Diagnosis and treatment of digital flexor tendon sheath pathology

Florent DAVID  DVM, MS, Dipl. ACVS, Dipl. ECVS, ECVDI Assoc., Dipl. ACVSMR

Board Certified Specialist in Equine Surgery
Board Certified Specialist in Equine Sports Medicine and Rehabilitation
Associate Member of the European College of Veterinary Diagnostic Imaging

Equine Veterinary Medical Center
Member of Qatar Foundation
Al Shaqab Street
Al Rayyan
Doha
Qatar
fdavid@qf.org.qa
www.evmc.qa

A- Functional anatomy

The superficial digital flexor tendon (SDFT) and deep digital flexor tendon (DDFT) run along the palmar/plantar aspect of the distal third of the cannon bone, fetlock joint and entire pastern region within a synovial cavity, called the digital flexor tendon sheath (DFTS). This simplified version of the anatomy was taught to me at vet school. Since this time, I have discovered a much more complex anatomic structure, with details very relevant to the diagnosis and treatment of DFTS pathology.

The DFTS can be divided into three continuous and communicating regions: Proximal to the fetlock, level with the fetlock, and distal to (pastern region) the fetlock. The DDFT is located dorsal to the SDFT for most of its path. In the pastern region, immediately distal to the fetlock joint, the SDFT divides into two branches. These SDFT branches are located lateral and medial to the DDFT, in a coronal plane, dorsal to the DDFT in their most distal aspect. The SDFT branches exit/fuse with the sheath wall to then blend with the proximal digital scutum. In this same region, the DDFT becomes bilobed and passes the distal aspect of the first phalanx, where it becomes the only tendon running through the DFTS.

The flexor tendons encounter, over their path, several bony prominences where marked changes in direction and friction occur. The proximal, middle and distal scuta are large, smooth, fibrocartilaginous surfaces, which assist in the gliding motion of the flexor tendons at the palmar/plantar aspect of the fetlock, pastern and coffin joints, respectively. Lubrication to the tendons is provided by the DFTS lining and synovial fluid.

The flexor tendons, coupled with the suspensory ligament, support the fetlock joint descent during the weight-bearing phase of the stride. This is only possible with the help of the palmar/plantar annular ligament (PAL), which maintains the flexor tendons against the
proximal scutum and within the meta-carpo/tarso-phalangeal canal. To avoid luxation or lateromedial instability of the flexor tendons, another proximal digital annular ligament, or retinaculum, provides stability immediately distal to the fetlock.

During digital flexion, the relaxed flexor tendons can deviate slightly from their loaded axis. To make sure the flexor tendons stay aligned to glide through the meta-carpo/tarso-phalangeal canal during digital extension, the horse has developed two tendinous rings, called manica flexoria. A strong proximal manica flexoria, originating from the medial and lateral borders of the SDFT, is present immediately proximal to the fetlock, forming a collar dorsal to the DDFT. This structure is made of an areolar portion proximally and a tendinous/fibrous portion distally. As shown by Findley et al. (2017), the fibrous manica flexoria is significantly longer, and the areolar portion shorter, in the forelimb when compared to hindlimb. In normal limbs, the distal aspect of the manica flexoria is very thin (less than 1mm) dorsally and is found at the level of the proximal aspect of the PAL. As shown by Kent et al. (2019), the distal aspect of the proximal manica flexoria is located more distally on hindlimbs than on forelimbs. When associated with conformational abnormality, or overloading of the limb, this structure is more at risk of getting caught on the proximal scutum and being torn. The proximal manica flexoria fuses proximally (and dorsally) with the sheath wall. A smaller distal, or digital, manica flexoria is present immediately distal to the fetlock, at the mid-level of the proximal phalanx. It originates/connects the distal branches of the SDFT and a significant amount of individual variation has been described by Jordana et al. (2016). It is also located dorsal to the DDFT, as for the proximal manica flexoria, but palmar/plantar to the distal sesamoidean ligaments.

Nutrients and oxygen are brought to the flexor tendons by a complex network of interconnected intrinsic and extrinsic blood vessels and by synovial fluid imbibition. Important to note, flexor tendons are deprived from paratenon within the DFTS, but the sheath wall is the equivalent to the paratenon for the non-sheathed tendons, as far as extrinsic blood supply is concerned. Extrinsic blood vessels enter/exit the flexor tendons via the proximal and distal reflection of the DFTS, and also by specific entering points called mesotenons, or vincula. They are condensation of areolar connective tissue where vessels travel from the sheath wall to the epitenon of the sheathed tendons. These blood vessels then enter the tendon core and connect, via the endotenon, to the intrinsic blood vessels. Proximal to the fetlock, the SDFT is nourished by a long palmar/plantar mesotenon that fades away proximal to the PAL. The DDFT is fed by medial and lateral mesotenons, located immediately proximal to the proximal manica flexoria. In the pastern region, vincula originate from the digital manica flexoria and connect with the DDFT. Certain areas are relatively avascular/hypovascular, such as the dorsal aspect of the DDFT at the fetlock level, as shown by Kraus et al. (1995). In this location, a large proportion of the tendon matrix is composed of fibrocartilage, thought to be more resilient to increased compressive and frictional forces.

Unlike the flexor carpal sheath, no nerves are running directly through the DFTS. In the distal pastern region, the DFTS is still present and extends dorsal to the DDFT, and comes in close proximity with synovial structures of the foot, i.e. the navicular bursa and the proximal palmar/plantar recess of the coffin joint.
Knowing the presence, variations, localization and function of all the structures detailed above can greatly assist in the understanding of the DFTS pathology and improve interpretation of diagnostic tests, as well as refine therapeutic approaches.

B- DFTS pathology and clinical findings

A combination of symptoms such as effusion, peripheral swelling, deformation, heat, pain on palpation, pain on flexion, and lameness, are common hallmarks of DFTS tenosynovitis, indicating DFTS pathology.

Effusion, as characterized by an increase in synovial fluid volume, is often noted in acute and chronic DFTS tenosynovitis. The severity of the distension is usually correlated to the severity of inflammation, with septic process typically inducing the most severe inflammation. The chronicity of the condition will usually lead to an enlargement of the proximal and distal recesses, highlighting a clear constricting band at the palmar/plantar aspect of the fetlock. Effusion can sometimes only be noted in the distal or the proximal recess, indicating a poor circulation of the synovial fluid through the meta-carpo/tarso-phalangeal canal. In these cases, a high suspicion of constriction syndrome exists. When combined effusion of the DFTS and fetlock joint is noted, particular attention should be paid to the proximal scutum, with osteitis of the axial border of the sesamoid bone at the top of the differential list.

Diffuse peripheral swelling can sometimes impair the detection of effused synovial recesses, and is typically noted with infectious process, with or without the presence of penetrating wounds.

The DFTS can appear asymmetric and deformed. This is typically true when a complete rupture of the proximal manica flexoria occurs, and the torn material coils up on one side of the sheath (often laterally for medial tears). This finding can also be noted with SDFT branch injuries or uni-axial DDFT lobe injury in the pastern region. Formation of synovial masses can also create distortion of the DFTS silhouette.

Focal heat and increased digital pulses, as a marker of inflammation, can be noted in acute cases. Fever can also be detected in septic cases.

Pain on palpation of the flexor tendons or deformed areas, or during passive flexion, is frequently detected during the acute/sub-acute phase. If a focal point of pain is noted during the clinical examination, this will usually help the identification of the lesion(s) during ultrasonography.

The degree of lameness usually varies with the severity of inflammation. Septic cases are typically lame at a walk but some acute and severe non-septic injury can also present lame at a walk. Most of the chronic non-septic cases are typically lame at a trot (grade 2-3/5 AAEP scale), with sometimes intermittent lameness noted. Dynamic flexion of the distal limb usually exacerbates the degree of lameness, and even more so when some degree of constriction is present.
C- Diagnostic tests

A series of diagnostic tests are available to characterize the DFTS pathology.

C1- Ultrasonography

This is the most useful diagnostic test for any acute or chronic DFTS tenosynovitis.

Several steps are necessary to perform a complete evaluation of the DFTS and to obtain as much information as possible:

1. 2D scan, from proximal to distal, focusing on one structure at a time; leg weight bearing.
2. 2D scan from proximal to distal with dynamic flexion/extension; leg non-weight bearing.
3. Doppler ultrasound on each tendon, mesotenon, and vincula; leg non-weight bearing.
4. Ultrasonoelastography; leg non-weight bearing
5. Optional: 2D scan after adding fluid/US contrast within the DFTS

1. 2D scan; leg weight bearing

**Technique:** This scan is performed in transverse fashion, either in the palmar/plantar plane or on the sides, and also in longitudinal fashion, with a 7.5-12MHz linear transducer, with or without a standoff. The leg is in a weight bearing position, or as much weight is placed on the leg as possible, to avoid relaxation artefacts and to fill the synovial recess properly. The hair is clipped and the skin washed with neutral soap and warm water, rinsed copiously, alcohol is applied to remove the remaining grease/sebum, and coupling gel is applied on the whole DFTS.

**Synovial fluid**
The volume of synovial fluid in the proximal and distal recesses should be evaluated. If the fluid is only present in one location, this usually indicates poor circulation of the fluid through the meta-carpo/tarso-phalangeal canal and this raises the suspicion of a constriction syndrome. A careful examination of meta-carpo/tarso-phalangeal canal is therefore warranted on those cases.

Effusion can sometimes be asymmetrically distributed. This warrants a close look at this level as there is usually a reason, such as a mass/coiled tissue filling the space, or the sheath wall is torn, allowing synovial fluid to accumulate under the skin (synoviocele).

For cases with significant and diffuse peripheral swelling, palpation can be unrewarding and the use of ultrasonography to locate synovial fluid accumulation can really help to select an appropriate synoviocentesis technique/site.

The echogenicity of the synovial fluid should be assessed carefully, as there is usually a good correlation between echogenicity and synovial sample cellularity. If the synovial fluid is
hypoechogenic, aspirating some fluid for cytology purposes may not provide any new information.

**SDFT and DDFT**

SDFT and DDFT are evaluated separately from proximal to distal. The core of the tendons is first scrutinized, rocking the probe to constantly keep the tissue on focus. Core hypo- or hyperchogenic lesions, although less frequently detected as for the tendons in the non-sheathed region, can be present. When the architecture appears modified, performing longitudinal scans to look at the fiber pattern alignment is recommended, as for any tendon scan. When scanning in transverse, tilting the probe by about 5-10 degree to perform an off-focus scanning can help to identify scar tissue within the tendons. Mineralizations with a classical acoustic shadow can be encountered in chronic cases, or when corticosteroids have previously been injected in core lesions, and are often at around the fetlock level. To make sure this area is not overlooked, it can be recommended to trim the ergot with a blade to ensure proper contact in this area. Although clearly abnormal within tendinous tissue and representing an advanced type of tissue scaring, mineralizations can also be incidental findings and not associated with lameness as suggested by O’Brien et al. (2018).

What is most commonly noted in sheathed tendons are clefts/tears, originating from the tendon periphery and extending either longitudinally or transverse-obliquely. A close-up evaluation of the flexor tendon contour is therefore mandatory. The DDFT is the most commonly affected structure, and its border can show irregular/non-sharp edges, with densely or loosely packed material around the area or between tendons, and with reduced echogenicity. When a suspicion is present, scrutinizing this region again with the leg in non-weight bearing position usually provides a great amount of information.

Mesotenons and vincula should also be scrutinized as some of the longitudinal tears originate close from those structures, particularly for the DDFT.

It is important in the distal pastern to assess each SDFT branches, and to remember that their fibrocartilaginous insertion within the middle scutum always looks heterogenous. In case you have a doubt, comparing with the opposite site can be rewarding.

In rare cases, tendon rupture, split or luxation can occur. This can be very disturbing. To ensure proper assessment of each tendon position, it is recommended to find a point more proximally on the leg and to follow the tendon down to help localizing which tendon is displaced.

When the limb has been recently wounded, gas artefacts in the vicinity of the DFTS may alter the quality of the ultrasound examination and serious damage to the flexor tendons can be easily missed.

**Proximal manica flexoria**

Evaluation of the proximal manica flexoria starts with the SDFT edges first in transverse. If the edges of the tendon are thick, heterogenous, or some tissue seems to be missing, or the tissue noted is heterogenous, or there is mass of heterogenous tissue, a high suspicion of complete or partial tear of the proximal manica flexoria can be raised. Scanning of the same area with the leg in a non-weight bearing position can help.

For complete tears, when applying the transducer in a longitudinal fashion in the sagittal plan immediately proximal to the fetlock, the proximal manica flexoria should be found as a triangle of tissue with the tip of the V pointing distally, between the dorsal aspect of the DDFT and the sheath wall. If some synovial fluid can be manually pushed here to separate the manica from the tendon or the sheath wall, this can help immensely. The distal end of the...
manica should be less than 1mm thick. If the tissue is very thick, or absent, serious/complete tearing and displacement can be suspected.

**PAL**
The palmar/plantar annular ligament and the meta-carpo/tarso-phalangeal canal should be evaluated carefully, especially when there is a suspicion of constriction syndrome. If the tendons are enlarged or the sheath wall is thickened, passing through this “ring” will become more and more difficult with time. The body usually responds by forming a thicker band of tissue (PAL and subcutaneous tissue) to counteract the forces. The insertion of the PAL on the sesamoid bones is usually reactive, with irregular new bone formation noted. The PAL should be 1mm thick in the normal horse. To find the dorsal and palmar/plantar margins of the PAL, it helps to move the probe to the sides and to identify the tissue at its bony insertion. In these insertions, the PAL is usually slightly thicker than 1mm in normal horses. If the PAL can’t be identified with certainty, the block of tissue including PAL, subcutaneous tissue and skin should be measured instead. If this is more than 6-8 mm thick, it is considered abnormal. These findings are suggestive of a constriction syndrome but more information can be obtained when scanning the leg non-weight bearing to identify tendon friction signs (see below).

**Sheath wall, mesotenons/plica and masses**
The sheath wall should be clearly delineated but is often covered by proliferated synovium. Defects/tears in the sheath wall can occur. Presence of fibrin instead of proliferated synovial membrane should be differentiated, with a negative doppler usually indicating the presence of fibrin. Possible adhesions between the sheath wall and the tendons can be suspected. Pressure on the sheath wall and movement of the tendon during further scanning can help to draw conclusions. Space-occupying masses can be found in the recesses or confined within the mesotenons. They can adopt very peculiar shapes and are usually made of fibrin and coiled/disrupted tendinous material. Some masses are extending outside the sheath wall, and present a well-defined contour. These masses are usually filled with synovial fluid or mixed echogenic material. They are called ganglia, synovial hernia or synoviocoele. Remember, mesotenons and vincula become thicker with inflammation. They are often only a representation of the degree of inflammation and should not be mistaken for adhesions. The sheath wall also becomes thicker with inflammation and these changes are usually secondary changes. The primary problem is likely somewhere else in the DFTS.

**Other structures**
The digital manica flexoria, although rarely affected, should be evaluated. A significant amount of variation has been reported by Jordana et al. (2016) so it can be wise to assess the contralateral limb in case of a doubt. The proximal and middle scutum should be scrutinized for surface irregularities and particular attention should be given to the axial border of the proximal sesamoid bones if the horse also present a fetlock joint synovitis. The proximal digital ligament is very similar to the PAL but inserts on the wings of P1. It provides stability to the flexor tendons in the pastern region. Avulsion of the insertion, usually with bone, leads to mild-to-moderate DFTS tenosynovitis signs, although this structure is anatomically extrathecal. Other extrathecal structures important to evaluate are the distal sesamoidean ligaments due to their close proximity with the DFTS.
2- 2D scan; leg non-weight bearing

Technique
This scan is performed with the leg up. The transducer is applied in transverse, or in longitudinal fashion. This reduces the tension on the flexor tendons and can be of great help if there is a suspicion raised at the time of the scan with the leg in weight bearing position. Picking the leg up redistributes the synovial fluid in areas where there was none when the leg was loaded. Performing different degrees of flexion/extension of the digit, with transducer remaining static, allow displacement of the flexor tendons relative to each other and relative to the sheath wall. It also allows evaluation of the level of friction in the metacarpo/tarsophalangeal canal.

Marginal tendinous tears
This technique helps to identify marginal tears as usually the torn tissue will get displaced/unpacked and the defect/tear becomes more obvious. Like a partially broken rope, when tension is released, the frayed material will become more obvious.

Manica flexoria tears
As suggested by Garcia da Fonseca et al. (2018), dynamic flexion/extension on the non-weight bearing limb is helpful for identifying manica flexoria tears. Signs such as medial displacement/subluxation of the SDFT in relation to the DDFT, presence of an anechoic gap between the flexor tendons at maximal flexion, reduced gliding of the SDFT in comparison to the DDFT, and loss of gliding of SDFT in relation to the subcutaneous tissue, were characteristics of significant tearing of the proximal manica flexoria.

Adhesions
Adhesions between tendons can be picked up with this technique. When only the coffin joint is flexed (manipulation at the toe level and coffin joint flexed mildly) the DDFT flexes and extends, while the SDFT should remain static. If the DDFT does not move, an adhesion to the sheath wall or to the SDFT should be suspected. If both tendons move together, this may indicate an adhesion between the tendons themselves. Pay careful attention not to over-flex the coffin joint because as soon as the fetlock flexes, the SDFT will start moving.

Constriction syndrome
The diagnosis of constriction syndrome is difficult to establish. As suggested by Digiovanni et al. (2016), the use of dynamic ultrasound can be a useful tool to detect restricted flexor tendon gliding motion relative to the PAL. The probe is applied in longitudinal fashion in the sagittal plan over the PAL and the digit is flexed. In a small number of abnormal horses, the authors subjectively detected abnormal gliding motion, and an increased angulation of the long linear echoes between 20–35°, while normal horses had normal gliding motion and an angulation of 0 to 13°. Once the US images are transferred to a PACS, on the still image acquired immediately proximal to the proximal sesamoid bones, two lines are created, each parallel to the long linear echoes of the superficial digital flexor tendon. The angle between the intersection of these lines is measured, and the angle recorded in degrees.
3- Doppler ultrasound, leg non weight bearing

**Technique:** This scan is performed with the leg non-weight bearing, ideally with the toe touching the ground with the leg unloaded. If the leg is over flexed by an assistant, the blood flow can be artificially reduced.

**Normal tendons**
Normal flexor tendons show no to very little Doppler activity. Mild increase in blood flow is usually detected at the vincula and mesotenon. Within the DDFT, where the tendon is bilobed in the pastern region, a well-defined artery is usually visible between the lobes in the mid-pastern level.

**Findings supportive of injury**
A pronounced vascular pattern is usually visible within a few days after injury, helping with the recognition of acute or subacute tendinopathy when sufficient tissue disruption materialized by hypoechogenicity may not be readily visible. This hypervascular status is a normal development in the natural healing process but should subside within 5-6 months as the healing progresses.

**Abnormal healing processes**
A lack of hypervascularization in injured tendons, especially during the first 2-3 months, is not normal and should be interpreted as a deficient repair response from the body. This problem is relatively frequent in the DFTS and medical intervention is warranted to stimulate the repair process.

There is a category of chronic and painful tendon lesions that do maintain a hypervascular state. The reason for this is unknown but as the 2D ultrasound findings become more discrete with time, the use of colored doppler truly helps to detect chronic lesions and this precious help should not be neglected by clinicians. The reason why certain chronic lesions fails to resolve this hypervascular state is unknown. Tissue biopsies performed in such lesions in human patients have revealed that these hypervascularized areas are also populated with abnormal nerve sprouts, likely contributing to the chronic pain reported in these patients. This same mechanism, although not proven, is plausible in horses too. The way to treat horses within this possible category remains open to debate.

**Tendon matrix degeneration**
There is a category of subclinical tendons in human patients where a rupture or tear is noted and preceding tendon matrix degenerative changes are identified on histology. Recreational human athletes seem particularly affected by this problem. No alteration in the blood vessels either within or around the tendon is detected in these patients prior to a tear/rupture. The body does not seems to be able to respond to the degenerative changes and this failure to elicit a vascular response may be one of the causative factors. The tendon degenerates and ultimately results in a weakened tissue, no repair attempt is made, and as there is no protective pain in these cases, a tear/rupture ultimately occurs. It is unclear if we encounter a similar mechanism in horses too. I suspect certain areas of the flexor tendons in the DFTS are subject to progressive degenerative changes, where no repair attempts are made, and
associated with high biomechanical constraints, are prone to tearing/fraying such as the lateral border of the DDFT in the proximal part of the DFTS.

Evaluation of the blood supply of the flexor tendons can provide clinicians with very valuable information on the healing process (normal vs. abnormal), allow detection of lesions (chronic) that could have been easily overlooked otherwise, and can guide medical/surgical intervention if the body fails to respond adequately, which is not uncommon for DFTS tendon lesions. The use of colored Doppler is also very useful to assess the horse going through a rehabilitation program. Some horses can progress faster, while others can progress slower. While 2D findings remain static after 4-6 months post-injury, a lack or a minimal increase in blood flow at the lesion and proximal to the lesion is a sign of good “tolerance” of the exercise program. Doppler ultrasound is used by the author before each exercise transition to ensure the repairing tissue is ready for the next level. On high level athletes, a recheck doppler ultrasound is also usually performed within days of the re-introduction of a new exercise known to be a critical point in the rehabilitation program as far as tendon re-injury is concerned.

4- Ultrasonoelastography, leg non-weight bearing

Strain and shear wave ultrasonoelastography are the two main techniques available to assess tissue elasticity. Good reviews have been produced by Sigrist et al. (2017) and Ryu et al. (2017) on this emergent ultrasound technique.

With strain elastography, the operator exerts manual compression on the tissue with the ultrasound transducer. Manual compression works fairly well for the SDFT but is challenging for assessing elasticity of the DDFT or SL. The compression results in displacement of the tissues and subsequently alters the sound waves as they travel through the tissue. This displacement is higher in soft, deformable tissues, such as damaged or disrupted tendons, and lower in hard, rigid tissues. Strain elastography is more operator-dependent with less reproducible results also.

With two-dimensional (2D) shear wave elastography, currently the newest elastography method that uses acoustic radiation force, shear waves are generated within the transducer and sent to the tissue in the parallel or perpendicular dimensions without the need to physically move the transducer. Multiple focal zones are interrogated in rapid succession, faster than the shear wave speed. This creates a near cylindrical shear wave cone, allowing real-time monitoring of shear waves in 2D. Shear wave speed, or Young’s modulus, are generated for quantitative evaluation. Elastograms are also produced in real-time for qualitative evaluation, superimposed on a B-mode image, enabling the clinician to receive information regarding the 2D ultrasound anatomy and tissue stiffness at the same time.

In human tendinopathy, shear wave ultrasonoelastography has been shown to be a reliable tool to differentiate between “diseased” and “healthy” tendons, as diseased tendons are significantly softer (60.3 kPa/4.48 m/s) than healthy ones (185 kPa/7.85 m/s). However, this technique not only helps in differentiating between “healthy” and “diseased” tendons, it also adds relevant diagnostic information suitable to quantitatively rate the degree of tendon impairment. In a study performed by Dirrichs et al. (2016), semiquantitative and quantitative
analysis of the shear wave ultrasonoelastograms correlated closely with patients' clinical symptoms. The overall sensitivity of conventional 2D ultrasound and power doppler in detecting tendinopathies was enhanced from 67.1% (94/140) to 94.3% (132/140) when combined with shear wave ultrasonoelastography.

Equine ultrasonoelastography is still in its infancy and strain ultrasonoelastography has been evaluated so far by Lustgarten et al. (2015) and Tamura et al. (2017) on a small number of clinical cases. The technique used was strain ultrasonography, known to be highly operator dependent and less reproducible. Qualitative and quantitative (color percentage analysis; strain ratio) assessments were performed, both on weight bearing and non-weight bearing legs. The authors concluded that ultrasonoelastography allowed better characterization of lesion chronicity and severity. After 3 months post-injury, gray scale ratios from 2D ultrasound remained unchanged on injured SDFT but they were able to monitor restoration of stiffness with elastography in the Tamura et al. (2017) paper. Sequential examinations, including elastography, may have the potential to optimize lesion management and guide the adjustment of rehabilitation protocols.

Currently I do not include ultrasonoelastography as part of my routine tendon/ligament evaluations but I am looking for more guidance during the mid-late term healing phase and this diagnostic tool could reveal itself to be very useful.

5- Optional: 2D scan after adding fluid/US contrast within the DFTS

As suggested by Daniel et al. (2019), injection of 30-35ml of saline in non-distended DFTS significantly improved delineation of the deep digital flexor tendon, manica flexoria, and straight distal sesamoidean ligament during ultrasonographic examination on cadaver legs. Bertuglia et al. (2014) were able to visualize better tears surgically-created within the DDFT on cadaver legs with the use of an ultrasound contrast agent containing stabilized microbubbles.

On clinical cases, the presence of effusion vastly improves the diagnostic ultrasound capacity to detect lesions. The synovial fluid, that can be manually pushed to certain areas of the sheath by squeezing the synovial recess symmetrically or just on one side, separates tendons, manica flexoria, mesotenons/vincula, sheath wall and abnormal structures, from the surrounding anatomy in such a way that margins are delineated.

This step is optional in my opinion, but if you need to block the DFTS to confirm pain/lameness originating from this structure, I often have a second look at the DFTS and in specific areas where a doubt still persists after the first detailed US examination. Usually local anesthetic is mixed with radiographic contrast material to combine diagnostic steps. Creating microbubbles by shaking the syringe with a cap of air at the top, in a sterile fashion, immediately prior to injection, is a simple way to generate an US contrast agent.

Practically speaking, timing is key to get all these tests done and to get the most out of them. Your x-ray machine should be ready for the post-injection radiograph (see below), taken immediately after having walked the horse a few steps. The following 4 minutes are dedicated
to perform a second US exam with the additional fluid/ultrasound contrast material. At 5 minutes post-injection, it is time to assess the response to the synovial block.

C2- Synoviocentesis

Normal DFTS synovial fluid is pale yellow, clear, and does not clot. It resembles to normal joint fluid, with normal cytologic values being similar. Macroscopic and cytologic parameters of the DFTS synovial fluid have been reported by Dykgraaf et al. (2007) and Malark et al. (1991).

The normal total volume of fluid within a normal DFTS in an adult horse varies between 3 to 5mL. Total erythrocyte counts are very low in normal DFTS (50 +/- 0 cells/L), but higher compared to what is observed in normal equine joint fluid. Total leukocyte count < 1 x10⁹ cells/L, percentage of neutrophils < 10% and protein concentration < 10 g/L are typically noted in normal DFTS, and these values are similar to what is recorded for normal joints.

Based on previous investigations in joints, values that are typically interpreted as evidence of sepsis include total leukocyte count > 30 x10⁹ cells/L, percentage of neutrophils > 80%, and protein concentration > 40 g/L. The presence of intracellular bacteria is also highly suggestive of a septic process.

As there is a wide gray zone based on the cytological values, Stack et al. (2019) showed that measurement of synovial SAA can help discriminating between inflamed but non-septic and septic synovial structures. Synovial SAA was significantly higher in the septic group. Correlation between ELISA and handheld test (Equicheck®) results were excellent and the cut-off that maximized sensitivity and specificity for the test was for ELISA SAA ≥ 23.95 µg/mL and for the handheld test was SAA ≥ moderate (1 or 2 lines visible on the test strip). The performance of the tests was highly accurate, and even better when synovial fluid samples were harvested >6h after the onset of clinical signs and if the horse did not receive antibiotics/anti-inflammatories prior to sampling.

For non-septic DFTS inflammation, another biological marker has been reported to be potentially useful in a clinical setting. Smith et al. (2011) showed that clinical cases with intrathecal tendon/ligament tearing had higher synovial fluid cartilage oligomeric matrix protein (COMP) than either clinical cases with other lesions, or normal horses. COMP is a non-collagenous extracellular matrix protein with very high concentration measured in tendons (up to 10 mg/g wet weight). With intrathecal tendon tearing, the protein is released into the synovial fluid but the horse’s age can also affect the intrasynovial content. Smith et al. (2011) suggested that in horses > 5 years old, when synovial fluid COMP concentration is >19.5 mg/mL, tenoscopic surgery is indicated to further investigate and treat DFTS tenosynovitis because intrathecal tendon/ligament tearing is very likely. Currently there is no commercially handheld test available. ELISA dosage in a laboratory is the only way to get COMP measured, making it unfortunately costly and unpractical in a clinical setting.

C3- Diagnostic analgesia
A volume of 60mL is usually necessary to perform good pre-surgical/tenoscopic distension prior to first cannula introduction. To my knowledge, no study has been carried out on the volume required to desensitize the DFTS wall and its structures, so common sense applies. Volumes varying between 5-20mL have been advocated. I personally do not think 5mL is enough to get local anesthetic contact in the entire tendon sheath if the sheath has been distended. After puncture, it is recommended to let excess of synovial fluid drain out before local anesthetic is injected to reduce the dilution effect. I usually prepare a 20ml syringe of 2% lidocaine or mepivacaine and inject a minimum of 10ml. If the resistance to injection is still low I typically inject a further 5-10mL, or more.

If local anesthetic is combined with radiographic contrast material, I still use the same total volume of injection.

Extravasation of fluid is likely when the sheath is pressurized, so it is recommended to keep pressure on the injection site for 10 seconds to prevent this problem.

Jordana et al. (2014) have shown that anesthesia of the palmar/plantar digital nerves with distal limb desensitization often occurs after DFTS analgesia. A higher chance of desensitization exists when injecting the proximal DFTS recess versus other approaches. It is advisable to verify skin sensitivity at the heel bulbs after DFTS analgesia to avoid false interpretations about the origin of pain causing lameness. Although they did not test skin sensation at the heel bulbs in their study, Harper et al. (2007) did not consider analgesia of the DFTS likely to abolish significant pain originating from the sole, navicular bursa or coffin joint based on their results and if the effect of block was checked within 20 minutes.

C4- Contrast tenosynoviography

As described by Fiske-Jackson et al. (2012), injection of iodinated contrast material in the DFTS can help diagnosis of conditions such as manica flexoria tears, DDFT tears and constriction syndrome.

Technique
An appropriate injection site is selected (see below) and prepared aseptically. 10ml mepivacaine plus 5–7 ml sodium meglumine diatrozoate (Urografin 370) are injected in the DFTS. It is important not to perform a subcutaneous injection as this badly affects the image interpretation.
Application of digital pressure at the injection site for 10 seconds is advised to avoid extravasation of fluid and appearance of a patch of contrast that would also affect the image quality.
To obtain adequate distribution of the contrast material, the horse is walked for 20 seconds. Alternatively, passive flexion and extension of the digit can be performed if the horse is too sedated (or lame) to walk.
The digital radiographic equipment should be set up before the injection is performed. Immediately after having walked the horse, perfect lateromedial radiographic views are taken, including the entire tendon sheath. It is recommended to take several views, and to
pay attention to the laterality although minor obliquity does not alter the interpretation significantly. The leg should be totally loaded when the radiograph is taken to make sure sufficient pressure is maintain within the DFTS and to produce an interpretable radiograph.

Interpretation
Kent et al. (2019) redefined the diagnostic criteria used by Fiske-Jackson et al. (2012).

Contrast tenosynovioigraphy has a very good sensitivity (92%) and acceptable specificity (56%) to diagnose proximal manica flexoria tears if a negative answer is given to one the following first 2 questions, or an affirmative answer is given to the last question.

a- Presence of 2 parallel lines delineating the proximal manica flexoria (except distally where it tapers) just proximal to the proximal sesamoid bones, at the dorsal border of DDFT?

b- Most dorsal parallel line delineating the proximal manica flexoria displaced proximally (should meet or overlie the proximal border of the proximal sesamoid bones)?

c- Isolated area of contrast overlying the dorsal border of the DDFT at the level of the proximal manica present?

The first criterion was found to be the most specific (true negative rate). The second criterion was found to be the most sensitive (true positive rate), with more complete disruption of the attachments of the proximal manica flexoria resulting in greater proximal displacement and thereby higher chance of being detected. The last criterion was found to be least specific and sensitive positive finding.

Contrast tenosynovioigraphy was not sensitive enough to diagnose DDFT tears or constriction syndrome. However, if a thin line of contrast extending proximally and obliquely from the outpouching of the DFTS distal to the proximal sesamoid bone, within the outline of the DDFT, this was found to be a very specific sign for DDFT tears.

CS- MRI & contrast-enhanced CT

There is no published information comparing the diagnostic accuracy of US vs. MRI for true DFTS tenosynovitis cases. On rare occasions, the author has discovered tendon lesions on MRI that were not detected on US. These lesions were mainly located within the core of the flexor tendons, and under the ergot, in an area where tendon lesions can be easily overlooked on US due to a lack of contact.

MRI has been mainly used in the context of uncharacterized distal limb lameness without obvious tenosynovitis signs, and when radiographic and ultrasonographic examinations were inconclusive. Although the vast majority of the lesions detected in these cases involve the distal sesamoidean ligaments, strain injuries and marginal tears of the SDFT or DDFT, intersesamoidean ligament desmitis, PAL desmitis, and proximal or distal digital annular desmitis were also identified with this imaging modality by Gonzalez et al. (2010), Dyson et al. (2011) and King et al. (2012).
Acute core lesions of the flexor tendons appear hyperintense in both T1- and T2-weighed sequences, whereas chronic tendon lesions appear hyperintense in T1-weighed sequences but hypointense in T2-weighed sequences. Longitudinal tears of the flexor tendons are characterized by an irregular tendon contour with a hyperintense area at the abaxial border of the tendon and partial separation of the tendon margins.

MRI can be very valuable in horses that US very poorly due to skin thickness issues (cobs) or where skin contact/pressure is painful and not abolished by sedation.

As suggested by Daniel et al. (2019), injection of saline in the DFTS improved delineation and visualization of the margins of the DDFT, SDFT, manica flexoria, and straight distal sesamoidean ligament on non-distended cadaver limbs on MRI.

Although less-adapted to soft tissue evaluation, contrast-enhanced CT has proved to be a good alternative when MRI examination is not available, and can be useful to monitor healing of DDFT lesions as suggested by Puchalski et al. (2009).

**D – Therapeutic approach**

Primary acute and subacute DFTS tenosynovitis, which are characterized by signs of tenosynovitis, without any tendon or manica damage diagnosed on US, are usually best treated with a conservative approach.

Any primary DFTS tenosynovitis not responsive to a conservative approach within 6 weeks should be taken to surgery.

DFTS tenosynovitis, either acute or chronic, but secondary to tendon or manica damage, are usually better treated with a surgical approach. Natural debridement of torn fibers floating in the sheath does not happen. These torn fibers generate ongoing inflammation. As a result, the body needs the help of mechanical debridement to resolve the inflammation spiral.

DFTS tenosynovitis, where a synovial mass, ganglia or synoviocoele is identified, is also usually better treated with a surgical approach.

PAL constriction syndrome, which is usually a manifestation of chronicity of inflammation, either primary (PAL lesions) or secondary (much more common), are also best treated surgically.

**D1 - Conservative approach**

Rest, local and systemic anti-inflammatory treatment, cryo/hydrotherapy, and bandaging, are the main avenues for the conservative approach.

For acute and primary DFTS tenosynovitis cases, rest (strict box rest initially followed by hand walking), ice therapy, systemic NSAIDs (flunixin, phenylbutazone), local application of
diclofenac or dexamethasone gel +/- DMSO, and bandaging, is advised for a period of 1-2 weeks.

For subacute primary DFTS tenosynovitis cases, where no tendon/manica damage is clearly detected on US, the same approach as above applies. In addition, intrathecal injection of short-mid duration corticosteroids and hyaluronic acid is recommended, as the above treatment is not sufficient to resolve or take control of the inflammation.

The selection of an appropriate injection site is based on clinician preferences. 4 approaches have been described and evaluated by Jordana et al. (2012). The presence of a wound or the absence of effusion may preclude the use of certain approaches.

- **Proximo-lateral approach**
  - Leg weight bearing if large effusion or slightly flexed otherwise
  - Needle inserted 1 cm proximal to the palmar annular ligament and 1 cm palmar or plantar to the lateral branch of the suspensory ligament.

- **Palmar/plantar axial sesamoidean approach**
  - Leg moderately flexed
  - Needle inserted at the level of the midbody of the lateral proximal sesamoid bone, through the palmar annular ligament, and 3 mm axial to the palpable palmar or plantar border of the lateral proximal sesamoid bone.

- **Base of proximal sesamoid bone approach**
  - Leg weight bearing
  - Needle inserted in palpable indentation just distal to the lateral proximal sesamoid bone, between the distal aspect of the palmar annular ligament and proximal aspect of the proximal digital annular ligament, palmar or plantar to the neurovascular bundle.

- **Palmar/plantar pastern approach**
  - Leg weight bearing
  - Needle inserted in the palmar or plantar aspect of the pastern region, in the outpouching of the digital flexor tendon sheath located between the proximal and distal digital annular ligaments.

Typically 5-15mg of triamcinolone acetonide and 40-80mg of hyaluronic acid are injected in the tendon sheath, after draining any excess of fluid. A slightly compressive bandage is usually applied immediately post-injection.

While it has been demonstrated by several groups that intrallesional injections of stem cells, PRP, or the combination of both, seems to enhance healing and reduce the risk of re-injury for extrathecal SDFT core lesions, there is currently no evidence that intrathecal injection of stem cells or PRP can help healing marginal lesions (once epitenon is damaged and fibers are exposed), such as longitudinal tears in the DDFT.

In sheep, Khan et al. (2018) created a longitudinal tear within an intrathecal portion of the DDFT. The tendon sheaths were treated with bone marrow-derived stem cells or with saline, as a control. Treated limbs indicated cellular distribution throughout the tendon synovial sheath but restricted to the synovial tissues, with no stem cells detected in the tendon or the surgically created lesion. Evaluation of both treated and control lesions showed no evidence
of healing of the lesion at 4, 12 and 24 weeks on gross and histological examination. Although the stem cells appeared capable of engrafting in the synovial membrane, they were not able to alter/improve the healing process in this model of intrathecal longitudinal tear.

**D2- Surgical Approach**

The possibility of a false negative diagnosis with ultrasonography of marginal tears, or manica flexoria damage, is possible, and tenoscopic exploration of the DFTS on recurrent or chronic tenosynovitis is strongly advised.

As the operating space is limited in the DFTS, the best cases are the ones that have shown effusion for several weeks so a progressive enlargement of the operating space comes along with the pathology. However, for cases where the synovium and/or the tendons are extremely swollen, tenoscopic exploration is difficult and may be traumatic.

Horses are placed in dorsal or lateral recumbency. If the diagnosis is clear from pre-operative ultrasound, positioning the side of the leg to operate on above facilitates the procedure but not necessarily the triangulation. Otherwise, if I have any doubt, I place the horse in dorsal recumbency, giving access easily to both sides of the leg.

The first portal in the DFTS is placed immediately distal to the PAL, midway between the ergot and the neurovascular bundle. A 4mm cannula and a blunt, or conical trocar, is introduced proximally. A thorough and methodical exploration is carried out first, evaluating each tendon (dorsal, palmar/plantar, medial and lateral aspects), manica flexoria (distal free border, edges, and areolar tissue proximally), mesotenons/vincula, and sheath wall. The proximal and middle scutum, as well as the PAL, are also assessed. Introduction of a palpation probe if often necessary to retract tendons, or if a tear is suspected on first look. Particular attention should be paid to the DDFT lateral border, where longitudinal tears are common within, and out of, the manica flexoria on front limbs. Also, the medial aspect of the manica flexoria should be scrutinized on hind limbs.

In cases where a constriction syndrome is suspected, or if the arthroscope/tenoscope travels with serious difficulty/friction from distal to proximal in the sheath through the metacarpo/tarso-phalangeal canal, the PAL is transected first, prior to the examination, to avoid damage to the flexor tendons. To perform this procedure, the author prefers the sickle-hook knife designed by Dr. Hans Wilderjans, which is available from Rita Leibinger company. As described by Wilderjans et al. (2003), a portal is made 2-3 cm proximal to the PAL. Care is taken not to introduce the hook knife through/into the manica flexoria. The knife is brought distally under visualization and the distal aspect of the PAL is hooked off midline and transection is performed under visualization by withdrawing the knife and applying external pressure to ensure the blade travels deep enough in the PAL and thickened subcutaneous tissue. An immediate tissue release is noted, allowing further exploration to be performed safely. It may be important on some cases to reverse scope and instrument portals to ensure the distal aspect of the PAL is entirely sectioned.
Other techniques have been described to transect the PAL, one with a slotted cannula used for carpal canal release in humans (Nixon et al. (1999)) and another one with a special equine hook knife designed for ultrasound-guided desmotomy (Espinosa et al. (2017)). Complications post PAL desmotomy, such as adhesion formation between the tendons and the PAL transected edges, have been reported. Surgeons performing this procedure should keep this in mind and have strict selection criteria.

When exposed tendon fibers or tears are detected, mechanical debridement with the shaver, is recommended. The debrided edges always appear rough at the end of the debridement. Coblation, or electrosurgical brushing, of the edges has been shown to be deleterious and should not be performed. If the epitenon is not ruptured, it is recommended not to debride these lesions, unless the shape of the tendon is severely affected and tendon movement is restricted relative to the other tendon, the PAL or the manica flexoria. Tears can be very extensive and a portal placed through the manica flexoria to deal with long DDFT tears is sometimes necessary.

For manica flexoria tears, incomplete and small tears are debrided. For more severe or complete tears with coiling and adhesions, the manica flexoria is completely resected, as suggested by Findley et al. (2012), using a 3-portal technique. The author uses a similar technique, but with a Wilderjans sickle-hook knife or electrosurgery, to transect the intact border and proximal aspect of the manica. Long term results obtained with DDFT tear debridement are not satisfactory as only 38% of the horses will resume soundness and return to the same level of pre-injury performance, as suggested by Arensburg et al. (2011). Some therapeutic improvements are therefore required for these lesions. Anecdotal reports of injection of PRP gel +/- stem cells at the end of the debridement under gas tenoscopy may improve the prognosis. Closure of the tear via tenoscopy is extremely difficult and technically challenging with the current instrumentation available. Open approach for tear suturing has been advocated but no critical analysis has been conducted. Postoperative care is more challenging with an open approach and the risk of sepsis or adhesion formation are also largely increased.

Long term results obtained with manica flexoria tears debrided or resected are satisfactory with 79% of the horses resuming soundness and returning to the same level of pre-injury performance, as suggested by Findley et al. (2012). Synovioceles or ganglion cysts, when associated with focal pain and lameness, as documented by Crawford et al. (2011), should be resected surgically if the conservative approach does not provide any results. An open approach directly over the “cyst” is advised. The sac and stalk are resected with a small amount of continuous sheath capsule. Closure of the sheath wall with absorbable suture is then performed.

D3- Rehabilitation

Orthopedic farriery
When a lesion of the DDFT is identified, trimming/shoeing to increase the angle between the solar surface of the pedal bone and the ground is recommended. Lateral radiographs of the
foot on a block in a neutral position (both feet on the blocks and at the same level) are taken. The focal point is the interface between the foot and the block, not the coronary band as for typical coffin joint radiographs. An angle elevation of 3 to 10 degrees, depending on the foot conformation, severity or the stage of the DDFT lesion is recommended immediately after the lesion is detected. This reduces the stress on the DDFT but the extra load will be shared between the SL and SDFT.

When a lesion of the SDFT is identified, trimming/shoeing to reduce the angle between the solar surface of the pedal bone and the ground is recommended. Again, the same lateral radiograph as described above is important in the case management. For severe lesions, the toe can be slightly elevated (3 degrees max) to recruit the DDFT to support the fetlock joint more and release the stress on the SDFT. Once a horse is walking or exercising on the giving ground, a suspensory shoe with wide toe and narrow heels will exacerbate the effect of the toe elevation in a dynamic way.

The modifications of the foot balance in the sagittal plane are reduced given time but often maintained for the rest of the athletic life of the horse, especially if a complicated healing has occurred and scar tissue formation is important.

Exercise
Rest and progressive return to exercise is advised, as for any soft tissue injury. The use of swimming and underwater treadmill can help to increase the level of muscular activity and general fitness without loading the tendon too quickly. Horses under exercise in water protocols do not get overweight and usually go through their rehabilitation faster. Although completely hypothetical at this stage, the author suspect the early mobilization in water allows increased blood flow to the leg and recruitment of better healing capacity in general.

For horses that do not take rest easily, the EqueStride rehabilitation device has been designed to control/prevent fetlock drop. In association with orthopedic shoeing, this rehabilitation device with its 4 positions can be very rewarding, allowing early remobilization.

For post-operative cases, walking in-hand immediately after the portals are sealed is advised with passive flexions/extensions. Usually, most of the horses start walking on day 4 post-surgery to prevent adhesion formation.

Physical therapy
Although there is a true lack of information on the use of therapeutic laser and magnetic diathermy therapy, these 2 modalities are commonly used by the author to encourage repair. Some evidence-base medicine is critically lacking in this area.

Viscosupplementation
Viscosupplementation is sometimes performed at the time of surgery but always after surgery. At 3 weeks, if the DFTS is still effused, injection of 40-80 mg hyaluronan is advised with a small dose of short acting corticosteroids. This treatment is repeated at 6 weeks post-surgery.
Others

Cushing’s disease has been associated with tendon degeneration. ACTH testing in horses with tendon injuries is recommended and pergolide treatment can be added if the horse is diagnosed with pituitary pars intermedia dysfunction.

Oral nutraceuticals such as Ramard Total Tendon Repair or Audevard Ekyflex Tendon are often advised to clients, although no scientific evidence has been produced by either of the companies, but some evidence does exist regarding some of the ingredients in each formulation.