Tendon and Ligament Injury

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1. Diagnosis of Tendon Injury

The most frequently injured tendons and ligaments in the horse are those on the palmar or plantar aspect of the distal limb. For this reason, this series of presentations will focus on these injuries. Diagnosis of strain-induced tendon injuries of the equine distal limb are based on history (usually a preceding period of exercise) and the development of the signs of inflammation (pain, heat, swelling, and lameness) over the affected structure. Confirmation and semi-objective assessment of severity is provided by diagnostic ultrasound.

2. Physical Examination

Lameness, which is often severe in the early stages, may not always be present when a patient is presented to a clinician, and it tends to be related to the degree of inflammation rather than the degree of damage. Similarly, after the inflammatory phase has passed in 1–2 wk, lameness usually resolves rapidly; however, the injury takes much longer to heal. Additionally, some tendon and ligament injuries do not follow this pattern. Deep digital flexor tendon (DDFT) overstrain injuries often remain persistently and markedly lame, and suspensory ligament (SL) desmitis, especially proximally in the hindlimb, can result in lower grade but persistent lameness.

Initial Examination—Non-Contact Observation

Observation of the limb before palpation can provide a considerable amount of information on the injured structure (nature and location of the swelling) and severity of the injury (alteration in the posture and function of the limb).

Swelling for superficial digital flexor tendon (SDFT) is most apparent when assessing the very palmar contour of the limb. It is often centered just distal to the mid-metacarpal region, but it can also be in the proximal metacarpal region (high bow) or distal within the digital sheath (low bow). In subtle cases, this swelling may only be apparent when the hair is clipped from the limb. Deep digital flexor tendinopathy rarely, if ever, occurs in the extra synovial portion of the tendon. Thus, injuries to this tendon are invariably associated with digital sheath distension and swelling in the pastern region. Desmitis of the accessory ligament of the DDFT (ALDDFT) occurs in the proximal one-half of the metacarpal region and is located immediately dorsal to the SDFT. It is often confused with DDFT enlargement, because it wraps around the tendon. Suspensory desmitis results in swelling over the affected area. It can occur proximally because of the presence of the splint bones; swelling may be minimal, especially in hindlimbs. It can also occur more distally in areas dorsal to the flexor tendons. Swelling of the body and branches of the SL is found

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medially and/or laterally and is immediately palmar to the metacarpal bone.

Resting metacarpophalangeal (MCP) joint angle is often normal with superficial digital flexor tendinopathy because of the action of the other supporters of this joint (SL and DDFT). Additionally, pain will result in a reduced loading of the limb. However, in cases of severe superficial digital flexor tendinopathy, the affected limb shows greater than normal overextension of the MCP joint when the load on the limb increases (e.g., when the contralateral limb is raised or when walking). Severe damage to the SL will have a greater effect on MCP joint extension. ALDDFT desmitis rarely affects limb posture unless adhesions occur between it and the flexor tendons. In that case, the limb can take on the appearance of a flexural deformity.

Palpation

In a case of suspected flexor tendon injury, careful palpation of the tendons and ligaments in the limb should be made both when the limb is bearing weight and not bearing weight (flexed). When weight bearing, enlargement is assessed by comparison with the contralateral limb; however, bilateral disease is common. With the limb raised, the flexor tendons become slack. Careful attention should be given to pain response, subtle enlargement, which often manifests as an indistinct border to the tendon, and consistency of the structure (soft after recent injury and firm after healing). The horse must be relaxed so that muscle activity does not tense the tendons and make them appear artificially firm. This assessment should also include the contralateral limb, because many strain-induced injuries are bilateral; however, one limb is usually more severely affected than the other limb.

Swelling of the ALDDFT is detected by proximal swelling, usually predominantly laterally, because this is where the body of the ligament is situated. Enlargement is best identified with the limb flexed and palpated between the flexor tendon bundle and the SL in the proximal metacarpal region.

The same evaluation should be made for the SL. Unfortunately, the proximal region is impossible to palpate in the weight-bearing limb, especially in the hindlimb, because it is covered by the heads of the splint bones and the taut flexor tendons. The proximal SL in the forelimb can be palpated in the raised limb by moving the flexor tendons to one side and pressing between the heads of the splint bones. A comparison should be made between sides, because some normal horses may respond.

Percutaneous tendon injuries are usually associated with moderate to severe lameness and may or may not have a concurrent wound. If a wound is present, it should be initially cleaned and then explored digitally with sterile gloves to find the damaged structures. Small wounds may hinder full evaluation, because the tendon laceration site, sustained under full weight-bearing load, is unlikely to be visible in the wound when the horse is severely lame. In such cases, concurrent ultrasonographic examination is very helpful. Penetration injuries or partial severance of a tendon will not alter the function of the tendon, and therefore, other than lameness, there will be little alteration in limb conformation. Complete transection, however, is associated with significant alterations in limb conformation under loading.

SDFT is the overextension of the MCP joint under weight-bearing load.
SDFT + DDFT is the overextension of the MCP joint at rest and when weight bearing; the toe is elevated from the ground when weight bearing.
SDFT + DDFT + SL is the MCP joint on the ground.

If the laceration is complete, the proximal part of a lacerated tendon often recoils and can become reflected on itself. It is also necessary to assess if any synovial structures have been penetrated. This is a common complication of trauma to the distal limbs and will frequently lead to synovial sepsis.

3. Ultrasonography

Indications for Ultrasonographic Evaluation of the Tendon and Ligament Injuries

1. Diagnosis

Although most metacarpal/metatarsal tendon and ligament injuries are easily detectable by palpation, palpation provides a poor objective assessment of the severity. A base-line scan can provide an assessment of severity that may relate to prognosis. It is usually performed 7–10 days after injury, because injuries can worsen initially. In the pastern, however, non-specific fibrosis that commonly accompanies soft tissue injuries in this region makes accurate determination of the injured structure difficult. Therefore, ultrasonography is essential for establishing an accurate diagnosis in this region.

2. Management

Follow-up ultrasonographic examinations (ideally every 2–3 mo) are used to optimize management decisions during the rehabilitation phase.

Ultrasonographic Technique

The limb should ideally be prepared by clipping a strip of hair from the palmar aspect of the limb. For the proximal SL in the hindlimb, it is useful to extend this clipped area to the medial aspect to increase the size of the ultrasonographic "window." The body of the SL is usually also evaluated from the palmar aspect; however, this only enables the axial one-third of the ligament to be examined. Therefore, a more complete examination can be achieved by increasing the clipped area for transducer place-

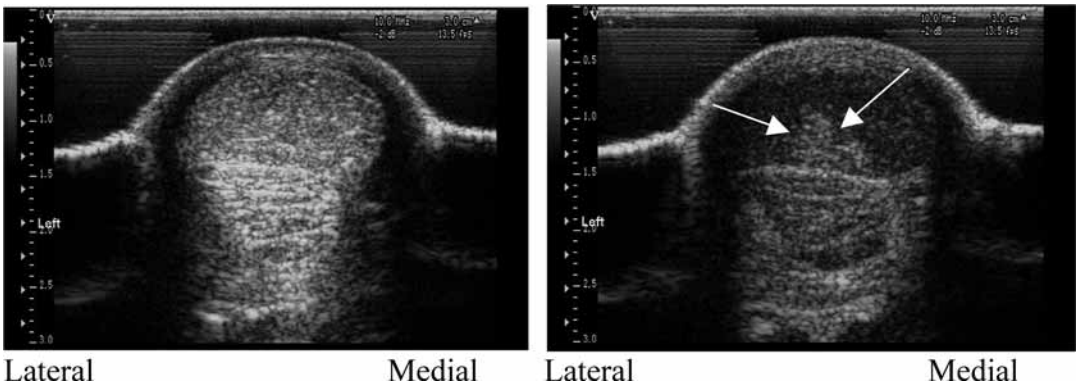

Fig. 1. Transverse ultrasonographs from the mid-metacarpal region showing the use of the off-incidence artefact and its ability to identify areas of poorly organized tissue post-healing. (Left) Normal on-incidence view. (Right) Transducer tilted by ~10°. Arrows show the retained echogenicity in the poorly organized scar tissue when the transducer is tilted.

ment to the medial and lateral aspects of the limb. Because the branches cannot be adequately examined from the palmar/plantar aspect of the limb, these are evaluated with the transducer placed directly over the branches on the medial and lateral aspects of the limb.

Careful preparation of the area is essential if good diagnostic images are to be obtained. After clipping, the area should be cleaned. Ideally, a surgical scrub should initially be used followed by surgical spirit, which degreases the skin and removes the bubbles created by the surgical scrub. Any excess is wiped from the limb, and then, high-viscosity contact gel is rubbed well into the skin. While scanning, the horse should be standing square so that both limbs are evenly loaded. Sedation may be necessary, although usually low doses of α-2 agonists (detomidine or romifidine) are used to minimize swaying. Both limbs should be examined, because many cases of strain-induced tendon injury have bilateral components. The contralateral limb can also serve as a comparison to help differentiate lesions from normal anatomical variants, which are usually bilaterally symmetrical.

There is no standardized technique, but a system of seven levels or zones is recommended; each has characteristic anatomical features.[1,2] The palmar/plantar pastern region is also divided into 3–5 levels or zones. The distal two zones correspond to the more distal position that can sometimes be achieved with a small footprint transducer; however, a more distal examination can be achieved with caudal limb position that hyperextends the distal interphalangeal joint. At least one longitudinal level is usually achievable with a linear transducer depending on the relative size of transducer and pastern. Easier access can be achieved by raising the foot on a block. Because a number of structures pass obliquely across the first phalanx, oblique 45° views should be used to perform a complete examination.

4. Principles of Interpretation—Ultrasonographic Pathology of Tendons and Ligaments

1. Echogenicity

For tendon injuries in general, hypoechoic change suggests an acute injury, whereas chronic pathology is characterized by a heterogeneous pattern of variable amounts of hypoechogenicity and hyperechogenicity. In chronic DDFT injuries (usually within the confines of the digital sheath), mineralization can frequently be found. Off-incidence transducer orientation can help to define areas of disorganized scar tissue in chronic injury, because it retains its echogenicity at greater transducer angles than normal tendon (Fig. 1).

2. Size

The SDFT cross-sectional area (CSA) is one of the most sensitive harbingers of impending reinjury because of excessive exercise during rehabilitation. There is large interindividual variation in CSA in normal horses. A recent study of a large number (n = 148) of National Hunt Thoroughbreds in the United Kingdom gave 80–130 mm^2 as the normal range for the mid-metacarpal region of the SDFT.[3] A >20% difference between limbs is considered a significant enlargement, although this may not be the case if both limbs are affected. When summing separately, the percentage of tendon damaged in the CSAs of the SDFT and the lesion from all seven levels is split into three levels: 0–15% damage is considered a mild injury, 15–25% damage is a moderate injury, and >25% damage is a severe injury. Sequential CSA measurements provide a more sensitive indicator of exercise to tendon healing mismatch during the rehabilitation phase. If the CSA at any level increases by >10%, it is advisable to maintain or lower the exercise level respectively.

The CSA on the other tendons and ligaments of the distal limb can also be used in this way. However, the CSA measurement of the proximal and

body regions of the SL is not possible, because the ultrasound "window" is narrower than the width of the ligament.

3. Pattern

In the longitudinal view, the tendon appears as a series of striations that relate to the linearity of the collagen fibers. Because tendon function relies heavily on this arrangement, the fiber alignment is important in assessing the current and, to some extent, the future functionality of healing tendon. The fiber alignment score (FAS) gives a semi-objective assessment between 0 (normal) and 3 (no striations visible).

4. Shape

Alterations in shape will occur with almost any tendon or ligament injury, but it can be an important indicator of subtle tendon pathology when the CSA is within the normal range. In addition, both percutaneous trauma, which tends to cause focal damage to the palmar surface of the SDFT, and focal adhesions in the tendon sheath can also distort the tendon shape.

5. Position

The SDFT becomes medially displaced with severe superficial digital flexor tendinopathy because of lengthening of the tendon. Adhesions can also alter the position of a tendon within tendon sheaths. In the case of the SDFT, complete transection of one branch in the pastern region results in a shift in position toward the side of the intact branch proximally (Fig. 2).

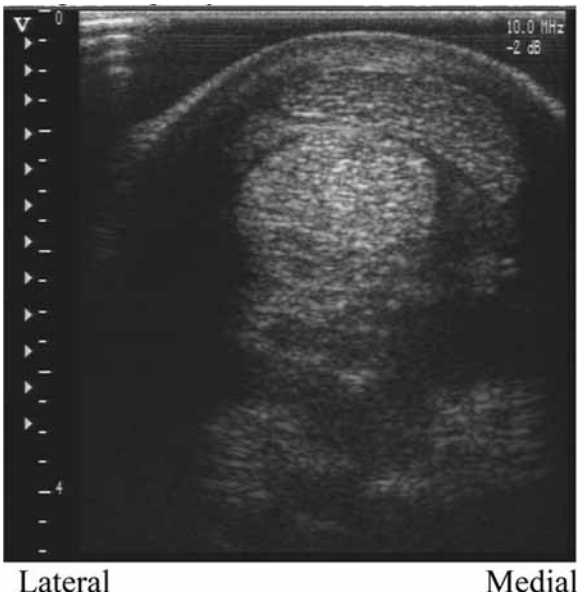

Fig. 2. Transverse ultrasonograph from the distal metatarsal region in a horse suffering a pastern laceration that had completely transacted the lateral branch of the SDFT. Note the altered medial position of the SDFT proximal to the metatarsophalangeal joint.

6. Margination

Within tendon sheaths, individual adhesions can sometimes be visualized when surrounded by fluid (normal mesotenon/synovial plicae). Poor tendon border definition has been suggested to be a sign of adhesions, but it can lead to their overestimation. Optimally, it should be determined by tenoscopy. Real-time imaging while the limb is flexed and extended will allow the ultrasonographer to assess the degree of the movement of the tendons and ligaments relative to one another and therefore, identify adhesion between adjacent structures.

Longitudinal tears in the DDFT within the digital sheath, easily observed tenoscopically, are often poorly discernible ultrasonographically. Greater sensitivity in detecting these tears can be made by using an oblique transducer position to assess the lateral and medial borders (Fig. 3). In contrast, some central defects may extend to the surface of the tendon without penetrating the epitenon and thus, may not be visible tenoscopically.

7. Vascularity

The blood flow within healing digital flexor tendons can be assessed using Doppler with the limb raised (Fig. 4). Normal digital flexor tendons usually have minimal discernible blood flow, whereas a pronounced vascular pattern is usually visible after injury. Hypervascularity is normal in the healing process but should subside as healing progresses.

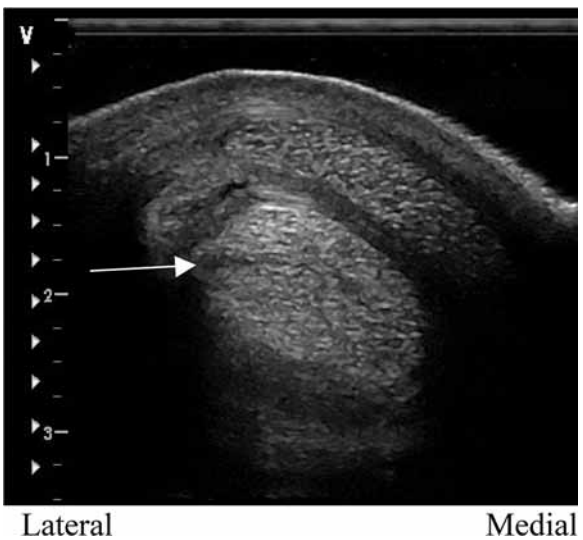

Fig. 3. Oblique transverse ultrasonograph from the palmarolateral aspect of the limb immediately proximal to the metacarpophalangeal joint showing a tear in the lateral margin of the DDFT. (Arrow) These are not always visible ultrasonographically. Note the echogenic material to the lateral side of the tendon that is a non-specific sign of such tears. This material can be torn tendon fibers or thicken synovial plicae, which can also be the site of fibrous mass formation is chronic cases.

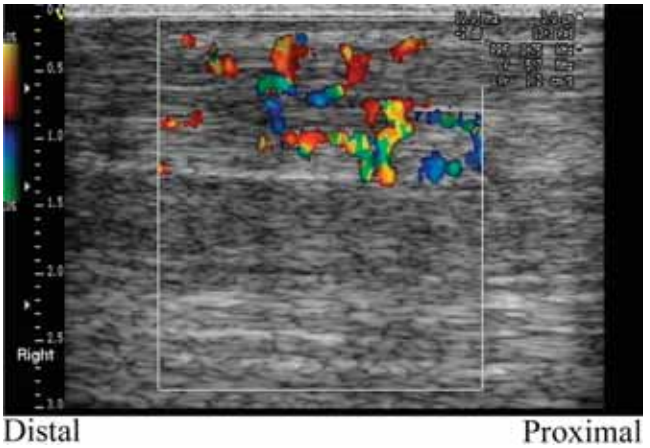

Fig. 4. Longitudinal ultrasonograph from the mid-metacarpal region from a horse with a damaged SDFT that showed increased vascularity with color-flow Doppler in a non–weight-bearing limb.

This technique is particularly useful for identifying exacerbations during the healing phase.

5. Ultrasonographic Appearance of the Metacarpal Region and Pastern Regions

SDFT

Proximally, the tendon lies within the carpal sheath as a semi-circular structure that is palmaromedial to the DDFT (Figs. 5 and 6). As the tendon runs distally, it reduces in the CSA and adopts a rounded medial contour and sharper lateral border. In the distal metacarpal region, it thins in a dorsopalmar direction and extends a ring of tissue around the DDFT (the manica flexoria). Tearing of the attachment of this structure to the SDFT can cause lameness (especially in hindlimbs), although diagnosing this ultrasonographically is difficult.

Distal to the fetlock, the SDFT continues as a thin structure that then divides into two branches in the mid-pastern region. Before its division, the distal "manica," another ring of the SDFT surrounding the DDFT, is usually visible deep to the DDFT. It is a useful landmark, but contrary to its more proximal sister, it is rarely significantly injured. The two SDFT branches run abaxially to insert through the thick fibrocartilagenous middle scutum onto the proximopalmar aspect of the middle phalanx. These branches are best observed ultrasonographically as comma-shaped structures with the transducer on the palmarolateral and palmaromedial aspects.

DDFT

In the proximal forelimb, the DDFT lies dorsolateral to the SDFT. As the tendon runs distally, it becomes more circular and also reduces in the CSA. In the mid-metacarpal level, the ALDDFT joins the DDFT on its dorsal surface and becomes enclosed in the one paratenon. However, the fibers of the ALDDFT can be identified, separated from the DDFT by a hypoechoic curved line, for an appreciable distance distally. In the distal metacarpal region, the DDFT increases in the CSA and becomes oval in shape at the level of the MCP joint. In the hindlimb, the dorsal surface of the DDFT usually has a well-circumscribed hypoechoic region within it in the proximal limit of the digital sheath that is normal.

Within the pastern region, the DDFT will frequently contain a dorsal hypoechoic region immediately distal to the ergot caused by off-incidence artefact from the change direction in the DDFT. As the DDFT runs distally, it adopts a bilobed appearance.

The DDFT can be examined further distally, but this requires a small footprint (e.g., curvilinear) probe that can be placed in the longitudinal plane between the bulbs of the heel. This allows identification of the DDFT distally to the level of the proximal border of the navicular bone, but it is off incidence. The DDFT overlying the navicular bone and inserting onto the solar surface of the distal phalanx can be seen when scanning through the frog; however, only the central portions of the tendon are visible.

ALDDFT

This ligament arises from the palmar carpal ligaments where it lies on the dorsal surface of the carpal sheath. It runs from a deep position proximally to a more superficial position distally where it joins onto the dorsal surface of the DDFT in the mid-metacarpal region. Proximally, it is a discrete structure that is separate from the other structures on the palmar aspect of the limb with a prominent longitudinal striated pattern. It runs in a slightly oblique angle compared with the flexor tendons, and its on-incidence echogenicity tends to be at a slightly different probe orientation to the flexor tendons. Thus, the flexor tendons or the ALDDFT can appear brighter than the other depending on probe orientation. As it runs distally, it starts to conform to the dorsal surface of the DDFT. The majority of the ligament is laterally positioned so that the transducer has to be moved to a palmarolateral position to view the entire ligament.

SL

At its origin, the echogenicity can be very variable, and it can include central hypoechoic regions. These normal variants are caused by areas of looser connective tissue within the ligament that contain fat and vascular elements. They are usually bilaterally symmetrical, but the presence of hypoechoic areas in this region should only be interpreted in the light of clinical examination and diagnostic analgesia. The dorsal border of the ligament is usually distinct and separated from the underlying palmar aspect of the metacarpus by a small anechoic gap. This hypoechoic area

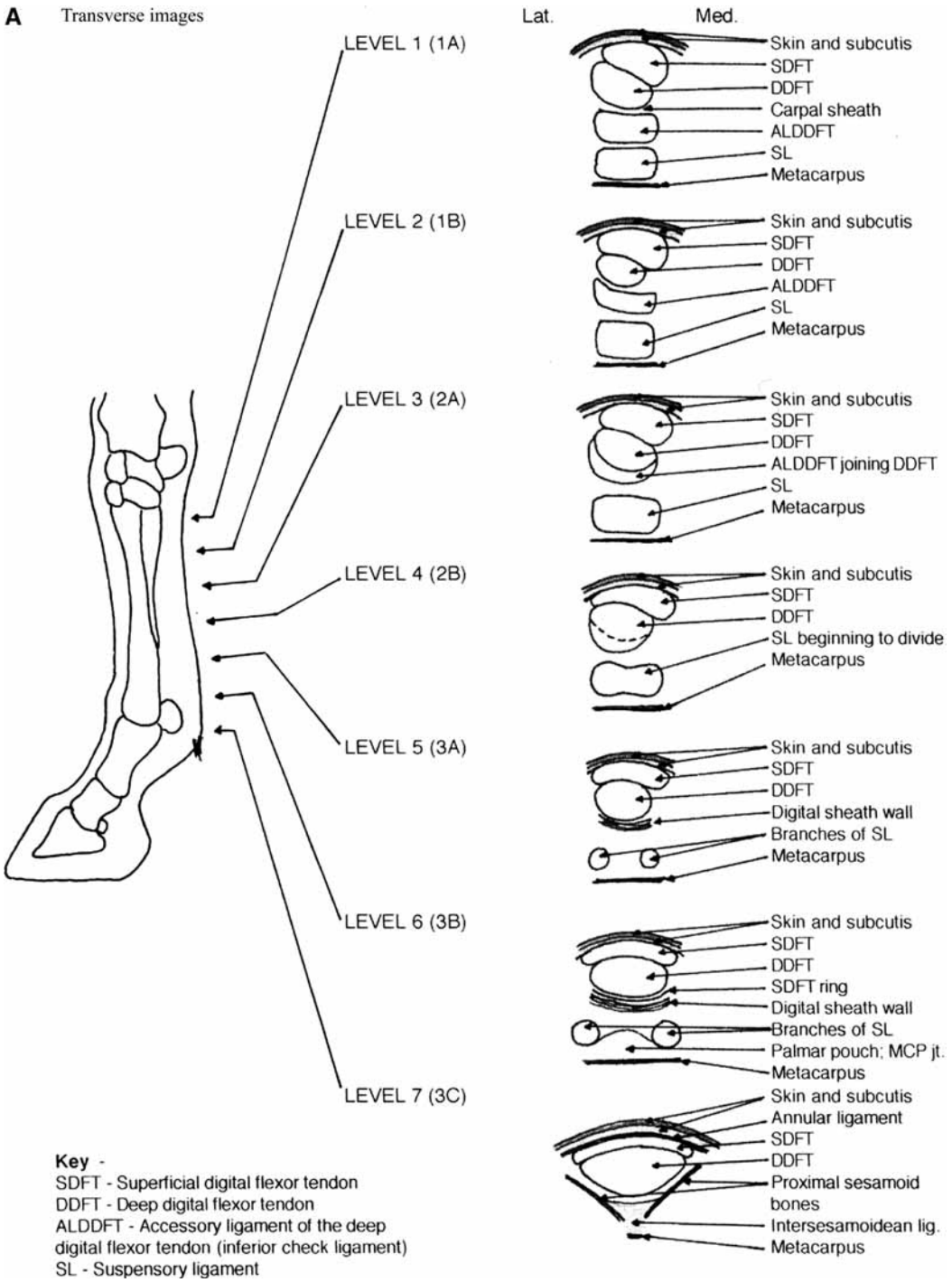

Fig. 5. Diagram representing the ultrasonographic anatomy of the metacarpal region. (A) Transverse images. (B) Longitudinal images. (From Smith RKW, Webbon PM. Diagnostic imaging—musculoskeletal ultrasonography. In: Hodgson DR, Rose R, eds. *The athletic horse*. 1992.)

becomes obliterated when the ligament is enlarged through pathology.

Both proximal and body regions of the forelimb SL are rectangular in shape in transverse images, but this only represents the middle one-third of the ligament because of the size of the ultrasonographic window. The medial and lateral borders can only be visualized by tilting the transducer onto the palmaromedial and palmarolateral aspects proximally and then positioning the transducer directly over the medial and lateral borders in the mid-metacarpal region where the splint bones are smaller. Because of the variable presence of muscle within the proximal and body (but not the branches) of the SL,

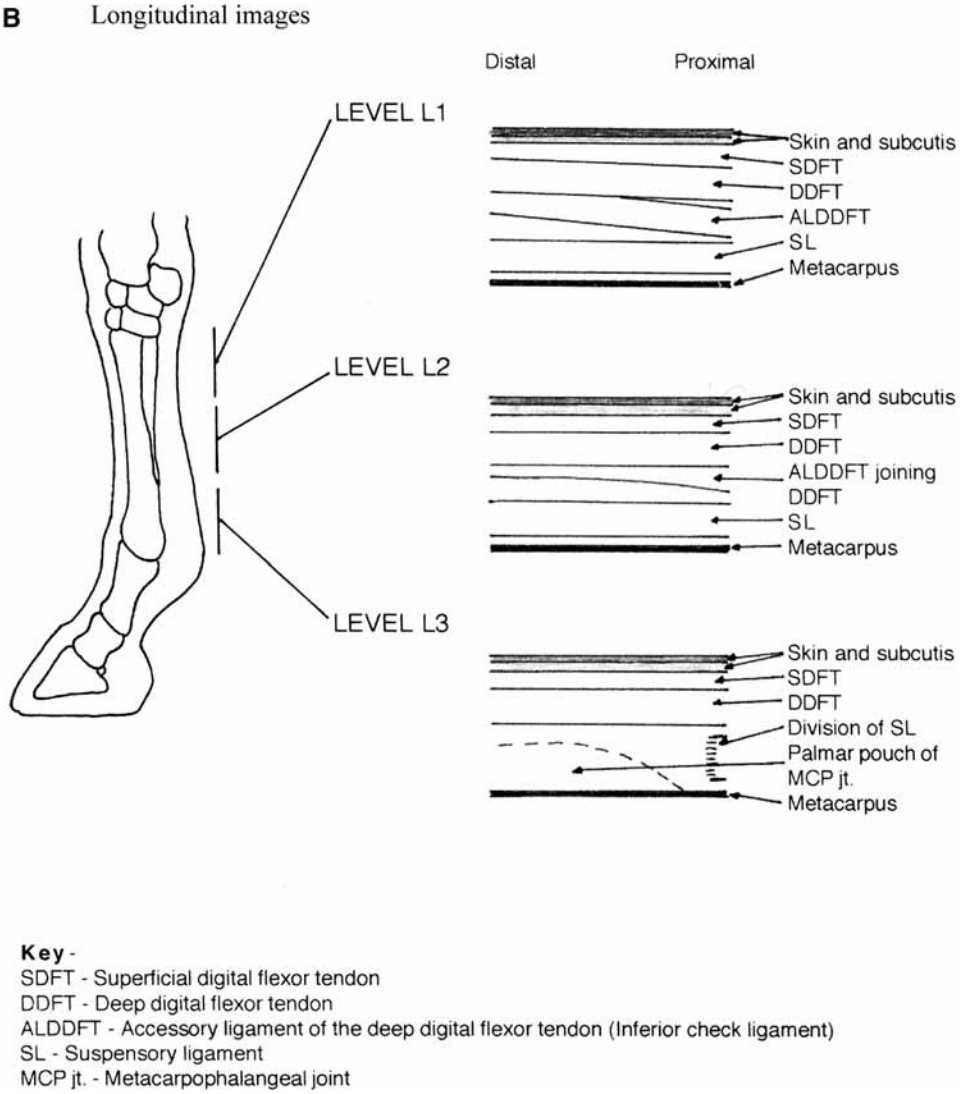

Fig. 5. (continued)

the longitudinal striated pattern of the SL is more coarse than seen in the flexor tendons.

The proximal SL in the hindlimb is more triangular in shape, and it is closely associated with the large head of the fourth metatarsal (lateral splint) bone and the smaller head of the second metatarsal (medial splint) bone. This area is difficult to evaluate and can be improved by one of the following two actions:

1. Move the transducer to the medial aspect of the limb. The ultrasonographic window is larger in this location because of the small head of the second metatarsal bone. A more complete evaluation of the proximal SL can be obtained in this location; however, edge refraction artefacts from the prominent blood vessels superficially in this region can induce shadows within the proximal SL.
2. Use a curvilinear transducer or "compounding," which provides a wider view of the deeper areas.

In longitudinal views, the proximal SL has a striated pattern, and the majority of the ligament is attached to the proximal palmar/plantar metacarpus/metatarsus. The most superficial portion of the ligament, however, continues and inserts more proximally.

In the distal one-third of the metacarpal region, the SL adopts a dumbbell shape in transverse images as it divides into two separate branches. Because of edge refraction shadowing from the borders of the flexor tendons, the branches cannot be visualized adequately from the palmar aspect of the limb, and therefore, the transducer needs to be moved so that it lies directly over the medial and lateral SL branches. These branches increase in the CSA in a proximodistal direction and are a teardrop shape. They lie immediately adjacent to the skin. Any pathology in these branches results in fibrosis between the branch and the skin, which effectively "moves" the branch away from the skin.

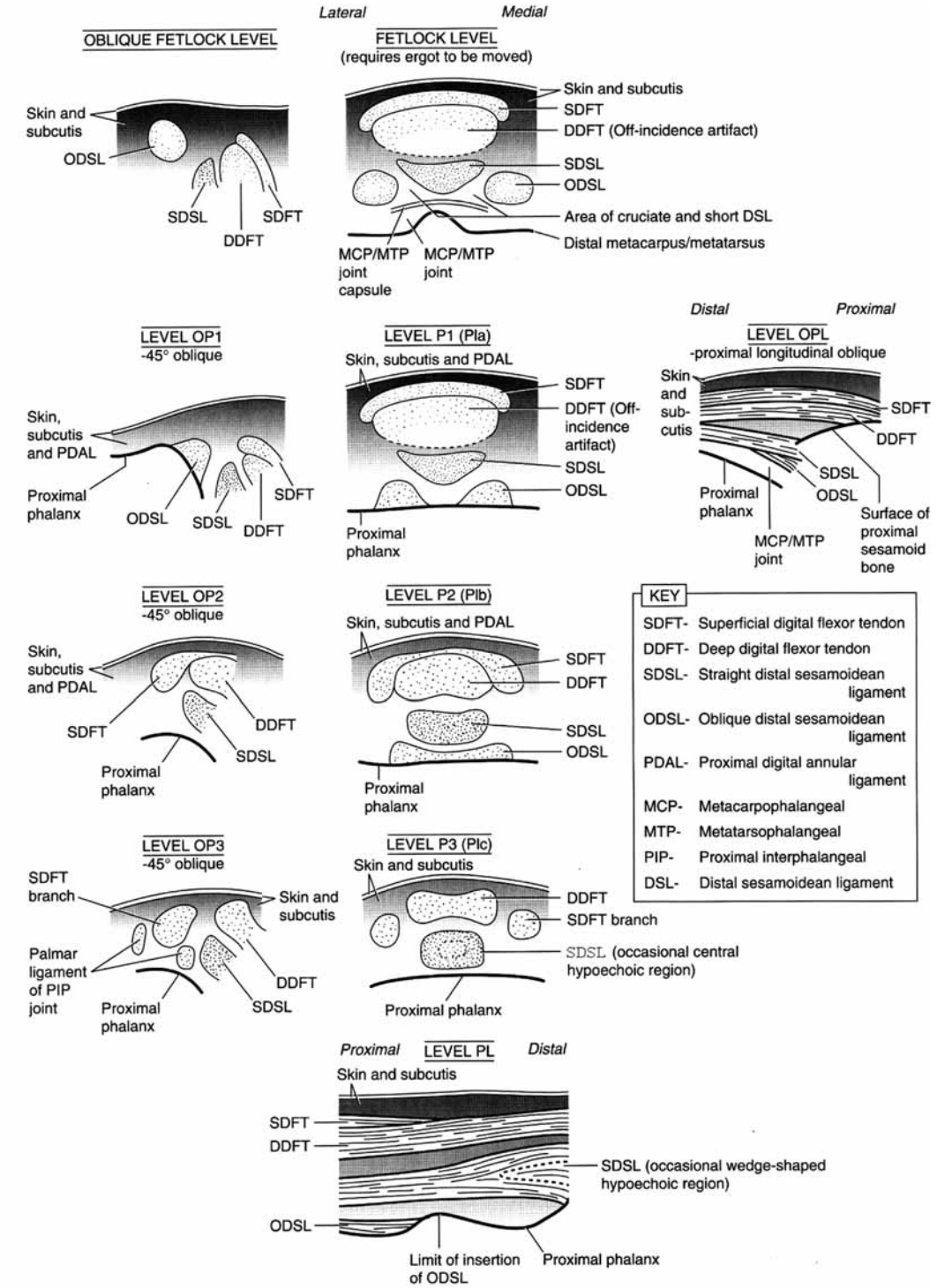

Fig. 6. Diagrammatic representation of ultrasonographic anatomy of the pastern region. (From Smith RKW, Webbon PM. Soft tissue injuries of the pastern. In: Robinson, NE, ed. *Current therapy in equine medicine*, 4th ed. Philadelphia: W.B. Saunders Co., 1997;61–69.)

Corresponding longitudinal images should also be obtained starting with the most distal of these longitudinal images where the attachment site of the SL branch onto the abaxial surface of the proximal sesamoid bone appears as an S-shaped surface; this has been termed, descriptively, the "ski-jump view."

The branches show similar fiber alignment to flexor tendons at this level.

Digital Sheath

The digital sheath extends from the distal metacarpal/metatarsal region to the foot on the palmar/plantar aspect of the limb. Therefore, abnormalities of this structure should include evaluation of this entire region. The digital sheath is commonly associated with pathology in the pastern region, although its involvement is more frequently secondary. In normal horses, the digital sheath contains only small amounts of synovial fluid, and its intrathecal architecture is often obscure. However, with effusion, more structures become visible. Outpouchings of the digital sheath can be seen proximally abaxial to the flexor tendons, immediately distal to the proximal sesamoid bones abaxially, and in the distal pastern region in the midline superficial to the DDFT. This is the best site to aspirate synovial fluid from the sheath.

In the distal metacarpal region within the proximal pouch of the digital sheath, abaxial synovial plicae connect the DDFT to the digital sheath wall both medially and laterally. Although not normally visible in the non-distended sheath, they are easily identified with the improved contrast associated with sheath distension. The plicae should not be confused with adhesions, but they are useful structures with which to assess the status of the synovial membrane.

In the distal pastern region, a normal thin mesotenon is sometimes visible in the midline between the DDFT and the digital sheath.

Palmar/Plantar Annular Ligament of the Fetlock

Identification of the palmar/plantar annular ligament of the fetlock (PAL) in normal horses is difficult because of its size (1–2 mm in thickness).[4–6] However, moving the probe medially or laterally away from the midline (where the annular ligament is joined to the SDFT by the vinculum) will improve definition of the ligament by the relatively hypoechogenic synovial lining (± synovial fluid) between it and the SDFT. If it still cannot be identified with confidence, the probe should be moved further medially or laterally to visualize its attachment to the very palmar/plantar border of the proximal sesamoid bones.

Some veterinarians prefer to assess the PAL by measuring the distance between the palmar/plantar surface of the SDFT and the skin surface, although this distance will include the skin, SC tissues, PAL, and synovial membrane. All of these can be affected to a variable degree in the condition of annular ligament syndrome (see below). A normal measurement of 3.6 ± 0.7 mm has been quoted; therefore, anything >5 mm should be considered significant.[4]

Digital Annular Ligaments

The digital annular ligaments (proximal and distal) cannot be easily visualized in the normal horse, because they are usually <1 mm in thickness. However, they can be seen when enlarged. They can be identified proximal to the distal out-pouching of the digital sheath, especially medially and laterally where they are more discrete structures grossly.

Distal Sesamoidean Ligaments

Both the oblique distal sesamoidean ligament (ODSL) and straight distal sesamoidean ligament (SDSL) can be identified ultrasonographically. The SDSLs are the most echogenic structures within this region and are often more easily assessed in the longitudinal images. The ODSLs require oblique views for adequate imaging. The short and cruciate distal sesamoidean ligaments (DSLs) cannot be distinguished but can sometimes be identified adjacent to the joint capsule in oblique views of the palmar/plantar aspect of the fetlock joint.

The insertion of the SDSLs onto the middle scutum on the palmar/plantar aspect of the proximal interphalangeal (PIP) joint frequently contains a hypoechoic "core" or "sandwich" in the transverse views (P3 only) and a hypoechoic "wedge" with its apex directly proximally in the longitudinal view. The hypoechoic region does not usually extend farther proximally than the distal limit of insertion of the ODSL. These are normal anatomical variations and should not be mistaken for pathology.

Differences in the Hindlimb

The ultrasonographic anatomy of the metatarsal region is similar to the metacarpal region, but there are a few differences:

- The SDFT is positioned laterally and the DDFT is positioned medially in the proximal metatarsal region.
- The subtarsal check ligament (ALDDFT) is a very thin structure lying on the dorsal surface of the distal tarsal sheath wall.
- The medial head of the DDFT, in its own tendon sheath, joins the DDFT on its medial border in the very proximal metatarsal region.
- The SL arises as a triangular structure adjacent to the third and fourth metatarsal bones (the latter is particularly prominent proximally).
- Proximal to the tarsometatarsal joint, three structures are visualized—the SDFT superficially, the DDFT deep to the SDFT and medially positioned, and the plantar ligament deep to the SDFT and laterally positioned.

6. Ultrasonographic Pathology

Superficial Digital Flexor Tendinopathy

A common manifestation of acute injury to this tendon is a concentric hypoechoic/anechoic lesion visible in the center of the tendon (thus, the usual term "core lesion"), usually centered in the mid-metacarpal region. It is accompanied by enlargement and SC edema in the acute stage. Lesions can also be localized eccentrically to the borders of the tendon—medially, laterally, dorsally, or palmarly. Often, dorsal lesions are thought to be associated with more lameness, presumably because of the direct pressure exerted by the DDFT onto the lesion under weight-bearing load.

In very subtle cases, often the only finding can be enlargement and/or change in shape of the tendon. This can be accompanied by peritendinous edema, which is not specific for tendonitis and can also result from local trauma. Providing that there is no evidence of tendon injury and the edema disappears, work can be recommenced after only a short period of rest. However, persistent edema suggests the presence of tendonitis.

Not all lesions involved local abnormalities, and another common manifestation is a generalized hypoechogenic tendon. This may represent either a tendon that is healing in which the core lesion has disappeared or, if the injury is recent, more diffuse damage to the tendon and/or intratendinous edema.

Injury can also occur to the SDFT in the fetlock (low bow) and pastern regions where it is associated with variable amounts of digital sheath effusion. Damage to the SDFT in the region of the fetlock canal appears ultrasonographically as a hypoechoic tendon with minimal enlargement because of the constraints of the palmar annular ligament. As a result, these injuries are often associated with secondary thickening of the palmar annular ligament. Injury to the branches of the SDFT is best identified by enlargement and hypoechogenicity of individual branches that is usually observed with the transducer positioned palmarolaterally or palmaromedially. There is usually secondary SC fibrosis with these injuries in contrast with those affecting the SDFT more proximally. If the injury is localized to the region of the MCP joint or distally, then there may be evidence of previous injury to the mid-metacarpal region.

Complete rupture of the SDFT is the most severe extreme of an overstrain injury, and it often results in an almost totally anechoic region of the SDFT surrounded by a thin echogenic line called the paratenon; this usually remains intact unless the injury has been caused by percutaneous trauma. Evidence of damage will also be apparent proximal and distal to the rupture. If the tendon ends have retracted, the outline of the paratenon at the site of the rupture may not be particularly enlarged but bunched up, retracted fibers will be identifiable proximal and distal to the rupture site. The SDFT also becomes medially displaced because of lengthening of the tendon.

Semi-Objective Assessment of Injury to the SDFT

Objective measurements potentially allow a better determination of prognosis and assessment of healing. The following measurements have been suggested: (1) the CSA (transverse image), (2) the percentage of damaged tendon (transverse image) for focal lesions, (3) the type of lesion, and (4) the FAS (longitudinal image).

1. There is a large interindividual variation in the CSA in normal horses—80–130 mm^2 for Thoroughbreds.[3,7] A >20% difference between limbs is considered a significant enlargement, although this may not be the case if both limbs are affected.[7]
2. The CSAs for both the size of the focal lesion and the total tendon CSA at each individual level can be summed for all seven levels or zones to give an approximation of the "volume" of the lesion over the volume of the tendon. This has been used to give what is thought to be the optimal assessment of severity: 0–15% of the tendon affected is a mild injury, 16–25% of the tendon affected is a moderate injury, and >25% of the tendon affected is a severe injury.[8] An alternative method is to consider the maximum injury zone only. A mild injury involves <10% of the CSA, a moderate injury involves 10–40% of the CSA, and a severe injury involves >40% of the CSA. However, this obviously does not take into account the length of the lesion.
3. There are four types of lesions or degrees of echogenicity. Type 1 is a hypoechoic lesion with more white than black. Type 2 is a hypoechoic lesion with the same amounts of white and black. Type 3 is a hypoechoic lesion with more black than white. Type 4 is an anechoic lesion with all black and no white.
4. The FAS (longitudinal image) is assessed subjectively on a scale from 0 (76%–100% parallel fibers; normal) to 3 (0–25% of parallel fibers).

Assessment of Healing

All tendon injuries should ideally be monitored ultrasonographically at 3-mo intervals or before and after a change in the exercise level. At each examination, the following indicates good progress:

1. A stable or decreasing CSA. Sequential CSA measurements provide the most sensitive indicator of exercise to tendon healing mismatch during the rehabilitation phase. If the CSA at any level increases by >10%, it is advisable to maintain or lower the exercise level.
2. An increase in the lesion echogenicity and a homogeneous texture.

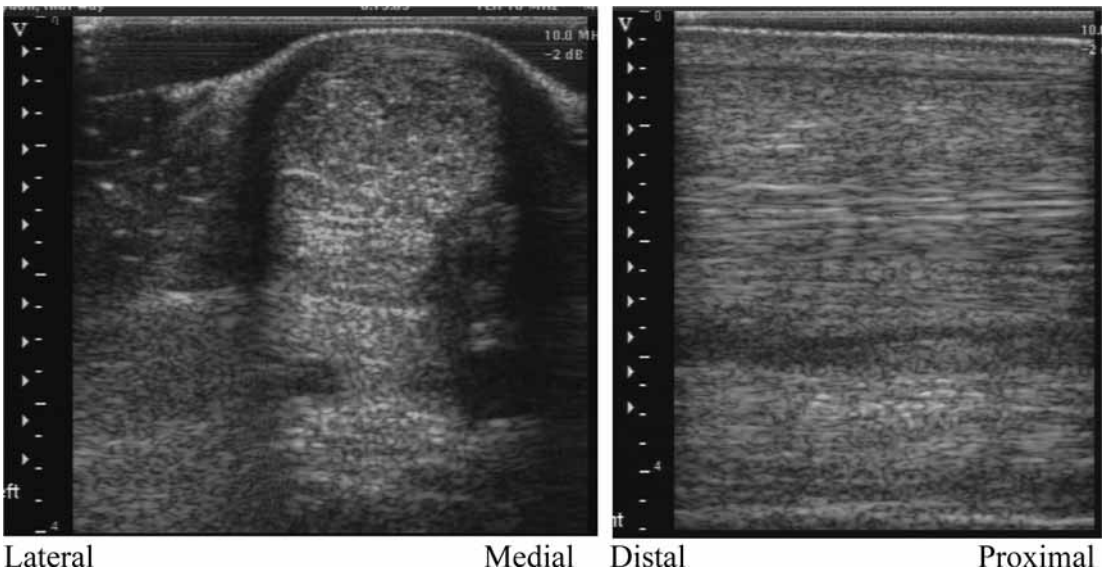

Fig. 7. (Left) Tranverse and (right) longitudinal ultrasonographs taken from the proximal metacarpal region of a horse with chronic superficial digital flexor tendinopathy. Note the enlarged SDFT with heterogeneous echogenicity and a poor longitudinal striated pattern. This is similar to the appearance of the tendon in the transverse image, which suggests the absence of normally aligned collagen fibers.

3. An improvement in the striated pattern seen longitudinally (fiber alignment).
4. An absence of peritendinous fibrosis and adhesions.[8]

More recently, the blood flow within healing digital flexor tendons can be assessed with the limb raised using Doppler (Fig. 4).[9] Normal digital flexor tendons usually have minimal discernible blood flow, whereas, after injury, a pronounced vascular pattern is usually visible. Hypervascularity is normal in the healing process. However, it should subside as healing progresses (normally between 3 and 6 mo after injury), and its reappearance can be an indication of reinjury.

Horses suffering from tendonitis are constantly at risk of reinjury. Healing, determined histologically, takes at least 15–18 mo.[10] The mean interval between injury and return to training in racehorses is dependent on the severity of the initial injury and varies between 9 and 18 mo.[10] Sports horses may be able to return to full work in a shorter time, but even the mildest ultrasonographically detectable injuries should have at least 6 mo to heal. Occasionally, horses are returned to full work before full resolution of the ultrasonographic lesion; however, this success may be caused by the horse being capable of sustaining work despite the presence of a tendon injury.

Chronic Tendinopathy

The ultrasound characteristics of chronic tendinopathy are more variable and can be subtle. The tendon is often enlarged, but its echogenicity varies from hypoechogenic through normoechogenic to hyperechogenic if the injury is severe and substantial fibrosis has occurred. The intratendinous pattern is usually more coarse and lacks striations in the longitudinal images (Fig. 7). In some cases, the outline of the original core lesion can still be seen. Mineralization may occur, which causes acoustic shadowing. However, if the calcification is florid, previous intratendinous injection of depot corticosteroids should be suspected. Off-incidence transducer orientation can help to define areas of disorganized scar tissue in chronic injury, because it retains its echogenicity at greater transducer angles than normal tendon (Fig. 1).

Local Trauma

Overstrain injuries need to be distinguished from local trauma caused by a bandage (so-called "bandage bow") or percutaneous trauma from, for example, a hindlimb. The effects of local trauma can vary from localized peritendinous edema with no evidence of intratendinous damage to localized hypoechoic/anechoic lesions on the palmar surface of the tendon (Fig. 8) to partial or complete transection. Local traumatic injuries do not extend far proximodistally. However, partial lacerations can be associated with longitudinal splits in the tendon that extend proximally or distally; these result from altered shear stresses. Partial lacerations can also be easily missed if the examination is restricted to the site of the wound, because they often occur when the tendon is fully loaded. Therefore, the site of injury moves more proximally in the resting or reduced weight-bearing limb. Ultrasound is, therefore, very useful to identify these sites of injuries not visible through the wound. Complete transection

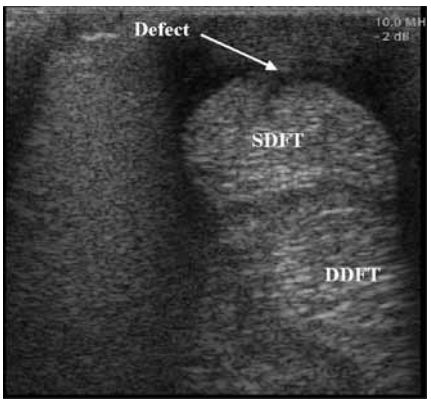

Fig. 8. Transverse ultrasonograph from the proximal metatarsal region in a horse that has suffered percutaneous trauma to the SDFT. Note the plantar surface defect (labeled).

of one branch of the SDFT in the pastern region results in a shift in position of the SDFT toward the side of the intact branch more proximally.

Sepsis after a penetrating injury (or occasionally, hematogenous spread) of the SDFT is rare. It usually gives an anechoic lesion, often with a communicating tract to the periphery of the tendon. Aspiration of the lesion will yield a sample containing large numbers of degenerate neutrophils. These lesions do not usually cause gross enlargement of the affected tendon and change rapidly in time compared with the core lesion in a tendon strain. If the lesion is present within a tendon sheath, there will usually be an accompanying septic tenosynovitis.

Manica Flexoria Tears

This is a common cause of digital sheath tenosynovitis, especially in hindlimbs.[11] Ultrasonographic diagnosis is difficult, but an altered position of the manica flexoria seen in a longitudinal scan in the midline immediately proximal to the metacarpo/metatarsophalangeal joint is probably the best indicator (Fig. 9). Tenoscopic assessment provides the definitive diagnosis.

Deep Digital Flexor Tendinopathy

DDFT injuries are extremely rare in the metacarpal region, but they do occur within the confines of the digital sheath. Of the strain-induced DDFT injuries, there are two forms—the intratendinous injury and surface tears.

Intratendinous Injury

Intratendinous injuries are frequently centered at the level of the MCP joint. They result from a sudden overextension of the distal interphalangeal joint when the MCP joint is fully extended and the limb is weight bearing. These injuries are frequently associated with considerable disruption of the tendon resulting in marked and persistent lameness. There is usually concurrent tenosynovitis and as with most soft tissue injuries in the phalangeal region, SC fibrosis. Other lesions are manifest by focal hypoechogenic lesions proximal[12] or distal to the MCP joint. Many central defects may extend to the surface of the tendon without penetrating the epitenon and therefore, may not be visible tenoscopically (Fig. 10).

Because of the location of the injuries within the digital sheath, healing is, at best, problematic. In the chronic stage, the lesions often persist as hypoechoic lesions with or without areas of calcification. Lameness usually persists, arising from tenalgia and/or adhesion formation within the sheath. Such adhesions can distort the tendon shape.

Border Tears

Damage to the surface of the DDFT can occur as a variant of overextension injury to the tendon.[13] This frequently occurs at the lateral and less commonly, medial borders of the DDFT in the region of the MCP joint. It occurs most commonly in the

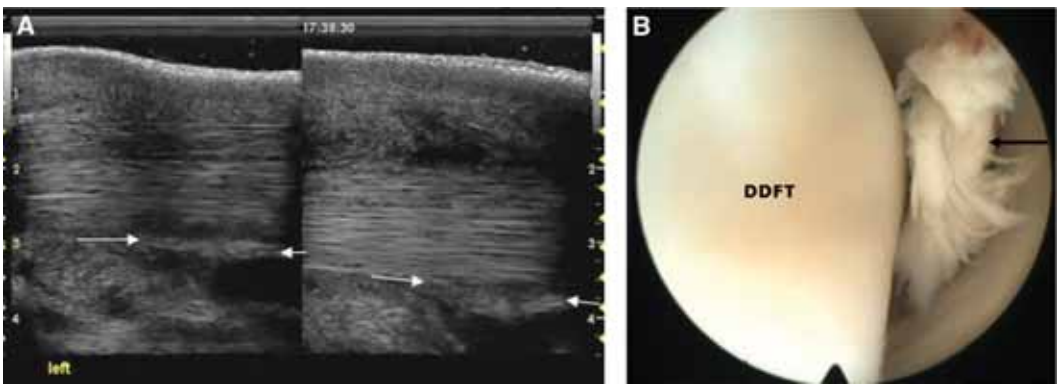

Fig. 9. The best method of diagnosing a manica flexoria tear ultrasonographically involves the identification of instability of the manica in midline longitudinal views in the distal metatarsal region. (A) The normal contralateral limb is on the left, and the torn manica flexoria is on the right. Note the wavy form to the manica (arrows). (B) Tenoscopic appearance. The arrow indicates torn manica.

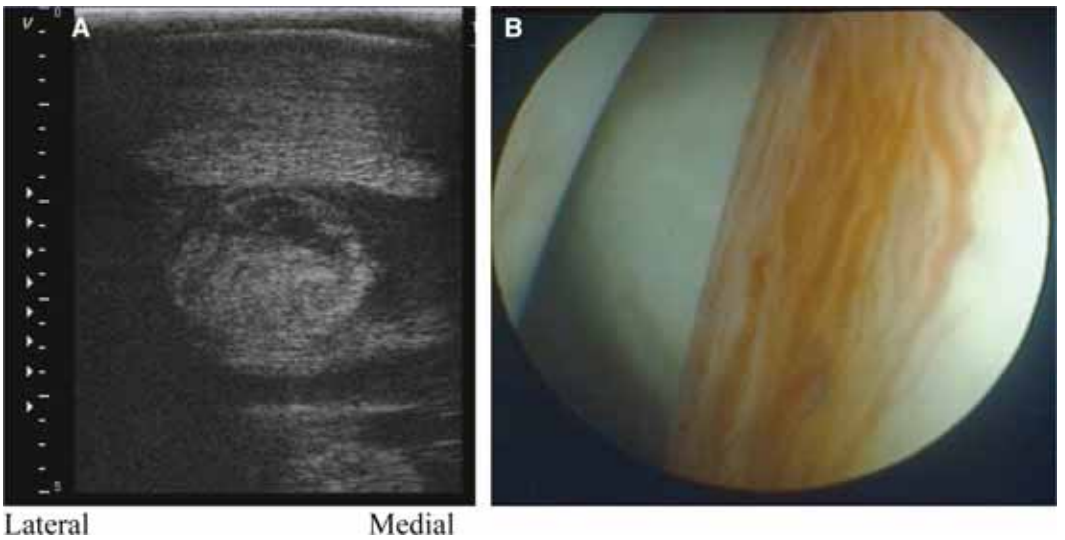

Fig. 10. The image on the left is a transverse ultrasonograph from the region of the proximal digital sheath in a horse suffering from deep digital flexor tendinopathy. Note the hypoechoic region on the palmar surface of the DDFT. Tenoscopic examination of this case (right) proved that the lesion did not communicate with the digital sheath, which has important consequences for management and healing.

forelimbs, presumably because of excessive forces during overextension that compress the tendon and cause a pressure-induced rupture.[11] Because of their intrasynovial location that is surrounded by synovial fluid, healing does not occur. These lesions often persist, being responsible for persistent digital sheath tenosynovitis and lameness.

Confident diagnosis of these tears using ultrasonography is difficult. Greater sensitivity in their detection can be made by using an oblique transducer positioned to assess the lateral and medial borders (Fig. 3). However, a negative finding on ultrasound does not rule out the presence of a tear. Tenoscopy is recommended to identify occult tears and should certainly be considered in those cases of tenosynovitis that have failed to respond or recurred after intrathecal medication.

Local Trauma

Local trauma to the palmar/plantar aspect of the pastern is common during overextension of the metacarpo/metatarsophalangeal joint at maximal exercise. Because of the close proximity to the skin, such injuries frequently damage the digital sheath and DDFT. Such combination injuries can result in digital sheath (and rarely, DDFT) sepsis if open, which gives rise to effusion and synovial thickening that is evident ultrasonographically. This synovial thickening also usually involves the epitenon surrounding the DDFT, which gives a "halo" appearance to the tendon. Such signs, although not pathognomic for sepsis, are strongly suggestive of it, and they should indicate synoviocentesis to confirm or refute the presence of sepsis. Local trauma will cause variably sized hypoechogenic lesions within the DDFT and enlargement, and it is often associated with adhesion formation between the damaged areas of the DDFT and sheath wall. Individual adhesions can sometimes be visualized ultrasonographically when surrounded by fluid (normal mesotenon/synovial plicae). Poor tendon border definition has been suggested as a sign of adhesions, but it can lead to overestimation of adhesions. When percutaneous trauma does not penetrate the skin, damage can still be induced in the underlying DDFT (blunt contusion), which may only become visible as a hypoechoic lesion over time. Therefore, if clinical signs persist, a repeat ultrasonographic examination is indicated after 2–4 wk.

7. Suspensory Ligament—Ultrasonographic Appearance and Pathology

Proximal Suspensory Desmitis

Proximal suspensory desmitis is also called high suspensory disease or proximal metacarpal syndrome. The ultrasonographic appearance of this injury has considerable overlap with the normal appearance. The presence of hypoechoic areas in the proximal SL is common in normal horses, and therefore, the significance of such findings must be interpreted in the light of clinical findings (swelling and pain on palpation) and diagnostic local analgesia. Those considered to be true lesions of SL desmitis will vary in time, and therefore, repeat examinations are useful to confirm their significance.

Ultrasonographic features of injury include enlargement of the SL, poor definition to the margins (especially dorsally), single or multiple poorly defined focal areas of hypoechoicity, diffuse hypoechoicity, and irregularity of the palmar surface of the proximal metacarpus/metatarsus, which is indicative of enthe-

siophytosis. In addition to the ultrasonographic abnormalities, radiographic changes, such as sclerosis and altered trabecular pattern at the origin of the SL, may also be present. They may occur with or without concurrent increased radionuclide uptake on gamma scintigraphy in the proximal metacarpal/metatarsal regions. Differential diagnoses include palmar cortical fractures, which usually have no abnormalities within the proximal SL and higher uptake on gamma scintigraphy, and avulsion fractures of the head of the SL, where abnormalities in the SL are often confined to an area immediately adjacent to the site of the avulsion fracture.

Desmitis of the Body of the SL

If this area is injured, there is usually generalized hypoechogenicity and enlargement to the ligament. In competition/sports horses, the injury often extends into the branches of the SL.

There is some controversy over the link between suspensory desmitis and splints. Some aggressive exostoses on the second or fourth metacarpal/metatarsal bones may impinge on the body of the SL and cause a localized suspensory desmitis, but this probably occurs in only the minority of cases. Many exostoses grow around rather than into the SL. Careful assessment by oblique positioning of the ultrasound transducer is necessary, because the ultrasound "window," when the transducer is placed on the palmar/plantar aspect, does not usually extend sufficiently abaxially to image these areas adjacent to the splint bones.

Desmitis of the SL Branches

This is the most common of the SL injuries in sports horses. In the forelimbs, biaxial desmitis has the highest incidence, whereas lateral branch desmitis is the most common manifestation in the hindlimbs. A core lesion or generalized involvement of the branch, together with enlargement, is seen ultrasonographically. The longitudinal image from the abaxial aspect gives an excellent assessment of the abaxial surface of the proximal sesamoid bones where any associated enthesiopathy is seen by steps in the S-shaped surface of the bone. The size of the SL branches should be compared with both contraaxial and contralateral branches at the same level, because the branches increase in size in a proximodistal direction. One of the most sensitive indicators of suspensory branch desmitis is periligamentar fibrosis, which is extremely common in this condition. It has the effect of "moving" the SL branch away from the skin (Fig. 11).

Clinical and radiographic examination of the metacarpo/metatarsophalangeal joint is also recommended in cases of SL desmitis. Concurrent pain and pathology in this joint is frequently present because of the nature of the injury (hyperextension). Furthermore, radiography will reveal bony pathology that is frequently associated with the SL body and branch desmitis, such as enthesiopathy of the proximal sesamoid bones ("sesamoiditis") and distal splint bone fractures.

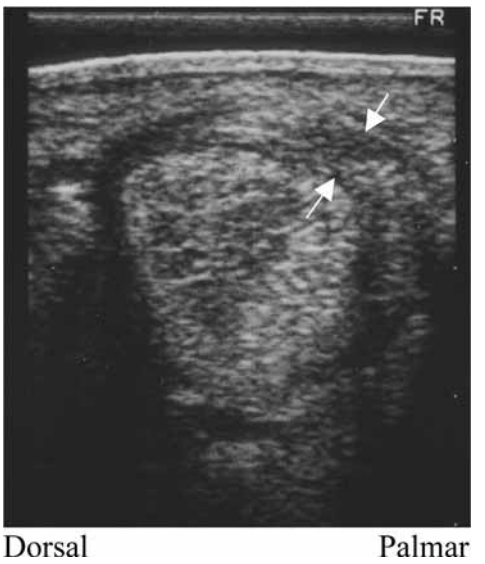

Fig. 11. Transverse ultrasonograph taken from the lateral aspect of the forelimb of a horse suffering from suspensory branch desmitis. Note the collar of periligamentar fibrosis (arrows) that is characteristic of this condition.

8. Other Diagnostic Techniques for Diagnosing Tendon and Ligament Injuries

In the ultrasonographic descriptions of pathology outlined above, other imaging techniques have been mentioned including radiography (for the identification of mineralization and/or enthesiopathy in chronic disease) and the pool phase of gamma scintigraphy. More recently, the advent of magnetic resonance imaging (MRI)[14] and computed tomography (CT), including contrast-enhanced CT,[15] for horses has enabled us to identify pathology in those areas where ultrasonography is limited. This has been particularly true for the foot—most commonly for injuries of the DDFT (Fig. 12) and the collateral ligaments of the distal interphalangeal joint. However, it is also beginning to help us differentiate pathologies in other areas where ultrasonography has frequently not provided sufficient evidence of pathology, such as the proximal suspensory region.

Finally, although not available clinically as yet, it is hoped that the use of molecular markers, assayed in blood or synovial fluid (for intrasynovial tendon and ligament injuries), may allow us to diagnose and monitor tendon injuries. This would provide an objective assessment of the efficacy of different treatments. One such marker is cartilage oligomeric matrix protein (COMP), which has shown good differentiation of digital sheaths containing injured tendons (e.g., tendon tears that are poorly identified by other means other than tenoscopy) compared with those without tendon pathology.[16]

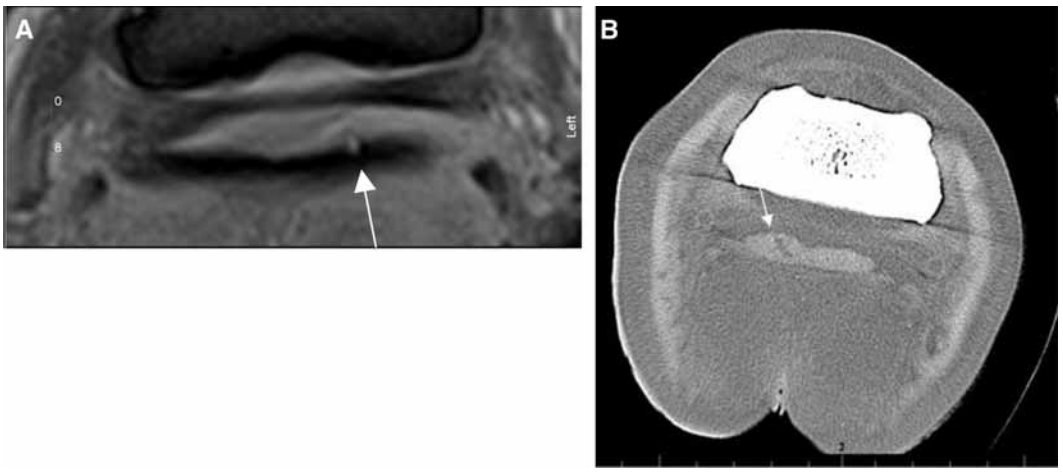

Fig. 12. (A) CT and (B) MRI images of DDFT tears (arrows) in the foot. (MRI image courtesy of Mr. Tim Mair, MRCVS.)

9. Treatment for Tendon and Ligament Injury (adapted from Davis and Smith[17])

Over the years, many treatment modalities have been tried, and most show equivocal or even deleterious effects on tendon healing. From our knowledge of the phases of tendon healing, the following summarizes those treatments that have at least a rationale for treating superficial digital flexor tendinopathy.

1. Physical Therapies

1. Cold, Pressure, and Support

In the acute inflammatory phase of tendon injury, cold therapy is an important aspect of treatment, because it is both anti-inflammatory and analgesic. This is because of its ability to cause vasoconstriction, decrease enzymatic activity, reduce the formation of inflammatory mediators, and slow down nerve conduction.[18] It should be applied several times a day. Cold hydrotherapy seems to be superior to the use of ice packs because of the increased contact time and evaporation.[19] It is also less likely to cause adverse effects such as superficial tissue damage because of local freezing. Prolonged exposure to cold temperatures can also cause a reflex vasodilatation that will potentially accentuate tissue swelling and edema. For this reason, it is recommended not to apply cold therapy for periods >30 min. One highly effective way of providing cold hydrotherapy is the use of equine spas, which are currently gaining popularity. They provide both cold and compression using hypertonic saline at 5–9°.

Cold can be combined with pressure applied to the affected limb. This will reduce inflammation and edema formation by increasing interstitial hydrostatic pressure. A modified Robert Jones bandage is suitable in most cases of acute tendon and ligament injury of the metacarpal/metatarsal region and pastern.

In acute stages, rest provided the easiest way of limiting loading on the damaged soft tissue. In severe injuries where there is hyperextension of the MCP joint, a palmar/plantar splint or cast may be applied to the bandaged limb to provide MCP joint support. A palmar splint can be fashioned from two rolls of 7.5-cm (5-in) casting tape. One roll is layered on top of itself over a length that matches the length of the limb from the carpus to the bulbs of the foot. This is placed on the palmar aspect of 2.5-cm thickness support bandage on the contralateral limb, which is used to provide the optimal degree of extension at the MCP joint. A second roll is wrapped around the palmar splint and bandage and left to set. The palmar splint is then cut away from the bandage using a cast saw, and it is applied to the palmar aspect of the bandaged affected limb. Research has shown this to be effective at reducing the extension of the MCP joint, and thus, it loads the palmar soft tissue structures at normal weight-bearing loads.[20] A similar effect can be achieved more easily with the specially designed boot, although this has yet to be available commercially.[20] A full distal limb cast may be used as an alternative in the most severe cases, such as SL rupture, when all MCP joint support has been lost.

2. Extracorporeal Shock Wave Therapy

Extracorporeal shock wave therapy (ESWT) was originally developed for the treatment of insertional desmopathies in man. ESWT involves the use of shock or pressure waves that are transmitted into the tissues to which the hand piece is applied. Both focused and non-focused units have been used, although there are little data comparing these machines to indicate a significant difference. The mechanism of action of ESWT on tissues is unclear. Significant effects have been shown in experimental studies,[21] but it is possible that these effects are deleterious. Therefore, this author does not consider ESWT to be appropriate for the management

of acute tendinopathy. One of the most convincing explanations for its effectiveness clinically is the induction of analgesia by an effect of sensory nerves.[22] The most frequently reported use of ESWT in horses has been for the treatment of proximal SL desmitis (PSLD) where it has shown a significant improvement in prognosis over conservative treatment for chronic hindlimb PSLD. Forty-one percent of hindlimb cases returned to full work within 6 mo of diagnosis[23] compared with the previously reported 13% for conservatively managed cases.[24]

3. Rehabilitation—The Use of Physical Forces to Influence Healing

Immediate controlled passive mobilization of the limb has been advocated in the acute phase of tendon and ligament injury in man to reduce inflammation and improve healing.[25] To be successful, the degree or frequency of mobilization must remain below the patient's pain threshold. This can be administered by a 15-min session of gentle physical therapy. The session should involve a series of 10–30 passive carpal and MCP joint flexions as long as they are tolerated by the horse.

Controlled exercise is an intrinsic part of the rehabilitation of tendon and ligament injuries, and it also helps to resolve residual inflammation, maintain gliding function, and promote optimal collagen remodeling.[26] Most SDFT injuries require at least 8–9 mo of rehabilitation before resuming full athletic function, although some may require up to 18 mo. It is rarely necessary to prolong rehabilitation longer than this, because healing seems complete by this stage.

A suitable exercise rehabilitation program should be created based on the severity of the ultrasonographic appearance of the lesion. The aim of the program is to provide a controlled and ascending exercise regimen that optimizes scar tissue function without causing further injury. This is difficult to predict because of the variability between animals. Therefore, the program should be adapted based on serial ultrasonographic monitoring and clinical signs such as lameness, heat, and swelling.

Ultrasonographic monitoring of the tendon/ligament should include measurements of tendon CSA. An increase in the CSA of >10% between examinations would suggest a degree of reinjury, and in such cases, the level of exercise should be reduced.[27] Because there is a sudden increase in strain levels in the SDFT and SL with an upward transition in gait from walk to trot, trot to canter, and canter to gallop, ultrasonographic examinations before and after these transitions can help to determine if the injured tendon/ligament can withstand the increased strain levels.

2. Pharmacological Management

1. Anti-Inflammatory Systemic Medication

Both systemic corticosteroids and non-steroidal anti-inflammatory drugs can be considered for the management of tendon/ligament inflammation in the acute stages. Phenylbutazone is commonly used at a dose of 2.2 mg/kg, q 12 h; however, the clinical effects of this drug seem to be more analgesic than anti-inflammatory.[28] Systemic steroids can be administered early in the acute inflammatory stage of the injury (usually within the first 24–48 h post-injury), and they are very effective. They should be avoided during the healing phase, because they also inhibit fibroplasia and therefore, repair of the tendon.[29,30] The induction of laminitis with systemic steroids represents a small, but nevertheless real, risk. In the author's opinion, this is less likely in Thoroughbreds compared with Warmbloods. Topical or IV dimeythl sulphoxide (DMSO) may reduce the inflammation, but a study has shown that 40–90% topical medical grade DMSO may weaken normal tendon tissue.[31]

Polysulphated glycosaminoglycans (PSGAGs) have been shown to inhibit collagenases and metaloproteinases as well as inhibit macrophage activation, but they were shown to have no effect on fibroblasts.[32] Thus, this drug can be viewed as a soft tissue anti-inflammatory agent. PSGAGs have been widely used for the treatment of tendinopathy and desmopathy. They are most commonly administered systemically through intramuscular injection, although they can also be given by intralesional injection.[33] Evidence for efficacy is limited; however, improved echogenicity of collagenase-induced superficial digital flexor tendinitis and faster resolution of core lesions treated with intralesional PSGAGs was demonstrated.[34] No significant difference in reinjury rates between horses treated with PSGAGs compared with controlled exercise alone has been shown.[35]

2. Intralesional Medication

Intralesional tendon and ligament treatment can be performed under standing sedation and local analgesia or under general anesthesia. It is preferred that it be performed with the leg bearing weight. Although the technique is frequently performed blindly by injecting where least resistance within the tendon is detected, accurate placement of the needle in the center of the lesion is best achieved using ultrasonographic guidance. The skin overlying the tendon or ligament to be injected should be clipped and aseptically prepared, and if ultrasonographic guidance is used, a sterile sleeve should be placed over the probe. A 2.5-cm, 23-g hypodermic needle can be used for most intratendinous treatments, but it varies with the viscosity of the agent. Intralesional treatment should not be administered until 3 days after the injury, because there is potential to increase hemorrhage. The volume injected into the tendon or ligament will depend on the extent of the lesion. Large volumes can be potentially damaging to the healing tendon.[36] In addition to PSGAGs, the other agents most frequently used for intralesional medication are hyaluronic acid (HA),

corticosteroids, and beta-aminoproprionitrile fumarate (BAPN).

HA is a component of tendon matrix and has been administered peritendinously, intralesionally, and systemically to treat tendinitis. In a study of collagenase-induced digital flexor tendinitis, HA was found to minimize tendon enlargement compared with controls; however, histopathological examination of the tendons failed to show a significant difference in the degree of inflammation.[37] Peritendinous HA has been shown to have no effect on ultrasonographic or histological appearance, biomechanical properties, or molecular composition of tendons in collagenase-induced tendinitis compared with controls, although it did appear to reduce lameness.[38] A review of the effectiveness of various medications has failed to show a significant difference between the reinjury rates of horses with SDFT tendinitis treated with intralesional HA compared with conservative treatment.[35] The drug is probably most appropriately used in the reduction of severity of adhesions after intrathecal injury.[39]

Corticosteroids, at least the depot preparations such as methylprednisolone, should not be injected directly into tendons or ligaments, because they have been shown to cause dystrophic tissue mineralization and tissue necrosis, most likely a consequence of the carrier.[40] Peritendinous or systemic use in the early stages are appropriate and can be used judiciously.

BAPN, a lathyrogen that inhibits the enzyme lysyl oxidase that normally forms cross-links between collagen fibers, has been used to treat tendinopathy. The rationale for its use is to allow exercise to promote alignment of newly formed collagen fibrils while preventing them being fixed in a haphazard fashion by cross-linking. BAPN does not hasten the resolution of the tendinitis, but it aims to improve the structure of the repaired tendon. Early experimental studies in collagenase-induced models of tendonitis appeared to show improvement in both the ultrasonographic appearance[41] and the histological collagen alignment.[42] However, more recently, concerns have been raised over its efficacy based on observations that BAPN reduces collagen synthesis[43] and showed no improvement over controls in a rabbit model of tendinitis.[44] Clinical studies have suggested that the reinjury rate of limbs treated with BAPN was reduced, although the rate for both limbs was no different from other treatments. This is caused by the fact that both limbs should be treated, because unilateral treatment increases the loading/reinjury risk on the contralateral limb. However, the drug has been withdrawn from the market and therefore, is now rarely used.

3. Surgery

1. Tendon Splitting

Tendon splitting was initially advocated as a treatment for chronic tendinitis to improve blood flow to damaged tendon tissue. The technique fell out of favor when subsequent research showed extensive granulation tissue formation, increased trauma to the tendon tissue, and persistent lameness post-treatment.[45] Tendon splitting is, therefore, no longer recommended for the treatment of chronic tendonitis. However, it is now thought to be more relevant for the management of acute cases where there is an anechoic core lesion evident on ultrasonographic examination that indicates the presence of a seroma or hematoma. It has been hypothesized that the presence of a core lesion within a tendon produces a "compartment syndrome," which results in decreased perfusion and ischemia of the region. The aim of tendon splitting in acute cases is to decompress the core lesion by evacuating the serum/hemorrhage and to facilitate vascular ingrowth. Removal of the fluid within the core lesion may also reduce proximodistal propagation of the lesion. In a collagenase-induced model of tendinitis in six horses, tendon splitting using the knife technique resulted in a faster resolution of the core lesion, a quicker revascularization of the lesion, and an increased collagen deposition relative to controls.[46]

Tendon splitting may be performed under standing sedation or under general anesthesia. It can be done blindly or using ultrasonographic guidance, which minimizes damage to normal tendon tissue by enabling the needle or knife to be inserted at a point where the core lesion is closest to the periphery of the tendon. A #11 scalpel blade or double-edged blade is inserted into the tendon and "fanned" proximally and distally. Alternatively, the procedure can be achieved with multiple insertions of a 23-g needle. This may cause less damage to the remaining, relatively intact tendon tissue. Furthermore, needle splitting may be combined with various intralesional treatments, although multiple needle injections may provoke leakage of the drug/agent out of the tendon.

After tendon splitting has been performed, a modified Robert Jones bandage should be applied. The horse should be rested in a box stall for 10–14 days, subsequent to which a controlled exercise program should be initiated.

2. Desmotomy of the Accessory Ligament of the SDFT

The aim of desmotomy of the accessory ligament of the superficial digital flexor tendon (or superior check ligament desmotomy (SCLD)) is to produce a functionally longer musculotendinous unit to reduce strain on the SDFT.[47] However, it has been shown in equine cadaver models that SCLD actually increases the strain on the SDFT and SL during loading because of increased extension of the MCP joint.[48] The biomechanical alterations of SCLD are complex, and it is recognized that studies using cadaver limbs may not represent the biomechanical events in a fatigued galloping racehorse. However, increased risk of injury of the SL after the SCLD has been performed has also been shown in vivo.[49]

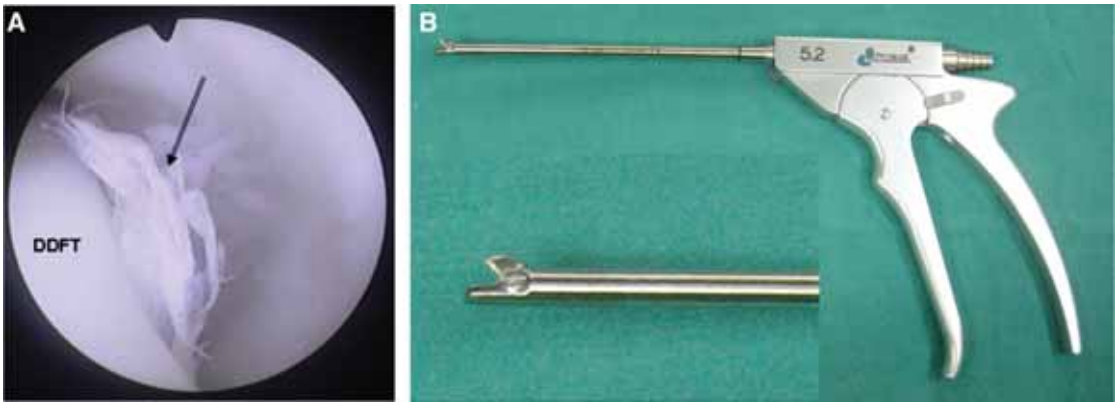

Fig. 13. Tenoscopic appearance of a tear in the lateral border of the DDFT. The torn fibers are debrided using a (A) mechanical synovial resector, arthroscopic scissors, or (B) suction punch biopsy forceps.

SCLD can be performed using a percutaneous approach[50] with the horse in lateral or dorsal recumbency. More recently, however, this procedure has been carried out tenoscopically through the carpal sheath.[51] With the affected limb uppermost and partially flexed, an arthroscopic portal is created into the carpal sheath 2 cm proximal to the distal radial physis on the lateral side of the limb. An instrument portal is made immediately proximal to the distal radial physis. With the limb in 90° flexion, the accessory ligament is visualized on the medial aspect of the sheath and cut using a #10 scalpel blade on a long handle or meniscectomy knife. The very proximal portion of the ligament cannot be visualized directly, but it is transected by careful dissection using punch biopsy forceps. Care must be taken to avoid perforating the blood vessel at the proximal limit of the accessory ligament.

3. Tenoscopy

The increasingly widespread use of tenoscopy has shown a high frequency of intrathecal tendon tears that are associated with tenosynovitis and lameness. Thus, horses presenting with tenosynovitis with pain on palpation of the proximal digital sheath and lameness should always be considered candidates for tenoscopic evaluation. Based on a recent survey of cases, tears in the DDFT (Fig. 13) are more commonly found in forelimb digital sheaths, whereas tears to the manica flexoria are more commonly found in hindlimbs. Ultrasonographic evaluation usually reveals non-specific changes of synovial hypertrophy, and it is frequently not possible to identify tears on ultrasonographic examination with confidence. In addition, tears can also be found and debrided tenoscopically in the SDFT and DSL within the digital sheath. Tears of other tendons and ligaments communicating with a synovial cavity have been seen associated with synovial distension and lameness (e.g., in the SL branches into the metacarpal/metatarsal joint).

Tenoscopy of the digital sheath requires general anesthesia and can be performed in lateral or dorsal recumbency. However, dorsal recumbency allows easier access to both sides of the digital sheath and is recommended. Arthroscopic portals are created immediately distal to the proximal sesamoid and 1–2 cm palmar/plantar to the neurovascular bundle.[52] This allows evaluation of the proximal and distal parts of the digital sheath, although distal visualization can sometimes be easier with the arthroscope inserted through a portal in the proximal digital sheath (e.g., as for a proximal instrument portal). Instrument portals are created where appropriate to allow debridement of any tendon tears with a mechanical resector, arthroscopic scissors, or suction punch biopsy forceps (Fig. 13). This may require the instrument portals to be extended through the synovial reflection that attaches to the proximal border of the manica flexoria to allow access to DDFT tears located within or proximal to the manica. Although manica tears can also be debrided in a similar fashion to the DDFT tears, in the author's opinion, this has resulted in a poorer outcome than with complete removal. Because no adverse effects have been observed with complete removal, this is recommended for all except for the most minor manica tears. Removal requires transaction of both medial and lateral attachments to the SDFT as well as the synovial attachment to the proximal border of the manica. This is facilitated by an assistant maintaining tension on the manica with rongeurs through the contra-axial proximal instrument portal. Transection can be achieved with arthroscopic scissors or a hook knife.

Adhesions can also be resected during tenoscopic evaluation, and the palmar or plantar annular ligament can be transected if it is believed to be involved in the pathology. Post-operatively, the horse should be strictly rested for at least 2 wk. After that period, hand walking can be started and gradually increased over a minimum period of 6 wk.

Thereafter, the duration of rehabilitation will depend on the severity of the injury. The prognosis for DDFT tears (~20%) is worse than for manica tears (~80%).[11]

4. Annular Ligament Desmotomy

Annular ligament desmotomy has been most frequently advocated as a treatment of annular ligament syndrome where there is a relative constriction of the tendons within the fetlock canal. This constriction can be either primary or secondary to other pathology within the digital sheath.[53–55] Consequently, desmotomy has been advocated for the management of DDFT and SDFT lesions in the region of the metacarpal/metatarsal joint. Usually, the procedure is indicated if the annular ligament is impeding the normal gliding function of the flexor tendons. It is probably rarely necessary, because the prognosis is closely related to the primary pathology. Desmotomy is, therefore, best performed tenoscopically using a hook knife rather than through closed or open techniques, because it is less traumatic, ensures accurate transection of only the PAL, and allows evaluation of the tendons to identify any surface defects.

5. Fasciotomy and Neurectomy of the Deep Branch of the Lateral Plantar Nerve for the Treatment of PSLD

Surgery has been advocated in cases of PSLD of the hindlimb that are unresponsive to conservative management. It has been reported that horses have returned to high-level competition after tibial neurectomy to treat PSLD.[34] However, a more specific neurectomy of the deep branch of the plantar nerve has been recently described.[56] This was performed under general anesthesia with the horse in dorsal recumbency. An incision 4–6 cm wide, which originates proximally from the level of the chestnut, is made adjacent to the lateral border of the SDFT. The plantar metatarsal fascia is incised, and the incision is extended deep to the SDFT by blunt dissection, which is facilitated by retraction of the SDFT. The lateral plantar nerve is located in connective tissue, and the deep branch is identified. It is then transected using a scalpel, and a 3-cm section is removed. The connective tissue fascia covering the SL is subsequently cut (fasciotomy) adjacent to the lateral splint bone to "decompress" the origin of the SL, because hindlimb PSLD is believed to be associated with a compressive "compartment" syndrome involving the plantar metatarsal nerves.

Post-operatively, only a short period of strict rest is needed (~2 wk) to allow the surgical incisions to heal. Thereafter, the horse can gradually begin a controlled, ascending exercise program.

Unpublished reports have suggested that this technique is associated with a high level of success (79%) in returning affected animals to full work with minimum risk of exacerbating the desmitis.

4. Alternatives

The effect of ultrasound, laser, and magnetic fields on tissues is not completely understood. It is thought that the main effect of ultrasound is the conversion of sound energy into thermal energy. Although there is a paucity of scientifically convincing research into the effects of ultrasound for the treatment of equine musculoskeletal disorders, a study by Morcos and Aswad[57] showed that the use of therapeutic ultrasound resulted in increased vascularization and fibroblastic proliferation compared with controls in experimentally split equine tendons.

Low-level laser therapy has been shown to stimulate cellular metabolism and enhance fibroblast proliferation and collagen synthesis in vitro.[58] There are, however, no clinical trials showing a significant difference between laser-treated and control cases of tendinopathy or desmopathy.

Despite its widespread use by horse owners and anecdotal reports of its efficacy, magnetic therapy has not been shown to enhance tendon or ligament healing in any clinical trials.

Counter irritation ("firing") has long been used in equine practice for the treatment of tendon and ligament injuries in the form of chemical or thermal cauterization. Topical iodine and mercurial-based compounds have been used for chemical cauterization or "blistering" of tendon injuries. Thermal cauterization or "firing" is performed under general anesthesia or standing sedation with local analgesia using heated bars or pins that are applied to the skin over the injured tendon or ligament. In some cases, the tendon is penetrated with the heated pins. Studies have shown that there is no histological difference between the collagen arrangement within the scar in cases of tendinopathy treated with firing compared with controls.[59] It has been postulated that any benefits from firing result from the enforced rest, local release of inflammatory cytokines, and/or a protective "bandage" of fibrous tissue/skin that will support the tendon. The limited controlled studies that have been performed on firing have concluded that it is not an effective treatment for tendon and ligament injuries.[59]

5. Tendon Lacerations

1. Emergency Treatment of Tendon Lacerations

The limb should be stabilized using a splint and a support bandage before the horse is moved to an appropriate place for treatment. It is important that the limb is supported to ensure the comfort of the horse. This may help to restore some biomechanical function of the limb and prevent further tissue damage. It is particularly important to avoid further trauma to the neurovascular structures of the distal limb. For flexor tendon lacerations, a palmar or dorsal splint or a commercial splint should be applied.

2. Surgical Repair

Surgical repair of flexor tendon lacerations involves debridement, with or without suturing of the tendon, and closure of the wound, usually performed under general anesthesia in lateral or dorsal recumbency. The aim of tenorraphy is to restore tendon gliding function, minimize gap formation between the tendon ends, minimize adhesion formation, and preserve functional vasculature. If the laceration is complete, the tendon may have recoiled, requiring proximal and distal extension of the skin wound in an elongated "S" to locate both tendon ends. Flexing of the metacarpal/metatarsal joint may facilitate locating of the distal tendon end. The wound and tendon ends should be debrided and lavaged. If the tendon ends can be apposed, tenorraphy can be performed using a monofilament absorbable suture (e.g., polydioxanone or polyglyconate). Non-absorbable materials should be avoided, because this can result in shearing between the healed tissue and the suture material. It may be responsible for persistent lameness. Two suture patterns have been commonly used—the three-loop pulley and the interlocking loop.[60] The three-loop pulley is strongest, and it prevents distraction of the ends of the tendon under loading ("gapping"). The interlocking loop has little suture material outside the tendon, and it is, therefore, the recommended technique for repair of intrathecal lacerations.

Frequently, however, the injury is associated with significant blunt trauma to the tendon ends, which precludes direct apposition of the tendon ends. In this situation, the tendon ends are left after debridement, the wound is closed, and the limb is cast, or an implant can be used to maintain the alignment of the tendon ends. The ideal tendon implant material would have similar biomechanical properties to normal tendon. Various implant materials have been used to repair lacerated flexor tendons including carbon fiber, terylene (polyester), autologous extensor tendon grafts, absorbable tendon splints, and poly-L-lactic acid (PLLA).[61] Carbon fiber implants were associated with persistent lameness postoperatively. This may have been caused by tenalgia that results from shear forces between inelastic carbon fibers and the healed tendon tissue. Autologous grafts with extensor tendons can be used to bridge the deficit between two ends of a lacerated tendon, but this technique has never gained popularity. PLLA has an advantage in that it supports fibroblast growth on its surface and loses its strength over several months. Therefore, it is able to match its mechanical properties with the tendon. Implants are anchored in each end of the lacerated tendon by fixing the ends in V incisions created in the tendon ends with sutures of monofilament absorbable sutures. The tendon splints have a semicircular cross-section and can be sutured to the tendon ends through holes in the splints. Implants are not recommended as a treatment for strain-induced tendinopathies.[62]

Partial lacerations involving $<50\%$ of the tendon may need only local debridement. Lacerations involving $>50\%$ of the tendon are probably best sutured, because this can prevent the generation of longitudinal splits between loaded and unloaded parts of the tendon or the failure of the remainder of the tendon under weight-bearing load.

Flexor tendon lacerations require a protracted rehabilitation period. A distal limb cast should be placed with forelimb lacerations post-operatively. In the hindlimb, a full limb cast is ideally required after flexor tendon laceration to immobilize the forces of the reciprocal apparatus. Distal limb casts can be used in the hindlimb to avoid the increased risk of complications with full limb casts. However, if an implant has been placed, this usually results in one end being pulled out of the tendon. Casting is required for a minimum of 6–8 wk and no more than 10–12 wk, because studies have shown that the breaking strength of the tendon repair at 6 wk approximates the body weight of the horse.[63] This means that usually at least one cast change under general anesthesia is needed.

Support of the metacarpal/metatarsal joint using a palmar/plantar splint with a modified Robert Jones bandage and caudal shoe extensions (for DDFT lacerations) can help protect the repair after cast removal. Continued box-stall rest is necessary for an additional 2–3 mo after which walking exercise followed by an ascending exercise regimen can be initiated. Ultrasonographic monitoring of tendon healing is useful to assess the integrity of the tendon repair. A minimum of 8–12 mo is usually required before full athletic function can be resumed.

The prognosis for flexor tendon injuries is guarded. In one study, ~45% returned to athletic function,[63] whereas in another study, the prognosis was 59% for flexor tendon lacerations.[64] In the second study, the prognosis for return to soundness was not increased if the DDFT and SDFT were simultaneously lacerated compared with if only a single structure was lacerated. Short-term complications include necrotic tendonitis, which occurs as a result of infection or damage to the vascular supply, concurrent synovial sepsis, cast complications, and exuberant granulation tissue formation. Long-term complications include adhesions, which result in continued pain and lameness, and occasionally, flexural deformity.

In contrast to the flexor tendons, extensor tendons heal remarkably successfully without tenorraphy, and they respond well to conservative management. The wound should be debrided, and the primary wound should be closed, if appropriate. If the extensor tendon has been lacerated within the confines of a tendon sheath (e.g., for extensor lacerations over the dorsal aspect of the carpus), lavage and elimination of sepsis from the tendon sheath also needs to be addressed. The prognosis for extensor tendon

lacerations is good with ~72% of horses returning to athletic function.[65] Other sources report an 80% good prognosis,[64] because the extensor tendon bears considerably less load. Additionally, it has a minimal effect on the gait, because most protraction of the limb and digit arises from the upper limb and momentum of the foot. Indeed, a recent publication reported the successful management of septic common digital extensor tenosynovitis by complete resection of the tendon.[66] Stumbling may be evident at the walk until the tendon has healed, but it can be reduced by shortening the toe of the hoof and rolling the toe of the shoe or by fitting a "Natural Balance" type shoe.

6. New Advances—the Evolution of Biologics

So far, none of the accepted treatment methods seems to be consistently more efficacious than any other for long-term return to racing without reinjury. After injury, tendon heals (repairs) well, but the scar tissue that replaces the damaged tendon is less functional than normal tendon tissue. This results in reduced performance and a substantial risk of reinjury. To avoid these adverse consequences, an ideal treatment should, therefore, aim to avoid the formation of excessive fibrous tissue and be able to regenerate normal tendon matrix. Spontaneous regeneration of tendon post-injury does not seem to occur. This is most likely because the endogenous stem cell response is inadequate: cells recovered from tendon show poorer differentiation capacity than bone-marrow cells.[67] In addition, the cellular infiltration, although initially dominated by blood-born cells derived from the bone marrow that are likely to be white blood cells involved in local debridement of damaged tissue, is later substituted by more long-lasting cellular infiltration, which is probably derived from local surrounding tissues like the paratenon.[68] This provides further support for a technique of exogenous administration of bone marrow-derived stem cells.

The aim of regenerating tendon tissue involves a tissue engineering approach that has been proposed to depend on four separate components: an appropriate mechanical environment, a scaffold, an anabolic stimulus, and a cell source.[69] Each of these components can be used individually, although the maximum effect is believed to occur with a combination of all four.

1. Scaffolds

Scaffolds can have a variety of potential beneficial effects. They can be used to carry or attract cells, help align reparative tissue by their structure, and protect the cells immediately after implantation and before new matrix has been synthesized. However, their influence can be both positive and negative.

ACell Vet is a novel intralesional treatment for tendinopathy/desmopathy using acellular tissue components derived from porcine urinary bladder submucosa. This preparation has been suggested to deliver appropriate growth factors to the injured tissue as well as attract mesenchymal stem cells. It is injected in liquid form as a reconstituted powder. However, it has been associated with significant inflammatory reaction after injection. It is recommended that horses are premedicated with anti-inflammatories and that cold is applied locally after treatment. It has mostly been used to treat chronic SL disease where there are anecdotal reports of benefit, although no objective data has yet been published of its efficacy.

2. Growth Factors

Insulin-like growth factor-1 (IGF-1) has been investigated to assess its effect on tendon healing both in vitro and in collagenase-induced models of tendinitis.[70] IGF-1 stimulates extracellular tendon matrix synthesis and is also a potent mitogen.[70] In collagenase-induced models of tendinitis, initial swelling was decreased after intralesional injections of IGF-1 compared with controls. However, no differences were found at later time points, and there was no difference between the quantities of type I and type III collagen synthesized.[70] Currently, there is no published long-term follow-up data regarding reinjury rates of tendinitis treated with IGF-1.

Equine recombinant growth hormone (rEGH) administered intramuscularly has shown a negative effect on the biomechanical properties (decreased yield point and ultimate tensile strength) of the SDFT during the early phases of healing in collagenase-induced tendinitis.[71] These properties were, however, assessed at 6 wk after treatment, which may have been too soon to detect any beneficial effects of the rEGH.

Transforming growth factor-beta (TGF-β) has been considered as another appropriate growth-factor treatment, although clinical experience has been limited. Treated horses showed significant enlargement of the tendon. Reinjury rates were similar to conservatively managed horses, but these reinjuries were all on contralateral, untreated limbs.[c]

Platelet-rich plasma (PRP) is becoming more commonly used for the treatment of tendon and ligament injuries. It is usually defined as having >4 times the platelet count of normal blood, and it can be prepared by either centrifugation or filtration (Fig. 14). PRP contains high levels of those growth factors sequestered in platelets like TGF-β1, IGF-1, and platelet-derived growth factor (PDGF). It is not known if these growth factors are optimum for tendon and ligament healing, but they do have anabolic effects[72,73] and are, therefore, logical factors to use. However, it may be that PRP will serve to promote an exaggerated fibrotic reaction rather than regeneration. Given this concern, PRP is probably most suited to the treatment of SL lesions. Evidence of efficacy is still lacking, but small clinical studies have been positive.[74]

Fig. 14. fPRP[a] system for preparing plasma rich in platelets. This system uses a filtration system for concentrating the platelets rather than centrifugation, and it is a closed system that can be used "horse side."

3. *Mesenchymal Stem Cells*

Stem cells have the potential of differentiating into a number of tissues.[75–79] Embryonic stem cells are truly pluripotential but have the disadvantages of being allogenic (although with greater immunological tolerance) and being associated with a risk of teratoma formation. Postnatally derived stem cells are thought to be multipotential or have a restricted number of cell lineages into which they can differentiate. They are subdivided into hemopoietic (blood-cell lines) and mesenchymal stem cells (MSCs), which can give rise to osteoblasts (bone), chondrocytes (cartilage), tenocytes (tendon and ligament), fibroblasts (scar tissue), adipocytes (fat), and myofibroblasts (myotubes). Furthermore, these stem cells can be recovered from adult tissue, and thus, there is the possibility of autologous reimplantation. This also has the added benefit that they do not incite an immune response from the host. Such stem cells are most easily recovered from bone marrow, although other workers are considering the use of fat-derived stem cells. These preparations are, however, less well characterized at present.

The differentiation of the MSCs into tenocytes is believed to be induced by a combination of mechanical (tension) cues, growth factors, and contact with "like" cells and matrix, most, if not all, of which are provided by implantation within the tissue.

There is currently much interest in the use of stem cells to "engineer" new tissue. Clinically, MSCs are currently being used for the treatment of ischemic heart disease in man with encouraging results of survival and increased cardiac output. The transplantation of MSCs into injured skeletal tissues has been shown to promote healing in a multitude of studies in experimental animals. In tendon, studies have used surgically created defects in tendons and ligaments of laboratory animals and have all shown significantly improved outcomes with the implantation of bone marrow (BM)-MSCs.[53–55]

The equine SDFT injury has a different etiopathogenesis. However, it is ideal for the implantation of MSCs, because there is usually an enclosed defect within the tendon that can retain implanted MSCs without the need for a scaffold.

There are several techniques currently employed clinically: direct administration of bone marrow, use of fat-derived stem cells, and autologous implantation of MSCs.

1. The direct intraligamentous (or intratendinous) administration of bone marrow has been reported to show promising results in the treatment of SL disease.[80] However, there are very few MSCs present in a bone-marrow aspirate (~1 MSC per 10^5 nucleated cells), which has led some to suggest that this treatment more resembles a growth-factor treatment than a true cell-based therapy. Certainly, the supernatant of bone marrow has been shown to contain a rich mix of anabolic factors.[81] Furthermore, the injection of large volumes (20–30 ml) cannot be fully retained in the structure, and together with the presence of other cell types and tissues (e.g., bone spicules), it can potentially be damaging to the healing tendon or ligament, especially by inducing ectopic calcification.
2. The above technique using BM-MSCs has received the most basic science research and aims to provide a pure source; however, VetStem[b] in the United States has used another source of stem cells from fat (Fig. 15). This technique involves collecting fat from the

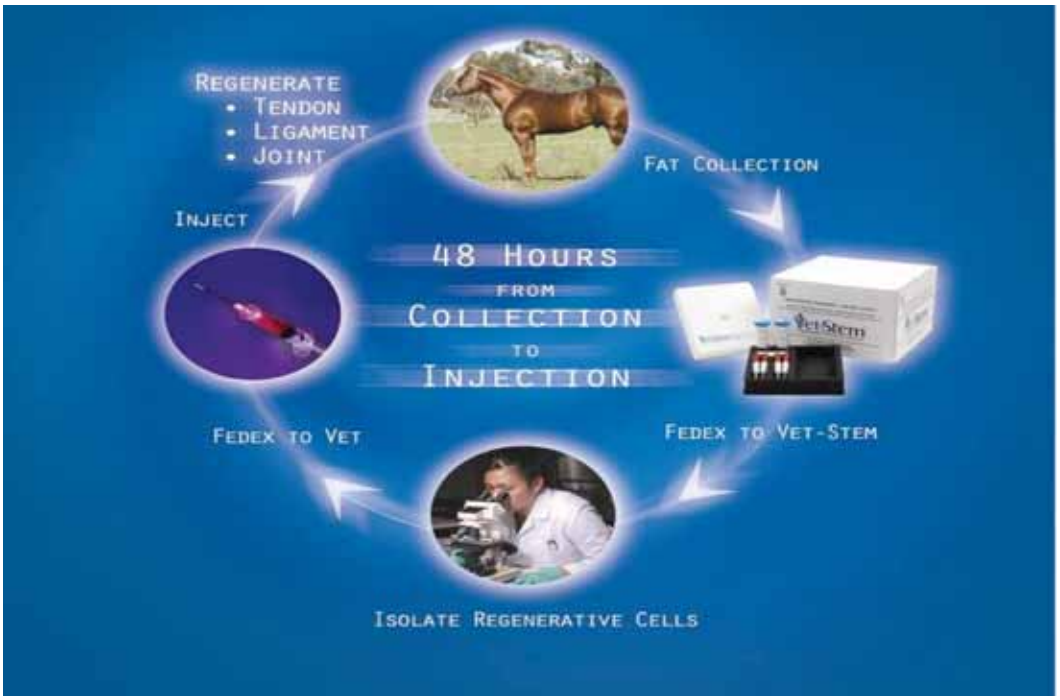

Fig. 15. The Vet-Stem[b] system for the treatment of tendon and ligament injuries with fat-derived cells.

tail head, digesting the tissue, and removing the fat cells. This leaves a mixture of cells that includes the adipose-derived stem cells (~2%), which are shipped back to the veterinarian without further purification for intratendinous injections.

3. In contrast, we have been developing an alternative approach involving the implan-

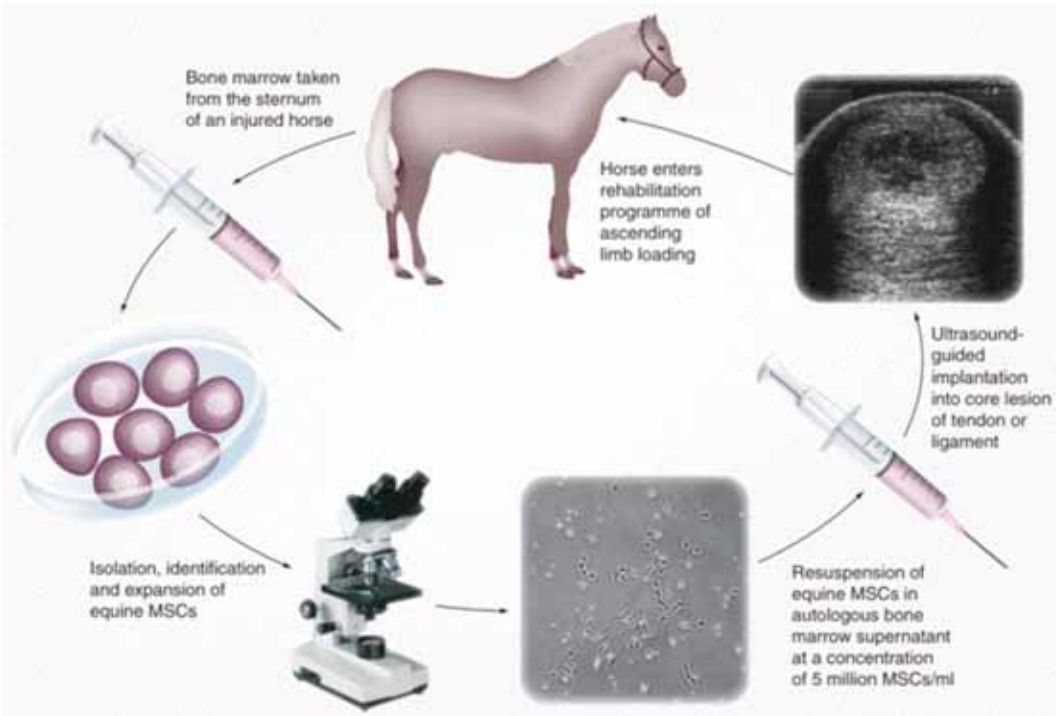

Fig. 16. The VetCell technique for the treatment of tendon and ligament injuries with autologous BM-MSCs.

Table 1. Typical Exercise Program After Tendon Injury (NB This program should be shortened or lengthened depending on the severity and progress of the case)

Exercise Level	Weeks	Duration and nature of exercise
0	0–2	Box rest
1	3	10 minutes walking
1	4	15 minutes walking
1	5	20 minutes walking
1	6	25 minutes walking
1	7	30 minutes walking
1	8	35 minutes walking
1	9	40 minutes walking
1	10–12	45 minutes walking daily
	Week 12	Repeat ultrasound examination.
2	13–16	40 minutes walking and 5 minutes trotting daily
2	17–20	35 minutes walking and 10 minutes trotting daily
2	21–24	30 minutes walking and 15 minutes trotting daily
	Week 24	Repeat ultrasound examination
2	25–28	25 minutes walking and 20 minutes trotting daily
2	29–32	20 minutes walking and 25 minutes trotting daily
	Week 32	Repeat ultrasound examination
3	33–36	45 minutes exercise daily with slow canter
3	37–40	45 minutes exercise daily with slow canter
3	41–44	45 minutes exercise daily with fast work three times a week
3	45–48	45 minutes exercise daily with fast work three times a week
	Week 48	Repeat ultrasound examination
4	From 48 wks	Return to full competition/race training

tation of large numbers of autologous MSCs derived from bone marrow and expanded in the laboratory (Fig. 16). We hypothesized that the implantation of marrow-derived stromal stem cells (BM-MSCs) directly into the central cavity of an injured SDFT would provide all four of the requirements for effective tissue engineering.

- The cells within the tendon would experience the tensional load placed on the tendon.
- They would be injected into a scaffold of granulation tissue that is highly vascularized. This provides maximal chance of cell survival, even with large lesions.
- The cells are implanted with the supernatant of the bone marrow from which they were derived. This has a pronounced anabolic effect on equine ligament cells that is greater than that of other biological fluids, including platelet-rich plasma.[81]
- The use of BM-MSCs would have the potential of synthesizing a matrix that more closely resembles tendon matrix than scar tissue. Thus, this would improve functional outcome because of the increased capacity for a successful return to performance without reinjury.

Our technique (developed in conjunction with Vet-Cell Bioscience Ltd.) involves the collection of bone marrow from the sternum in the standing sedated horse followed by isolation and expansion of the nucleated adherent cell population (containing the MSCs) in the laboratory.[77] After ~3 wk, in excess of 10×10^6 cells are available for implantation. The cells are suspended in bone-marrow supernatant, because this has been found to have a beneficial mix of growth factors. Then, they are implanted under ultrasonographic guidance into the core lesion of the tendon or ligament under standing sedation.

The horses then enter an ascending exercise regimen aimed at providing a controlled, mechanically appropriate (tension) environment for the cells. This consists of initial rest for 7 days (for the cells to establish themselves) and is followed by walking exercise for 3 mo. Thereafter, trotting is introduced after 3 mo and is followed by cantering after 9 mo and full work at 12 mo. Regular ultrasonographic monitoring is advised at 1, 3, 6, 9, and 12 mo post-implantation.

Initially, a Phase I trial was performed to ensure safety. This consisted of six horses with large core lesions in their SDFTs that were followed for up to 12 mo. Results indicated that the technique did not cause any worsening of the injury, and there was no reaction or enlargement of the tendon post-implantation. Furthermore, no bone or cartilage was formed based on gamma scintigraphy and ultrasonography after 3 mo. Core lesions filled in quickly when a hypoechoic lesion was still visible at the time of implantation. The longitudinal pattern,

however, remained inferior to normal tendon but improved with exercise.

Since the initial trial, in excess of 500 horses have been treated with this technique. At the most recent evaluation of clinical outcome (September 2007), 172 racehorses had been treated with >1 yr follow-up. For National Hunt racehorses (n = 145), the reinjury rate was 18% (23% when injuries to untreated contralateral limbs were included). When only those horses that had entered full training were included, the reinjury rate rose slightly to 24% (33% with contralateral reinjuries). These percentages have remained relatively constant for up to 3 yr after treatment, although numbers are small for the longest follow-up. However, these data compare favorably with previous analyses for the same category of horse (56% reinjury rate for National Hunt horses[14]) for analysis of horses used for the same discipline followed for 2 yr after a return to full work. In further support for this improvement in outcome, reinjury rates for sports horses (all disciplines combined; n = 109 with >1 yr follow-up) was improved by a similar degree (13% compared with 23–43% reported for different sport-horse disciplines[35]).

We proposed that the optimum time to implant the cells is after the initial inflammatory phase but before fibrous tissue formation. It was hypothesized that the presence of mature fibrous tissue within the tendon would (1) make implantation more difficult and (2) reduce the benefits of the stem-cell therapy because of its persistence. Both have been supported by clinical experience of delayed implantation of BM-MSCs and outcome. Successes had an average interval between injury and implantation of 44 days, whereas horses suffering reinjury had an average interval of 83 days (p = 0.0035). Current recommendations are that bone marrow is aspirated within 1 mo of injury. For the same reason, known recurrent injuries are not considered ideal cases, because significant fibrosis would already be present. The time of implantation may be further optimized by pre-injury storage of cells.

Five cases that died through unrelated causes have been analyzed histologically and showed excellent healing with minimal inflammatory cells and crimped organized collagen fibers. In contrast, a contralateral untreated SL injury in one of these horses, which was clinically silent at the time of implantation, showed persistent inflammatory cells and poorly organized collagen fibers.

A more limited number of cases have been treated with injuries to other tendons and ligaments. For lesions present within a tendon sheath, the implantation is done after tenoscopic evaluation to ensure that there are no surface defects through which the cells could leak.

10. Conclusions

The extensive number of treatments available for the treatment of tendon and ligament injuries provides strong evidence that none are universally effective. Because of the natural variability of the disease, careful and objective assessment of large numbers of cases is essential to prove efficacy of a treatment. Treatment should be selected by stage and severity of disease, use of horse (racing is still the most severe test for an effective treatment), and follow-up time (reinjuries tend to occur when the horse is back racing but not before). This means that strong evidence-based treatments cannot be provided. The two most important aspects are to (1) obtain an accurate diagnosis of which structure is injured, including the stage and severity, and (2) apply treatments based on a strong scientific rationale with respect to the pathophysiology. Currently, combinations of treatments may offer the best approach (e.g., stem-cell treatment and SCLD). However, in many cases, cost implications may also influence the choice of combination treatments.

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