
PAL - Palmar/plantar annular ligament
PDAL - Proximal digital annular ligament
P - P-value
p - Proportion of positives
pp. - Pages
PSB - Proximal sesamoid bone

RF - Right forelimb
RH - Right hindlimb
SDFT- Superficial digital flexor tendon
SL - Suspensory ligament
SSL - Straight sesamoidean ligament
SW - Sheath wall
“T” ligament - Transverse lamina ligament
TNCC - Total nucleated cell count
TP - Total protein
USG - Ultrasonography
VEH - Valley Equine Hospital

Externship Reports

As part of the Integrated Masters Degree in Veterinary Medicine from the Faculty of Veterinary Medicine - University of Lisbon, seven months of clinical training in equine medicine were completed. During these, five hospital externships in England, one in Belgium, one in France and one in Portugal were undertaken.

In England, the extra-mural studies started in Rossdales Equine Hospital from 18th of September 2016 to 9th October (three weeks) and continued in Donnington Grove Veterinary Group (DGVG), from 16th October to 12th November (four weeks), Valley Equine Hospital (VEH), from 13th to 26th November (two weeks), Bell Equine Veterinary Clinic (BEVC), from 27th November to 11th December (two weeks) and Newmarket Equine Hospital (NEH), from 12th December to 7th January (four weeks). In all five clinics, interns and senior clinicians supervised the student's involvement in the admission, treatment and management of both elective and emergency, medical and surgical cases. It was given the opportunity of being present in several surgeries and scrubbing in in various surgical procedures such as arthroscopy, tenoscopy, fracture repair, fetlock arthrodesis, standing dorsal spine process removal, fasciotomy and neurectomy of the deep branch of the lateral plantar nerve, neurectomy of the palmar digital nerve, colic surgery, castration, tie-forward, tie-back, resection of the aryepiglottic folds and resection of tumours (keratomas, sarcoids and melanomas). Alongside this, it was allowed to assist the interns during induction, preparing the horse (placing urinary catheter, clipping and scrubbing), anaesthesia and recovery. Hospital duties were assigned, including out of hours shifts, monitoring and feeding horses, preparing drugs and intravenous perfusions, walking and cold hosing horses, cleaning the facilities and re-stocking rooms. Aside the hospital duties, it was possible to assist and follow the cases, from the horse's arrival to the diagnostic procedure (sample collection, radiographs, ultrasound, scintigraphy, CT, MRI and diagnostic analgesia) and the treatment, allowing the development of clinical and communication skills.

In Portugal, the externship started the 30th January and finished the 29th of March (two months), during which there was a two week break for attending the externships in Belgium and France. The two months were spent in the Equine Military Veterinarian Hospital - Escola das Armas, Mafra, under the supervision of the Captain Med. Vet. Gonçalo Paixão. A more practical training was possible, as the students were given the opportunity to collect samples, administer drugs (oral and parenteral), manage wounds, change bandages, and walk and lunge horses. As part of the teaching, it was allowed to perform lameness evaluations (flexion tests, diagnostic analgesia, radiographs and ultrasounds), colic examination (colic check, rectal palpation and nasogastric tubing) and reproduction assessment (rectal ultrasonography, palpation and standing castration).

In Belgium, the externship was attended at Dierenkliniek De Morette, between 12th and 18th of March (one week) and in France at Clinique Equine de Meslay, from 20th to 27th of March (one week). The extern duties in both clinics included morning checks and treatment of inpatients, drug administration, setting of intravenous perfusions, wound management, assisting in consultations, lameness workups and emergencies, and helping in induction and recovery from surgery. As in the other externships, it was possible to assist and follow the cases, from the horse's arrival to the diagnostic procedure and the treatment.

LITERATURE REVIEW

1. THE TENDON

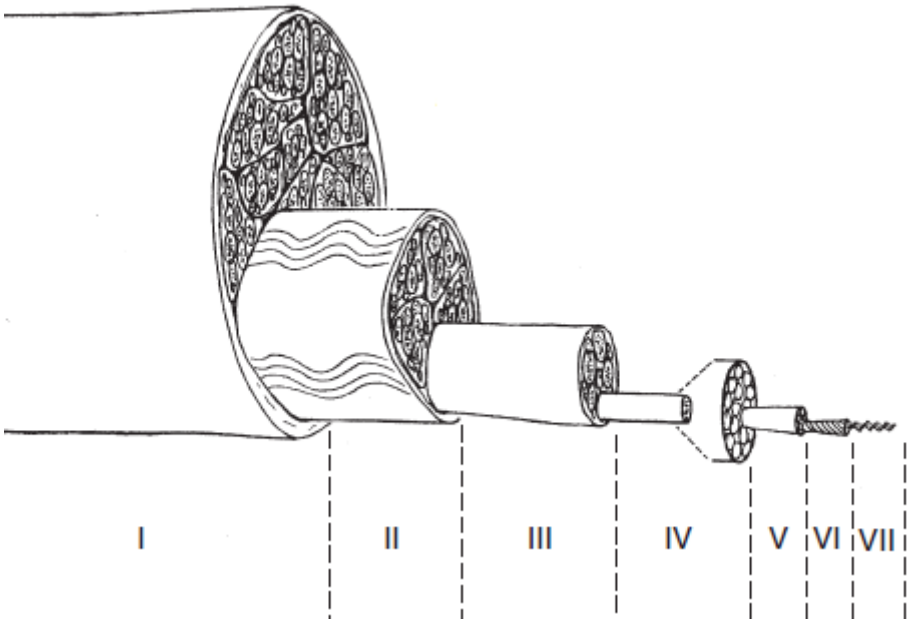
1.1. Structure

Tendons and ligaments are connective tissues of the skeletal system that play a critical role in the transmission of forces between muscles and bones and between bones, respectively. A tendon is a though band of fibrous tissue that connects muscle to bone being able to withstand tension and allow the movement of joints. Certain tendons developed a specialized role acting as springs to store energy for locomotion (Birch, Sinclair, Goodship & Smith, 2014; Thorpe, Udeze, Birch, Clegg & Screen, 2013; Thorpe *et al.*, 2016). This role is particularly important in the horse, where the superficial digital flexor tendon (SDFT), the deep digital flexor tendon (DDFT) and the suspensory ligament (SL) on the palmar/plantar aspect of the metacarpal/metatarsal region act to support the hyper-extended metacarpo/ metatarsophalangeal joint during weight bearing (Birch *et al.*, 2014).

All tendons are composed by highly parallel aligned type I collagen fibers grouped to form collagen bundles of increasing diameter: fibril, fiber and fascicle. The fascicles are held together by the loose connective tissue, the endotenon, which is confluent with the epitenon, the outside of the tendon. When the tendon is not surrounded by a tendon sheath, a thick fibrous layer, the paratenon, surrounds it (Smith, 2003; Thorpe *et al.*, 2013). In gross inspection tendons have a white homogenous appearance although they are composed by a complex arrangement of fibroblast-like cells (tenocytes), extracellular matrix proteins, blood vessels, lymphatic vessels and nerves. The tenocytes reside between the parallel aligned fibers and are responsible for synthetizing and degrading the matrix components of the tendon (Birch *et al.*, 2014; Thorpe *et al.*, 2013).

The functional properties of the tendons rely on the extracellular matrix, which is composed by two-thirds of water and one-third of collagen type I and other proteins (dry weight). About 80% of the dry weight refers to collagen while the remaining 20% is comprised of non-collagenous glycoproteins such as proteoglycans, cartilage oligomeric matrix protein, elastin, fibronectin and lubricin. These proteins are vital for the structure and function of the tissue as they provide resistance, regulate the collagen fibril diameters and organization, and facilitate the sliding between fascicles. The blood vessels along with the synovial fluid provide the nutrients for the tendon cells. The tendon blood supply arises from the musculo-tendinous junction, the osseous insertion and via mesotendon attachments within tendon sheaths. It has been shown that the blood flow to the tendons is higher in foals and increases during exercise (about 200%) and when injuries are present (more than 300%) (Birch *et al.*, 2014; Smith, 2003; Thorpe *et al.*, 2013).

Figure 1. Structural hierarchy of the tendon. (*I*) Tendon unit surrounded by paratenon in extrasynovial locations and epitenon in synovial locations; (*II*) Third-degree fascicle (1 to 3 mm diameter); (*III*) second-degree fascicle (400 to 1000 µm diameter); (*IV*) first degree fascicle (15 to 400 µm diameter); (*V*) collagen fiber (1 to 20 µm diameter); (*VI*) colagem fibril (20 to 150 nm diameter); (*VII*) collagen triple hélix (1 nm diameter) (adapted from Avella & Smith, 2012).

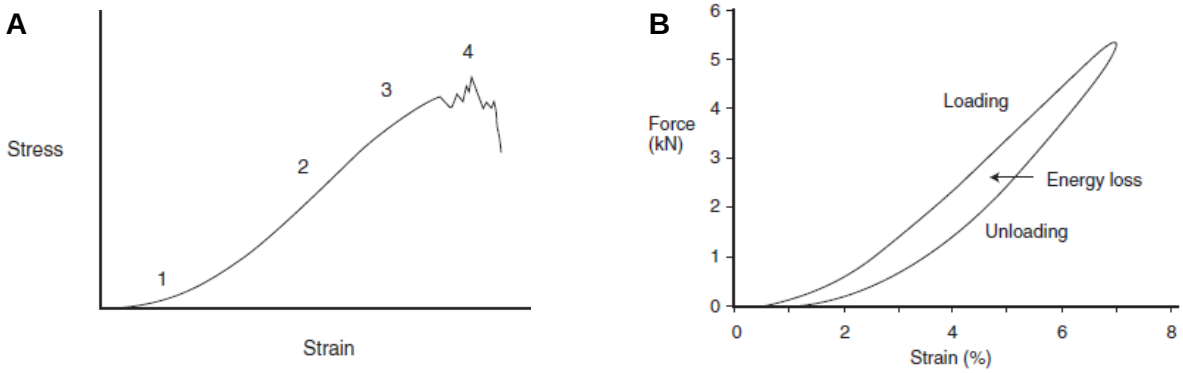


1.2. Functional characteristics

The predominant function of most tendons is to transfer the forces generated by the muscles to the bones, resulting in correct movement (Smith, 2003; Thorpe *et al.*, 2013). The biomechanical properties of a tendon can be defined *in vitro* by its structural or material properties. These experiments generate a load-deformation curve (Graphic 1, A) from which the ultimate tensile strength (kN) and the stiffness (N/mm) can be derived. The curve has four regions: the “toe” region where there is a non-linear stretch of the tendon, representing the initial elongation at low loads; the linear deformation, area of the curve from which the stiffness is determined; the yield region, that represents irreversible lengthening of the tendon; and the region of rupture (Birch *et al.*, 2014; Smith, 2003).

The tendon ultimate tensile stress (force per unit area at the point at which the tendon breaks, N/mm^2) can be calculated by knowing the cross-sectional area of the tendon and its length, the stress (force per unit area) and the strain (percentage of elongation). In the horse, the ultimate tensile strength of the SDFT is 12 kN and the ultimate tensile stress is close to 100 MPa. *In vitro* tests show that the ultimate tendon strain (percentage of extension of the tendon at its breaking point) may not be constant along the length of the SDFT, it is usually 10 to 12% but can go up to 20% before rupture, which may explain why these are more prone to injury (Birch *et al.*, 2014; Smith, 2003).

Graphic 1. (A) Simplified stress-strain curve for tendon. Zone 1 refers to the toe region, initial elongation at low loads; zone 2 to the linear deformation, area of the curve from which the stiffness is determined; zone 3 to the yield region after which irreversible damage occurs; and zone 4 to where tendon fibers rupture. (B) Hysteresis loop for tendon (adapted from Smith, 2003).



When a tendon is loaded the curve obtained is different than when unloaded, demonstrating the hysteresis property (Graphic 1, B). The area between both curves represents the energy lost during the loading cycle, which is usually 5%. The energy is mostly lost as heat and can rise the tendon temperature up to 46ºC. The high temperatures could be harmful to the tenocytes but these cells remain viable when subjected to temperatures of this magnitude (Birch *et al.*, 2014).

During tendon loading the energy is stored in its extension, resulting in a reduction in the energetic cost of locomotion. In order to allow this energy to return to its usable form, the rate of unloading needs to be tuned to stride frequency since there is little tolerance in the system and the tendon is prone to overstrain injury. When the limb impacts the ground the loads rise the quickest in the SDFT, being considered a weight-bearing and energy storing tendon. During galloping exercise the strain of SDFT is 16%, while the strain of the common digital extensor tendon (CDET) is 3%, indicating that it is a positional tendon. In energy storing tendons the properties of the interfascicular matrix influence directly the mechanical properties of the whole structure, implying that interfascicular sliding is critical for these tendons (Birch *et al.*, 2014; Smith, 2003; Thorpe *et al.*, 2013).

2. ANATOMY OF THE DISTAL LIMB

2.1. Bones and joints

The equine digit is comprised by the foot and pastern, including the navicular bone, proximal phalanx (P1), middle phalanx (P2), distal phalanx or coffin bone (P3) and all their joints and associated structures. Proximally to the digit, the metacarpophalangeal (MCP)/ metatarsophalangeal (MTP) joint, the proximal sesamoid bones (PSBs) and the components

surrounding them form the fetlock in both thoracic and pelvic limbs (Kainer & Fails, 2011; Budras, Sack, Röck, Horowitz & Berg, 2009).

In the horse, only the metacarpal/metatarsal bones two (Mc2/Mt2), three (Mc3/Mt3) and four (Mc4/Mt4) are present. Depending if localized on thoracic or pelvic limbs, they will be referred as metacarpal or metatarsal bones, respectively. The Mc2 and Mc4 or splint bones are rudimental and the Mc3, also known as cannon bone, carries the entire weight assigned to the limb. In the thoracic limbs (forelimbs) the proximal bases of the Mc3 articulate with the carpal bones (carpometacarpal joint) while in the pelvic limbs (hindlimbs) the metatarsal 3 (Mt3) articulates with the distal row of tarsal bones (tarsometatarsal joint). The distal end of the Mc3/Mt3 presents a sagittal ridge that engages a groove in P1 originating the MCP/MTP joint. The distal surface of P1 and the proximal surface of P2 form the proximal interphalangeal joint (IPJ) or pastern joint (Kainer & Fails, 2011; Budras *et al.*, 2009).

The proximal and distal sesamoid bones are of extreme importance in the horse. The PSBs articulate with the Mc3/Mt3, while the distal sesamoid bone, also known as navicular bone, lies within the hoof and articulates with both P2 and P3. The PSBs are part of the fetlock and the digital flexor tendons slide between them, through the fetlock canal. The navicular bone has two articular surfaces that articulate with P2 and P3 and a flexor surface, where the DDFT glides before inserting in P3. The articular facets of the navicular bone, the distal articular surface of P2 and the articular surface of P3 form the distal IPJ or coffin joint (Kainer & Fails, 2011; Budras *et al.*, 2009). Associated to P3, two ungular cartilages provide support to the palmar/plantar aspect of the foot, dissipate forces of the foot's impact with the ground and are involved in venous return from the digit (Dyson, Brown, Collins & Murray, 2010). These cartilages are large rhomboid curved plates that lie under the corium of the hoof and extend from each palmar process of the bone proximal to the coronary border of the hoof (Kainer & Fails, 2011; Mair & Sherlock, 2008).

2.2. Arteries, veins and nerves

The blood supply of the distal limb relies on the medial and lateral palmar/plantar arteries. In the thoracic limb, the medial palmar vein, artery and nerve lie next to each other in this dorsopalmar sequence (Figure 2). The artery and nerve pass through the carpal canal to the metacarpus, where they run medial to the SL and the DDFT. The vein crosses the carpus superficial to the flexor retinaculum, joining the artery and the nerve in the metacarpus. The lateral palmar vein, artery and nerve pass the carpus near the extremity of the accessory carpal bone. The vein and the nerve lie next to each other and deep to them is a small artery (Budras *et al.*, 2009).

In the fetlock region, the medial and lateral palmar vessels and nerves turn into the medial and lateral digital vessels and nerves (Figure 2) (Budras *et al.*, 2009). The digital vessels and

nerves become superficial on the proximal part of the fetlock, covered by superficial fascia. As each digital artery crosses over the fetlock it gives off branches to the fetlock joint, digital extensor and flexor tendons, digital synovial sheath, ligaments, fascia and skin (Kainer & Fails, 2011).

Around the middle of P1, a short artery of P1 arises from the medial and lateral digital arteries, originating an anastomotic circle that divides into dorsal and palmar branches. The palmar branch extends between P1 and the DDFT, while the dorsal branch lies deep to the CDET. At the level of the proximal IPJ the artery of the digital cushion (bulbar artery) arises from each digital artery, supplying the hoof with its branches (Kainer & Fails, 2011; Budras *et al.*, 2009).

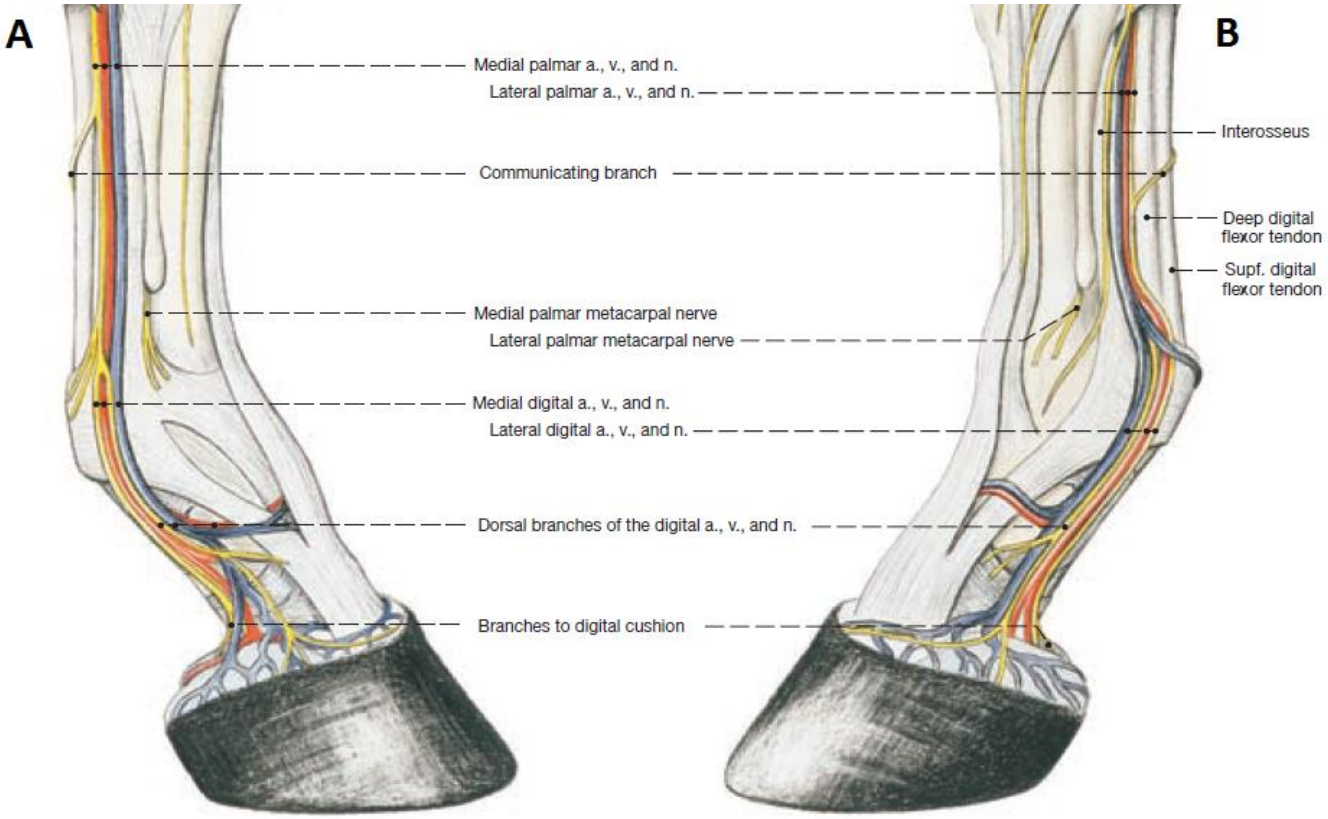
The venous drainage of the foot is assured by two parallel veins in the solar canal that come together at the level of the navicular bone to form the medial and lateral terminal veins. These veins join with branches of an inner plexus becoming the digital veins that carry the blood from the digit (Kainer & Fails, 2011).

At the carpal level, the median nerve splits into medial and lateral palmar nerves. The ulnar nerve also divides in two branches: a dorsal branch that supplies the skin over the dorsolateral aspect of carpus and metacarpus, and a palmar branch that joins the lateral palmar nerve. After joining with the ulnar nerve, the lateral palmar nerve gives off a deep branch that innervates the proximal attachment of the SL and is continued by the medial and lateral palmar metacarpal nerves (Budras *et al.*, 2009). These run along the axial surface of the splint bones and emerge immediately on their distal extremity, ramifying in the superficial fascia of the pastern (Kainer & Fails, 2011).

In the middle of the metacarpus, the lateral and medial palmar nerves form a communicating branch, before descending to the fetlock. In the fetlock, these nerves continue as lateral and medial palmar digital nerves and each one gives off a dorsal branch that crosses between the digital vein and artery (Figure 2). In one third of the cases, an intermediate branch originates from the dorsal branch and together they supply sensory and vasomotor innervation to the fetlock and to the dorsal structures of the digit. The palmar continuations of the nerves supply the fetlock and the palmar structures of the digit. The palmar digital nerves terminate in a fine branch accompanied by a small artery, establishing a neurovascular bundle that descends adjacent to the synovial membrane of the distal IPJ to enter P3 (Kainer & Fails, 2011).

In the pelvic limb, the lateral and medial plantar arteries, veins and nerves continue the caudal branches of the saphenous artery, medial saphenous vein and tibial nerve, respectively. The medial and lateral plantar metatarsal nerves are analogous to the thoracic limb and the medial and lateral dorsal metatarsal nerves are terminal branches of the deep peroneal nerve (Budras *et al.*, 2009). Anatomically, the vessels and nerves of the distal pelvic limb are arranged in a similar way to the distal thoracic limb, with minor differences.

Figure 2. (A) Medial and (B) lateral views of the distal thoracic limb arteries, veins and nerves (adapted from Budras *et al*., 2009).



2.3. Tendons

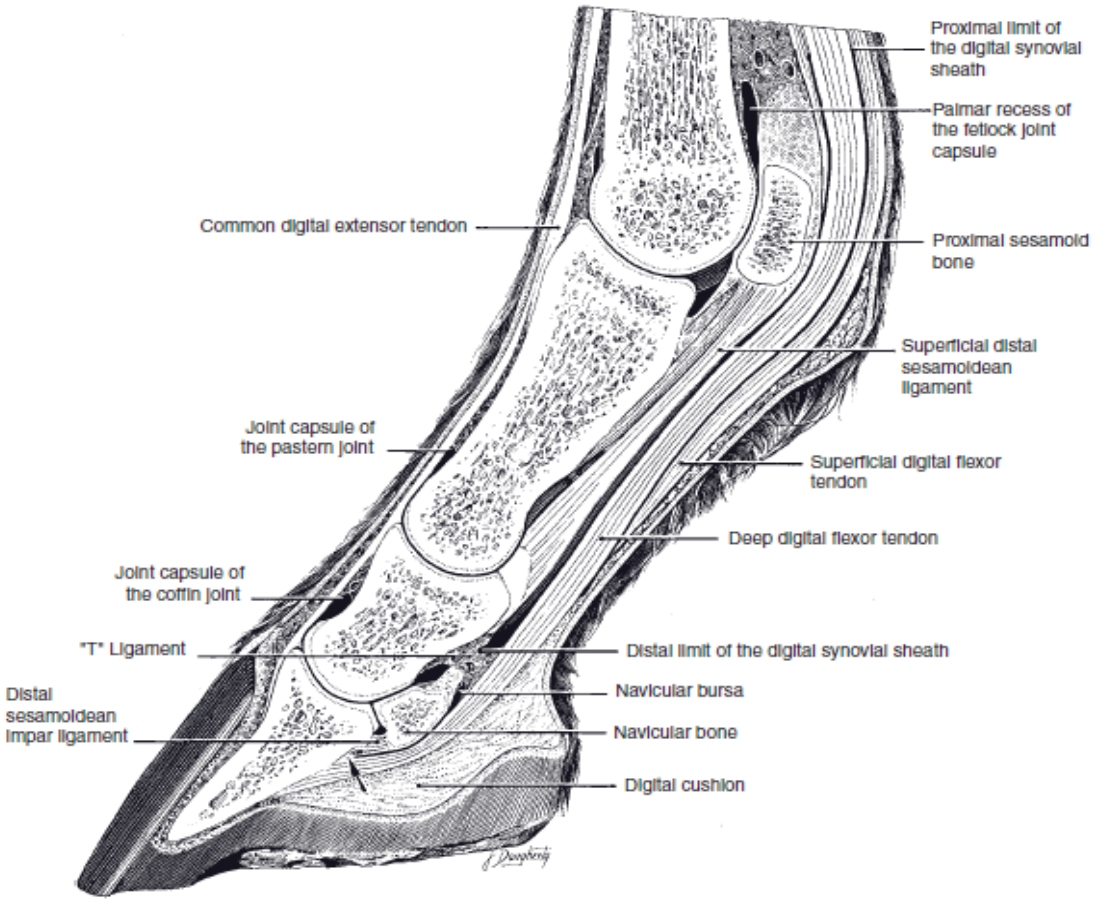
The movements of the digits are assured by the joints, tendons and ligaments. The thickenings of the deep fascia of the equine limb originates the extensor and flexor retinaculums at the level of the carpus. The extensor retinaculum, found in the dorsal surface of the carpus, guides the extensor tendons, while the flexor retinaculum, located in the palmar surface of the carpus, forms the carpal canal by extending from the carpal bones on the medial side to the accessory carpal bone (Budras *et al.*, 2009).

The palmar/plantar surface has an arrangement of three structures: the SDFT and DDFT with their accessory ligaments and the SL. On the dorsal aspect are two or three digital extensor tendons (Budras *et al.*, 2009; Birch *et al.*, 2014). The CDET and the lateral digital extensor tendon (LDET) pass over the dorsal aspect of the fetlock where a bursa is interposed between each tendon and the joint. The tendon of insertion of the common digital extensor muscle inserts partially on the middorsal aspect of the proximal extremities of P1 and P2 on its way to a final insertion on the extensor process of P3 (Figure 3), attaching to the cartilages of P3 by two ligaments. The tendon of insertion of the lateral digital extensor muscle inserts lateral to the partial insertion of the CDET on the proximal middorsal surface of P1 (Kainer & Fails, 2011).

In the equine limb, the tendon of insertion of the superficial digital flexor muscle is located in the palmar surface of the cannon bone, fetlock and pastern (Figure 3). The SDFT terminates by bifurcating into two branches that insert on the proximal extremity of P2, palmar to the collateral ligaments of the proximal IPJ. The tendon of insertion of the deep digital flexor muscle is positioned dorsally to the SDFT, passing between its two branches and inserting in the flexor surface of P3 (Figure 3) (Kainer & Fails, 2011; Budras *et al.*, 2009). Proximal and distal to the MCP/MTP joint the SDFT encircles the DDFT forming the manica flexoria (MF) and the digital manica (DM), respectively (Fiske-Jackson, Barker, Eliashar, Foy & Smith, 2013). The MF extends over 30 mm from the most distal part of the metacarpal/metatarsal region to the apex of the PSBs, being thicker proximally and becoming thinner distally (Seignour, Coudry, Norris & Denoix, 2011). This structure has a free distal margin, the proximal margin is continuous with the tendon sheath and abaxially blends with the SDFT (McIlwraith, Nixon & Wright, 2015). Its function is to maintain the flexor tendons in a central position within the digital flexor tendon sheath (DFTS) (Redding, 1993).

Both flexor tendons have accessory ligaments to attach them directly to bone providing a direct bone to bone connection. Proximally to the carpus the accessory ligament of the SDFT (superior check ligament) makes this connection, while distally it is achieved by the accessory ligament of the DDFT (inferior check ligament) localized in the proximal third of the metacarpus (Birch *et al.*, 2014).

Figure 3. Sagittal section of equine fetlock and digit (adapted from Kainer & Fails, 2011).



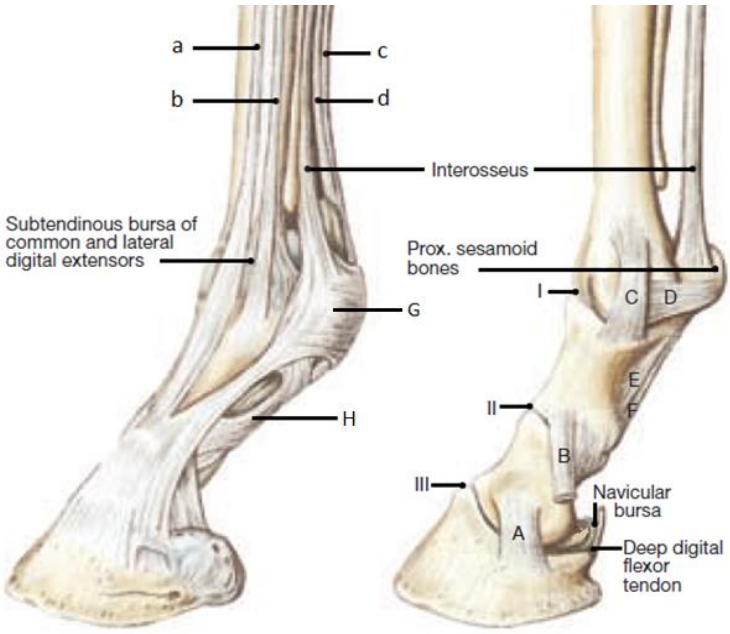
2.4. Ligaments

The ligaments stabilize the joints by transmitting forces between bones. The fetlock joint needs to be stabilized to prevent it from overextending when the weight of the horse is supported by the limb. This is achieved by the suspensory apparatus and the digital flexor tendons. The suspensory apparatus is composed by the PSBs, the SL and the sesamoidean ligaments (Figure 4). The *musculus interosseous medius* tendon (suspensory ligament; SL) arises from the carpus and proximal end of the cannon bone and inserts on the PSBs, where it sends two extensor branches around P1 to the CDET. There are two collateral ligaments in the fetlock that extend distally from the cannon bone and attach to the edge of the articular surface of P1 and to the abaxial surface of the PSBs, maintaining them in position. Between the PSBs, the intersesamoidean ligament (ISL) covers the flexor surfaces of these bones and forms a smooth depression through which the digital flexor tendons pass. The tension in the SL is continued distal to the joint by the short, cruciate, oblique and straight sesamoidean ligaments. The short sesamoidean ligaments extend from the base of both PSBs to the palmar edge of the articular surface of P1, they are the deepest ligaments. The cruciate sesamoidean ligaments (CSLs) cross each other inserting distally to the contralateral eminence on the proximal extremity of P1. The oblique sesamoidean ligaments (OSLs) attach distally to the palmar surface of P1, while the straight sesamoidean ligament (SSL) attaches to the proximal extremity of the palmar surface of P2. The digital flexor tendons assist the suspensory apparatus providing a tendinous support via their accessory ligaments (Kainer & Fails, 2011; Budras *et al.*, 2009).

The thickenings of the deep fascia originate three annular ligaments (Budras *et al.*, 2009). These are the most superficial ligaments and collectively function as a retinaculum in order to hold the flexor tendons in the DFTS at the level of the MCP joint (Cohen, Schneider, Zubrod, Sampson & Tucker, 2008; McGhee, White & Goodrich, 2005). At the level of the fetlock joint, the fascia forms the palmar annular ligament (PAL) (Figure 4), that has transverse fiber orientation and lays immediately beneath the skin and subcutaneous tissue (Seignour *et al.*, 2011). The PAL arises from the abaxial aspect of the PSBs and is most prominent at the palmar aspect, constituting the palmar border of the fetlock canal. The fetlock canal is an inelastic canal formed by the PSBs, the ISL and the PAL, maintaining the flexor tendons in place (Budras *et al.*, 2009; Cohen *et al.*, 2008, Fiske-Jackson *et al.*, 2013; Wilderjans, Boussauw, Madder & Simon, 2003). Distally, the proximal digital annular ligament (PDAL) (Figure 4) is located on the palmar surface of P1 and has an *X* shape, adhering to the SDFT and extending to the medial and lateral borders of the distal aspect of P1 (Budras *et al.*, 2009; Cohen *et al.*, 2008). This ligament covers the SDFT as it bifurcates and aids in holding the DDFT (Kainer & Fails, 2011). The distal digital annular ligament (DDAL) originates between the medial and lateral borders of the distal aspect of P1 and inserts on the palmar

aspect of P3, between the DDFT and the digital cushion. This ligament is crossed medial and laterally by ligaments of the ergot, which diverge from beneath the horny ergot on the palmar surface of the fetlock and reach the DDAL, connecting it to the hoof cartilage (Kainer & Fails, 2011; Budras *et al.*, 2009; Cohen *et al.*, 2008).

Figure 4. Lateral view of equine fetlock and digit. (*a*) Common digital extensor tendon, (*b*) lateral digital extensor tendon, (*c*) superficial digital flexor tendon, (*d*) deep digital flexor tendon; (*I*) fetlock joint, (*II*) proximal interphalangeal joint; (*III*) distal interphalangeal/coffin joint; (*A*) collateral ligament of coffin joint, (*B*) collateral ligament of navicular bone, (*C*) collateral ligament of fetlock joint, (*D*) collateral ligament of proximal sesamoid bones, (*E*) oblique sesamoidean ligament, (*F*) straight sesamoidean ligament, (*G*) palmar annular ligament and (*H*) proximal digital annular ligament (adapted from Budras *et al.*, 2009).



The bones of the proximal IPJ are held together by two short collateral ligaments and four palmar ligaments (axial and abaxial). Oriented vertically, the collateral ligaments join the distal part of P1 to the proximal part P2 on their lateral and medial surfaces. The abaxial palmar ligaments extend from P1 to the palmar surface of P2, while the axial palmar ligaments originate on the triangular surface of P1 and insert in the palmar margin of the proximal part of P2, where they blend with the SDFT and the SSL. The SSL and the digital flexor tendons give additional support to this joint (Kainer & Fails, 2011; Budras *et al.*, 2009).

The distal IPJ is constituted by the articular surfaces of P2, P3 and navicular bone. On its palmar surface, the navicular bone is supported by the navicular suspensory apparatus that consists in three ligaments. Two collateral sesamoidean (suspensory navicular) ligaments arise from the lateral and medial distal ends of P1 and attach to the proximal border of the navicular bone joining each other. Distally, a distal sesamoidean impar ligament supports the navicular bone and extends from its distal border to intersect with the DDFT (Kainer & Fails, 2011). Two short and strong collateral ligaments originate from the lateral and medial

collateral fossae of P2 and are placed on the dorsomedial and dorsolateral aspects of the joint. These ligaments are orientated obliquely, inserting on the collateral fossae of P3 and the dorsal border of the ungular cartilages (Kainer & Fails, 2011; Denoix, Bertoni, Heitzmann, Werpy & Audigié, 2011).

In the pelvic limbs, the fetlock and interphalangeal joints are supported as in the thoracic limbs by the SDFT, DDFT, SL, sesamoidean ligaments and collateral ligaments. There are two differences from the arrangement in the distal part of the thoracic limbs: the inferior check ligament is thinner and may be missing, and the superior check ligament is absent, being compensated by the SDFT firm attachment on the calcanean tuber (Kainer & Fails, 2011).

2.5. Synovial sheaths

As a protective mechanism, the digital flexor tendons are contained within synovial sheaths in regions where they pass over high motion joints: the carpal sheath proximally and the digital sheath distally. The DFTS surrounds the flexor tendons protecting them from shear damage and facilitating their movements. The anatomy of the sheath has been well described and is similar in thoracic and pelvic limbs. It extends from the distal third of the metacarpal region (4 to 7 cm proximal to the PSBs) to the palmar pouch of the distal IPJ as far as the transverse lamina ligament ("T" ligament) containing a very small amount of fluid (Kainer & Fails, 2011; Fiske-Jackson *et al.*, 2013; Schramme & Smith, 2003; Seignour *et al.*, 2012; Wilderjans, Boussauw, Madder & Simon, 2003).

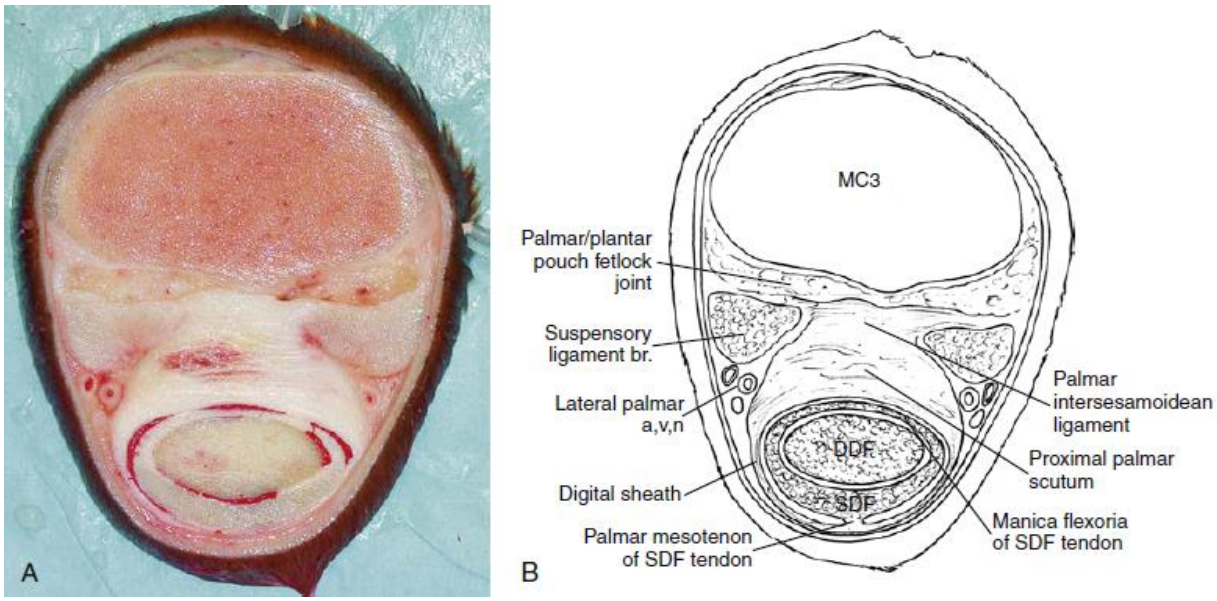
The DFTS is composed of two layers, an outer fibrous layer and an inner synovial layer. The dorsal wall of the sheath is formed by the proximal and middle scutum and distal sesamoidean ligaments, while the palmar wall incorporates the three annular ligaments (Schramme & Smith, 2003). The DFTS is surrounded by the PAL at the level of the fetlock canal (Figure 5), the PDAL along the palmar border of P1 and the DDAL that adheres to the palmar surface of the distal part of the sheath (Wilderjans *et al.*, 2003). Proximal to the MF, the DDFT is attached to the DFTS medially and laterally by the mesotenon or synovial plica. Distal to the MF further mesotenons (vinculae) attach the DDFT to the dorsal sheath wall (SW) (Wright & McMahon, 1999). The SDFT is also attached to the sheath by a thin mesotenon on the palmar aspect of the fetlock (Figure 5) (Wilderjans *et al.*, 2003). The mesotenon between the SDFT and the PAL is the so-called vinculum of the SDFT (Schramme & Smith, 2003).

In case of inflammation the sheath can accumulate fluid and pouch out in any of the places where it is not attached to the annular ligaments: three paired proximal pouches (proximal, proximal collateral and distal collateral pouches) and one distal/palmar pouch. The proximal pouch is proximal to the PAL and palmar to the branches of the SL, the proximal collateral pouch lies between the PAL and the PDAL, and the distal collateral pouch occurs between

the proximal and distal branches of the PDAL. The distal/palmar pouch is found between the distal branches of the PDAL and the proximal border of the DDAL (Davies & Philip, 2007; Liebich, König & Maierl, 2004; Schramme & Smith, 2003).

The “T” ligament is a wide, thin, elastic sheet of fibrous material that originates on the DDFT and inserts on P2, separating the DFTS from the navicular bursa and the palmar pouch of the distal IPJ (Figure 3) (Davies & Philip, 2007). The navicular bursa is a synovial sheath localized between the DDFT and the fibrocartilaginous distal scutum covering the flexor surface of the navicular bone. It provides frictionless movement of the DDFT over the navicular bone, protecting the tendon from wear (Budras *et al.*, 2009; Kainer & Fails, 2011).

Figure 5. (A and B) Cross section of a forelimb 4 cm proximal to the apices of the proximal sesamoid bones, showing the digital flexor tendon sheath and related structures. *DDF*: deep digital flexor tendon; *MC3*: third metacarpal bone; *SDF*: superficial digital flexor tendon (adapted from McIlwraith *et al.*, 2015).



3. TENDON PATHOLOGY

3.1. Factors affecting tendon pathophysiology

Tendons are influenced by several factors, especially age and exercise. The age of the horse and the exercise program can lead to changes in the extracellular matrix that will predispose to tendon degeneration. In long-term exercised older horses regional differences in collagen fibril diameter can be seen, but not in young or short-term exercised ones. Studies showed that exercise accelerates a degenerative change that occurs inevitably with aging, suggesting that cumulative fatigue damage weakens the tendon matrix and allows the initiation of clinical tendonitis when loading overcomes the resistive strength of the tendon (Smith, 2003).

Being submitted to stress during aging leads to a gradual decrease in the mechanical integrity of the tendons. Various stimuli could result in the synthesis, release or activation of proteolytic enzymes that originate an imbalance between the synthesis and degradation of the extracellular matrix and result in accumulation of partially cleaved collagen. The actual mechanism of degeneration of the tendon is currently unknown but the decline of collagen turnover, the energy transmitted to the tendon under weight-bearing load and the rise of the temperature within the tendon can produce direct damage to the matrix by disrupting the tendon ultrastructure (Birch *et al.*, 2014; Smith, 2003; Thorpe *et al.*, 2013).

Thorpe *et al.* (2013) suggested that alongside aging a significant stiffening of the SDFT interfascicular matrix occurs, leading to a decreased capacity of sliding between fascicles. The area occupied by the matrix decreases causing tendons to be more tightly packed in aged individuals. In addition, the improper repair of any tendon damage may result in the formation of adhesions.

Besides the exercise and the age of the horse, there are other risk factors that predispose for tendon injury such as the weight the horse is carrying, its body weight, fatigue, shoeing, working surface (especially hard surfaces) and speed of the horse (the faster the horse is going, the greater the risk of tendinitis) (Birch *et al.*, 2014).

3.2. Tendon injury and healing

Tendons and ligaments can be injured by overstrain or percutaneous penetration/laceration. Overstrain injuries can result from a sudden overloading or continuous degeneration. Clinical injuries occur when the stress encountered by the tendon overwhelms its structural integrity leading to irreversible damage. These strain-induced tendinopathies are usually bilateral with one limb more severely affected than the other (Birch *et al.*, 2014).

Once the tendon suffers clinical injury with disruption of its matrix, there is an acute inflammatory reaction with increased blood-flow, oedema, infiltration of cells (neutrophils, monocytes and macrophages) and release of proteolytic enzymes. This first phase of repair allows the removal of the damaged tendon tissue, lasting one to two weeks. After this, the reparative phase begins, with strong angiogenic response and accumulation of fibroblasts to synthetize scar tissue. The scar tissue is mainly composed by collagen type III, being weaker than the normal tendon tissue. Several months after the injury, the remodeling phase starts and the collagen type III is gradually replaced by collagen type I. This matured scar tissue is stronger than the normal tendon tissue but has poor elasticity, resulting in increased strain in adjacent undamaged regions of the tendon. Consequently, re-injure in the same tendon can occur, but frequently at adjacent or remote sites of the original injury (Birch *et al.*, 2014; Smith, 2003).

3.3. Tendonitis

Clinical overstrain injuries, known as tendonitis, are about 10% of lameness cases in the athletic horses (Kalisiak, 2012). Thoroughbreds and upper level event horses have an increased risk of SDFT injury due to the high speed associated with jumping. Most SDFT injuries caused by athletic use occur in the forelimb mid-metacarpal region (zones 2B to 3B) and appear as a convex "bow" on the side view (Avella & Smith, 2012; Bertone, 2011; Gillis, 2014; Jorgensen & Genovese, 2003). Lesions in the distal cannon bone can be associated with digital sheath tenosynovitis or constriction of the PAL (Bertone, 2011). Regions of the SDFT enclosed within tendon sheaths are less affected, in contrast with the DDFT, which is more frequently injured within the DFTS (Avella & Smith, 2012; Gillis, 2014).

The clinical signs of SDFT injury vary considerably depending on the location, type, severity and timing of the injury. Clinical signs of SDF tendonitis include lameness, swelling, thickening, heat and sensitivity to direct digital palpation. A high palmar nerve block usually resolves the lameness but ultrasonography (USG) is the recommended diagnostic method for tendonitis. It is important to evaluate the echogenicity of the tendons, perform area measurements and compare it with the contralateral limb. One of the greatest manifestations of SDF tendonitis is a central injury ("core" lesion) seen ultrasonographically. The cross-section area (CSA) of the hypoechogenic lesions is the most used parameter to describe tendonitis severity, along with an increase in the maximal tendon CSA and total tendon CSA (Avella & Smith, 2012; Kalisiak, 2012; Jorgensen & Genovese, 2003).

Tendonitis of the DDFT is generally associated to unilateral DFTS effusion and mild to moderately severe lameness. During clinical evaluation, pain can be elicited by palpation and passive flexion of the lower limb. Intrathecal analgesia of the DFTS usually results in significant improvement but low palmar/plantar perineural analgesia shows better results. The definitive diagnosis requires ultrasonographic evaluation that can be difficult to perform with DFTS effusion. DDF tendonitis can display four types of lesions: enlargement and change in tendon shape, focal hypoechogenic lesions within the tendon or on its border, mineralization within the tendon and marginal tears. In some cases, surgical exploration (tenoscopy) may be required for a definitive diagnosis (Dyson, 2003).

The treatment of strain-induced tendinopathies can be nonsurgical and/or surgical. The decision of whether to manage a horse conservatively or surgically should take in account the history, clinical evaluation, response to previous treatments, use of the horse and some other factors. As a nonsurgical approach, pharmacological treatment (systemic and intralesional) and physical therapy such as cold therapy, compression, controlled exercise, extracorporeal shock wave therapy and therapeutic ultrasound are of extreme importance for a successful return to work. The surgical approach allows the exploration of the DFTS with direct observation of the lesions and possibility of debriding adhesions and tears (Avella & Smith, 2012).

3.4. Tendon tears

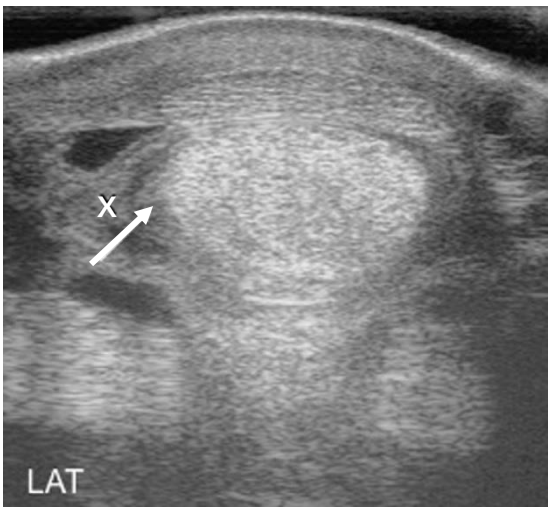
Tendon tears within the DFTS usually occur in the DDFT (predominantly in the forelimbs) and the MF of the SDFT (mainly in the hindlimbs) (Fiske-Jackson *et al.*, 2013; Smith & Wright, 2006). DDFT tears occur when higher pressures within the compressed portion of the tendon passes over the MCP joint leading to its "bursting", more commonly of its lateral border. Manica tears may occur when the MF gets trapped in the fetlock canal, and be exacerbated by synovial hypertrophy, adhesions and/or PAL constriction within the sheath (Avella & Smith, 2012).

MF tears are more common in the hindlimbs and associated to DFTS effusion (Findley, Oliveira & Bladon, 2012; Smith & Wright, 2006). Ultrasonographic diagnosis has a low sensitivity, showing more than one abnormality, such as sheath effusion, PAL thickening and synovial proliferation (Findley *et al.*, 2012; Smith & Wright, 2006; Wilderjans *et al.*, 2003). According to the study undertaken by Fiske-Jackson *et al.* (2013) MF tears are particularly hard to identify ultrasonographically hence contrast radiography performed at the same time as intrathecal analgesia of the DFTS can provide valuable information to an accurate diagnose. Tears of the MF are more frequent on the medial attachment to the SDFT and can be classified as acute or chronic, complete or partial and longitudinal or transverse. This classification is based on appearance during tenoscopic observation (Findley *et al.*, 2012; Smith & Wright, 2006).

Longitudinal tears (LTs) of the digital flexor tendons were first described by Wright and McMahon (1999) as an underlying cause of tenosynovitis of the DFTS. These are seen predominantly in the forelimbs. The most common site of LTs is the lateral margin of the DDFT, followed by the medial margin and the dorsal and palmar/plantar margins in a minority of cases (Arensburg, Wilderjans, Simon, Dewulf & Boussauw, 2011; Smith & Wright, 2006; Wilderjands *et al.*, 2003; Wright & McMahon, 1999). LTs of the SDFT seem to be much less common than the ones in the DDFT (Arensburg *et al.*, 2011; Smith & Wright, 2006).

Horses with DDFT tears are more easily diagnosed with USG and intrathecal analgesia, since they are more likely to show a positive improvement in lameness after diagnostic analgesia than horses with a MF tear (Fiske-Jackson *et al.*, 2013). Typical ultrasonographic changes indicative of LTs were defined as irregular borders of the tendon, hypoechogenic foci and echogenic masses continuous with the tendon's border (Figure 6) (Arensburg *et al.*, 2011; Smith & Wright, 2006; Wilderjands *et al.*, 2003; Wright & McMahon, 1999). Tenoscopy is the most reliable technique used to confirm the presence of LTs and is recommended in all cases (Arensburg *et al.*, 2011; Wilderjands *et al.*, 2003). Since disrupted tendon fibrils cannot be removed from the tendon sheath by intrinsic mechanisms (Wright & McMahon, 1999), tenoscopic debridement and removal of torn fibrils is the most effective treatment, although the long-term prognosis is guarded (Arensburg *et al.*, 2011).

Figure 6. Ultrasonographic image of an acute case of a longitudinal tear (LT) in the lateral border of the deep digital flexor tendon (DDFT). Typical ultrasonographic changes indicative of LTs are visible: irregular lateral border of the DDFT (*arrow*), hypoechogenic foci and an echogenic mass (*x*) continuous with the DDFT border. *LAT* = lateral (adapted from Arensburg *et al.*, 2011).



3.5. Percutaneous tendon injury

The equine distal limb has minimal soft tissue cover, being prone to tendon injuries by trauma. The extensor tendons and the SDFT are positioned directly under the skin therefore small wounds can transect these tendons. Traumatic injuries such as overreaching, wires, landing on sharp objects, jumping and kicking injuries are the most common causes to percutaneous tendon damage (Avella & Smith, 2012; Dyson & Bertone, 2003).

It is of extreme importance to palpate, shave, clean, debride and evaluate the wound, especially its position relative to the synovial structures, since concurrent synovial contamination or sepsis requires specific emergency treatment. If suspected of DFTS contamination synovial fluid can be obtained by synoviocentesis and submitted for cytological and biochemical evaluation. Ultrasonographic examination of the wound and tendons can help to determine the presence of foreign material, the degree of tendon damage and if there is involvement of synovial structures (Dyson & Bertone, 2003).

As medical management, all horses with tendon lacerations need wound cleansing debridement and broad-spectrum antibiotics (Dyson & Bertone, 2003). If the DFTS is involved, surgery should be performed in order to further evaluate the lesions, remove debris, adhesions or foreign bodies, lavage, repair and perform intrathecal administration of antimicrobial drugs. Close monitoring of sheath fluid cytological condition, longer use of systemic and intrathecal antibiotics and immobilization of the limb should be continued post-surgery (Fraser & Bladon, 2004; Dyson & Bertone, 2003).

4. LIGAMENT PATHOLOGY

4.1. Palmar/plantar annular ligament syndrome and desmitis

The PAL is composed of transverse fibers that attach to the PSBs, forming the fetlock canal together with the ISL. PAL syndrome refers to the different diseases that have characteristic clinical manifestation that include PAL thickening and distension of the DFTS. Enlargement of the DFTS or the flexor tendons can lead to constriction within the fetlock canal with a relatively normal PAL. The clinical signs of PAL syndrome consist in persistent lameness accompanying a concave notch at the level of the PAL with the DFTS protruding proximally (Figure 7). In these cases, tenoscopic evaluation for further examination and treatment is advised (McGhee *et al.*, 2005; Schramme & Smith, 2003).

Primary desmitis of the PAL can be caused by traumatic injury, hyperextension of the fetlock with excessive tension on the PAL (Owen *et al.*, 2008; Schramme & Smith, 2003), pressure caused by swelling of the flexor tendons and chronic tenosynovitis with adhesion and fibrosis. Thickening of the PAL with no pathological conditions of the structures within the DFTS can cause lameness in horses since it leads to stenosis of the fetlock canal and constriction of the flexor tendons. This constriction and continuous pressure originate further inflammation, fibrosis, thickening and persistent pain (Schramme & Smith, 2003).

Figure 7. Lateral view of a limb with plantar annular ligament (PAL) syndrome. Note the digital sheath distension with characteristic concave notch of the tendons proximal to the PAL (*arrow*) (adapted from Nixon, 2011).

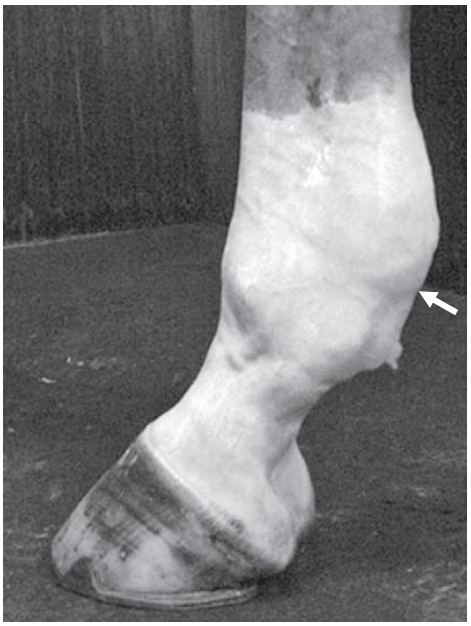
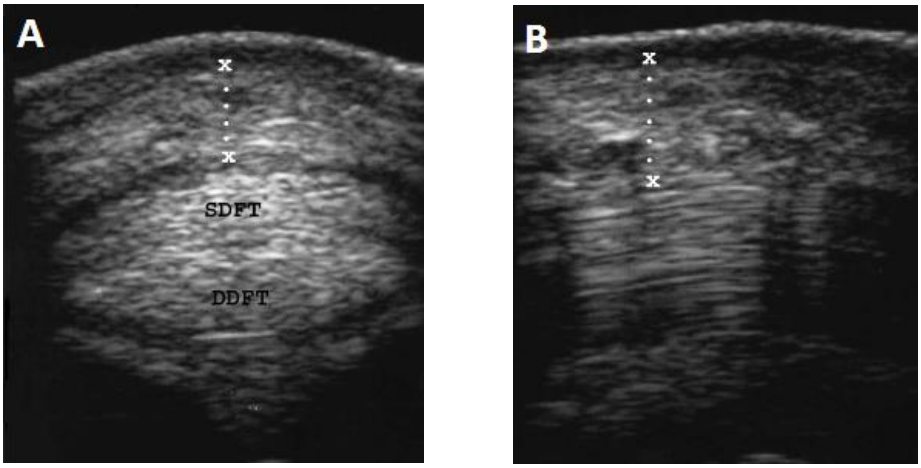


Figure 8. Lateral view of a hindlimb with primary desmitis of the plantar annular ligament. There is a convex contour of the plantar aspect of the fetlock (*arrow*) due to ligament thickening (adapted from Owen *et al.*, 2008).

PAL desmitis and thickening is more common in hindlimbs and results in a convex contour of the palmar/plantar aspect of the fetlock with distension of the DFTS, especially proximal to the PAL (Figure 8). Horses usually have a history of mild to moderate persistent lameness and pain can be elicited by palpation and manipulation of the fetlock. Intrathecal and perineural analgesia of the palmar/plantar nerves can improve lameness associated with this condition (Owen *et al.*, 2008; Schramme & Smith, 2003).

USG is widely used as a diagnostic method for PAL desmitis and should be performed in the lame and the contralateral nonlame limb as comparison. An unaffected PAL can be hard to identify because it is only 1 to 2 mm thick, while a thickened PAL has more than 2 mm (Figure 9) and can have a diffuse or focal decrease in echogenicity, loss of fiber pattern and subcutaneous fibrosis associated. In acute cases, controlled exercise, anti-inflammatory medication and correct farriery can be an effective treatment. In chronic, unresponsive cases and/or with lesions identified within the DFTS, surgery is recommended (Owen *et al.*, 2008; Schramme & Smith, 2003). A minimally invasive approach through a small skin incision and subcutaneous transection of the PAL blindly is quick and simple but tenoscopically guided PAL desmotomy allows a complete examination of the DFTS and the treatment of concomitant pathologies (Schramme & Smith, 2003).

Figure 9. (A) Transverse and (B) longitudinal ultrasonographic images of the fetlock region of a horse with primary desmitis of the palmar annular ligament (PAL). The PAL was 9 mm thick in the transverse image and 10 mm thick in the longitudinal image. *DDFT*: deep digital flexor tendon; *SDFT*: superficial digital flexor tendon (adapted from McGhee *et al.*, 2005).



4.2. Diseases of the intersesamoidean ligament

Damage to the ISL as a cause of lameness in horses is uncommon. When the fetlock overextends, the distal and extensor branches of the SL induce high tension in the PSBs leading to high tension in the ISL. Described diseases of this ligament include desmitis (infectious and non-infectious), rupture, avulsion fracture from the PSBs and enthesopathy.

Injuries in the ISL usually cause acute moderate to severe lameness that can be improved with intrasynovial analgesia of the fetlock joint or DFTS and eliminated by a low four-point nerve block. Ultrasonographic examination can display abnormalities such as enlargement of the space between the PSBs in case of rupture, and enlargement and alteration of echogenicity within the ligament in case of desmitis and tears (Dyson & Genovese, 2003).
Desmitis of the ISL is usually accompanied by axial osteitis of the PSBs, characterized by focal areas of bone lysis and wearing of the ISL in its insertions. This pathology can be associated with infectious tenosynovitis and/or arthritis (Brommer *et al.*, 2014).
Tears in this ligament are rare and particularly difficult to diagnose with USG. A definitive diagnose and treatment can only be achieved by arthroscopy of the palmar/plantar pouch of the MCP/MTP joint and/or tenoscopy of the DFTS. Horses with desmitis and/or focal tears of the ISL have poor response to the surgical treatment even when associated with conservative therapy (Dyson & Genovese, 2003).

4.3. Straight and oblique sesamoidean ligaments desmitis

The SSL and the OSLs are part of the suspensory apparatus, holding the PSBs to P1 and P2 (Sampson *et al.*, 2007; Smith, Dyson & Murray, 2008). Desmitis of the SSL as cause of lameness in horses is rare and can occur together with desmitis of one or both OSLs (Dyson & Genovese, 2003).
Clinical signs include acute onset of lameness with no palpable abnormalities in the region and usually without swelling, although swelling and DFTS effusion can develop after a few days. Lameness can be eliminated with an abaxial sesamoid nerve block (Gillis, 2014; Dyson & Genovese, 2003; Sampson *et al.*, 2007; Smith *et al.*, 2008).
The diagnosis is based on USG even though the overlying of the digital flexor tendons impairs the ultrasound image (Sampson *et al.*, 2007; Smith *et al.*, 2008). Desmitis of these ligaments can be recognised in USG as enlargement of the ligament, diffuse reduction in echogenicity, focal hypoechogenic areas, poor delineation of the margins of the ligament, reduction of the distance between the ligament and the DDFT/SDFT and sheath effusion (Dyson & Genovese, 2003). Radiography might reveal bone changes in the insertions of the ligaments (Gillis, 2014). For an accurate diagnose, magnetic resonance imaging seems to be the best diagnostic method. Treatment for desmitis of the SSL and/or the OSL consists of box rest and corrective farriery followed by a rehabilitation program with controlled exercise (Dyson & Genovese, 2003; Sampson *et al.*, 2007). Occasionally, lesions of the SSL can disrupt the palmar/plantar surface of the ligament and covering synovium, leading to tenosynovitis. In these cases, tenoscopic examination is indicated for debridement and treatment (McIlwraith *et al.*, 2015).

5. DIGITAL FLEXOR TENDON SHEATH PATHOLOGY

5.1. Ganglion cyst

Ganglion cysts are small cysts (1.5 to 3 cm diameter) composed of fibrous or fibromyxoid tissue that does not hold synovium. Originated from degenerated connective tissue, these cysts are connected to a joint or tendon sheath. Their common localization is proximally in the DFTS just proximal to the PAL. Lameness and DFTS effusion caused by ganglion cysts in horses is an uncommon condition. To establish the clinical significance of the cyst, ultrasound, perineural anaesthesia and intrathecal analgesia with contrast radiographs are required. Tenoscopy and surgical removal of the cyst has a good prognosis for soundness if there is no concurrent pathology of the tendons (Crawford, O'Donnell, Crowe, Eliashar & Smith, 2010; Schramme & Smith, 2003).

5.2. Non-infectious tenosynovitis

DFTS effusion is common in working horses, frequently idiopathic in origin and affecting both limbs without causing lameness. In some cases this effusion is seen in only one limb along with lameness. The term tenosynovitis refers to inflammation of the synovial membrane of a tendon sheath and may be a single clinical entity or be associated to a tendon injury (Schramme & Smith, 2003; Wright & McMahon, 1999).

Non-infectious tenosynovitis of the DFTS has been attributed to many causes, for example tendonitis of the SDFT and DDFT, desmitis of the PAL and PDAL, LTs of the SDFT and DDFT, MF tears and chronic synovitis of the DFTS of unknown cause (Smith & Wright, 2006; Wilderjans *et al.*, 2003). These conditions are likely to result in chronic tenosynovitis due to continuous irritation to the sheath by the exposed torn collagenous tissue (Wright & McMahon, 1999). Complex tenosynovitis has been described as tenosynovitis with thickening of the PAL, synovial distension and adhesions/synovial masses (Schramme & Smith, 2003).

Diagnostic techniques that localize disease to the sheath include synoviocentesis and synovial fluid analysis, intrathecal analgesia and positive contrast radiography (Schramme & Smith, 2003). USG is the most commonly used technique for evaluating the DFTS, although it has poor accuracy for lesion prediction, especially for the presence of marginal tears of the DDFT, SDFT and MF (Smith & Wright, 2006). Tenosynovitis has been described in three ultrasonographic stages of progression: stage 1, with symmetrical distention of the DFTS and without evidence of synovial proliferation; stage 2, often asymmetrical distension of the proximal pouch with synovial proliferation; and stage 3, extensive synovial proliferation with adhesions and synovial masses in the sheath (Schramme & Smith, 2003).

The treatment of horses with acute tenosynovitis consists of systemic anti-inflammatory medication, bandage immobilization, cold therapy and rest. In the chronic cases, aspiration of the synovial fluid and intra-thecal administration of hyaluronan and corticosteroids might be necessary. In unresponsive cases and in the ones of complex tenosynovitis tenoscopic exploration is indicated (Schramme & Smith, 2003).Tenoscopy is necessary to identify accurately the presence of lesions and their morphologic features, allowing an appropriate treatment and a better prognosis (Smith & Wright, 2006).

5.3. Infectious tenosynovitis

Infectious tenosynovitis is a critical condition in the horse that can lead to death or euthanasia. Horses with DFTS infection usually present with severe, non-weight-bearing lameness with heat, pain and diffuse swelling. Contamination results from the introduction of microorganisms usually by penetrating wounds, although it may also develop following intra-thecal injections or as result of extension from adjacent tissues. Rarely haematogenous spread to the digital sheath may result from bacteremia (Bertone, 2011; Schramme & Smith, 2003; Wereszka, White & Furr, 2007). Infection follows when the microorganisms proliferate and colonize the sheath, leading to an acute inflammatory response with rapid influx of neutrophils and other inflammatory cells (McIlwraith *et al.*, 2015).

An early recognition and prompt treatment are crucial, since it is difficult to eliminate infection from the DFTS and there is a high risk of long-term sequela that contributes to permanent lameness. Clinical examination, radiography and USG are useful to recognize complicating factors such as foreign bodies, concurrent tendon and ligament injuries, osteomyelitis and infectious tendinitis. Synoviocentesis should be performed as early as possible since it allows collection of a synovial fluid sample for culture and sensitivity testing, and cytological analysis (Bertone, 2011; Schramme & Smith, 2003; Wereszka *et al.*, 2007). Negative bacteria culture results do not necessarily rule out infection because bacteria can be sequestered in neutrophils, fibrin or synovium, and previous treatments can inhibit their growth (Schramme & Smith, 2003; Wereszka *et al.*, 2007).

Treatment of infectious tenosynovitis must consist of aggressive systemic and intrathecal broad-spectrum antibiotics with lavage of the sheath. Tenoscopy should be performed in order to lavage the sheath (either with plain fluids or diluted antimicrobials), debride fibrin and adhesions, remove foreign bodies and sometimes transect the PAL to provide pain relieve in cases where there is excessive fluid distension and thickening of the SW. The portals are usually closed but in specific cases can be left open for drainage. Bandaging and immobilization of the limb promote wound healing and reduce inflammation; on the other hand, it limits drainage and promotes the formation of scar tissue and adhesions (Schramme & Smith, 2003; Wereszka *et al.*, 2007).

In horses with extensive contaminated wounds, these can be cleaned and kept under sterile bandages to allow healing by second intension. It is of extreme importance to maintain the intrathecal antimicrobial concentration within therapeutic concentrations over an extended period of time. That can be achieved with intrathecal administration, intravenous regional perfusion of antibiotics, antibiotic infusion pumps and slow release antibiotic-depot systems in collagen or polymethylmetacrylate (Schramme & Smith, 2003). According to Fraser and Bladon (2004), treatment within 36 hr of initial injury leads to a better prognosis for the horse's return to athletic function.

6. DIAGNOSTIC METHODS

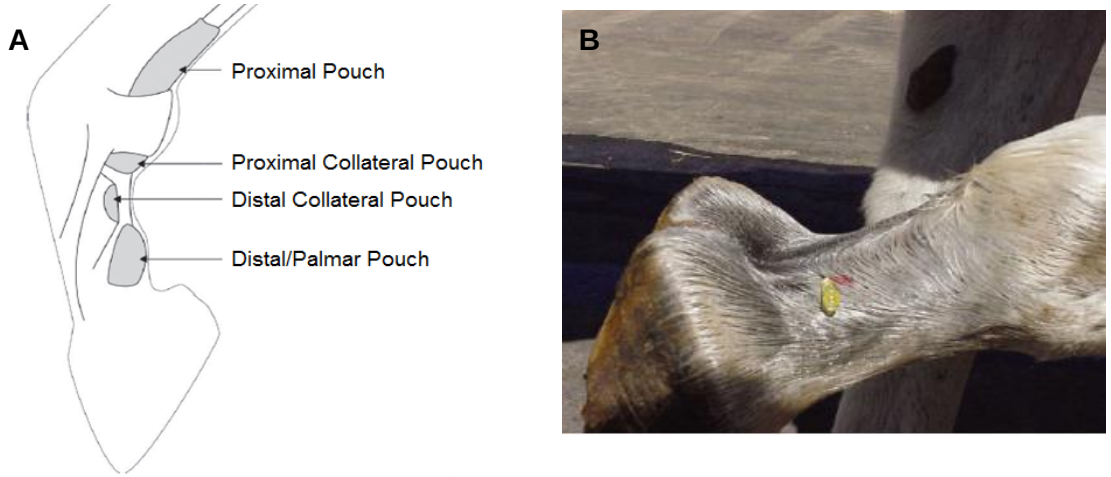
6.1. Synoviocentesis

Synoviocentesis or tenovaginocentesis is usually performed for intrathecal administrations and to determine if a wound has penetrated the sheath, especially soon after injury or in small self-closing wounds. This technique can be done in one of the DFTS pouches (Figure 10, A), being easier if the sheath is distended (Dykgraaf *et al.*, 2007; Schramme & Smith, 2003).

The easiest access to the sheath is through its distal/palmar pouch (Figure 10, B), localized between the distal branches of the PDAL and the proximal border of the DDAL, along the palmar surface of the DDFT. When the sheath is distended, access to the proximal pouch is possible by introducing a needle along the dorsal aspect of the DDFT, between the DDFT and the lateral branch of the SL, proximal to the lateral PSB and PAL. Synoviocentesis of the DFTS can also be done from its proximal or distal collateral pouches (Schumacher, Schumacher, Schramme, Degraves & Smith, 2007; Moyer, Schumacher & Schumacher, 2007; Schramme & Smith, 2003). Recent studies show that this technique is more successful when performed in an axial (axial to the PSB) or a distal (at the pastern) approaches (Jordana, Oosterlinck, Pille, Valère & Martens, 2012).

After a sterile preparation of the sheath (clipping and surgical scrub), a 19 or 20 gauge, 2.5 cm (1 inch) needle is introduced, the fluid is collected with a syringe and placed into a sodium EDTA tube for cytological analysis (Mahaffey, 2002; Dykgraaf *et al.*, 2007; Schumacher *et al.*, 2007). The fluid can also be placed into a plain tube for culture and sensitivity testing. The normal DFTS synovial fluid is clear yellow with total protein (TP) concentration values less than 10 g/L and total nucleated cell count (TNCC) inferior than 2 x 10^9cells/L (Mahaffey, 2002; Schramme & Smith, 2003). A TNCC higher than 30 x 10^9 cells/L with more than 80 to 90% neutrophils and TP greater or equal to 40 g/L is considered pathognomonic of infection (Dykgraaf *et al.*, 2007; Schramme & Smith, 2003; Wereszka *et al.*, 2007).

Figure 10. (A) Representation of the digital flexor tendon sheath (DFTS) pouches. In this image, the distal and the lateral proximal pouches are distended. (B) Access to the DFTS through the distal pouch. The needle should be aimed between the deep digital flexor tendon and the ipsilateral distal branch of the superficial digital flexor tendon (adapted from Schumacher *et al.*, 2007).



6.2. Diagnostic analgesia

Diagnostic analgesia is one of the most valuable tools to localize lameness. The technique can be performed on site with minimal equipment and expense, allowing a fast assessment of the outcome (Bassage II & Ross, 2003).

To accurately localize lameness to the DFTS, perineural (nerve blocks) and intrathecal analgesia are of extreme importance. Because of the DFTS extension, regional analgesia of the medial and lateral palmar/plantar digital nerves proximal to the DFTS (abaxial sesamoid nerve block) can be non-specific, resulting in partial responses (Baxter & Stashak, 2011; Fiske-Jackson *et al.*, 2013). Presently, mepivacaine hydrochloride 2% is the most frequently used anaesthetic for regional and intrasynovial anaesthesia due to its long lasting activity and low tissue irritation (Jordana *et al.*, 2016).

Analgesia of the sheath is considered more specific and efficient since it desensitizes the structures within, localizing the source of pain to the DFTS. This can be achieved by intrathecal administration of 10 to 15 mL of mepivacaine hydrochloride 2% in one of the sites where synoviocentesis can be performed (Baxter & Stashak, 2011; Moyer *et al.*, 2007; Schramme & Smith, 2003). Even when the pathology is localized within the sheath, partial responses to intrathecal analgesia are common, especially in horses with MF tears as shown by Findley *et al.* (2012) and Fiske-Jackson *et al.* (2013). Furthermore, it has been shown that diffusion of mepivacaine from the DFTS to adjacent synovial structures occurs but the concentrations are insufficient for synovial analgesia (Jordana *et al.*, 2016), suggesting that the improvement of lameness in horses that undergo intrathecal analgesia of the DFTS is due to analgesia of the structures within the sheath and not adjacent to it (Harper, Schumacher, Degraves, Schramme & Schumacher, 2007).

6.3. Radiography

Radiography of the DFTS is an important diagnostic method for tendon sheath pathology. It is usually performed to make evident the existence of intrathecal air when a penetrating wound is present, to visualize foreign materials, soft-tissue swelling and metaplastic mineralization of structures usually associated with sheath pathology, and to study concomitant bone injury (Farrow, 2006; McIlwraith *et al.*, 2015; Schramme & Smith, 2003).

Contrast radiography of the DFTS (negative and positive-contrast tenography) is an easy technique to perform and by outlining the interior of the sheath, it provides information regarding tendon sheath perforation, thickening, stenosis, adhesion, intra and extrasynovial masses, mass effects and foreign bodies (Farrow, 2006). Positive-contrast tenography can be achieved by injecting 5 to 10 mL of sterile iodine solution in the DFTS while performing intrathecal diagnostic analgesia, followed by a lateromedial radiograph of the distal limb including the whole length of the DFTS (Fiske-Jackson *et al.*, 2013; Schramme & Smith, 2003). The contrast is uptaken by the proximal pouch dorsal to the DDFT, and distal to it, the MF, DDFT and SDFT are visible as filling defects outlined by contrast. Distal to the PSBs the contrast accumulates in the collateral pouches and extends from the distal pouch to the "T" ligament (Fiske-Jackson *et al.*, 2013).

Contrast radiography was a widely used diagnostic method before the advances and widespread of USG (Valdéz-Martinéz & Park, 2011). With this diagnostic technique it is possible to identify pathologies such as MF and DDFT tears (Fiske-Jackson *et al.*, 2013), synovial herniations, intrasynovial communications (Valdéz-Martinéz & Park, 2011), ganglion cysts (Crawford *et al.*, 2010) and perforation of the sheath (Schramme & Smith, 2003). According to Fiske-Jackson *et al.* (2013) positive contrast radiography delineates the border of the MF accurately allowing the diagnosis of MF tears with a sensitivity of 96%, exceeding that of USG. Currently, the use of contrast radiography has declined but it still remains a useful method for the preoperative diagnosis of DFTS pathology, especially if combined with USG and the results of diagnostic analgesia (Fiske-Jackson *et al.*, 2013).

6.4. Ultrasonography

Diagnostic USG was introduced in the early 1980s to equine veterinarians as a practical diagnostic technique to evaluate soft tissue injuries. Over the last decades ultrasound technology has improved, becoming the imaging modality of choice for soft tissue evaluation, especially tendons and ligaments. A correct knowledge of the normal anatomy, adequate equipment, limb preparation and scanning skills of the ultrasonographer are critical for a reliable diagnose (Rantanen, Jorgensen & Genovese, 2003; Redding, 2011).

After localization of the lameness by clinical examination and, if necessary, the use of nerve blocks, USG should be done. In some cases, diagnostic analgesia might interfere with the exam by causing air artifact and inflammation (Redding, 2011). USG can be performed any time after the injury, but the optimal reference scan for assessing severity is about 1 week after the onset of the clinical signs since many lesions expand during the initial few days leading to a less accurate diagnosis (Avella & Smith, 2012).

The horse's restrain and preparation are crucial for good-quality images. The hair should be clipped using a No. 40 blade, then the limb cleaned and scrubbed, followed by application of acoustic coupling gel. When clipping of the hair is not possible, the area should be cleaned and washed before topical application of alcohol, which enhances the sound transmission. For ultrasonographic examination of the soft tissues of the equine limb, a focus linear array transducer (probe) with a frequency of at least 7.5 MHz or higher should be used, since it decreases distortion and artefact creation, giving superior images at tissue depths of 2 cm or less. Linear probes facilitate the evaluation of longitudinal tendon and ligament fiber alignment parallel to the skin surface, and have strand-off pads available that are essential to image the most superficial structures (Rantanen *et al.*, 2003; Redding, 2011).

As the horse stands squared, a thorough and schematic examination of both limbs is advised, so that each structure is visualised and compared with the contralateral limb in its complete length, proximal to distal in longitudinal and transverse planes. The imaging protocol of the metacarpus is based on its length being about 3 hands-widths (8 cm/hand breadth), so it is divided in 3 zones, each zone subdivided into 2 zones of 4 cm each: 1A, 1B, 2A, 2B, 3A and 3B (Figure 11). The metatarsus has approximately 4 hands-widths length, being divided in 4 zones, each zone subdivided into 2 zones of 4 cm each: 1A, 1B, 2A, 2B, 3A, 3B, 4A and 4B. The area associated with the PSBs is considered zone 3C in the forelimb and 4C in the hindlimb. Some authors prefer to use a simple numerical scheme from 1 to 7 in the forelimb and 1 to 9 in the hindlimb. At the level of P1, the pastern is divided in 3 zones, each with 2 cm: P1A, P1B and P1C. The P2 is shorter, being divided only in 2 zones with 2 cm each: P2A and P2B (Figure 12) (Rantanen *et al.*, 2003; Redding, 2011).

At every metacarpal/metatarsal level the skin, subcutaneous tissue, SDFT, DDFT, SL and surface of Mc3/Mt3 can be scanned. Their position, shape and size vary along the levels (Figure 11), reinforcing the idea that correct anatomy knowledge is needed (Rantanen *et al.*, 2003). In the forelimbs, the DFTS is found at the level 3A continuing to level P1C. Its thickness can be accessed at levels 3A and 3B, where the capsule is an echogenic band dorsal to the DDFT and MF. The MF can be found in zone 3B, where the SL bifurcates into its medial and lateral branches. At level 3C, the PAL is visualized as a thin (1 to 2 mm) echogenic band immediately adjacent to the palmar surface of the SDFT (Rantanen *et al.*, 2003; Schramme & Smith, 2003).

Figure 11. Ultrasonographic imaging protocol of the metacarpus. Schematic representation of its 7 levels (1 to 7): 1A, 1B, 2A, 2B, 3A and 3B. The area associated with the proximal sesamoid bones is considered zone 3C. The structures identified from the probe to the palmar metacarpus are the superficial digital flexor tendon (*SDFT*), deep digital flexor tendon (*DDFT*), suspensory ligament (*SL*) and accessory ligament of the DDFT (*ALDDFT*) (adapted from Redding, 2011).

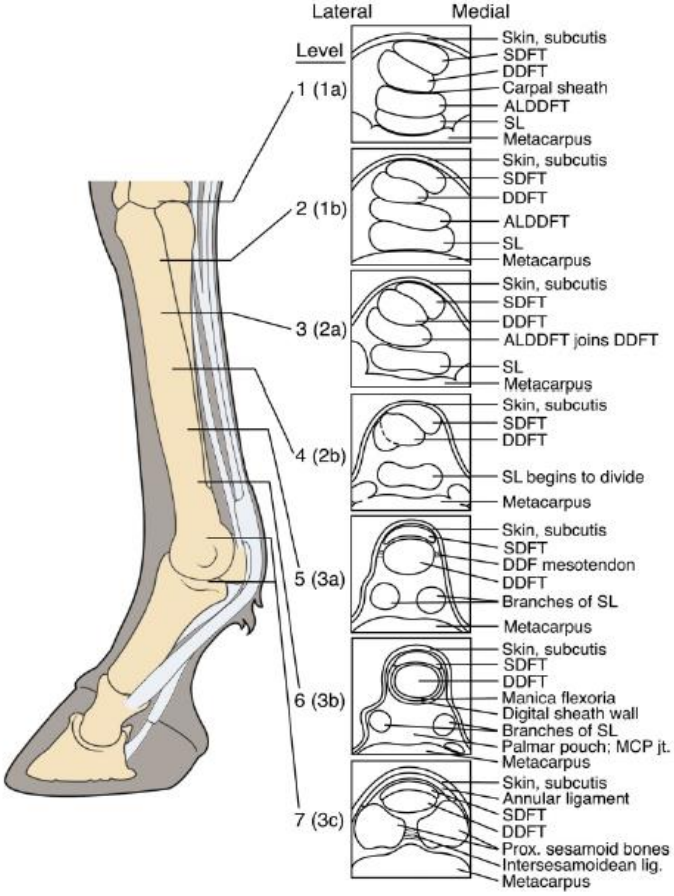
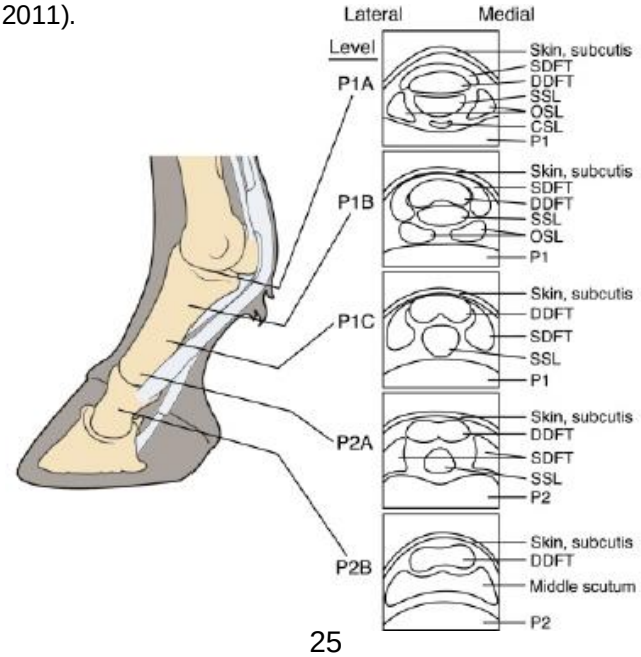


Figure 12. Ultrasonographic imaging protocol of the proximal and middle phalanxes. Schematic representation of the levels P1A, P1B, P1C, P2A and P2B. The structures identified from the probe are the superficial digital flexor tendon (*SDFT*), deep digital flexor tendon (*DDFT*), cruciate sesamoidean ligaments (*CSL*), straight sesamoidean ligament (*SSL*) and oblique sesamoidean ligaments (*OSL*) (adapted from Redding, 2011).



In the pastern zones (Figure 12), the PDAL can be seen in the palmar/plantar surface of the DDFT in zone P1A, where the SSL and a small portion of the CSLs may be identified. The OSLs are visualized in close proximity to the palmar/plantar surface of P1, dorsal to the SSL in zone P1B. The SSL continues to zone P1C, being found on the dorsal surface of the DDFT and having its fibrocartilaginous insertion in zone P2A. In zone P2A a thin DDAL is seen in the palmar/plantar border of the DDFT (Rantanen *et al.*, 2003).

The prognosis of tendinopathies is most dependent on the severity of the initial damage, being prudent to evaluate, describe and classify the injuries during the lameness assessment (Avella & Smith, 2012). A systematic approach to the defined zones including both qualitative and quantitative analysis should be used to define the injuries and to compare with follow-up examinations (Rantanen *et al.*, 2003). The use of dynamic USG is beneficial in improving the diagnostic certainty, being achieved by maximally flexing and extending the MCP and the IPJs while scanning (DiGiovanni, Rademacher, Riggs, Baumruck & Gaschen, 2016).

Tendon and ligament injury can be recognised ultrasonographically by changes in size, shape, position, echogenicity, fiber pattern (echotexture) and surrounding inflammatory reaction. The lesions should be classified based on their location (core, peripheral, radial and diffuse), length, alteration in echogenicity, pattern of altered echogenicity (focal, diffuse, homo and heterogenous), alteration of fiber pattern (in longitudinal section), percent CSA of tendon injury and changes of the lesion over time. These findings should be analysed in conjunction with the clinical signs, comparing with images of the contralateral limb (Farrow, 2006; Rantanen *et al.*, 2003). The CSA measurements are considered a very sensitive indicator of inflammation and can be assessed from a frozen image on the screen (Redding, 2011). The CSA of the hypoechogenic lesions, maximal tendon and total tendon CSAs are the parameters used to describe injury severity (Avella & Smith, 2012; Kalisiak, 2012; Jorgensen & Genovese, 2003). Acute tendinopathy can exhibit enlargement, hypoechogenicity (focal or generalized), reduced fiber pattern, changes in position, margins and/or shape, and associated edema and effusion in the DFTS. Furthermore, chronic tendon pathology is associated with irregular striated pattern (indicative of fibrosis) and variable changes in echogenicity and size of the tendon (Avella & Smith, 2012).

Even though USG is currently the widest used diagnostic tool for the evaluation of equine tendons and ligaments, it is not without limitations. The quality of the image is directly related to the anatomical area being examined, the equipment settings and the operator skills in positioning, defining the appropriate settings and recognizing structures and artifacts (Redding, 2011). It has been determined that tenoscopy reveals tendon lesions that are not detected on ultrasonographic evaluation (Smith & Wright, 2006; Wilderjans *et al.*, 2003).

Presently, USG remains a practical, inexpensive and accessible imaging technique for soft tissue injuries in the horse but magnetic resonance imaging is now considered the gold standard diagnostic technique for lameness with origin in the distal limbs (Redding, 2011).

6.5. Surgical procedure

Endoscopy of a synovial tendon sheath, the so-called tenoscopy, is a minimal invasive surgical technique that offers comprehensive evaluation of the DFTS. This technique has many advantages by permitting minimally invasive surgical approaches leading to decreased morbidity, simplified aftercare, rapid return to normal function of the sheath and reduced risks of complications such as infection, fibrosis, ankylosis and wound breakdown (Cauvin, 2003; McIlwraith *et al.*, 2015). Recently, this surgical approach has become a routine technique in equine surgical practice, making a major contribution to the understanding and diagnosis of tendon sheath pathology (McIlwraith *et al.*, 2015).

The DFTS is the most common site of tenosynovitis (Cauvin, 2003); hence horses presenting it with pain on palpation and associated lameness should be considered candidates for tenoscopic examination (Avella & Smith, 2011). A tenoscopic approach to the DFTS allows the exploration of the structures within the sheath with direct observation of previously unreported lesions, confirmation of lesions diagnosed by other diagnostic methods and early treatment of the previous (McIlwraith *et al.*, 2015).

6.5.1. General technique and equipment

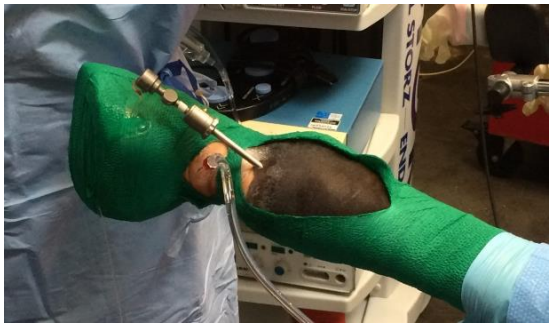
Tenoscopy requires general anaesthesia and can be performed with the horse in lateral or dorsal recumbency. Lateral recumbency is preferred in most cases, with the affected limb uppermost for a lateral approach and the distal limb free to permit variation of joint angles. The use of an Esmarch bandage and tourniquet applied to the proximal metacarpal/ metatarsal level and moderate flexion of the distal joints facilitates the entry into the sheath (McIlwraith *et al.*, 2015).

A standard arthroscopic equipment is used, including a rigid 4 mm diameter with a 25º to 30º lens videoarthroscope or direct-view arthroscope, video camera, digital image capture and storage device, fiberoptic light cable and light source, fluid irrigation system (usually a motorized pump), 5 to 6 mm diameter cannula and conical obturator, 2 to 3 mm diameter egress cannula and locking trocar (sharp stylet or conical obturator), probes, Dyonics 5.2 mm suction punch rongeurs, Ferris-Smith arthroscopic rongeurs, biopsy punch rongeurs, arthroscopic scissors, hook knives and motorized synovial resectors (McIlwraith *et al.*, 2015).

Nixon's (1990) tenoscopic approach using a single portal for evaluating the DFTS has been adopted as the standard. The term palmar is used in the following description for either palmar or plantar, since the approach is similar in both fore and hindlimbs. When the tendon sheath is not distended enough, 10 to 20 mL of sterile saline solution should be administered through a needle inserted in the palmar aspect of the mid-pastern region in order to safely entering the sheath (Cauvin, 2003; McIlwraith *et al.*, 2015). The standard tenoscopic portal is

created immediately distal to the PSB and about 6 mm palmar to the neurovascular bundle, between the PAL and the PDAL (Figure 13). First, a small skin incision is made with a scalpel, continued by a stab incision using a No. 11 or 15 blade to penetrate only the SW. Following this, the arthroscopic cannula is introduced through the portal using a conical obturator, and directed proximally through the fetlock canal between the DDFT and the SW, avoiding iatrogenic trauma to the tendons. The obturator is then replaced by the arthroscope, the light source and fluid lines are connected and systematic examination is carried out (Cauvin, 2003; McIlwraith *et al.*, 2015). As the fluids are running, the sheath is lavaged through an 18 gauge needle inserted in the proximal pouch (Cauvin, 2003). The instrument portals are created where appropriate by passing a spinal needle for accurate location before the incision, avoiding the annular ligaments (McIlwraith *et al.*, 2015).

Figure 13. Standard tenoscopic approach to the digital flexor tendon sheath. Left hindlimb with a fluid line inserted in the plantar aspect of the mid-pastern region and standard lateral arthroscopic portal with a cannula placed, before inserting the arthroscope (Original illustration).



A detailed knowledge of the normal tenoscopic anatomy of the DFTS and its structures is vital, since normal anatomical structures such as vinculae, endotenon and synovial folds should not be damaged or confused with pathological changes (Cauvin, 2003).

A systematic “layer-by-layer” approach is recommended by McIlwraith, Nixon & Wright (2015), to completely evaluate the sheath. After creating the tenoscopic portal, the arthroscope should be positioned in the proximal recess of the sheath and by rotation of the lens and gradual withdrawal, the dorsal SW, dorsal surface of the MF and its distal free margin can be visualized (Figure 14). Further withdraw of the arthroscope maintaining it in the dorsal region will show the dorsal surface of the DDFT when viewing palmarly, and the PSBs and ISL when viewing dorsally (Figure 14). Then, the scope is repositioned to place its tip distally and the DDFT, DM and paired vinculae come to image (Figure 15).

The arthroscope should be reoriented proximally for a second visualization of the sheath, but this time the tip of the scope should be passed between the free margin of the MF and the DDFT, showing the medial and lateral mesotenon of the DDFT. After that, the arthroscope can be rotated over the abaxial margin of the DDFT leading its tip to the space between the DDFT and the SDFT. In this position, by rotating the lens and moving slightly the scope it is

possible to assess both medial and lateral reflections of the MF from the SDFT and the surfaces of both tendons. The tip of the scope is then pointed distally to evaluate the dorsal and the palmar surfaces of the SDFT and the DDFT, respectively (Figure 15). In this pathway, close to the midpoint of P1 it is possible to visualize the free margin of the SDFT isthmus and the dorsal and axial surfaces of the insertion branches of the SDFT. The DDFT is further inspected as far as the reflection of the SW onto the tendon's surface, lining the proximal margin of the DDAL. Rotating the scope over the abaxial margin of the DDFT allows the visualization of the distal cul-de-sac of the sheath, which is typically covered by synovium superimposing the "T" ligament. While in this position, withdrawal and angulation of the lens images the synovium overlying the proximal IP scutum, the SSL and the branches of insertion of the SDFT (Figure 15). To complete the sheath examination, the scope should be positioned again proximal to the fetlock to run outside of the MF on the abaxial surface of the SDFT to visualize it as far as its vinculum. For visualization of specific lesions the arthroscope may need to be introduced through a proximal instrument portal, leaving the tenoscopic portal free for instrument entry (McIlwraith *et al.*, 2015).

Figure 14. Tenoscopic views looking proximally, showing multiple regions from dorsal to the flexor tendons to the palmar surface of the sheath. *S*: superficial digital flexor tendon; *D*: deep digital flexor tendon; *MF*: manica flexoria; *AL*: palmar annular ligament; *Ses*: lateral proximal sesamoid bone; *M*: midline attachment of the mesotenon (adapted from McIlwraith *et al.*, 2015).

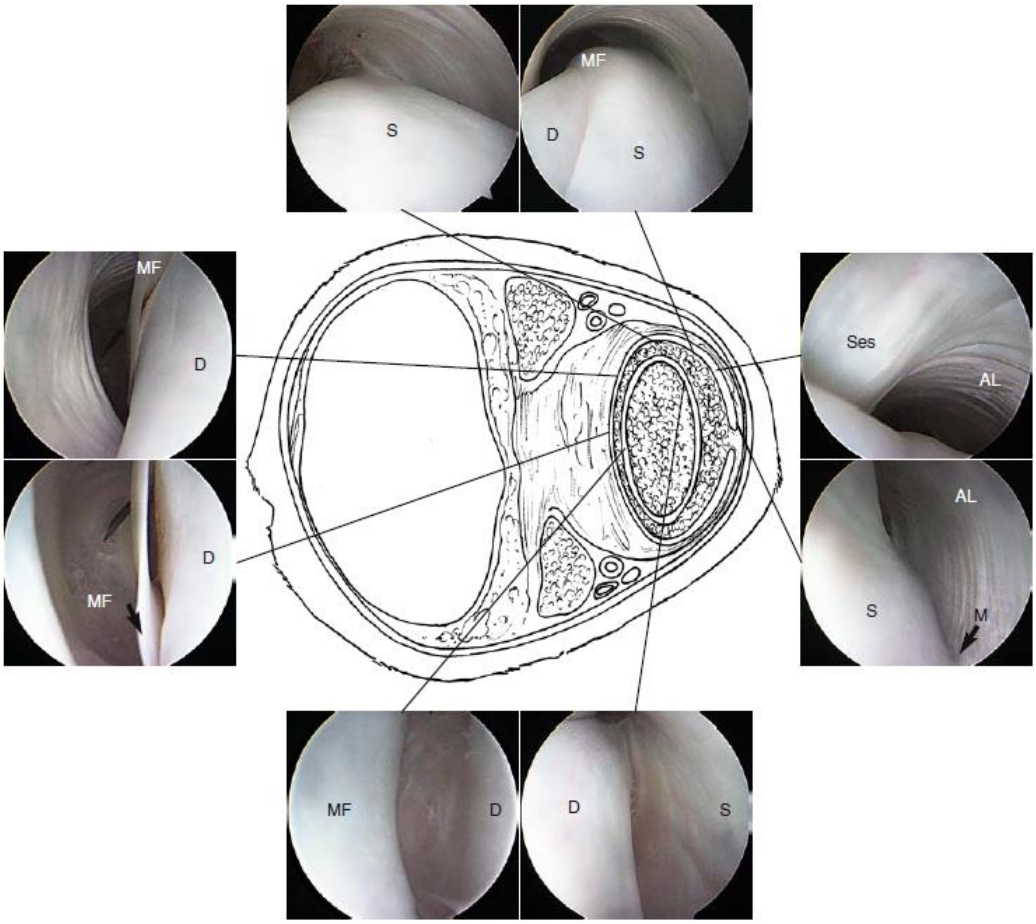
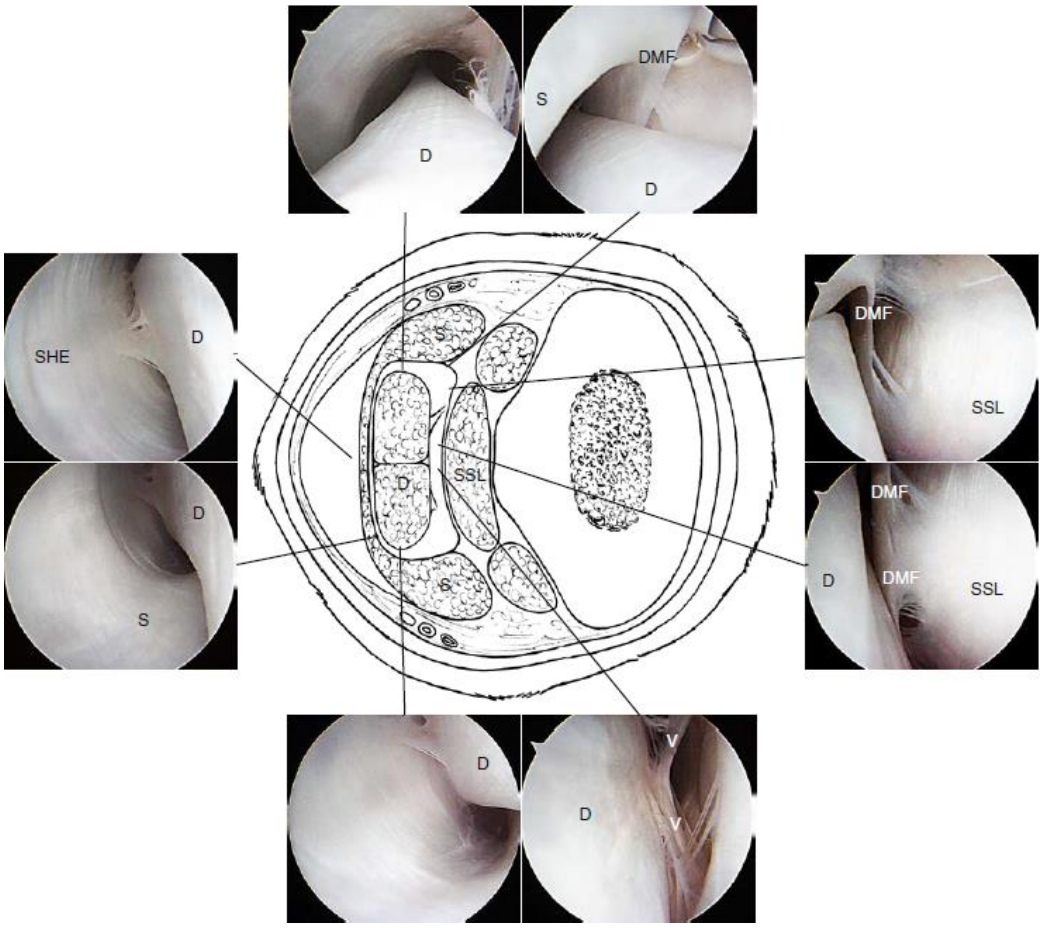


Figure 15. Tenoscopic views with the arthroscope reversed to visualize distal regions of the digital sheath. *S*: superficial digital flexor tendon; *D*: deep digital flexor tendon; *SHE*: sheath; *DMF*: digital manica flexoria; *SSL*: straight sesamoidean ligament; *V*: vinculae (adapted from McIlwraith *et al.*, 2015).



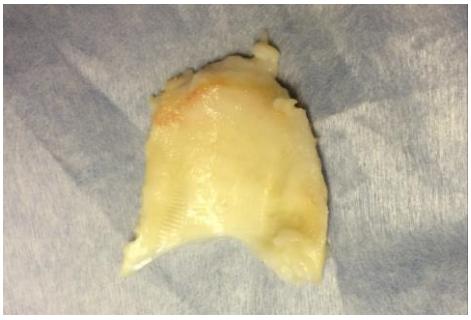
When finishing the surgery, the sheath is lavaged and evacuated through a large-bore cannula. The portals can be closed with simple interrupted sutures using material of the surgeon's choice, and a pressure bandage should be applied for 5 to 7 days post-operatively (Cauvin, 2003).

6.5.2. Tears of the manica flexoria

Tears of the MF can occur as solitary lesions or with concomitant marginal tears of the DDFT or SDFT, and are more frequently found in the hindlimb medial attachment to the SDFT (Findley *et al.*, 2012; Smith & Wright, 2006). During tenoscopic examination the tear can be classified as acute, detached MF with no substantial adhesions, or chronic, thickened and deformed on the site of the tear and adhered to the sheath (Findley *et al.*, 2012). When a complete tear occurs, the MF usually recoils, folding into the intact contralateral attachment to the SDFT and in some cases becoming adherent to the SW. If the tear is partial, the free distal margin of the MF may wrinkle and a space between this structure and the DDFT may be observed (McIlwraith *et al.*, 2015).

When a partial tear is present, debridement of the torn fibrils should be done with suction punch biopsy rongeurs, Ferris-Smith arthroscopic rongeurs and/or a motorized synovial sector in oscillating mode with suction applied. If the tear is complete, total resection of the MF is advised by separating its intact border from the SDFT using arthroscopic scissors or meniscectomy knives, before section of its proximal synovial attachment. Most of the times another portal is created so that the MF can be grabbed with Ferris-Smith arthroscopic rongeurs, stabilized and pulled (Findley *et al.*, 2012; McIlwraith *et al.*, 2015).
The prognosis for the majority of horses that undergo tenoscopic surgery due to MF tears is considered reasonable (Smith & Wright, 2006) and a complete removal has been associated with a better outcome (Avella & Smith, 2011).

Figure 16. Completely removed manica flexoria. The lateral border of the manica flexoria was acutely torn and partially separated from the superficial digital flexor tendon (Original illustration).



6.5.3. Longitudinal tears of the digital flexor tendons

As described in the tendon pathology section, LTs occur more frequently in the lateral margin of the DDFT (Arensburg *et al.*, 2011; Smith & Wright, 2006; Wilderjans *et al.*, 2003; Wright & McMahon, 1999), being more easily diagnosed by USG than MF tears (Fiske-Jackson *et al.*, 2013). Tenoscopic examination of the DFTS is recommended to confirm the presence of LTs and to treat them. LTs can be classified as superficial (< 5 mm) or deep (> 5 mm) (Arensburg *et al.*, 2011; Wilderjans *et al.*, 2003) and localized proximal, distal or in the fetlock canal, being considered long if extending for two regions. Long tears are more common in the fetlock canal and proximal to it, while short tears are more frequent distally (Smith & Wright, 2006).
The goal of the surgical treatment is the removal of the torn fibrils, which reduces the surface area of exposed disrupted collagenous tissue to the synovial sheath, decreasing the inflammatory response and endorsing the development of a viable scar. The torn fibers are managed by the same principles and techniques as the partial MF tears. The margin of the tears can be covered by granulomata (granulation tissue with varying degrees of organization) and in those cases, the large masses should be dissected free with arthroscopic scissors before removal with Ferris-Smith arthroscopic rongeurs. The

instrument portals are created according to the lesion location, for proximal lesions the access has to be from a portal abaxial to the MF (McIlwraith *et al.*, 2015).
The prognosis for LTs after tenoscopical intervention appears to be reasonably fair, but considered worse than for other lesions. Negative prognostic features include obvious pre-operative distension of the DFTS, long duration of clinical signs, presence of a long tear and persistence of post-operative distension, indicating incomplete healing (Smith & Wright, 2006).

6.5.4. Complex tenosynovitis

Complex tenosynovitis has been described as lesions involving two or more structures within or adjacent to the DFTS with usually the PAL being one of them (McIlwraith *et al.*, 2015). This pathology may result from chronic tenosynovitis with low grade inflammation, synovial distension, thickening and fibrosis of the sheath, PAL syndrome and tendon deformation. Adhesions and synovial masses secondary to SW or tendon disruption are a common feature (McIlwraith *et al.*, 2015; Schramme & Smith, 2003).
The treatment of complex tenosynovitis is achieved by removing tenoscopically the synovial masses and adhesions, and performing a PAL desmotomy to facilitate the movement through the fetlock canal. Synovial masses can be resected using arthroscopic scissors, biopsy punch rongeurs, straight and curved motorized synovial resectors and some other instruments. A second instrument portal might be needed if the mass is large, to provide tension on the mass while it is separated from the synovium. Adhesions are commonly synoviosynovial and their removal is achieved using biopsy punch rongeurs and/or motorized resectors (McIlwraith *et al.*, 2015). After performing a PAL desmotomy and removing the synovial masses and adhesions, the prognosis for horses with complex tenosynovitis is favourable, unless there are associated tendon injuries, which are a negative feature (Schramme & Smith, 2003).

6.5.5. Palmar/Plantar annular ligament desmotomy

PAL syndrome and primary desmitis have been associated with tenosynovitis as primary or secondary to it (McIlwraith *et al.*, 2015). When the PAL is causing sheath constriction and impeding the normal gliding between the tendons a desmotomy is advised (Avella & Smith, 2011). It is believed that desmotomy of the PAL releases the sheath constriction and decreases inflammation, allowing a better tenoscopic access and examination of the structures within the DFTS (McIlwraith *et al.*, 2015).
Tenoscopically, desmotomy of the PAL can be performed by a “free-hand” division or using a slotted cannula, being done abaxially between the PSB and the palmar/plantar reflection of the sheath (McIlwraith *et al.*, 2015). Extrathecal techniques such as an approach through a small skin incision and blind subcutaneous transection of the PAL have been described as

quick and simple, but the tenoscopically guided technique is considered better since it allows direct visualization of the sheath and treatment of concurrent pathologies (Schramme & Smith, 2003). The decision between “free-hand” or slotted cannula techniques can be done during the initial examination of the sheath, considering that the use of slotted cannula is advantageous for the most chronic and constricted fetlock canals. The “free-hand” technique can be achieved by creating a standard arthroscopic portal and an ipsilateral proximal instrument portal abaxial to the MF (the portals can be interchanged for a better visualization). The desmotomy should be done using a curved meniscectomy knife or a right-angled blade, in a distoproximal direction and between the PSB and the reflection of the sheath to the SDFT (McIlwraith *et al.*, 2015).

Tenoscopic desmotomy of the PAL, with the aim of relieving intrathecal restriction, is considered a nonharmful procedure and has been encouraged in the treatment of complex tenosynovitis (Nixon, 2003), MF tears and LTs of the digital flexor tendons, exposing the sheath contents and improving its tenoscopic evaluation (Wilderjans *et al.*, 2003; Wright & McMahon, 1999). Constriction of the PAL is often secondary to chronic inflammation and distension of the sheath; hence since 2003 desmotomy should be reserved for chronic cases or cases with obvious thickening of the ligament (Arensburg *et al.*, 2011; Smith & Wright, 2006).

6.5.6. Contaminated and infected tendon sheath

Infectious tenosynovitis is a critical condition in the horse that has high risk of long-term sequela and permanent lameness (Bertone, 2011; Schramme & Smith, 2003). The treatment of a contaminated and infected sheath consists in the removal of foreign material, debridement of torn and infected tissue, elimination of microorganisms, effective lavage and restoration of a normal intrathecal environment (Wright, Smith, Humphrey, Eaton-Evans & Hillyer, 2003).

In most cases, initial high pressure lavage (500 mL/min) of the sheath is needed (McIlwraith *et al.*, 2015) in order to wash the inflammatory exudates, cellular debris and fibrin (Wereszka, *et al.*, 2007). This can be achieved by creating multiple ingress and egress portals, and moving the scope around and between the tendons. The fluid of choice is sterile buffered polyionic solution, but antimicrobial drugs, antiseptics, dimethyl sulfoxide and fibrinolytics can be added to it. After the lavage, a thorough and prompt examination of the sheath is important since foreign material can migrate and deposit at any site. Standard tenoscopic portals can be used, and when wounds or punctures are present, they can work as instrument and egress portals (McIlwraith *et al.*, 2015).

Removal of foreign material is of extreme importance as it acts as a substrate for bacterial growth and causes physical and biochemical irritation to the sheath, perpetuating the

inflammation. Ferris-Smith rongeurs for large pieces and motorized synovial resectors for small pieces are used for the removal of foreign material and detached contaminated tissue, and debridement of torn fibers, pannus and wounds (Wright *et al.*, 2003). A final lavage should be performed and traumatic wounds must be debrided or excised to a clean state so that they can be closed, minimizing the risk of further contamination and/or infection. If needed, the limb can be immobilized by a cast to improve wound healing (McIlwraith *et al.*, 2015).

6.5.7. Post-operative management

After a tenoscopic surgery, a pressure bandage from foot to distal carpus/tarsus should be applied to reduce motion and prevent sheath swelling and contamination (Cauvin, 2003). It is advised that the bandaging continues between 2 to 4 weeks after surgery. Perioperative administration of antibiotics and nonsteroidal anti-inflammatory drugs is usually performed to the surgeon's preference. In the case of infectious tenosynovitis and presence of wounds the antibiotics sometimes have to be continued for more days or changed according to sensitivity testing. Preferably, microbiological samples should be taken before antimicrobial therapy and empirical treatment implemented until the results are available. Monitoring the synovial fluid and clinical signs, such as improvement of lameness, reduced swelling and surface temperature, helps to determine the appropriate duration of the antimicrobial treatment (McIlwraith *et al.*, 2015).

The horse should be restricted to box rest in a clean environment until the sutures are removed around 10 to 14 days postoperatively (Avella & Smith, 2011). Hand-walking can be started and gradually increased according to the surgeon's discretion. The duration of the rehabilitation protocol depends on the severity of the injury and clinical response (Avella & Smith, 2011; McIlwraith *et al.*, 2015).

TENOSCOPIC EXAMINATION OF THE DIGITAL FLEXOR TENDON SHEATH: A RETROSPECTIVE ANALYSIS OF 86 HORSES (2016)

MATERIALS AND METHODS

Tenoscopy has become a routine procedure in equine surgical practice since it is a minimal invasive surgical technique that offers comprehensive evaluation of the DFTS, carpal and tarsal sheaths, and carpal and tarsal extensor tendon sheaths. Injuries to the digital flexor tendons are among the leading causes of clinical disability in horses, therefore tenoscopic examination of the DFTS is of great relevance. In spite of all this, there are relatively few publications regarding this subject.
In this study, medical records regarding tenoscopic examination of the DFTS were analysed for non-infectious and infectious tenosynovitis, and traumatic injuries to the sheath.

1. Inclusion criteria

The medical records from four British equine hospitals (BEVC, DGVG, NEH and VEH) were reviewed for horses that underwent tenoscopic examination of the DFTS in 2016.
Ninety-three tenoscopies were performed in 86 horses and the author was present in 12 of them. Two horses had two surgeries and one horse had three, nevertheless, for statistical significance only the first surgery of each horse was included in this study. In most cases data was collected regarding the horse's age, gender, breed, history, clinical signs and clinical findings: affected limb, diagnostic analgesia (perineural and intrathecal), cytological analysis of synovial fluid, radiographic and USG abnormalities, tenoscopic findings, surgical treatment and peri-operative treatments.
Since some of the cases were referred to the Hospitals for surgery, all information was obtained from the medical records. Therefore, clinical follow-up information was not available and for most cases it was not possible to collect the data regarding the current use of the horse, diagnostic methods and long term outcome.

2. Data classification and analysis

Diagnostic analgesia and synovial fluid analysis results were reviewed. Radiologic, ultrasonographic and tenoscopic findings were studied and summarized.
All horses were evaluated tenoscopically under general anaesthesia. Standard arthroscopic material and surgical techniques as previously described in the chapter "Surgical procedure" (pp. 27-34) were used, with small variations according to the surgeon performing the surgery.

MF tears were classified in acute (detached with no substantial adhesions) or chronic (thickened, deformed and adhered to the sheath wall), partial or complete and regarding the affected border (medial, lateral and distal). Concerning DDFT LTs, these were categorized according to their location (proximal, distal or within the fetlock canal), length (long or short), depth (superficial or deep) and affected border (medial, lateral, dorsal or palmar/plantar). PAL constriction was studied as caused by primary PAL disease or secondary to DFTS and/or tendon pathology. Additionally, traumatic injuries were characterized by affecting only the SW or also the ligaments and/or tendons. In the latest, the location of the injuries was recorded (medial, lateral, palmar or dorsal).

In the cases where synoviocentesis was performed, samples were considered positive if at least two of the analysed parameters were pathognomonic of contamination.

For analysis, 5 age groups were defined: 0-5, 6-10, 11-15, 16-20 and >20. Gender definition included: male (colts and stallions), gelding and female. Horse breeds were grouped into Thoroughbreds, Warmbloods, Coldbloods, Ponies and unknown. Specific information regarding each horse can be conferred in Annex 1. Comparisons were made concerning different pathologies and between the affected limb (forelimb versus hindlimb), characteristics of specific lesions, age, gender and breed of the horses for each diagnosis.

To determine whether various factors were associated with the diagnosed pathologies, analyses were made in R version 3.3.2 (R Development Core Team, 2010). Pearson's Chi-squared tests were performed to identify significant associations between the pathologies diagnosed and the dependant variables: affected limb (forelimb versus hindlimb), age class, gender and breed group of the horse. A significance level of $P < 0.05$ was assumed for all tests.

The sensitivity of some the diagnostic tests was determined and the 95% confidence interval (CI) for the proportion of positives (*p*) obtained from all the horses submitted to the test (*n*) was calculated using the normal approximation with the following formula:

$$p \pm 1{,}96\sqrt{\frac{p(1-p)}{n}}$$

RESULTS

1. Case details and clinical features

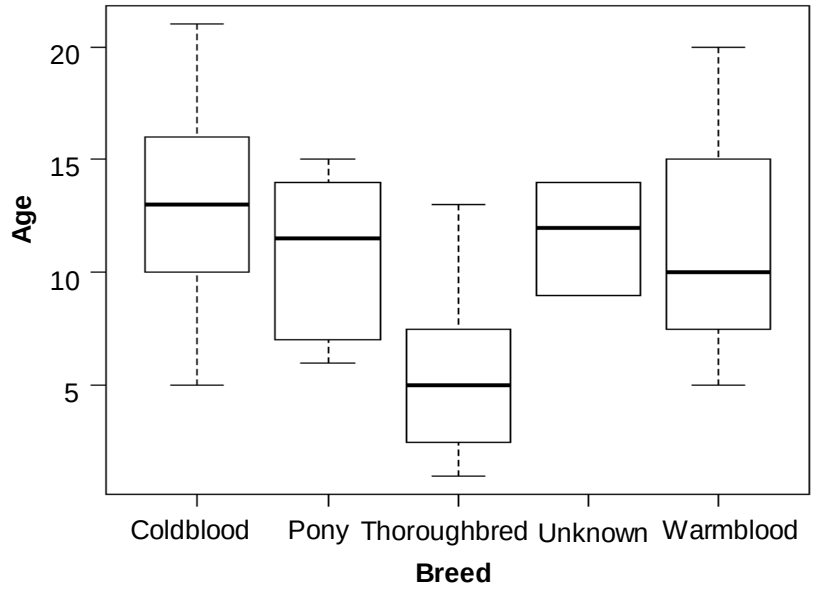
Eighty-six horses were included in this study. The age range was 0.5-21 years, with a mean of 9.8 years and median of 10 years. Nine percent were males (n = 8), 55% geldings (n = 47) and 36% females (n = 31). Thirty-one percent were Thoroughbreds (n = 27), 28% Warmbloods (n = 24), 20% Coldbloods (n = 17), 12% Ponies (n = 10) and 9% of unknown breed (n = 8). The age, gender, breed and affected limb are summarised in Table 1. Age distribution according to breed is represented in Graphic 2.

Seventy-nine (92%) horses had surgery in only 1 limb, 2 (2%) had it in both forelimbs, 4 (5%) in both hindlimbs and 1 (1%) in the right forelimb and hindlimb. Thus, 93 DFTS were affected: 24% right forelimbs (RFs) (n = 22), 15% left forelimbs (LFs) (n = 14), 30% right hindlimbs (RHs) (n = 28) and 31% left hindlimbs (LHs) (n = 29).

Table 1. Summary of age, gender, breed and affected limb of 86 horses that underwent tenoscopic examination of the digital flexor tendon sheath.

Age (years)	No. (%)	Gender	No. (%)	Breed	No. (%)	Limb	No. (%)
0-5	16 (19%)	Male	8 (9%)	Thoroughbred	27 (31%)	RF	22 (24%)
6-10	31 (36%)	Gelding	47 (55%)	Warmblood	24 (28%)	LF	14 (15%)
11-15	26 (30%)	Female	31 (36%)	Coldblood	17 (20%)	RH	28 (30%)
16-20	12 (14%)			Pony	10 (12%)	LH	29 (31%)
> 20	1 (1%)			Unknown	8 (9%)		
Total	86 (100%)		86 (100%)		86 (100%)		93 (100%)

Graphic 2. Age distribution according to breed.



Eighty-four (98%) horses were lame at the time of clinical examination and 87 (94%) limbs had a visible distended DFTS. Flexion tests were recorded for 24 limbs and considered positive in 21 (88%).

Three horses (4%) were euthanized, 2 while still under anaesthesia due to extensive tendon damage, and 1 after its third surgery given the lack of response to aggressive surgical and medical therapy.

2. Diagnostic analgesia

Diagnostic analgesia for the assessment of the lameness source was recorded in 23 horses. The results are represented in Table 2.

Perineural analgesia of the palmar/plantar nerves (abaxial sesamoid) resulted in improvement in lameness in 4 out of 8 (50%) horses. This nerve block was performed in 1 forelimb and 7 hindlimbs, being positive in 4 hindlimbs. The 4 horses that had no significant improvement of lameness after the abaxial sesamoid nerve block all improved after a low 4 point nerve block. Positive response to perineural analgesia was seen in horses with PAL thickening (n = 6), SDFT tears (n = 1) and ganglion cysts (n = 1).

Intrathecal analgesia of the DFTS was performed in 22 horses and 19 (86%) were thought to have a positive response. Three forelimbs and 19 hindlimbs were submitted to intrathecal analgesia, from which the 3 forelimbs and 16 hindlimbs had a positive response. These included horses diagnosed with MF tears (n = 10), PAL thickening (n = 5), SDFT tears (n = 2) and DDFT tears (n = 2).

Seven horses had the abaxial sesamoid and the intrathecal blocks, 3 (43%) were positive to both, 2 (29%) were only positive to the intrathecal analgesia, 1 (14%) was only positive to the abaxial sesamoid block and 1 (14%) was negative to both.

Table 2. Results of diagnostic analgesia performed in 23 horses.

Response to Diagnostic Analgesia	Abaxial Sesamoid Nerve Block		Low 4 Point Nerve Block		Intrathecal Analgesia	
	Forelimb	Hindlimb	Forelimb	Hindlimb	Forelimb	Hindlimb
Positive	-	4	1	3	3	16
Negative	1	3	-	-	-	3
Total	8		4		22	

3. Synoviocentesis

The results of synovial fluid analysis were available for 22 horses and are represented in Table 3. Mean TNCC was 45.9 x 10^9 cells/L (range, 0.6 to 161.5 x 10^9 cells/L), mean TP was 38.9 g/L (range, 8 to 70 g/L) and mean proportion of neutrophils was 82.2% (range 10 to 97.4%). For these calculations values expressed as an interval were excluded. Eight (36%) samples contained ≥ 30 x 10^9 nucleated cells/L, 11 (50%) had TP ≥ 40 g/L and 14 (64%) had ≥ 80% neutrophils.

Sixteen of the 21 horses (76%) had synoviocentesis performed after a traumatic injury. Nine horses had it done in the first 24 hr after injury, 5 only after 24 hr and there is no information regarding 2 horses.

Table 3. Results of synovial fluid analysis and bacteria culture of 22 horses.

Horse ID	TNCC (x 10^9 cells/L)	TP (g/L)	Neutrophils (%)	Appearance	Bacteria Culture
4	3.6	38	-	as blood	-
6	8.4	22	75	bloodstained	-
7	10.3	8	97	-	No growth
14	3.4	36	89	slightly cloudy ambar	-
16	61	50	-	bloodstained	No growth
28	5.8	13	72	slightly bloodstained	-
31	153	52	95	cloudy straw	Moderate growth of β-Hemolytic Streptococci and Coagulase-Negative Staphylococci
39	84.4	50	-	-	-
40	40.4	20	89	-	-
41	161.5	50	91	-	No growth
42	139	-	-	-	-
43	51.9	40	97.4	-	-
49	20-50	70	95	slightly bloody	-
50	0.6	32	10	clear	-
52	25-50	42	90	slightly cloudy	-
57	20-50	37	80	cloudy	No growth
62	< 5	55	90	slightly cloudy	Moderate growth of *Pasteurella* spp.
68	6,4	31	60	sanguineous	No growth
71	> 50	46	95	cloudy	No growth
76	27.4	49	90	cloudy yellow	No growth
79	10.6	26	80	bloodstained	-
84	12.3	49	85	cloudy yellow	No growth

4. Radiography

In the records, 47 horses were radiographed before surgery. Thirty-three (70%) had plain radiographs, 9 (19%) had contrast radiographs after intrathecal administration of contrast and 5 (11%) had both. No significant abnormalities were present in 27 of the 38 (71%) plain radiographs. Identified lesions included gas shadows within the DFTS (n = 8) (Figure 19), small radiopacity zones in the superficial soft tissues in the region of the PAL (n = 2) (Figure 17), foreign bodies (n = 1) (Figure 18) and fragmentation of the lateral PSB (n = 1).

Fourteen contrast tenograms were assessed and no significant abnormalities were seen in 5 (36%). Eight horses had variations in the normal distribution of the contrast, such as abnormalities in the orientation of the MF (n = 6) (Figure 20), no uptake of contrast up past the fetlock (n = 2) (Figure 21) and atypical contrast uptake in the location of the SDFT distal to the MF (n = 1).

Table 4. Radiographic findings of 47 horses.

Radiography	No.	Contrast Tenogram	No.
No significant abnormalities	27	No significant abnormalities	5
Gas shadows	8	Abnormalities in the orientation of the MF	6
Small radiopacity zones	2	No uptake of contrast up past the fetlock	2
Fragmentation of the lateral PSB	1	Atypical contrast uptake in the location of the SDFT	1
Foreign bodies	1		
Total	39		14

Figure 17. Lateromedial radiography of Horse 85. It is possible to visualize two small radiopacity zones in the superficial soft tissues in the region of the plantar annular ligament (courtesy of NEH).

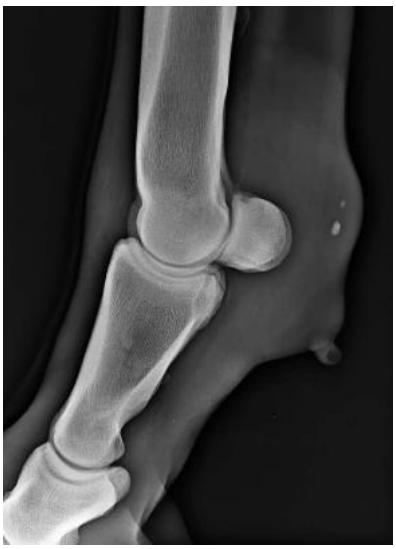


Figure 18. Lateromedial radiography of Horse 51. There is a piece of wire embedded within a circumferential wound in the pastern (courtesy of NEH).

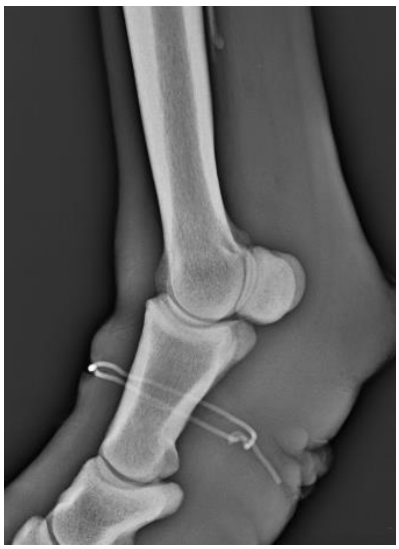


Figure 19. (A) Lateromedial and (B) oblique dorsomedial-palmarolateral projections of Horse 28 fetlock. The horse presented with a laceration of the tendon sheath that was confirmed radiographically by the presence of gas within the sheath (courtesy of DGVG).

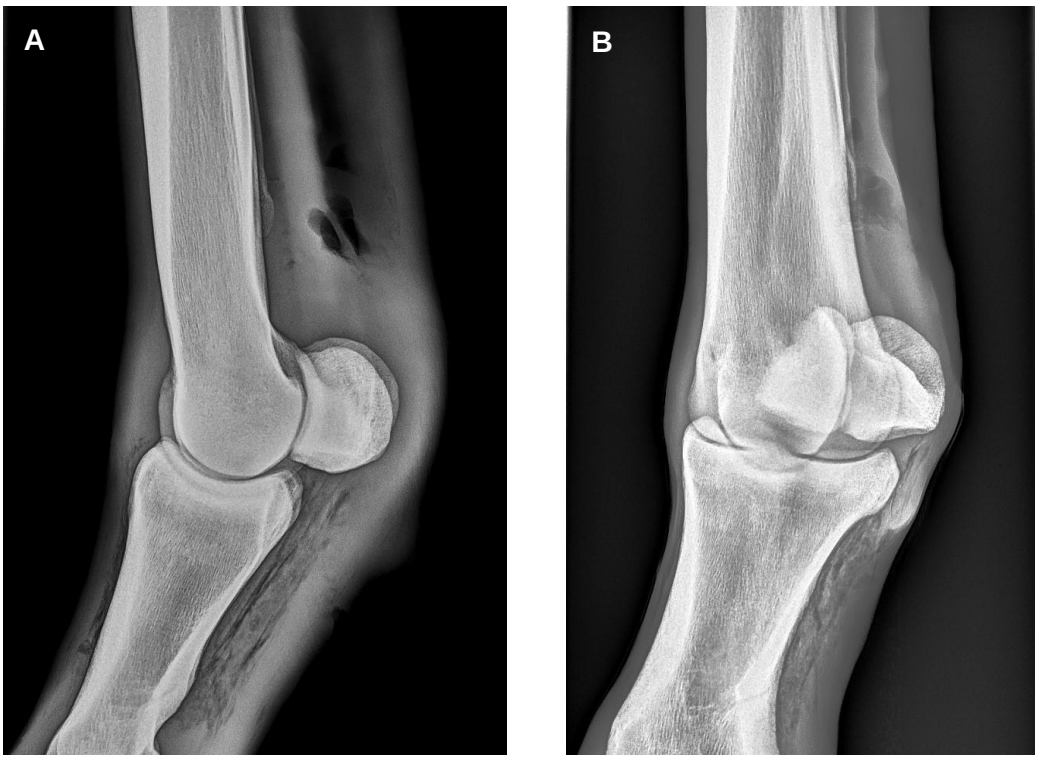


Figure 20. Contrast tenogram of Horse 35. The manica flexoria is thickened, shortened and in the proximal digital flexor tendon sheath only (*arrow*) (courtesy of DGVG).

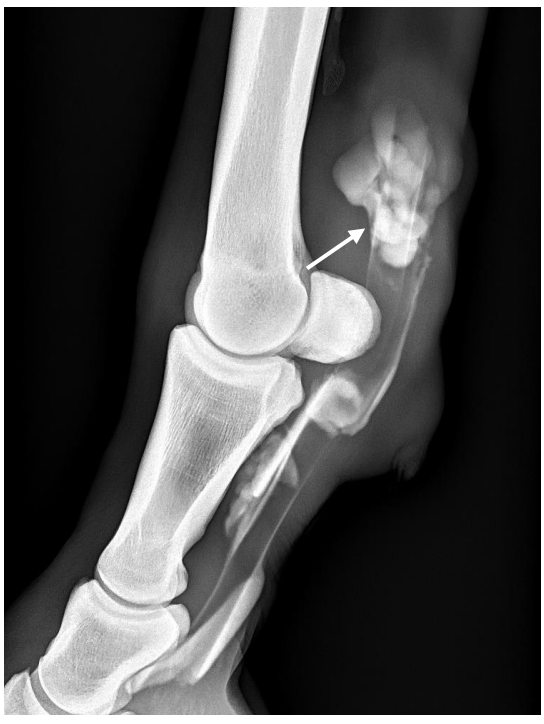
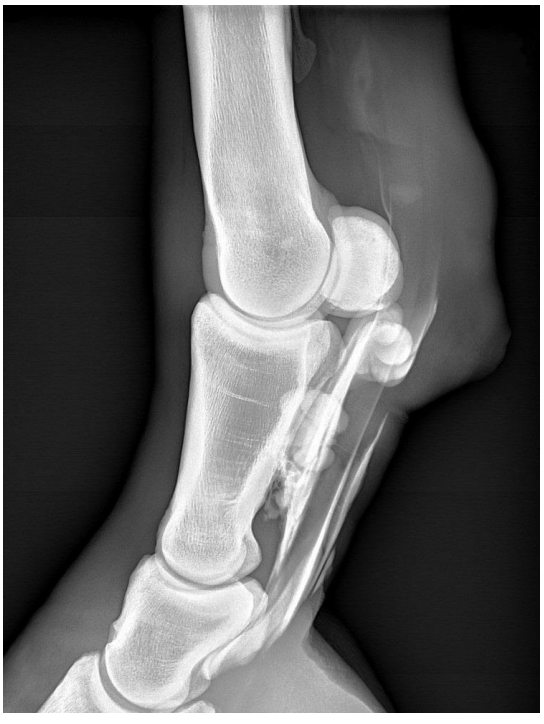


Figure 21. Contrast tenogram of Horse 44. There is no uptake of contrast up past the fetlock suggesting plantar annular ligament constriction (courtesy of BEVC).



5. Ultrasonography

Ultrasonographic images were available for retrospective evaluation in 72 horses. USG was performed in 28 (39%) forelimbs and 44 (61%) hindlimbs.

Eleven (15%) limbs had limited ultrasonographic abnormalities. Findings included PAL thickening (n = 16), PAL disruption (n = 4) (Figure 22), hypoechogenic lesion within the PAL (n = 3) (Figure 23, A), SDFT disruption (n = 8), hypoechogenic lesion within the SDFT (n = 3) (Figure 24), DDFT thickening (n = 3), DDFT disruption (n = 6), hypoechogenic lesion within the DDFT (n = 7), MF thickening (n = 4), MF disruption (n = 8) (Figure 24), SSL thickening with an hypoechogenic lesion within (n = 1), intrathecal echogenic material (n = 12), air within the DFTS (n = 4), adhesion formation (n = 3), fluid filled structure (n = 2) (Figure 23, B), tenosynovitis (n = 2) and foreign material (n = 1) (Table 5).

The ultrasonographic findings were consistent with primary PAL disease (n = 20), SDFT tears (n = 4), DDFT tears (n = 14), MF tears (n = 9), SW perforation (n = 4), SDFT laceration (n = 4), DDFT laceration (n = 2), ganglion cysts (n = 2), DDFT fibrosis (n = 1) and SSL desmitis (n = 1).

Table 5. Ultrasonographic findings of 72 horses.

Findings	No.	Findings	No.
Limited findings	11	MF thickening	4
PAL thickening	16	MF disruption	8
PAL disruption	4	SSL thickening + hypoechogenic lesion	1
Hypoechogenic lesion within the PAL	3	Intrathecal echogenic material	12
SDFT disruption	8	Air within the DFTS	4
Hypoechogenic lesion within the SDFT	3	Adhesion formation	3
DDFT thickening	3	Fluid filled structure	2
DDFT disruption	6	Foreign material	1
Hypoechogenic lesion within the DDFT	7		

Figure 22. (A) Transverse and (B) longitudinal ultrasonographic images of the right hindlimb of Horse 56. The plantar annular ligament is markedly thickened and disrupted (*x*) (courtesy of NEH).

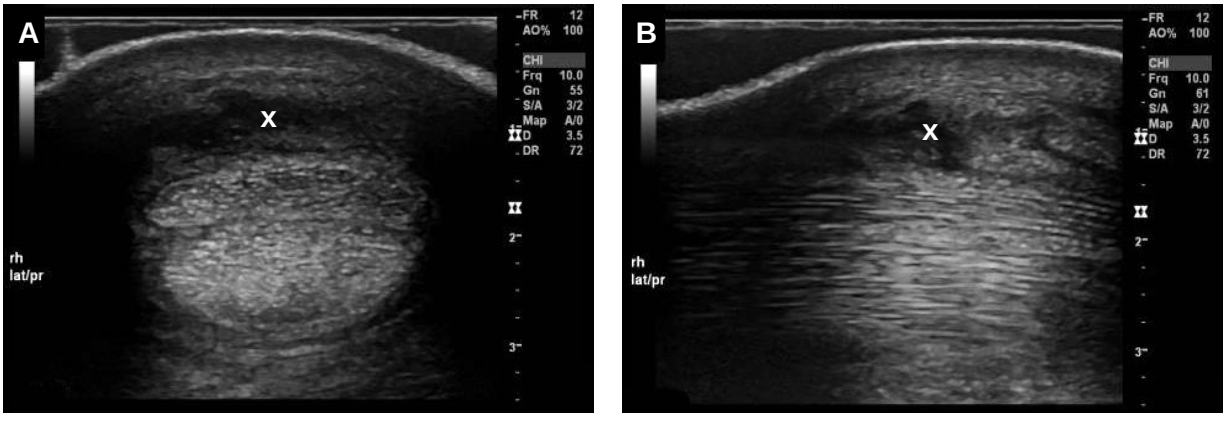


Figure 23. Ultrasonographic images of Horse 11. (A) Transverse image of the right forelimb palmar annular ligament demonstrating thickening of the ligament (0.6 cm) associated with a focal hypoechogenic area within the medial aspect of the ligament itself. (B) A transverse image of the right hindlimb fetlock revealed a multilocular hypoechogenic subcutaneous area within the medial plantar aspect of the fetlock, consistent with a ganglion (courtesy of DGVG).

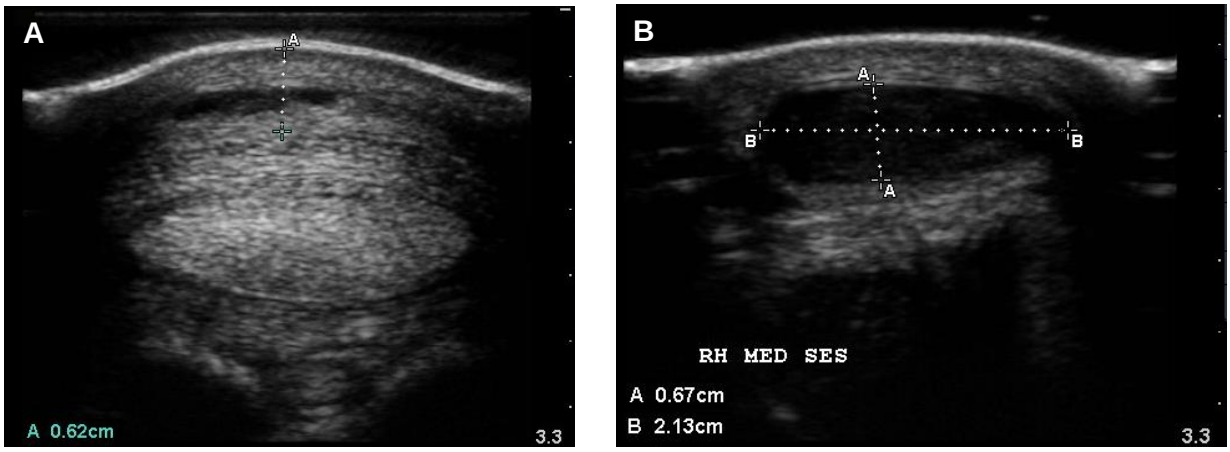
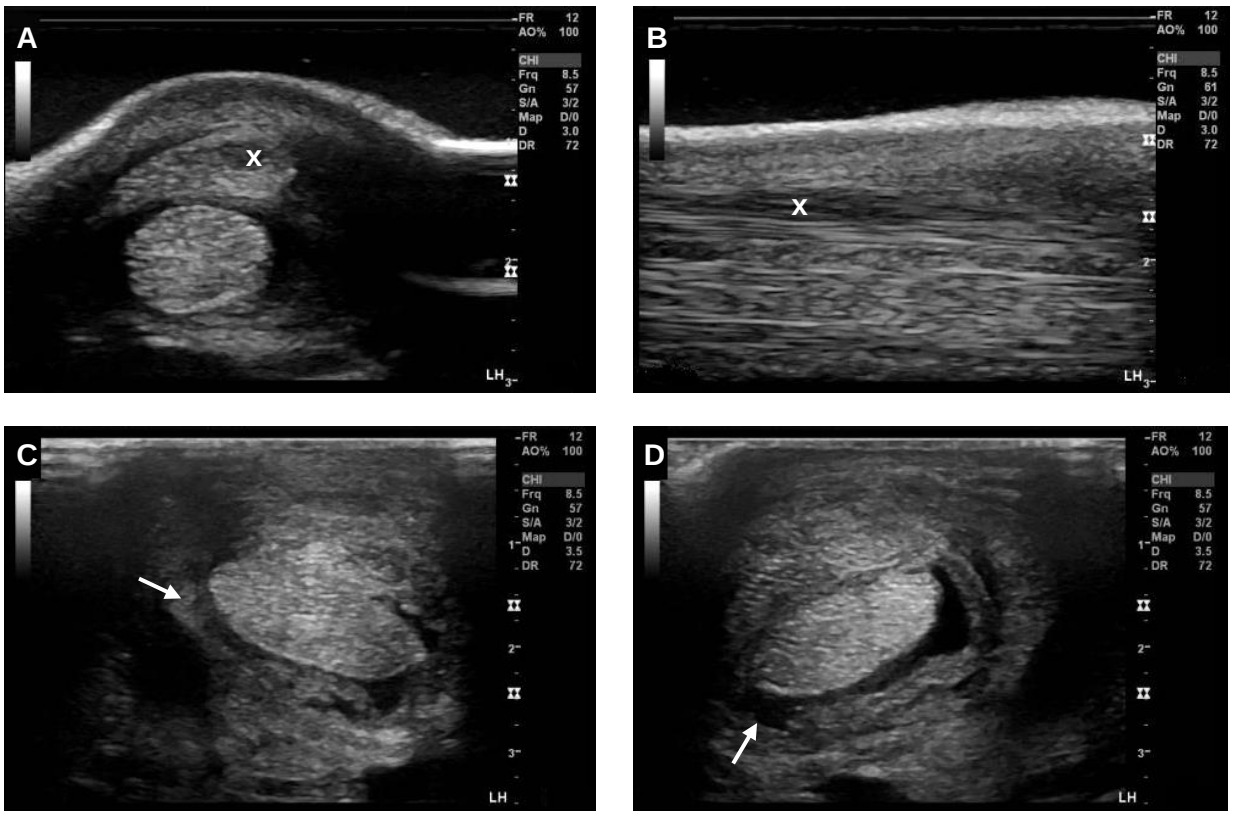


Figure 24. Ultrasonographic images of Horse 66 left hindlimb. (A) Transverse and (B) longitudinal ultrasonographs of the proximal portion of the digital flexor tendon sheath (zone 3A). There is a hypoechogenic zone (*x*) in the plantar lateral aspect of the superficial digital flexor tendon proximal to the sheath. (C and D) Transverse ultrasonographic images of the proximal fetlock region (zone 3B). It is possible to visualize a thickened and medially disrupted manica flexoria adherent to the sheath wall (*arrows*) (courtesy of NEH).



6. Surgical treatment

Eighty-six horses were submitted to tenoscopic examination. Two were euthanized while still under anaesthesia due to extensive tendon damage.

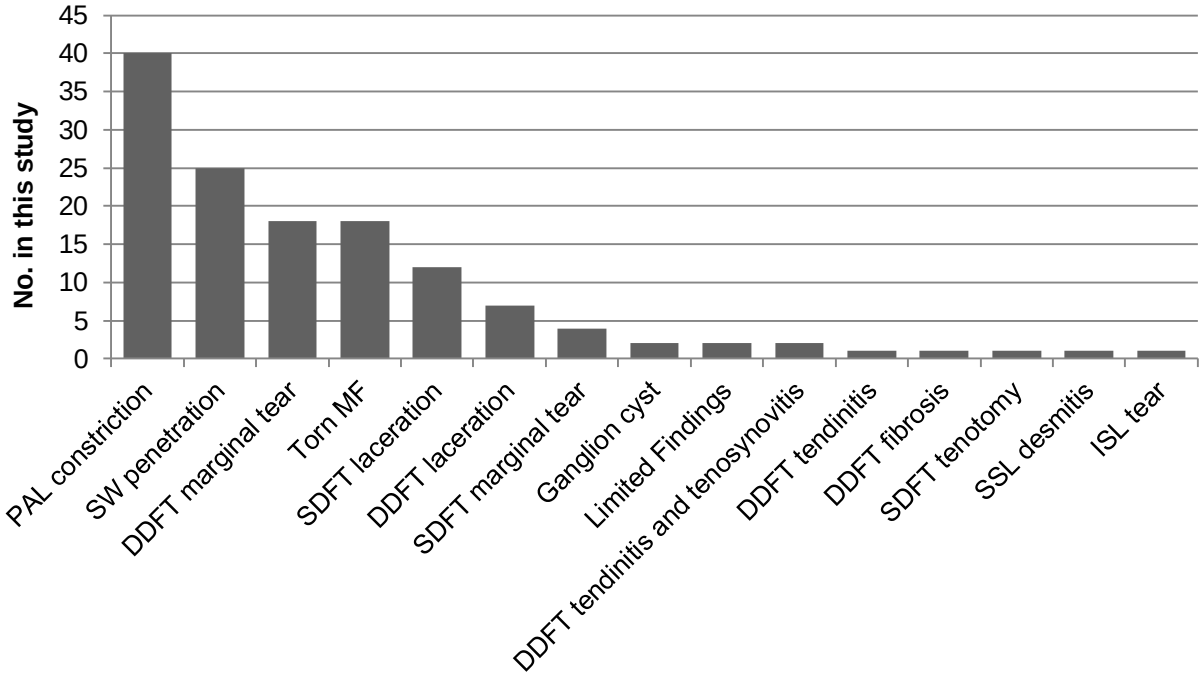
Ninety-three DFTS were examined, 36 (39%) forelimbs and 57 (61%) hindlimbs. The tenoscopic diagnoses are summarized in Table 6 and Graphic 3.

Table 6. Tenoscopic diagnoses in 93 digital flexor tendon sheaths.

Diagnosis	Forelimb	Hindlimb	Total
Torn MF			18
Torn MF only	-	2	2
Torn MF + other*	2	14	16
SDFT marginal tear			4
Marginal tear only	-	-	0
Marginal tear + other*	-	4	4
DDFT marginal tear			18
Marginal tear only	7	5	12
Marginal tear + other*	1	5	6
PAL constriction			40
PAL constriction only	8	11	19
PAL constriction + other*	4	17	21
SW penetration			25
SW penetration only	4	5	9
SW penetration + other*	11	5	16
SDFT laceration + other*	8	4	12
DDFT laceration + other*	4	3	7
Ganglion cyst	-	2	2
Limited findings	-	2	2
DDFT tendonitis and tenosynovitis	-	2	2
DDFT tendonitis	1	-	1
DDFT fibrosis	1	-	1
SDFT tenotomy	1	-	1
SSL desmitis	-	1	1
ISL tear	-	1	1

*The concomitant pathologies are described further in the text.

Graphic 3. Tenoscopic diagnoses in 93 digital flexor tendon sheaths.



Eighteen torn MF were found, from which 15 had concomitant pathologies. From the 15 horses, 14 had a torn MF associated with PAL constriction and 1 had a medial tear in the MF with concomitant medial tears to the SDFT and the DDFT, and PAL constriction (Figure 26).

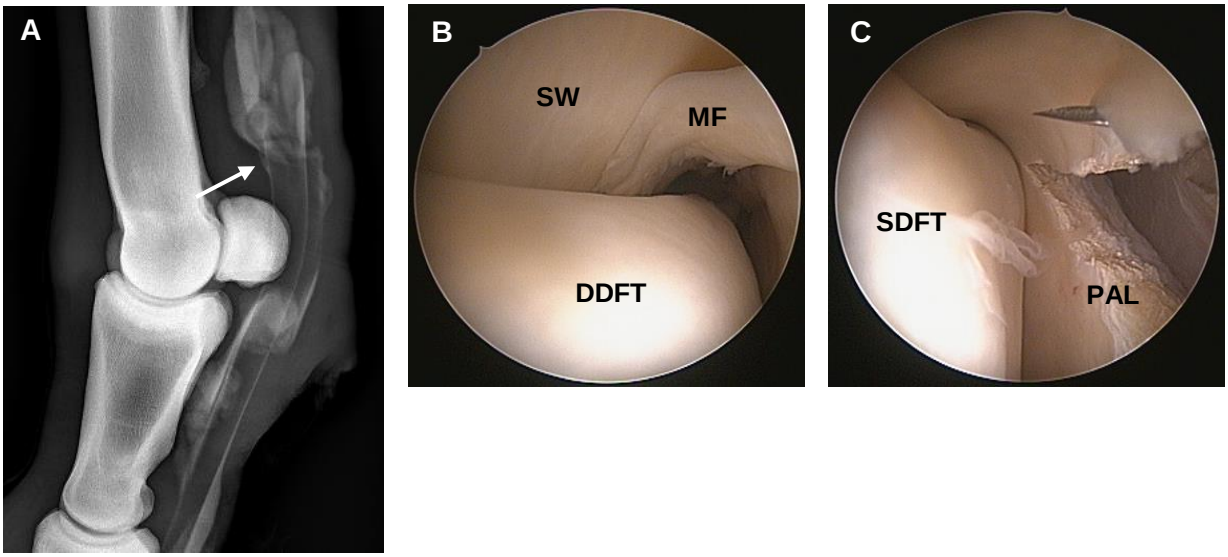
There were 21 tears in 18 MF, since 2 had a medial and a lateral tear and 1 had a medial and a horizontal tear. Ten (47%) tears occurred in the medial attachment to the SDFT, Nine 9 (43%) in the lateral attachment (Figure 25), 1 (5%) was seen in the distal margin and 1 (5%) in the horizontal plane (Table 7). The hindlimbs (n = 16, 89%) were significantly (P < 0.01) more commonly affected. Chronic (n = 10, 47%) and complete (n = 12, 57%) tears were more frequent than acute (n = 6, 29%) and partial (n = 4, 19%), and there was no information regarding 5 (24%) tears. All MF were completely removed except for 1.

In the 18 horses diagnosed with MF tears the majority (n = 10, 56%) were aged 11-15 years (Graphic 4). This pathology occurred predominantly in Coldbloods (n = 10, 56%), followed by Ponies (n = 4, 22%), Warmbloods (n = 3, 17%) and a horse of unknown breed (n = 1, 5%) (Graphic 5). There were significant associations between the occurrence of a MF tear and the horse's age class (P < 0.01) and breed group (P < 0.001), and no significant association with the gender (P = 0.8).

Table 7. Distribution of manica flexoria tears.

	Medial	Lateral	Distal	Horizontal	Total
Forelimb	1	-	1	-	2 (10%)
Hindlimb	9	9	-	1	19 (90%)
Total	10 (47%)	9 (43%)	1 (5%)	1 (5%)	21 (100%)

Figure 25. Contrast tenogram and tenoscopic images of Horse 29 left hindlimb. (A) Contrast tenogram suggestive of disruption to the manica flexoria (*arrow*). (B) Tenoscopic view of the torn manica flexoria. There is a complete separation from its lateral attachment to the superficial digital flexor tendon. (C) A plantar annular ligament desmotomy using a No. 12 blade was performed because the ligament was grossly thickened. *DDFT*: deep digital flexor tendon; *MF*: manica flexoria; *PAL*: plantar annular ligament; *SDFT*: superficial digital flexor tendon; *SW*: sheath wall (courtesy of DGVG).



There were 4 SDFT marginal tears, all in hindlimbs: 1 medial, 1 lateral, 1 plantar and 1 to the mesotenon. All 4 horses with SDFT marginal tears had concomitant PAL constriction and 1 of them had simultaneous medial tears to the MF and the DDFT (Figure 26). The horses affected were 2 Warmbloods and 2 Coldbloods (Graphic 5) with ages between 7-16 years (Graphic 4). A tenoscopic guided tenotomy of the SDFT was performed in the RF of a 15 years old Warmblood gelding with no other pathologies associated.

Marginal tears of the DDFT were identified in 8 (44%) forelimbs and 10 (56%) hindlimbs of 18 horses. Tears to the lateral border were the most common finding (n = 9, 50%) whereas medial (n = 3, 17.3%), dorsal (n = 3, 17.3%) and palmar/plantar (n = 3, 17.3%) tears were equally frequent (Table 8). The length of the tear was described in 16 of the 18 cases: 8 (44%) long and 8 (44%) short tears. Four long tears extended from beneath the MF to halfway the pastern, 2 from the proximal sheath to the level of the DM and 2 from the proximal sheath to the fetlock. Short tears were found mainly distal to the fetlock canal (n = 6), but also within (n = 1) and proximal to it (n = 1). Proximal tears were predominantly lateral (n = 7) with the remaining being medial (n = 2). Tears further distal affected mainly the palmar/plantar (n = 3) surface of the DDFT and less commonly the lateral (n = 1), medial (n = 1) and dorsal (n = 1) surfaces. The defects were classified as superficial in 3 cases and deep in 2 cases.

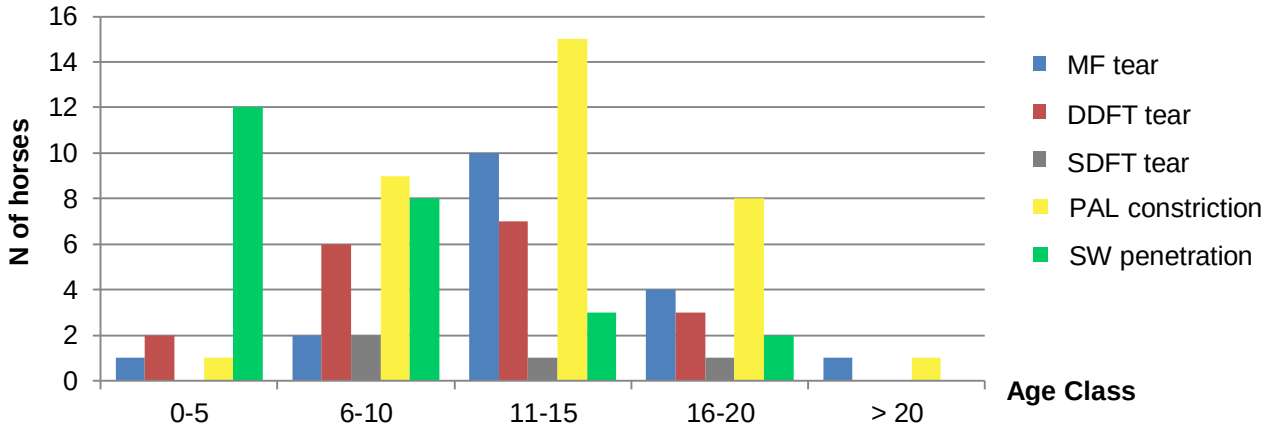
In all DDFT defects torn herniated fibrils were visualized. Other tenoscopic findings included granulomatous masses (n = 6) (Figure 27), hemosiderin deposits (n = 6), adhesions to the SW (n = 2), pannus deposits (n = 2) and fibrin deposits (n = 2).

Table 8. Distribution of deep digital flexor tendon tears.

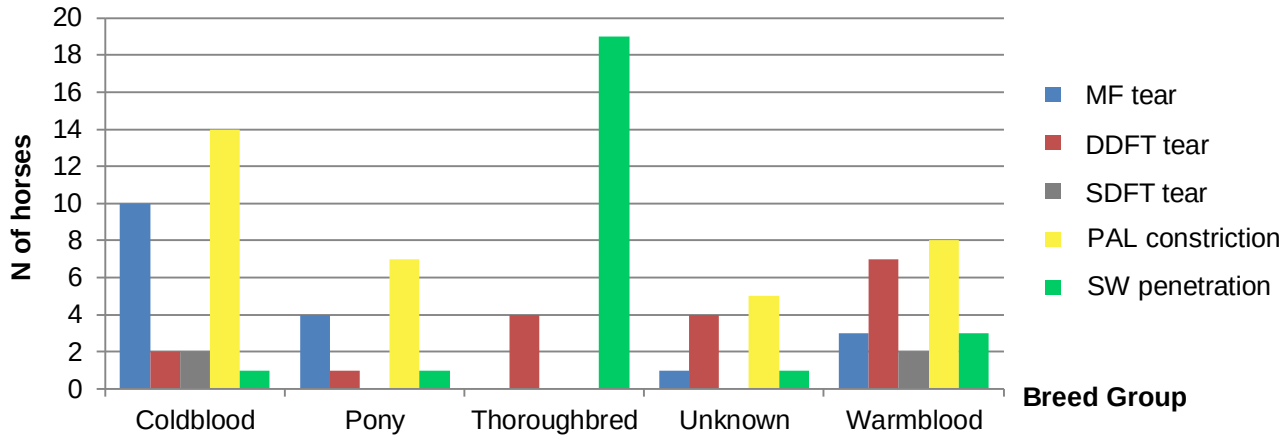
	Medial	Lateral	Dorsal	Palmar/Plantar	Total
Forelimb	-	6	-	2	8 (44%)
Hindlimb	3	3	3	1	10 (56%)
Total	3 (17.3%)	9 (50%)	3 (17.3%)	3 (17.3%)	18 (100%)

From the 18 horses, 6 had PAL constriction associated to the DDFT tear, and 1 had additional medial tears to the MF and SDFT. DDFT tears occurred more frequently in geldings (n = 12, 67%) (Graphic 6), Warmbloods (n = 7, 39%) (Graphic 5) and horses aged between 11-15 years (n = 7, 39%) (Graphic 4). No significant associations were seen between the occurrence of SDFT or DDFT tears and the affected limb (SDFT: *P* = 0.098; DDFT: *P* = 0.632), age class (SDFT: *P* = 0.834; DDFT: *P* = 0.794), gender (SDFT: *P* = 0.729; DDFT: *P* = 0.502) and breed group (SDFT: *P* = 0.301; DDFT: *P* = 0.123).

Graphic 4. Age distribution of 18 horses diagnosed with manica flexoria (MF) tear, 18 with deep digital flexor tendon (DDFT) tear, 4 with superficial digital flexor tendon (SDFT) tear, 34 with palmar/plantar annular ligament (PAL) constriction and 25 with sheath wall (SW) penetration.



Graphic 5. Breed distribution of 18 horse diagnosed with manica flexoria (MF) tear, 18 with deep digital flexor tendon (DDFT) tear, 4 with superficial digital flexor tendon (SDFT) tear, 34 with palmar/plantar annular ligament (PAL) constriction and 25 with sheath wall (SW) penetration.



Graphic 6. Gender distribution of 18 horses diagnosed with manica flexoria (MF) tear, 18 with deep digital flexor tendon (DDFT) tear, 4 with superficial digital flexor tendon (SDFT) tear, 34 with palmar/plantar annular ligament (PAL) constriction and 25 with sheath wall (SW) penetration.

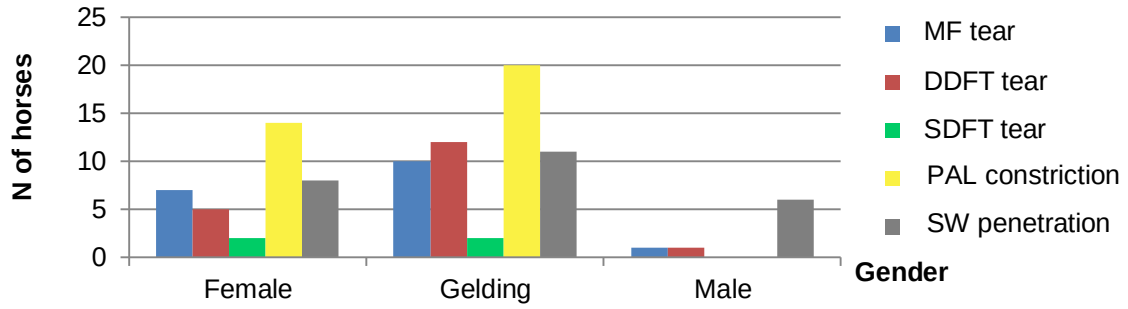


Figure 26. Ultrasonographic, tenoscopic and manica flexoria images of the right hindlimb of Horse 47. (A and B) Transverse ultrasonographs confirming the fluid distension of the digital flexor tendon sheath. The manica flexoria is grossly thickened and completely torn medially from its attachments to the superficial digital flexor tendon (*arrow*). There was irregular margination of the medial border of the deep digital flexor tendon at the base of the fetlock canal consistent with a marginal tear (*x*). (C and D) Tenoscopic view of the complete tear of the manica flexoria from its medial attachment to the superficial digital flexor tendon. It is possible to visualize a mass of disrupted fibrils. (E) Long longitudinal tears on the medial margin of both superficial and deep digital flexor tendons. (F) Plantar and (G) dorsal surfaces of the completely removed manica flexoria. *DDFT*: deep digital flexor tendon; *MF*: manica flexoria; *SDFT*: superficial digital flexor tendon; *SW*: sheath wall (courtesy of NEH).

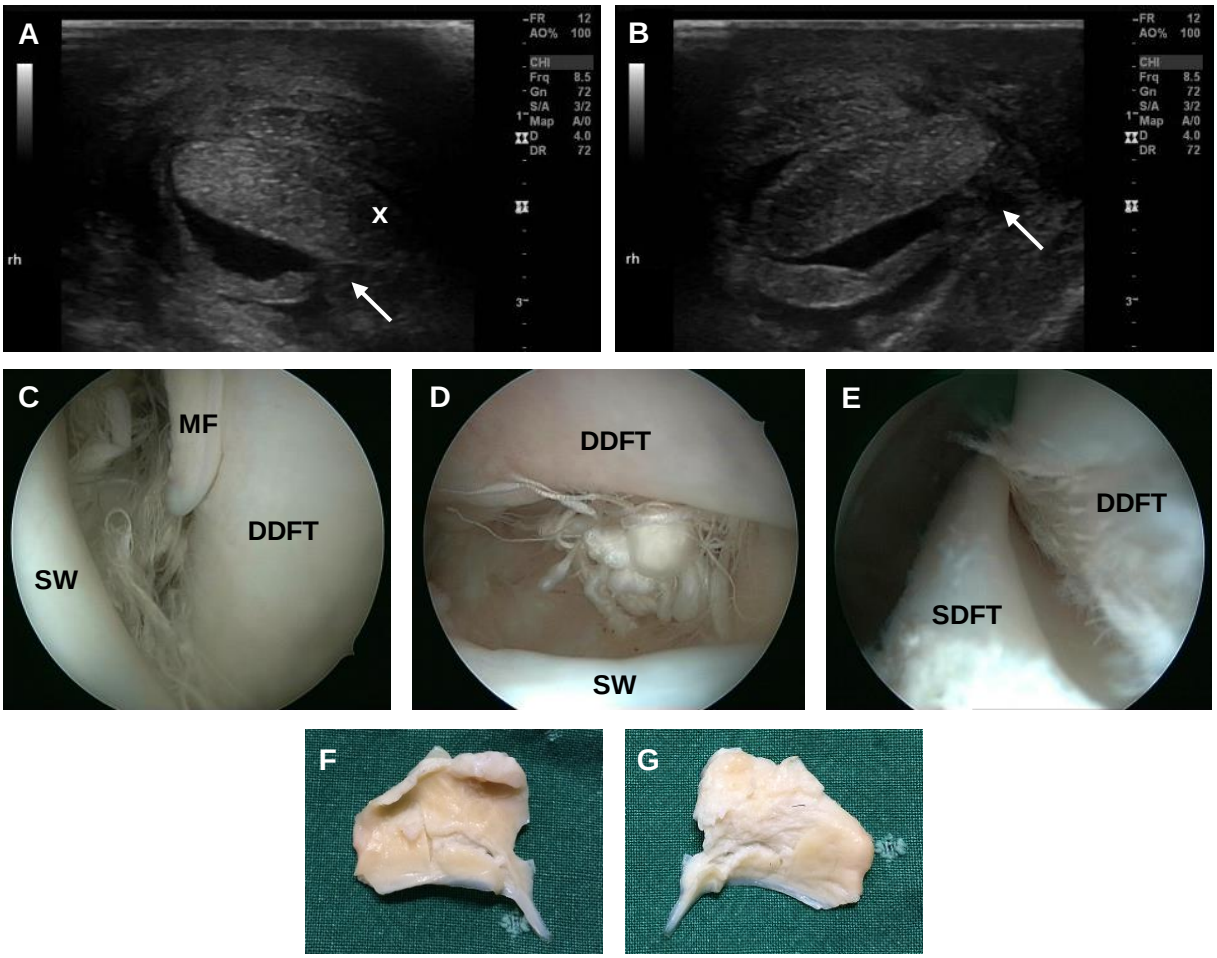
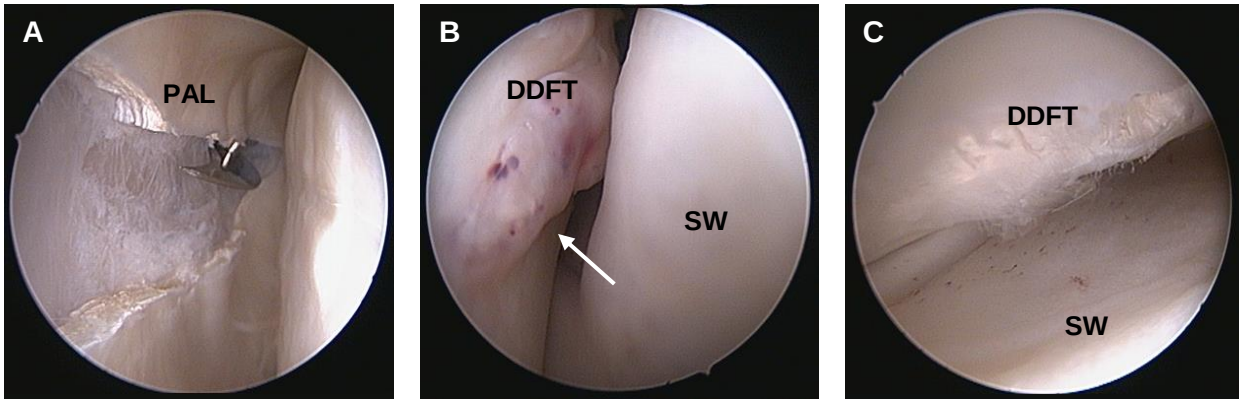


Figure 27. Tenoscopic views of Horse 34 right hindlimb digital flexor tendon sheath. (A) Plantar annular ligament desmotomy using a No. 12 blade. (B) A granulomatous mass (*arrow*) was present on the plantar border of the deep digital flexor tendon just medial to the midline. (C) View of the DDFT following debridement of the mass. *DDFT*: deep digital flexor tendon; *PAL*: plantar annular ligament; *SW*: sheath wall (courtesy of DGVG).



Besides the presence of marginal tears, other single pathologies regarding the DDFT were diagnosed. In the RF of a 7 years old Warmblood gelding the presence of a scar (fibrosis) in the palmar surface of the DDFT was visualized (Figure 28). There was 1 case of tendinitis (Figure 29) of the RF of a 3 years old Thoroughbred mare and 2 cases of infectious tenosynovitis: the RH of an 8 year old female and the LH of a 6 years old gelding, both Thoroughbreds. Regarding the last case, tenosynovitis occurred consequently to an abscess in the RH. This horse was submitted to 3 tenoscopies (Figure 30) before euthanasia was performed due to the absence of response to aggressive surgical and medical therapy.

Figure 28. Tenoscopic examination of the digital flexor tendon sheath of Horse 18. It is possible to visualize the presence of fibrosis in the palmar surface of the deep digital flexor tendon close to the distal reflection of the sheath (courtesy of DGVG).

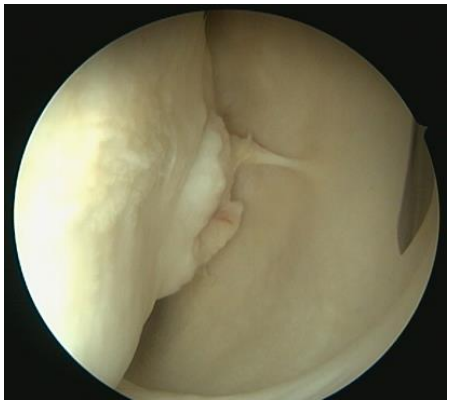


Figure 29. Tenoscopic view of the deep digital flexor tendon (DDFT) of Horse 68. There is marked inflammation in the proximal lateral epitenon of the DDFT. *DDFT*: deep digital flexor tendon (courtesy of NEH).

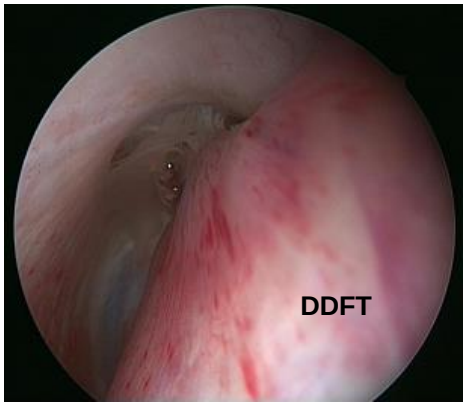
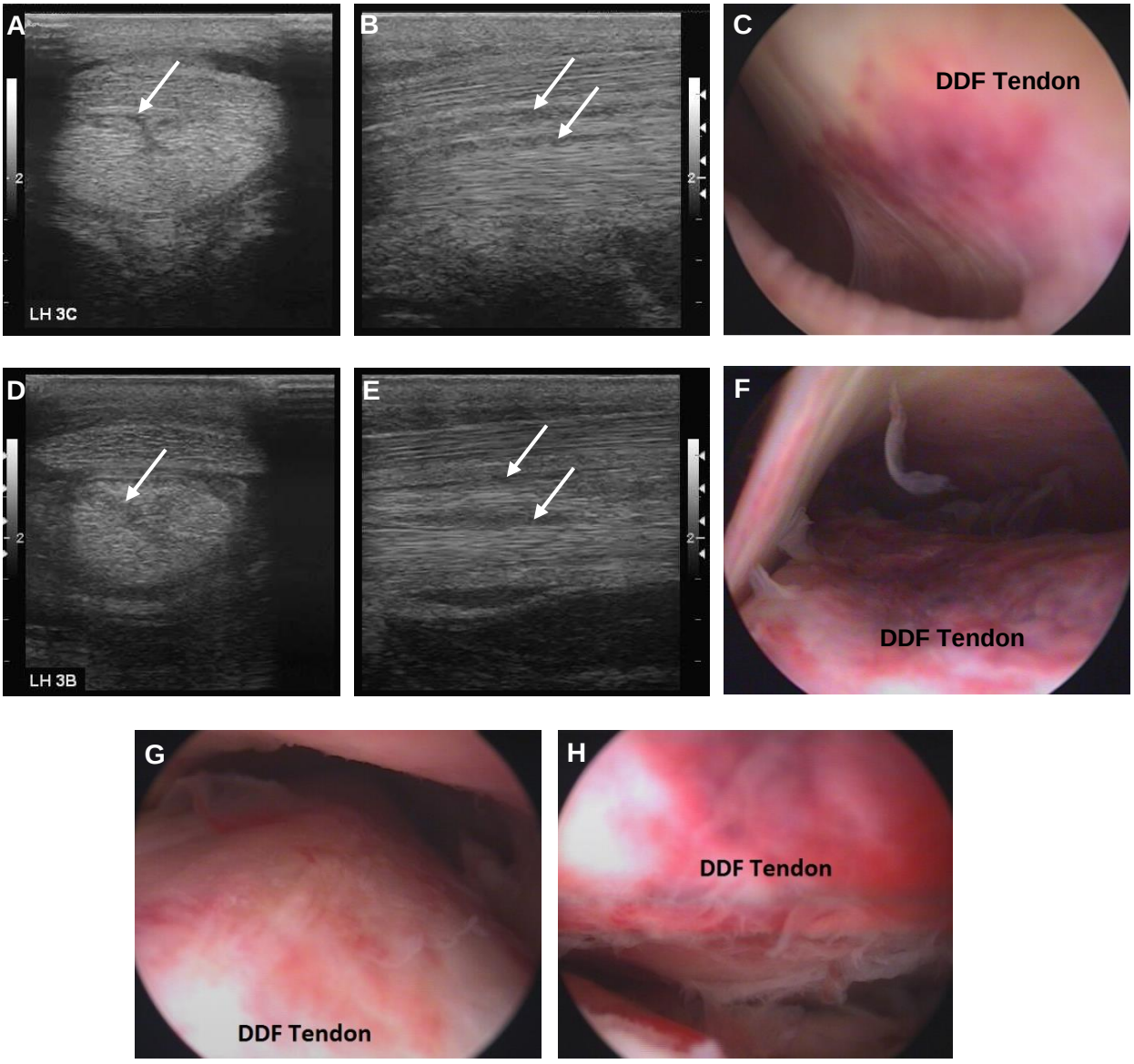


Figure 30. Ultrasonographic images and tenoscopic views of the three tenoscopies performed to the left hindlimb of Horse 41. (A) Transverse and (B) longitudinal ultrasonographic images revealing irregular fiber pattern and hypoechogenicity (*arrow*s) of the deep digital flexor tendon (DDFT). (C) Tenoscopic view of the digital flexor tendon sheath (DFTS) in the first surgery. There was mild sheath wall (SW) inflammation and superficial haemorrhage under the intact epitenon of the DDFT. (D) Transverse and (E) longitudinal ultrasonographic images showing more severe irregularity regions of hypoechogenicity with loss of fiber pattern (*arrows*) in the DDFT. (F) Tenoscopic view of the DFTS in the second surgery. Severe DDFT tenosynovitis with marked pannus and sub-epitenon haemorrhage was seen. (G and H) Tenoscopic views of the DFTS during the third surgery. The DDFT appeared similar in gross appearance after removal of pannus and fibrinous debris. *DDF Tendon*: deep digital flexor tendon (courtesy of VEH).



A “free-hand” PAL desmotomy (Figures 25C and 27A) was the most frequent surgical technique performed in this study. From the 86 horses, 38 (44%) had a PAL desmotomy done in 44 PALs. Thirty two horses had this technique done in a single limb, 4 had it in both hindlimbs and 2 in both forelimbs. The hindlimbs (n = 33, 75%) were significantly ($P < 0.001$) more commonly intervened. PAL desmotomy was performed alone in 19 limbs. In the other limbs it was associated to other pathologies such as MF tears (n = 17) and marginal tears of the SDFT (n = 4) and the DDFT (n = 6).

PAL constriction caused by primary PAL disease was diagnosed in 25 (57%) limbs. In 15 (34%) limbs the constriction was secondary to DFTS and/or tendon pathology. The remaining 4 (9%) had a PAL desmotomy performed as part of treatment even though there was no evident constriction to the fetlock canal (Table 9). Thirty-four horses had PAL constriction, from which 20 (59%) were found to have primary PAL disease (25 limbs). This was more frequently found in the hindlimbs (n = 16, 64%) and thickening of the ligament was seen in all 25 limbs, sometimes associated with tissue calcifications (n = 2) (Figure 17), fiber disruption (n = 2) (Figure 22) and presence of a cyst (n = 1) (Figure 23).

Geldings (n = 20, 59%) (Graphic 6), Coldbloods (n = 14, 41%) (Graphic 5) and horses aged 11-15 years (n = 15, 44%) (Graphic 4) were more frequently diagnosed with PAL constriction. There were significant associations between this pathology and the horse’s age class ($P = 0.001$) and breed group ($P < 0.001$).

Table 9. Summary of 43 limbs submitted to PAL desmotomy.

	Primary PAL disease	Secondary PAL constriction	No evident constriction	Total
Forelimb	9	2	-	11 (25%)
Hindlimb	16	13	4	33 (75%)
Total	25 (57%)	15 (34%)	4 (9%)	44 (100%)

Twenty-five horses were presented after a traumatic injury, from which horses with less than 10 years (n = 20, 80%) (Graphic 4), Thoroughbreds (n = 19, 76 %) (Graphic 5) and geldings (n = 11, 44%) (Graphic 6) were significantly more affected ($P < 0.001$, $P < 0.001$ and $P = 0.01$, respectively). Forelimbs (n = 15, 60%) were significantly ($P < 0.05$) more injured. A SW penetration as single pathology was seen in 9 (36%) cases and in association with tendon and ligament lesions in the 16 (64%) remaining cases. The last included lacerations to the SDFT (n = 7), DDFT (n = 2), SDFT and DDFT simultaneously (n = 5), and to the PAL (n = 2). Regarding the SDFT lacerations, these were identified in the medial (n = 4), lateral (n = 4), palmar (n = 3) and dorsal (n = 1) borders. DDFT lacerations were seen in the medial (n = 3), lateral (n = 3) and palmar (n = 1) surfaces of the tendon (Table 10).

Table 10. Distribution of lacerations to the superficial and deep digital flexor tendons.

	Medial	Lateral	Dorsal	Palmar	Total
SDFT	4	4	1	3	12 (63%)
DDFT	3	3	-	1	7 (37%)
Total	7 (37%)	7 (37%)	1 (5%)	4 (21%)	19 (100%)

The presence of a ganglion cyst was visualized in 2 horses. A 15 years old Pony mare was diagnosed with PAL desmitis and a cyst in the RF, and a ganglion cyst in the RH (Figure 23). In the other horse, a 7 year old Warmblood gelding, the ganglion cyst was identified in the LH. Pathology of the sesamoidean ligaments was recognised in the RH of 2 horses: a 13 years old Thoroughbred with SSL desmitis and a 10 years old Warmblood with a tear to the ISL. The most frequent tenoscopic findings, diagnostic methods performed, and sensitivity and 95% CI for radiography, contrast tenography and USG can be consulted in Table11.

Table 11. Summary of the most frequent tenoscopic findings and diagnostic methods performed. The sensitivity and 95% CI were calculated for radiography, contrast tenography and ultrasonography.

		Primary PAL Disease	MF tear	SDFT tear	DDFT tear	SW Perforation
Perineural Analgesia (Abaxial Sesamoid)	**NA**	21	18	3	17	25
	+	1	-	1	0	-
	-	3	-	0	1	-
Intrathecal Analgesia	**NA**	14	8	2	16	25
	+	6	10	2	2	-
	-	3	0	0	0	-
Synoviocentesis	**NA**	24	17	4	17	10
	+	0	-	-	1	10
	-	1	1	-	0	5
Radiography	**NA**	18	15	2	13	12
	+	2	0	0	0	9
	-	5	3	2	5	4
	Sens	30%	0%	0%	0%	70%
	95%CI	± 34%	-	-	-	± 25%
Contrast Tenography	**NA**	20	10	2	16	25
	+	2	6	0	0	-
	-	3	2	2	2	-
	Sens	40%	75%	0%	0%	-
	95%CI	± 43%	±30%	-	-	-
Ultrasonography	**NA**	4	2	-	2	5
	+	20	9	1	13	18
	-	1	7	3	3	2
	Sens	95%	56%	25%	81%	90%
	95%CI	± 9%	± 24%	± 42%	± 19%	± 13%
Tenoscopy	**No.**	25	18	4	18	25

NA: not accessed; +: positive; -: negative; *Sens*: sensitivity; *CI*: confidence interval; *No.*: number.

DISCUSSION

In this series, 27 Thoroughbreds, 24 Warmbloods, 17 Coldbloods, 10 Ponies and 8 horses of unknown breed were examined tenoscopically. The over-representation of Warmbloods (including Thoroughbreds) and young horses reflects the case load of the 3 Hospitals located in important places of the racehorse industry: Newmarket, Newbury and Lambourn.

The typical clinical findings of horses presented with chronic tenosynovitis were lameness, distension of the DFTS and positive distal limb flexion test, which is in accordance with ones presented by Wright and McMahon (1999), Wilderjans *et al.* (2003), Smith and Wright (2006), Owen *et al.* (2008) and Arensburg *et al.* (2011).

The most common finding in horses with noninfected tenosynovitis was PAL constriction. This was caused by primary PAL disease in 57% of the cases and in the remaining the constriction was secondary to DFTS and/or tendon pathology. Regarding tendon pathology, MF tears and DDFT marginal tears were the most common findings. Other diagnosis included SDFT marginal tears, DDFT tendonitis, DDFT infectious tenosynovitis, DDFT fibrosis, desmitis/tears of sesamoidean ligaments and ganglion cysts.

Diagnostic analgesia is an important tool to determine the source of lameness (Bassage II & Ross, 2003). There were only records of 23 horses regarding this, but it was probably performed in most horses presented with chronic lameness. Regional analgesia can be non-specific because of the DFTS extension, resulting in partial responses (Baxter & Stashak, 2011; Fiske-Jackson *et al.*, 2013). This was confirmed in this study as 50% of the abaxial sesamoid nerve blocks performed had a negative response, but when a low 4 point block followed the response was positive. Intrathecal analgesia was shown to be more specific, since a positive response was seen in 86% of cases. Even though it is more efficient, partial responses can still be seen, especially in horses with MF tears (Findley *et al.*, 2012; Fiske-Jackson *et al.*, 2013). In this series, the horses that did not show improvement in lameness following intrathecal analgesia all had primary PAL constriction.

USG is currently the most commonly used non-invasive diagnostic method for identification of tendon and ligament lesions (Redding, 2011). In some cases, it confirms the typical changes seen in a chronic inflamed sheath but it fails to reveal the primary cause behind the lameness. In this study, ultrasonographic examination was the most used diagnostic method. When performed, predicted the lesions identified by tenoscopy with a sensitivity of 81% (95% CI: 62–100) for DDFT tears, 56% (95% CI: 32–80) for MF tears, 95% (95% CI: 86–100) for primary PAL disease and 90% (95% CI: 77–100) for SW perforation. There was a poor sensitivity in the diagnostic accuracy of DDFT and MF tears using USG, being in keeping with previous observations (Wilderjans *et al.*, 2003; Smith & Wright, 2006; Arensburg *et al.*, 2011; Findley *et al.*, 2012; Fiske-Jackson *et al.*, 2013). PAL thickening, intrathecal echogenic material, SDFT and DDFT disruption, hypoechogenic lesions within the DDFT, and MF

disruption and thickening were all common findings in this series. When ultrasonographic changes indicate the presence of LTs of the flexor tendons or MF tears, tenoscopy of the DFTS should be recommended to remove tears as the underlying cause for non-infectious chronic tenosynovitis. In refractory cases or extensive tears, an open approach with direct suturing of the tendon, as described by Wright and McMahon (1999), may be adopted.

Radiography and contrast tenography associated with intrathecal analgesia were performed in a smaller number of cases. According to Fiske-Jackson *et al.* (2013), MF tears are predicted with a higher sensitivity by contrast tenography than by USG and the opposite occurs for DDFT tears. Contrast tenography was performed in 44% of cases diagnosed with MF tears tenoscopically, being indicative of this pathology in 75% (95% CI: 45–100) of contrast studies, which is greater compared to the sensitivity of 56% (95% CI: 32–80) with USG. The low sensitivity of USG in predicting MF tears can be related to the thick hairy skin and prominent folds of the most affected breed group (Coldbloods) associated with the convex swelling of the DFTS, which reduces the contact area of the probe. In this series, DDFT tears were not identified by contrast tenography but the sensitivity of USG was 81% (95% CI: 62–100), comparable with the 71% (95% CI: 60–82) reported by Smith and Wright (2006) and 63% (95% CI: 53–72) by Arensburg *et al.* (2011).

Smith and Wright (2006), Findley *et al.* (2012) and Fiske-Jackson *et al.* (2013) reported a greater prevalence of MF tears in the hindlimbs. A breed association was only evidenced by Findley *et al.* (2012), considering Cobs and Ponies predisposed to MF tears. The results of this study are in concordance with the previous reported associations, adding the finding that this pathology affects more frequently horses aged 11-15 years. MF tears were predominantly medial, chronic and complete, as showed in Findley *et al.* (2012) study.

In series of 17, 46 and 91 LTs of the DDFT, Wilderjans *et al.* (2003), Smith and Wright (2006) and Arensburg *et al.* (2011), respectively, noted a greater prevalence of DDFT marginal tears in forelimbs, but in this study the hindlimbs were affected in 56% of the cases. It is believed that this distribution was random and the number of cases is too small to draw conclusions.

In a retrospective analysis of 76 cases of noninfected tenosynovitis, Smith & Wright (2006) found tears of the DDFT to be more commonly proximal. Regarding their length, long tears involved the proximal portion of the DFTS and short ones were found more frequently distal to the fetlock canal. Proximal tears were predominantly in the lateral border of the tendon and distal tears were palmar/plantar. This study verified the exact same results.

The most consistent tenoscopic findings were disrupted herniated fibrils, considered the most probable cause of chronic tenosynovitis (Wright & McMahon, 1999). The presence of granulomatous masses (granulomata) frequently containing areas of hemosiderin staining was often seen and these were debrided and removed to allow tendon healing. Comparing with Wilderjans *et al.* (2003) series, adhesions were uncommon.

Marginal tears to the SDFT were less identified than ones in the DDFT. The same was true for series reported previously (Arensburg *et al.*, 2011; Smith and Wright, 2006). It is known that SDF tendonitis is common in the mid-carpal region, appearing ultrasonographically as a "core" lesion (Avella & Smith, 2012). Thus, one explanation for this might be that the components of the extracellular matrix of the SDFT are more stable inside the sheath, conferring resistance to degeneration, which might justify the low incidence of marginal tears seen in this tendon. The DDFT might be prone to LTs inside the sheath since it is compressed dorsally by the SL and intersesamoidean ligament, and palmarly by the SDFT, leading to marginal ruptures when passing through the fetlock canal (R. Smith and A. Fiske-Jackson, personal communication).

The PAL is an inelastic ligament that attaches to the PSBs forming the fetlock canal. Primary PAL disease, ligament/tendon pathology and chronic tenosynovitis can lead to stenosis of the fetlock canal (McGhee *et al.*, 2005; Schramme & Smith, 2003). Previously, when tenosynovitis occurred, PAL desmotomy was advised in order to relieve the pressure within the sheath and consequently decrease the pain. It was also beneficial to the tenoscopic examination as it decompresses the sheath and a better access to the structures is achieved (Wilderjans *et al.*, 2003). The same authors do not longer consider PAL desmotomy to help the case management, although it enhances the tenoscopic examination. Consequently, it was advocated that PAL transection should only be performed when PAL thickening is causing the constriction. In this study, PAL desmotomy was the most frequent surgical technique. It was performed in 44 limbs, even though thickening of this ligament was only evident in 25 cases. In the remaining cases, desmotomy was done when necessary as part of the management of LTs of the flexor tendons and MF, since the constriction within the fetlock canal was not allowing access to the lesions.

Traumatic injuries are quite common in horses, especially in the distal limb. The flexor tendons are enfolded by the DFTS and positioned under the skin, being prone to injuries after a trauma (Avella & Smith, 2012; Dyson & Bertone, 2003). The determination of whether the SW was penetrated and contaminated relies on radiography, ultrasonography and synoviocentesis (Bertone, 2011; Schramme & Smith, 2003; Wereszka *et al.*, 2007). From horses that were presented with a traumatic injury radiography was performed in 50%, ultrasonography in 80% and synoviocentesis in 60%. The presence of gas within the soft tissues and the sheath was found in 70% (95% CI: 45–95) of the radiographs, indicating SW perforation. Ultrasonography predicted penetration to the sheath in 90% (95% CI: 77–100) of cases, regularly identifying injuries to the SDFT. In a few cases no complementary diagnostic method was performed after clinical examination due to obvious perforation and indication for surgery. Cytological analysis revealed contamination and possible infection in 10 out of 15 samples. Samples were considered positive if at least two of the analysed parameters were indicative of contamination. The horses that presented with traumatic injuries were more

frequently young Thoroughbreds and injuries were seen especially in forelimbs. This is a result of the case load and location of the Hospitals, since some horses presented after racing, especially with overreaching injuries. Traumatic injuries sustained while turned out were also frequent.

Infectious tenosynovitis can be consequence of an injury or caused by intrathecal injections, extension from adjacent tissues and haematogenous spread (Bertone, 2011; Schramme & Smith, 2003; Wereszka *et al.*, 2007). Two cases of infectious tenosynovitis with no evidence of a penetrating wound to the DFTS were diagnosed by cytological analysis and ultrasonography. Both presented extremely lame with pain and distension of the DFTS, the typical signs of infectious tenosynovitis (Bertone, 2011; Schramme & Smith, 2003; Wereszka *et al.*, 2007). The samples were submitted for bacterial culture and sensitivity, yielding a *Pasteurella* spp. with broad sensitivity to antimicrobials in one case, and no growth in the second case. The first horse progressively improved after tenoscopic lavage, debridement and instillation of antimicrobials associated with intravenous antibiotics. Concerning the second horse, several surgeries were performed and multiple samples were analysed but no bacterial or fungal growth was obtained. As the horse was progressively deteriorating, the intravenous antimicrobials were changed and three surgeries were done until euthanasia was decided. Necropsy confirmed chronic DDFT tendonitis and tenosynovitis in the affected limb and presence of a granulating wound secondary to a suspected foreign body penetration in the contralateral limb. Since there were no signs of a wound in the affected limb, haematogenous spread from one limb to the other was the most probable cause, even though it is rare.

Ganglion cysts and diseases of the ISL and SSL as cause of lameness in horses are uncommon (Dyson & Genovese, 2003; Schramme & Smith, 2003). Regarding the case of SSL desmitis, the conservative treatment was not successful so a tenoscopic approach was advocated as advised by McIlwraith *et al.* (2015). The debridement and removal of torn fibrils resulted in significant clinical progress. There is no previous information about the horse that was presented with an ISL tear, so it can only be reported that the tear was debrided. Ganglion cysts were identified by clinical examination and confirmed by USG. In both cases was proven difficult to isolate the cyst from the surrounding neuro-vascular structures. Careful sharp dissection was made and the defect debrided and sutured. DFTS examination revealed no abnormalities other than a focal defect in the fibrocartilage wall of the fetlock canal which was debrided and repaired with a single suture. Since there was no other pathology, the prognosis was considered very good in both cases.

As limitations to this study, the absence of long term follow-up was considered the most important. All surgeries were performed in order to promote the horse's return to the previous athletic function. Since there was no access to that information, it was not possible to determine the success rate of the tenoscopies. Other important factor to have in mind is the

subjectivity of reports interpretation. In spite of a thorough and precise medical records revision, misinterpretation is a possibility that should be taken in account.

In this series, age and breed predispositions were identified for MF tears, PAL constriction and traumatic injuries to the sheath. Gender predisposition was only true for traumatic injuries. Additionally, hindlimbs were significantly more affected by MF tears and PAL constriction, while forelimbs by traumatic injuries. It would be important to correlate the horse's current use to the diagnosis, as it is a determinant factor in tendon pathophysiology.

CONCLUSION

In this study, a retrospective analysis of 86 horses submitted to tenoscopic examination of the DFTS in 2016 was made. As expected, PAL constriction and tears of the flexor tendons and MF were the most frequent diagnosis. Tenoscopy was of extreme importance as a therapeutic and diagnostic technique, considering that MF tears were only identified by other methods in 61% of the cases, flexor tendon tears in 67% and PAL constriction in 53%.

The correlation study of various factors with the diagnosed pathologies determined important associations and predispositions. This analysis was undertaken for the horse's age, gender, breed and affected limb. The strongest relation was seen between traumatic injuries and forelimbs of young Thoroughbreds. Furthermore, hindlimbs of middle aged Coldbloods were significantly more diagnosed with MF tears.

In conclusion, tenoscopy was proven to be a simple and useful surgical technique, even though the long term follow-up of analysed horses was not studied.

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ANNEXES

Annex 1. Horse identification (ID) and respective age, breed, gender, affected limb(s) and diagnosis.

Horse ID	Age	Breed	Gender	Affected Limb(s)	Diagnosis					
					PAL Constriction	MF Tear	SDFT Tear	DDFT Tear	Traumatic injury	Other
1	17	Cob x Thoroughbred	Female	LF	X					
2	11	Connemara	Gelding	RH + LH	X X					
3	15	Welsh Cob Section C	Gelding	LH	X	X				
4	9	Unknown	Female	LF	X					
5	17	Andalusian	Male	LH		X				
6	3	Thoroughbred	Gelding	RH					X	
7	6	Thoroughbred	Gelding	LF					X	
8	7	Irish Sport Horse	Gelding	LH						X
9	14	Unknown	Gelding	RF	X			X		
10	3	Thoroughbred	Male	RF					X	
11	15	Polo Pony	Female	RF + RH	X					X
12	13	Cob	Gelding	RF + LF	X X					
13	12	New Zealand Thoroughbred	Gelding	LH				X		
14	1	Thoroughbred	Female	RH					X	
15	8	Connemara x Thoroughbred	Gelding	RH						X
16	16	Hungarian Warmblood	Gelding	RH					X	
17	14	New Forest Pony	Female	LH	X	X				
18	7	Irish Sports Horse	Gelding	RF						X
19	11	Welsh Cob Section D	Gelding	LH	X	X				
20	16	KWPN	Gelding	RH	X			X		
21	7	KWPN	Female	LH			X			
22	10	Sports Horse	Female	LH	X					
23	12	New Forest Pony	Gelding	RF	X	X				
24	9	KWPN	Gelding	RH + LH	X X					
25	9	Irish Draught x Thoroughbred	Female	RH + LH	X X		X			
26	8	Skewbald	Gelding	LH	X					
27	9	Welsh Cob Section D	Female	RH + LH	X X	X				
28	3	Thoroughbred	Gelding	RF					X	
29	14	Unknown	Female	LH	X	X				
30	16	Welsh Cob Section C	Female	LH	X					
31	15	Polish Riding Horse	Female	RH					X	
32	19	Skewbald	Gelding	LH	X	X				
33	13	Unknown	Gelding	RH	X			X		
34	11	Unknown	Gelding	RH				X		
35	12	Lusitano	Gelding	LH	X	X				
36	5	Welsh Cob Section C	Gelding	RF	X	X				
37	6	Thoroughbred	Gelding	LF					X	
38	12	Thoroughbred	Gelding	LH					X	
39	16	Thoroughbred	Gelding	RF					X	

40	5	Thoroughbred	Female	RF						X	
41	6	Thoroughbred	Gelding	LH							X
42	1	Thoroughbred	Male	RH						X	
43	10	Cob	Female	RH						X	
44	20	Cob	Female	LH	X						
45	12	Welsh Cob	Gelding	RH	X		X				
46	13	Connemara	Gelding	RH	X	X					
47	16	Cob (Dale x Fell)	Gelding	RH	X	X	X	X			
48	6	Thoroughbred	Female	LF						X	
49	1	Thoroughbred	Female	LF						X	
50	14	Fell Pony	Female	RH	X	X					
51	6	Shetland Pony	Gelding	RH						X	
52	10	Irish Draught x Thoroughbred	Gelding	LF						X	
53	9	Unknown	Female	RF				X			
54	1	Thoroughbred	Male	RF						X	
55	16	Polish Cob	Female	LH	X	X					
56	14	Belgian Warmblood	Female	RH	X	X					
57	1	Thoroughbred	Male	LF						X	
58	21	Irish Draught x	Female	RH	X	X					
59	8	KWPN	Gelding	RH				X			
60	11	Welsh Cob Sec D	Female	RF				X			
61	5	Hannoverian x Thoroughbred	Gelding	RF				X			
62	8	Thoroughbred	Female	RH							X
63	9	Unknown	Gelding	RF + LF	X X						
64	9	Thoroughbred	Gelding	LH				X			
65	14	Unknown	Gelding	LF						X	
66	13	Welsh Cob Sec C	Gelding	LH		X					
67	10	Danish Warmblood	Gelding	LF				X			
68	3	Thoroughbred	Female	RF							X
69	13	Thoroughbred	Gelding	RH							X
70	7	Thoroughbred	Gelding	RF						X	
71	2	Thoroughbred	Female	LH						X	
72	10	KWPN x Thoroughbred	Gelding	RH							X
73	6	Pony	Gelding	RF	X						
74	7	Polo Pony	Female	RF				X			
75	6	Thoroughbred	Female	RF						X	
76	3	Thoroughbred	Male	LH				X			
77	15	Irish Sports Horse	Gelding	RF							X
78	8	Cob	Gelding	RH	X	X					
79	1	Thoroughbred	Male	LH						X	
80	6	Warmblood	Female	LF				X			
81	12	Warmblood	Female	RF				X			
82	6	Irish Sports Horse	Female	RH							X
83	12	Hanoverian	Gelding	LH				X			
84	3	Thoroughbred	Male	LF						X	
85	20	Arabian	Gelding	LH	X						
86	16	Thoroughbred	Gelding	LH				X			