Biomechanical Testing of Double Hemitenotomy for Equine Deep Digital Flexor Tendon Lengthening

by

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Abstract

Deep digital flexor (DDF) tenotomy is performed in horses with laminitis to diminish the force of the DDF on the third phalanx (P3) which causes P3 rotation due to loss of laminar integrity counteracting the DDF pull. DDF tenotomy results in a guarded prognosis for athletic use. Consequently, a surgical procedure for DDFT lengthening rather than complete transection may be advantageous and allow for better recovery of tendon function compared to traditional tenotomy. The objective of this study was to develop a double hemitenotomy (DHT) technique to lengthen the equine DDFT. Models were used to 1) evaluate the DDFT tensile strength following DHT; 2) evaluate the DDFT lengthening following DHT compared to complete tenotomy (CT) and; 3) compare the reduction in DDFT load following DHT to CT. We hypothesized that DHT would result in less than 3 mm lengthening of the DDFT and at least half the load reduction of CT.

The study was divided into two parts using cadaveric models. In part 1, cadaveric DDFT pairs (n=30) were utilized. Two hemitenotomies were created in one tendon while the other served as an intact control. Monotonic tensile load was applied to a maximum of 500 kg using an Instron Universal Testing Machine. Tendon lengthening, load reduction with hemitenotomy and load at failure were recorded by the Instron. In part 2, cadaveric forelimb pairs (n=16) were subjected to DHT followed by CT under monotonic compressive load applied by the Instron. Interactions between independent and dependent variables were assessed with statistical tests including the Wilcoxon Signed Ranks test and Friedman test.

DHT resulted in significant DDFT lengthening and load reduction in both isolated tendons and intact cadaveric forelimbs. Less lengthening was achieved with DHT compared to CT (P=0.008). No difference in load reduction between DHT and CT was observed (P=1). A smaller

load reduction was observed following the first hemitenotomy incision when compared to the second hemitenotomy incision (P=0.022). Isolated DHT tendons failed at a tensile load of 195 kg compared with none of the intact tendons (P=0.0001). None of the tendons failed in part 2.

In conclusion, DHT was comparable to CT in load reduction. DHT reduced tensile strength, but load at failure was similar to or exceeded the estimated load on the DDFT in vivo at stance.

DHT may be a useful alternative for surgical management of horses with laminitis, but in vivo studies are needed to confirm these findings.

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List of Abbreviations

ALDDFT	Accessory Ligament of the Deep Digital Flexor Tendon
cm	Centimeters
CT	Complete Tenotomy
DDFT	Deep Digital Flexor Tendon
DHT	Double Hemitenotomy
kg	Kilograms
mm	Millimeters
MRI	Magnetic resonance imaging
P3	Third Phalanx
SDFT	Superficial Digital Flexor Tendon

I. Introduction and Literature Review

a. Laminitis

i. Importance

Laminitis is a disease of the foot that can result in chronic lameness in horses and cause severe, intractable pain necessitating humane euthanasia.[1] Currently, it is the second leading cause of death in horses, and to date there is no cure.[2] Several etiologies have been proposed but irrespective of the origin, the disease process results in lamellar degradation and failure of the suspensory apparatus for the third phalanx (P3), allowing P3 to be displaced (rotate and/or sink) within the hoof capsule.[3] The disease has afflicted the equine population for centuries [2] and affects approximately 16% of horse facilities in the United States.[4] The economic cost of lameness (including laminitis) in 1998 was estimated to be $678 million to $1.1 billion with 110 days of lost use per lameness event.[5] Given the economic and emotional consequences as well as the lack of a universally accepted treatment for the prevention of P3 rotation, the need for continued investigation is clear.

ii. Forces Placed on P3

The biomechanical function of the equine digit is to absorb, transmit, and disperse loads from the ground and musculoskeletal system to the soft tissues and bones of the distal limb.[6] Vertical loads of up to 1.7 times the horse´s body weight occur on a single forelimb at a gallop.[7] All structures in the equine digit, including the laminae, work in concert to absorb and dissipate these extreme loads (Figure 1).[8]

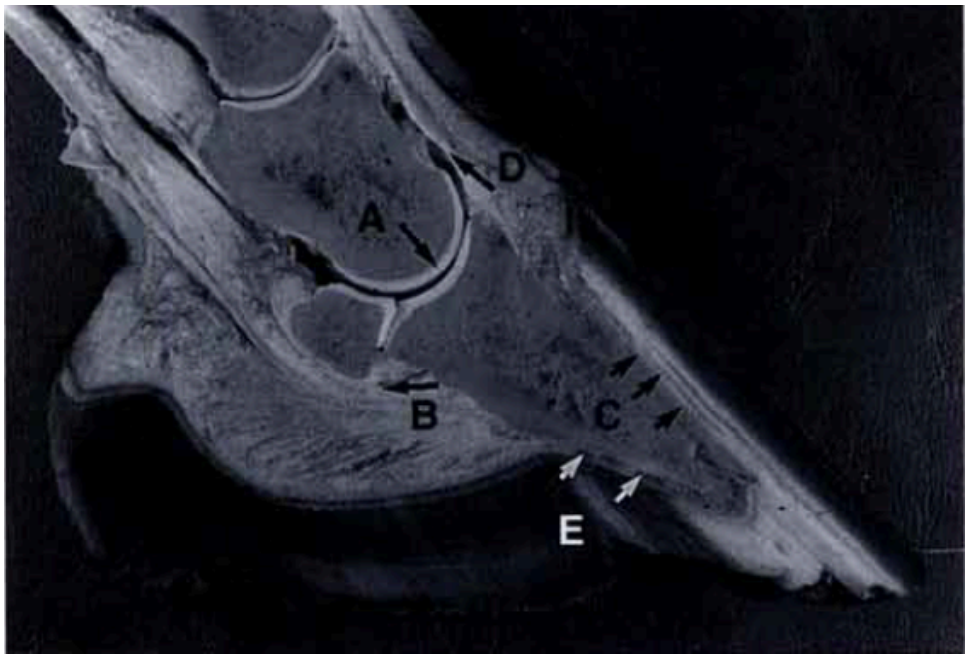


Figure 1. The five main forces acting on a normal P3 include compressive load as a result of the weight of the horse (A), tensile load as a result of the deep digital flexor tendon attaching on the palmar surface of the P3 (B), tensile loads generated by the laminar interface (C), tensile loads as a result of the common digital extensor tendon attaching on the extensor process of the P3 (D), and compressive loads against the sole (E), taken from Hood 1999.

The lamellae are leaf-like structures that attach the hoof capsule to P3.[8,9] There are approximately 600 primary dermal laminae originating from stratum internum or lamellar corium, which is contiguous with the subcutis and periosteum covering the parietal surface of P3. These interdigitate with the insensitive, or horny, laminae of the stratum internum, the deepest layer of the hoof wall (Figure 2). Each of the primary laminae gives off approximately 100 secondary laminae increasing the functional surface area of the laminar junction.[10]

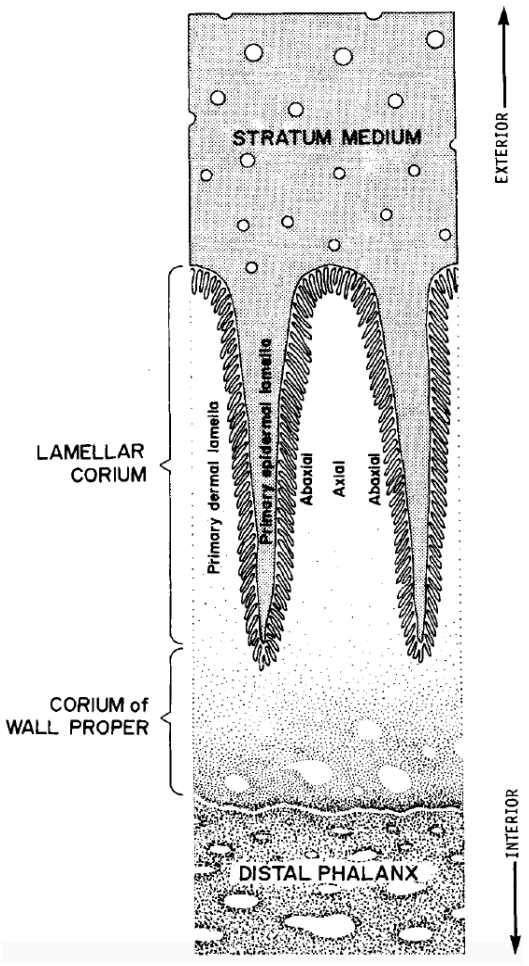


Figure 2. Schematic illustration of a cross section through the hoof wall showing primary and secondary lamellae. The lamellar corium forms the deepest layer of the hoof wall, also called stratum internum, taken from Mishra and Leach 1983.

Loads on the ground/hoof interface are transmitted through the hoof wall, to the laminae, the P3, and finally to the bony column of the limb. At higher loads and speed, the frog, an elastic V-shaped structure of the horse's hoof, additionally dissipates load.[11] Forces that act on the hoof during weight bearing include a vertical compressive load due to the weight of the horse, a tensile load resulting from the deep digital flexor tendon (DDFT) attaching on the palmar surface of P3, tensile loads generated by the laminar interface and the common digital extensor tendon

attaching on the extensor process of P3, as well as compressive loads against the sole.[8] The important role of the laminae in dampening locomotory forces has been previously demonstrated. The laminar structures were found to dampen 67% of the impact vibration in an in vitro study.[12]

iii. Phases of Laminitis

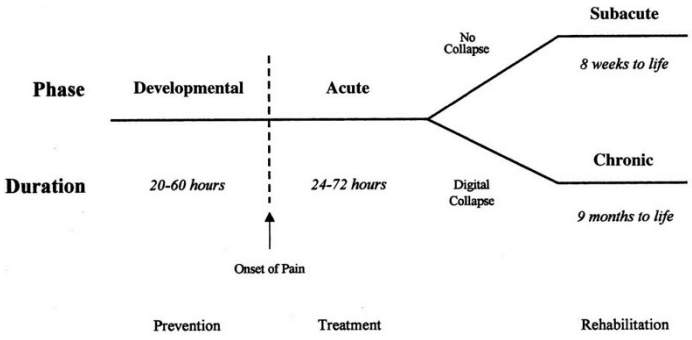
There are four phases of laminitis: the delopmental phase, the subacute phase, the acute phase, and the chronic phase. Developmental laminitis signifies the first phase defined as the period between the causative insult and development of lamellar pathology.[13] This phase lasts a maximum of 72 hours and occurs when lamellar destruction has been triggered but the horse is not yet painful.[14] In the second, acute phase of laminitis, the laminae undergo further microscopic changes that alter their biomechanical function and the first clinical signs are apparent (Figure 3A). The initial histologic findings include endothelial cell swelling followed by erythrocyte congestion and occlusion of laminar capillaries occuring within 8 hours after onset of lameness. Microvascular thrombi and worsened edema is followed by hemorrhage in the primary dermal laminae within 72 hours. The resulting ischemia causes epithelial cell edema, vacuolization, nuclear swelling and pyknosis of the secondary epidermal laminae (Figure 3B).[14] Detachment of the basement membrane from the basal cells of the secondary epidermal lamellae occurs. Mechanical integrity of the basement membrane is critical for stable laminar attachment to P3, and loss of it's integrity further contributes to mechanical breakdown of the hoof .[8]

Horses with mild damage to the laminar interface can recover following healing of microscopic lamellar collapse. If mechanical failure of the foot does not occur within the first 72 hours after onset of clinical signs (acute phase), the horse is considered to enter the subacute phase of laminitis. Rotational and/or vertical displacement of P3 relative to the hoof wall is a

hallmark sign for mechanical collapse of the foot (chronic phase of laminitis). Rotation of P3 occurs in horses with sufficient laminar interface damage to unoppose the tension applied by the DDFT (tensile force) (Figure 4A).[8] Horses with chronic laminar changes have reduced strenght of the laminar junction compared to normal horses. When these horses undergo medical treatment for acute laminar inflammation, horses that respond to treatment have greater total laminar strength than horses that do not respond to treatment (60% of normal laminar strength in responders vs. 42% of normal laminar strength in non-responders).[15] In horses with more circumferential damage to the laminae, distal displacement (i.e. sinking) of P3 may occur as a result of the vertical load down the bony column from the horse's body weight (shear force) (Figure 4B). Distal displacement of P3 can occur in addition or in place of rotation.[8]

Laminitis is more common in the front feet as a result of horses bearing approximately 60% of their total body weight on their front limbs.[16] However, laminitis of a single limb or all four feet can occur.[16,17]

(A)



(B)

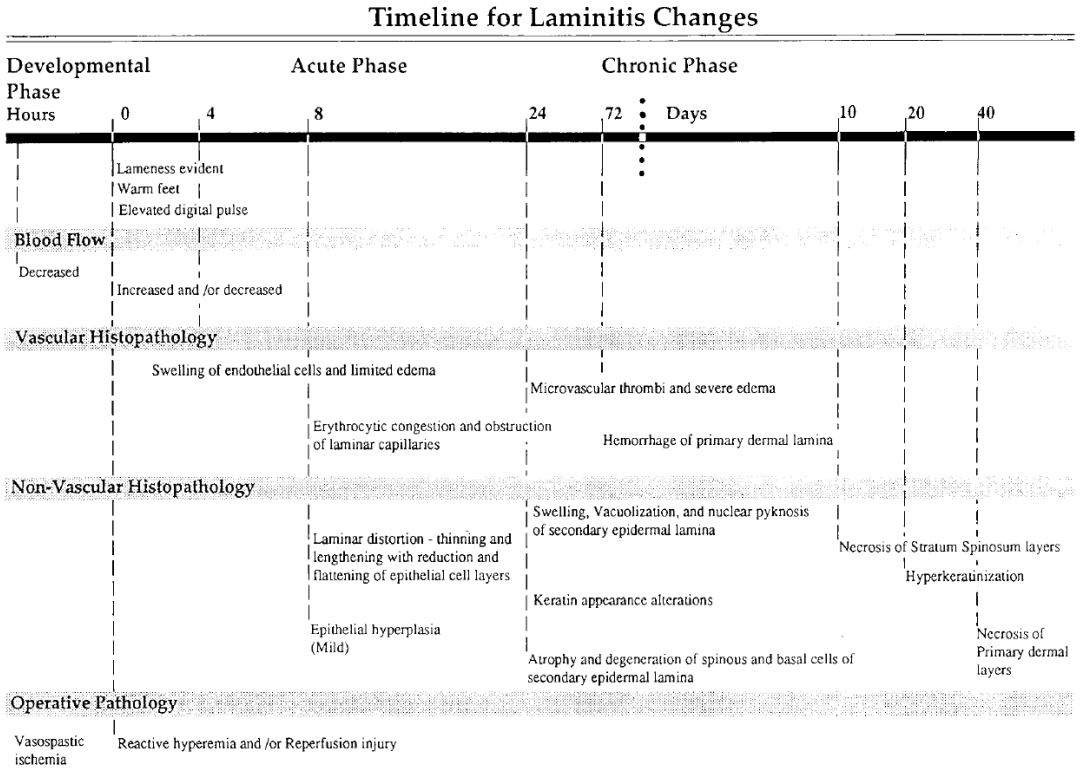


Figure 3. Laminitic phases. A, Duration and therapeutic goals, taken from Hood 1999. B, Clinical, hemodynamic, and histopathologic changes occurring in horses with laminitis, taken from Hood 1993.

(A) **(B)**

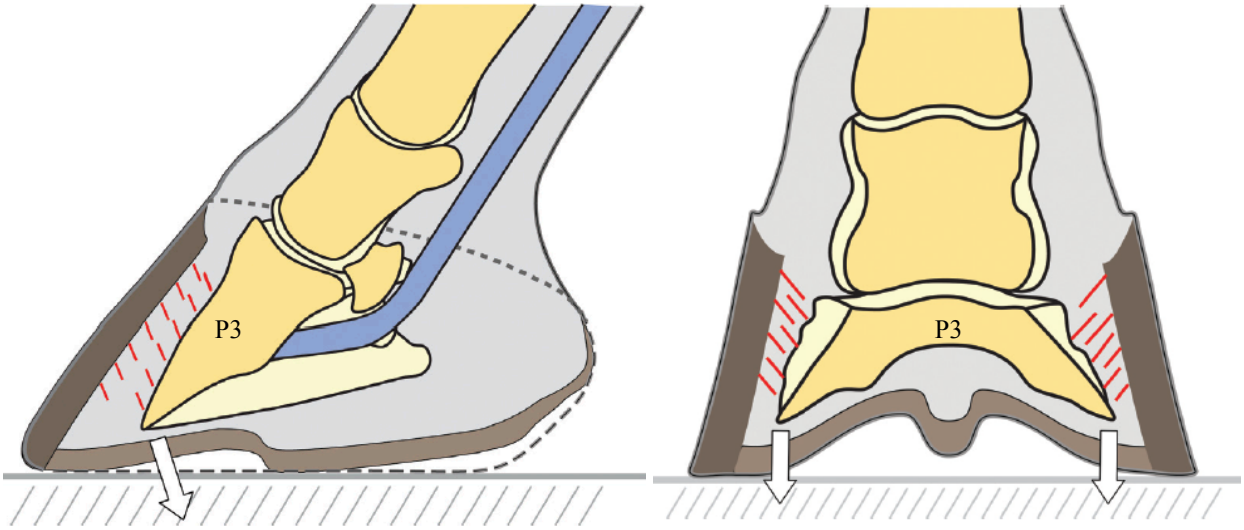


Figure 4. Displacement of the third phalanx (P3) within the hoof capsule. A, In horses with rotational displacement failure of the lamellae supporting the P3 is greatest dorsally, while the heels may be relatively unaffected B, In horses with vertical displacement disruption of the lamellae supporting the distal phalanx appears to occur around the circumference of the foot. The deep digital flexor tendon is outlined in blue and the hoof capsule in brown, taken from Equine Laminitis, 1st edition.

b. Anatomy of the forelimb DDFT and ALDDFT

The deep digital flexor (DDF) muscle has three heads of origin in the forelimb; the medial epicondyle of the humerus (humeral head, largest), the middle aspect of the medial radius (radial head) and the caudal aspect of the proximal ulna (ulnar head). Tendons of all three heads fuse at the level of the carpus to form the DDFT, which courses distally, deep to the SDFT and superficial to the suspensory ligament (Figure 5). The DDFT inserts on the palmar aspect of P3 in the facies flexoria. The accessory ligament of the deep digital flexor tendon (ALDDFT) originates from the palmar carpal ligament and fuses with the DDFT in the middle third of the metacarpal region. The SDFT and DDFT are surrounded by the digital flexor tendon sheath as they pass over the palmar fetlock joint, providing a smooth gliding surface.[18]

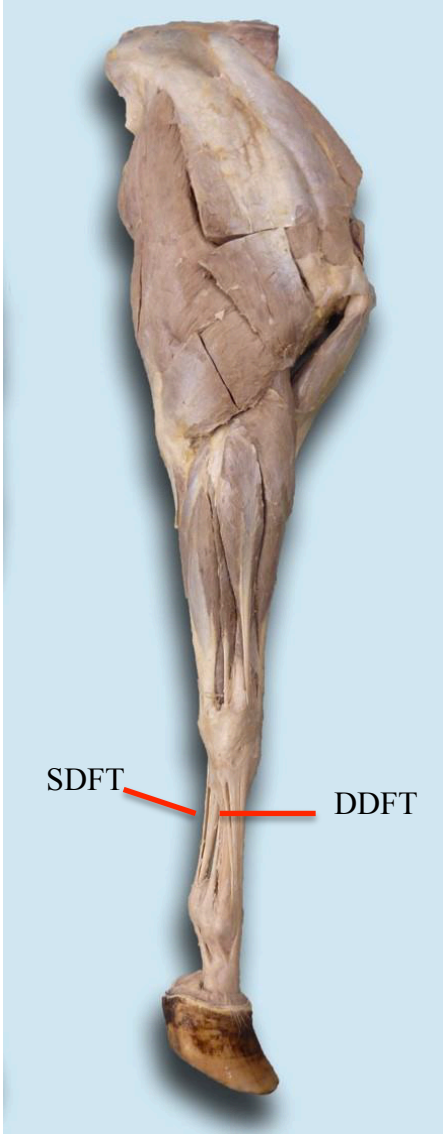


Figure 5. Schematic of the muscles and tendons of the forelimb of the horse. DDFT, Deep Digital Flexor Tendon. SDFT, Superficial Digital Flexor Tendon. Image provided by R. Wilhite.

c. Current treatments of equine laminitis

i. Developmental and Acute Phases

Laminitis often develops as a sequel to 4 different clinical entities including disease associated with sepsis/endotoxemia, excessive overloading of a limb due to injury to the opposite limb, pars

pituitary intermedia dysfunction (PPID, i.e. Cushing´s disease), or equine metabolic syndrome (EMS) including pasture associated laminitis. Thus it is of paramount importance that the primary disease is treated urgently and effectively.[19]

Treatment of laminitis will differ within each of the three distinct phases.[14] Prevention of predisposing factors and/or inciting cause is important and would include treatment of conditions causing systemic inflammation from endotoxemia, appropriate structural support in a horse that is unequally bearing weight due to a musculoskeletal injury, and/or treatment of underlying metabolic disorders (i.e. PPID or EMS). However, laminitis can be difficult to predict and the inciting cause can be multifactorial therefore aggressive medical and/or surgical treatment is necessary if a horse is suspected to be in the developmental or acute phases of laminitis.[20] In addition to elimination or minimization of any predisposing factors, the goals of treatment in the developmental and acute phase of laminitis include reduction of pain, reduction or prevention of permanent laminar damage, improvement or reversing of deleterious digital or laminar hemodynamics, as well as mechanical support to prevent further displacement of P3 within the hoof capsule.[16] Cryotherapy (i.e. soaking the feet in crushed ice or cold water) is one reportedly effective method to prevent vasodilation and reduce the activity of tissue-degrading enzymes. To mechanically support the diseased laminae, stall confinement with deep bedding, and methods to transfer the load of weight-bearing from the hoof wall to the frog (shoe removal, trimming of the hoof and sole support) should be instituted.[21] Numerous other treatments including primary medication and/or supplements are used at the discretion of the clinician.[22]

ii. Chronic Phase: Medical and Surgical Treatments to Manipulate DDFT Tension

Once chronic laminitis has occurred, the main goals of treatment are to correct residual foot problems and prevent recurrence of systemic disease. Various medical, biomechanical, and

surgical treatments are used to achieve these goals.[16] Tension within the DDF muscle tendon unit constitutes one of the major forces causing rotational displacement of P3.[23] In an approximately 500 kg horse, the tensile force in the DDFT required to maintain the distal interphalangeal joint in a neutral position during walking was measured to be approximately 415 kg.[24] To date, there is no universally accepted treatment for the prevention of P3 rotation. However, repair of laminar tissue occurs rapidly if no physical disruption has occurred[25] and recovery is therefore likely if the anatomical integrity of the hoof could be maintained during early stages of laminitis.

There is general agreement that heel elevation to decrease the tensile force of the DDFT acting on P3 is important.[26] Non-surgical treatments that aim to reduce the tension within the DDFT include application of a heel wedge or injection of the DDF muscle with clostridium botulinum toxin.[27,28] The main goal of surgery in refractory laminitis is to reduce or eliminate the force of the DDFT on P3 to prevent further rotation and/or sinking. Currently used techniques include DDF tenotomy and desmotomy of the ALDDFT.[29-31]

1. Corrective Trimming and Therapeutic Shoeing

In a laminitic horse, application of a shoe is performed following appropriate trimming of the hoof to reduce the load on the most severely affected lamellae, increase the base of support, facilitate breakover of the foot, and provide heel elevation if necessary. The trim is performed with the aim to restore normal spatial alignment between the solar surface of P3 and the hoof capsule and should be guided by radiographs.[32] The goals of therapeutic shoeing in horses with chronic laminitis are several.[33] The first includes stabilization and protection of the mechanically collapsed digit to allow healing of submural and subsolar tissues. The second goal is to increase comfort by reducing undesirable biomechanical forces (from the DDFT or the hoof wall). Lastly

it is used to restore the normal conformation of the foot. Several types of shoes have been used for these purposes including egg-bar shoes, heart-bar shoes, reverse shoes, aluminum shoes, and wooden shoes. A single shoe type will not benefit all horses with laminitis.[32]

Elevating the heels is believed to decrease the load on the laminae by decreasing the tensile stress on P3 by the DDFT.[34] An 18º heel wedge was successful in the majority of 29 cases in which it had been used on the basis of improvement in the level of the horse's comfort, but no biomechanical assessment was included.[34] Another study using a hoof cast with a 15-20º heel wedge documented a 59% decrease in strain on the dorsal hoof wall, but a 34% increase in strain on the lateral hoof wall, using strain gauges instrumented on cadaver specimens.[27]

2. Botulinum Toxin Type A Injection

Clostridium botulinum toxin type A reduces muscle activity and has a spasmolytic effect in horses.[28] Injecting the DDF muscle of a horse with laminitis with C. botulinum toxin type A results in reduced DDF muscle activity without causing lameness.[35] The theory is that the injection could therefore diminish DDF pull on the P3 and prevent P3 displacement in horses with acute laminitis.[28]

In one study, C. botulinum toxin type A injections (100-200 IU per limb) into the DDF muscle of 7 laminitic horses resulted in radiographic stabilization of P3 displacement and clinical improvement in lameness.[28]

It has been theorized that C. botulinum toxin type A could have a place in the treatment of laminitis without the risks involved with tenotomy including contracture of the DDF.[28,36]

3. Deep Digital Flexor Tenotomy

DDF tenotomy is the most commonly described surgical technique for treatment of severe or refractory laminitis.[29,37] It is considered a salvage procedure, and the technique was first described by Allen and colleagues in 1986. The technique was performed at the level of the pastern, requiring entrance into the digital flexor tendon sheath.[37] Hunt and colleagues later described a technique for DDF tenotomy performed at the level of the mid-metacarpal diaphysis distant from synovial structures.[29] Both sites are considered equally effective, but use of the mid-metacarpus technique is typically considered easier and safer for the equine practitioner.[29]

Mid-metacarpal tenotomy is described in the standing, sedated horse (Figure 6). Regional analgesia is provided with a high palmar nerve block,[29] median and ulnar nerve block,[38] or via local infiltration around the DDFT proximal to the surgical site.[29] For the minimally invasive technique, a vertical stab incision is made laterally between the superficial and DDFT through the skin and subcutaneous tissues at the mid-metacarpus level. A mosquito hemostatic forcep is guided between the superficial and DDFT in a medial direction to separate the two tendons and create a tract for the tendon bistoury. A single edge tendon bistoury is introduced with the cutting edge oriented proximally between the superficial and DDFT and is subsequently turned 90 degrees to transect the DDFT with a dorsal cutting motion. The DDFT tenotomy results in approximately 2 cm gap formation between the transected ends of the DDFT. The skin incision is left open or sutured with a simple interrupted pattern.[29] A more traditional open approach is also described which exposes the DDFT through a longer skin incision prior to its elevation and transection. Regardless of surgical method, a shoe with a heel extension is required following DDFT tenotomy for a minimum of 10 weeks to reduce the chances of distal interphalangeal joint (DIP) subluxation.[36]

DDF tenotomy generally has a variable success rate. Seventy seven percent of horses survived for at least 6 months and 59% for over 2 years after DDFT in a study by Eastman et al. including 35 cases between 1988 and 1997.[39] In the study by Allen et al., five of thirteen (38.5%) horses returned to light athletic activity, six were pasture sound and two were subsequently euthanized due to deterioration of the patient or economic constraints.[37] In the study by Hunt et al., 11 horses survived less than 1 month, six survived more than 6 months and 3 of those remained lame. None of the horses in that study returned to athletic performance.[29] Morrison reported the results of 245 cases. Sixteen of 124 cases (13%) returned to some form of athletic soundness following DDF tenotomy. Disease of P3, characterized as radiographic signs of demineralization or remodeling, and clinical or radiographic P3 sinking had a negative influence on outcome in that study. On the contrary, the degree of P3 rotation did not influence the long-term prognosis.[36] It is unknown whether the poor athletic return rate reported reflects poor recovery of tendon function[40] or the severity of laminitis.[29]

Morrison reported four criteria for a successful DDF tenotomy including careful case selection, timing of the procedure (before P3 disease occurs), importance of the combination with realignment shoeing and physical therapy to encourage loading of the limb and prevent excessive scarring and DDFT contracture.[36]

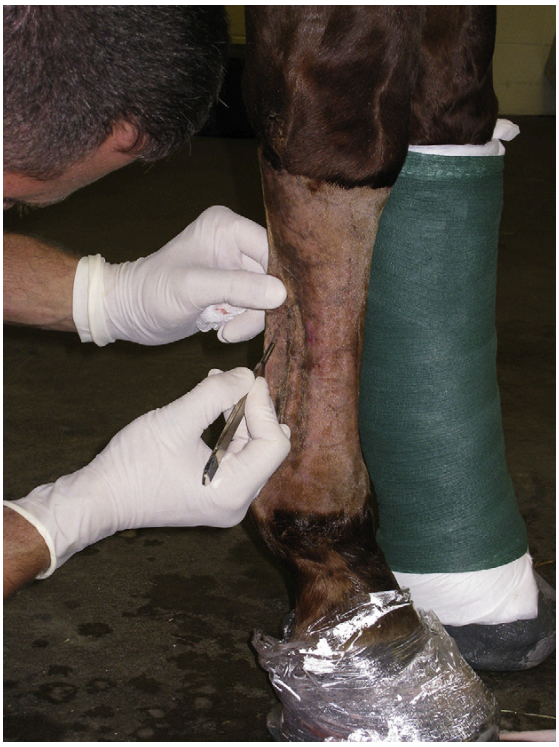


Figure 6. DDFT tenotomy performed at the mid metacarpal region in the standing and sedated horse following regional anesthesia and realignment shoeing, taken from Morrison 2011.

4. Desmotomy of the Accessory Ligament of the Deep Digital Flexor Tendon

Desmotomy of the accessory ligament of the deep digital flexor tendon (ALDDFT) has been reported as an alternative to DDF tenotomy for treatment of refractory laminitis cases. The procedure is associated with less morbidity and improved prognosis for return to athletic use compared to DDF tenotomy. Its use over DDF tenotomy appears related to the severity of P3 rotation and tension of the DDFT.[31] However, documented increases in the peak load of the DDFT at certain stance times following desmotomy of the ALDDFT[41] contributes to questionable benefits for using this procedure in chronic, refractory cases of laminitis.

Desmotomy of the ALDDFT can be performed using an open technique with or without ultrasound guidance, or via a tenoscopic approach.[31,42,43]

Milner first reported bilateral desmotomy of the ALDDFT in a laminitic pony.[31] The pony was anaesthetized and placed in dorsal recumbency. An open approach was used and a 3-4 cm vertical incision was made through the skin and subcutaneous tissues over the lateral aspect of the mid to proximal metacarpus over the dorsal DDFT. The ALDDFT was identified, isolated using artery forceps, and exteriorized through the skin incision (Figure 7). Sharp horizontal complete transection of the ALDDFT was performed using an #11-scalpel blade. The paratenon and subcutaneous tissues were sutured separately in a simple continuous pattern and the skin was closed using surgical staples.[31]

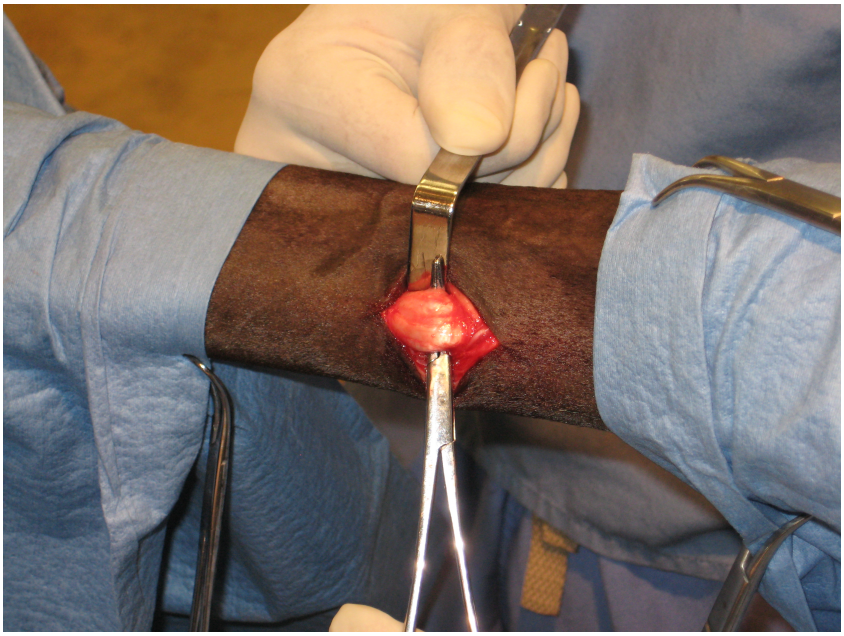


Figure 7. The ALDDFT is isolated and exteriorized above artery forceps prior to sharp complete horizontal transection of the ligament. Image provided by F. Caldwell.

5. Lag-screw through the dorsal hoof wall into P3

Recently, a lag-screw technique was described using a 5.5-mm-diameter cortical bone screw through the dorsal hoof wall and into P3 to counteract the DDFT pull on P3.[30] The technique was

used in 40 limbs from 10 equine cadavers and 16 clinically normal horses. Laminitis was induced using oligofructose in ten of the horses prior to screw placement. The screw was placed through the midline of the dorsal hoof wall two cm distal to the coronary band (Figure 8). A standard lag-screw technique was used, including a 5.5-mm glide hole through the dorsal hoof wall followed by a unicortical 4.0-mm hole through the majority of P3. The palmar or plantar cortex of the bone was avoided to prevent inadvertent DDFT damage (Figure 9). The screw was tightened until dorsal hoof wall compression was seen and was subsequently backed out by one-quarter turn by the screwdriver. Radiographs were used to guide the screw placement. Ten horses had a washer placed and six did not. The purpose of the washer was to prevent rotation of P3 before the screw head completely engaged the hoof horn.

The technique was demonstrated to provide sufficient holding power to counteract the pull of the DDFT in approximately 500-kg horses. In clinically normal horses without induced laminitis, the technique was well tolerated and did not cause lameness. This technique was able to stabilize P3 in horses with induced laminitis, but no reduction in pain was noted with mechanical stabilization. Its use has not yet been reported in horses with naturally occurring laminitis.[30]

Figure 8. Photograph showing a 5.5-mm-diameter cortical bone screw with a washer aseptically placed through the midline of the dorsal hoof wall into P3 two cm distal to the coronary band, taken from Carmalt et al. 2019.

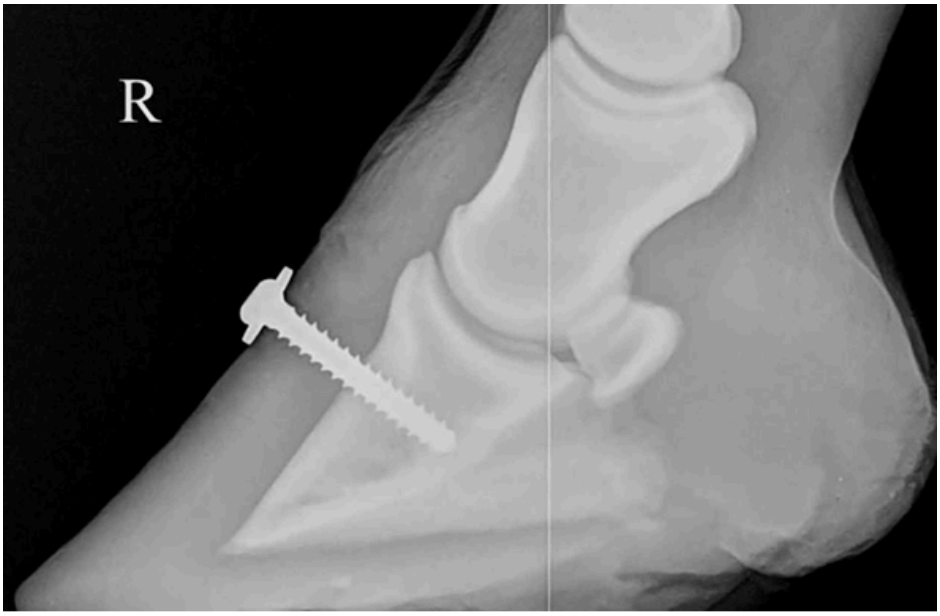


Figure 9. Lateromedial radiograph illustrating a right front unicortically placed screw with a washer through the dorsal hoof wall and P3, taken from Carmalt et al. 2019.

Without a generally accepted, well proven therapeutic strategy, better treatment than the current standard DDF tenotomy is needed to preserve DDFT function if the horse recovers from laminitis.

d. Tendon lengthening in humans

Surgical techniques for lengthening of the gastrocnemius-soleus complex have been used to treat equinus deformity in the human orthopedic field for several decades.[44] Equinus deformity is a condition in which ankle dorsiflexion is restricted.[44-47] It has been speculated to result from

failure of longitudinal growth of the gastrocnemius-soleus complex relative to tibial growth[48] and has been associated with a number of clinical conditions including cerebral palsy.[44] The prevalence in athletes is unknown and studies of the athletic potential following Achilles tendon hemitenotomy are lacking.[49] A variety of procedures have been described for treatment including intramuscular lengthening of the gastrocnemius and soleus muscles, gastrocsoleus recession, and double- and triple Achilles hemitenotomy. Intramuscular lengthening of the gastrocnemius and soleus muscles involve varying the number of surgical incisions in the fascia overlying the two muscles, whereas gastrocsoleus recession involves division of the conjoined gastrocnemius aponeurosis-soleus fascia. Of those, Achilles tendon hemitenotomy has been reported to result in the greatest lengthening.[44]

i. Hemitenotomy

The triple hemitenotomy procedure for lengthening of the Achilles tendon was first described by Hoke in 1931.[50] In 1943, a double hemitenotomy technique was reported by White.[51] It has since been modified and used to successfully correct equinus deformity in humans.

Two or three offset hemitenotomies are performed across 50% of tendon width. Subsequently, the foot is manipulated in dorsiflexion until gaps are observed at the hemitenotomy site and slide lengthening of the tendon occurs (Figure 10).[46] Varying distances between hemitenotomies have been described, but commonly a 2.5-5 cm distance is reported.[45-47] Hoefnagles et al. (2007) used a triple Achilles hemitenotomy technique with the incisions created 2.5 cm apart in 20 cadaver specimens resulting in a 5% (1/20) tendon rupture rate following slide lengthening. An increased distance between hemitenotomies protects against total rupture of the tendon as a consequence of sliding.[45]

Postoperative care typically includes a walking cast or ankle-foot orthosis in neutral dorsiflexion for 6 weeks. Immediate weightbearing is encouraged to prevent muscle atrophy.[45]

A mean Achilles tendon lengthening of 30 mm and 32.9 mm has been reported using a double- or triple- hemitenotomy technique respectively.[44]

(A) **(B)**

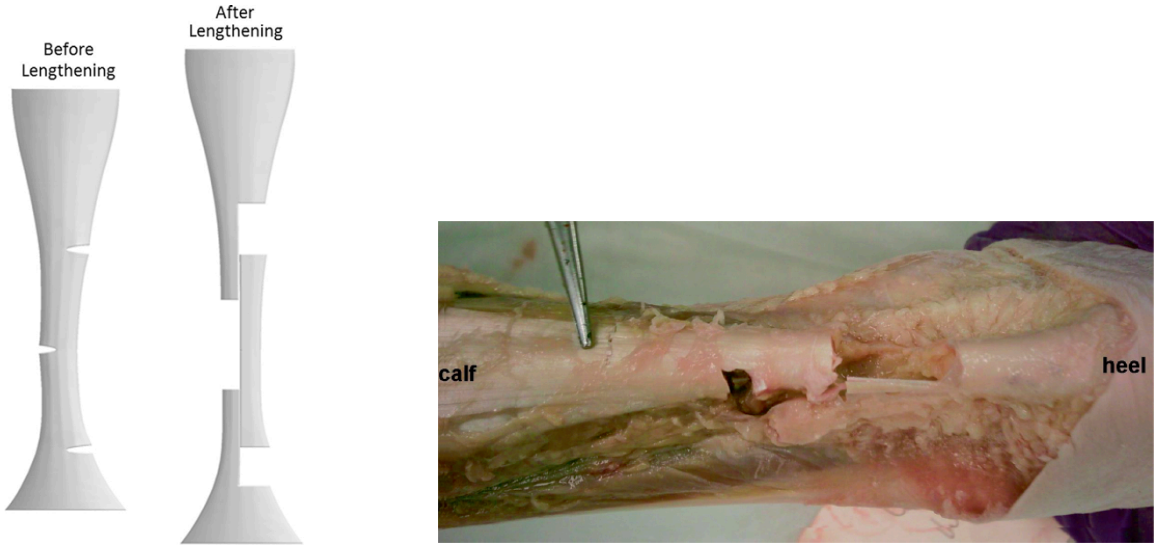


Figure 10. Triple hemitenotomy for Achilles lengthening. A, Illustration of the tendon lengthening process before and after dorsiflexion for slide lengthening, taken from Von Forell and Bowden 2014. B, Failure of tendon lengthening. The proximal cut (distal to scissor) did not slide, and the distal cut ruptured during the sliding procedure, taken from Hoefnagels et al. 2007.

Cadaveric studies have demonstrated that variations in the hemitenotomies significantly affect tendon lengthening.[47] When offset hemitenotomies are not adequately long to sever all the connecting fibers, weakening of the connecting fibers will need to take place before sliding occurs.[46] Salamon et al. (2006) evaluated the accuracy of three hemitenotomies for lengthening of the Achilles tendon in 15 cadavers. Overall surgeon accuracy was relatively high. The widths of the tendon at the level of hemitenotomy one, two, and three from distal to proximal were

found to average 61, 50, and 55%, respectively.[52] In contrast, Hoefnagles et al. (2007) found that one-third of hemitenotomies resulted in failure to lengthen the tendon due to inadequate transection.[47] Importantly, the fibers in the human Achilles tendon twist along their axis at an angle of 11 to 65 degrees.[53] When the twist of fibers is maximal, transverse hemitenotomies will fail to sever neighboring fibers and sliding will be incomplete or nonexistent (Figure 11).[45,47] In order to adjust for the varying degree of Achilles torsion, an open surgical approach can be used.[46]

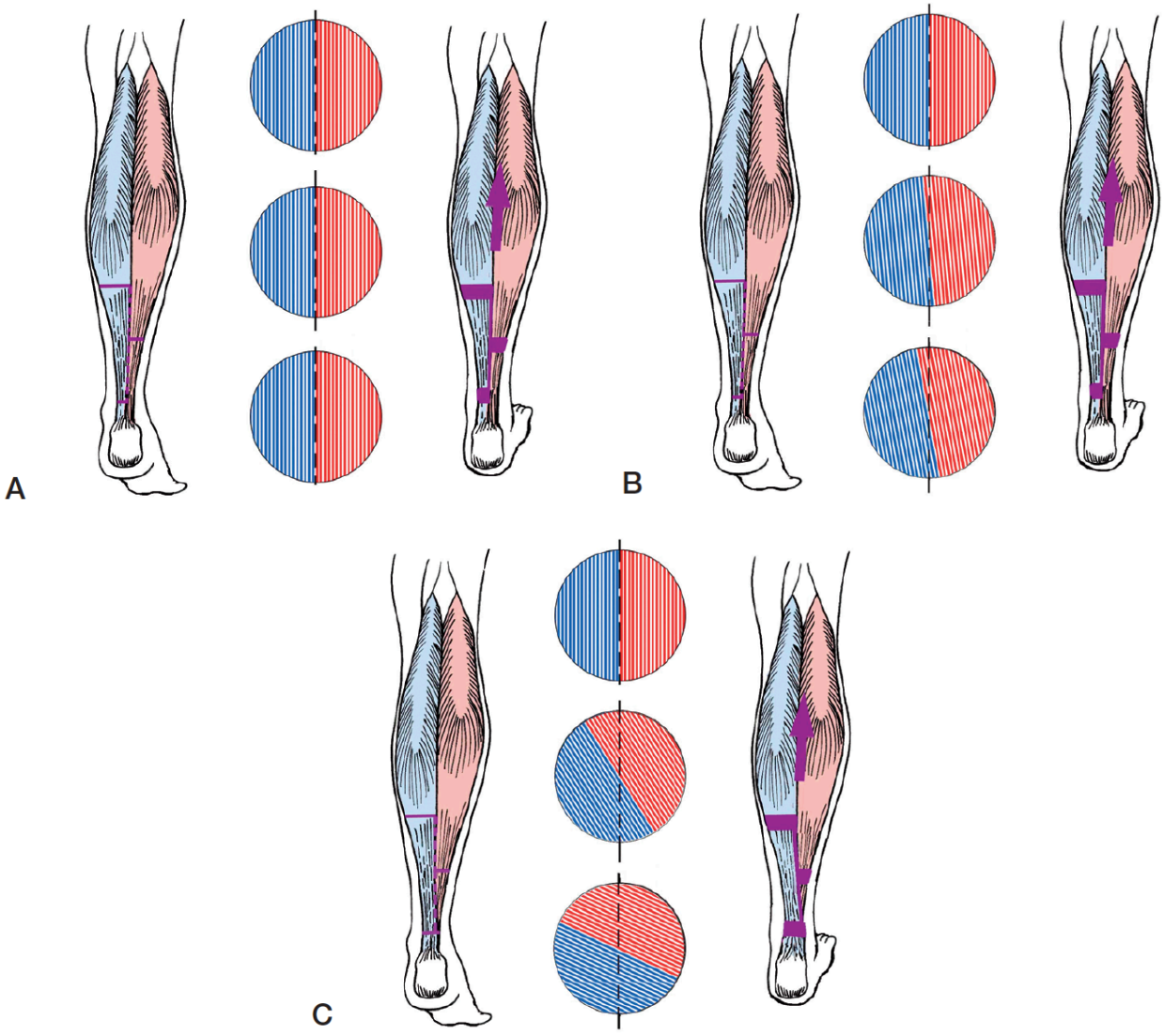


Figure 11. Illustration of triple-hemitenotomy Achilles tendon lengthening with cross sections. A, 0° torsion. Three hemisections will sever neighboring fibers and the fiber bundles will glide along each other. B, 11° torsion. The tendon fibers are still almost parallel and the fibers will still glide along each other after 3 hemisections. C, 65° torsion. The three hemisections do not sever neighboring fibers, and the gliding mechanism will fail or remain incomplete, taken from Hoefnagels et al. 2007.

e. Tendon healing

i. Healthy Tendon

Tendons, bands of parallel fibers, are made of highly aligned collagen fibrils. Fibers and tendon fibroblasts are organized into fascicles. Fascicles are bound by connective tissue sheaths, endotenon, to form a tendon (Figure 12). The tendon is covered by a thin sheet called epitenon. The endotenon and epitenon are largely responsible for the blood and nerve supply to the tendon. A tendon's primary role is mechanical translation of muscular contractions into joint movement. They prevent stress concentrations onto bone that would have resulted from direct muscle-to-bone contact and passively store and release energy during movement.[54]

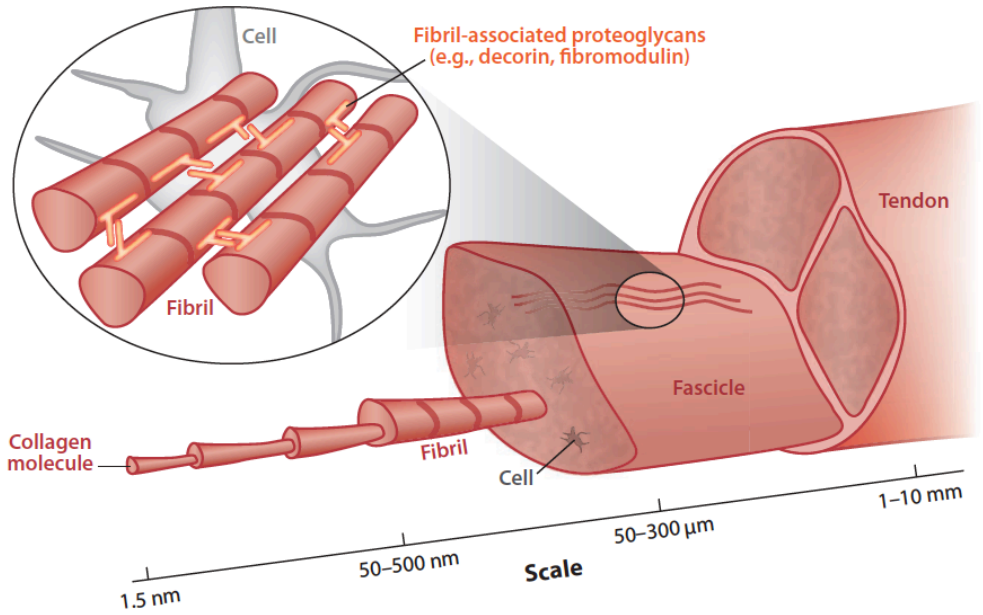


Figure 12. Hierarchial structure of a tendon, taken from Voleti et al. 2012.

Development of the tendon appears to occur in at least three steps; 1) immature fibril intermediates form as collagen molecules assemble extracellularly close to fibroblasts, 2) intermediates assemble resulting in linear fibril growth, 3) fibrils fuse laterally to generate large diameter fibrils as seen in fully developed tendons. Mature tendons consist of approximately 70% type I collagen in addition to proteoglycans, glycoproteins, and minor collagens.[54,55]

ii. Phases of tendon healing

Both extrinsic and intrinsic processes are thought to be involved in the healing process. Extrinsic repair results from cellular infiltration from the paratenon or tendon sheath. Intrinsic repair is a result of migration and proliferation of cells from the endotenon and epitenon into the site of injury. In cases of severe tendon disruption or transection, extrinsic repair may predominate. This can result in increased peritendinous adhesions with a negative impact on the gliding function of a tendon.[56]

The process of tendon healing is complex and occurs in three overlapping phases; 1) inflammatory, 2) proliferative, and 3) remodeling phase. The inflammatory phase lasts a few days and is characterized by haematoma formation, infiltration of inflammatory cells, fibrin deposition, fibroblasts and synthesis of Types I, III, and V collagen as well as non collagenous proteins. The proliferative or repair phase, initiated after approximately two days, is characterized by angiogenesis and fibroblast proliferation. Collagen fibers are abundant but have a random arrangement. In the remodelling phase, occuring 1-2 months after injury, a gradual decrease in cellularity is observed. Collagen I synthesis dominates and the extracellular matrix is more organized. The tendon matures for at least a year, but will never regain pre-injury morphologic characteristics nor mechanical properties.[54,55] In horses, collagen-fibril diameter and crimp remained reduced 14 months after experimetally induced tendon injury.[57]

The healing capacity of a tendon varies with location (anatomical, intrasynovial versus extrasynovial) and is likely a result of environmental, nutritional and functional differences.[55] Gap formation between repaired tendon edges as small as 1-2 mm have been associated with a poor functional outcome in people.[58] In dogs, strength and stiffness of severed tendons with gaps of three millimeters or less increased significantly with time.[40] To the authors knowledge, this has not been investigated in the horse.

f. Methods of evaluation of tendon biomechanics

Tendons possess a unique structure and composition and therefore show a characteristic mechanical behavior, reflected by a typical stress-strain curve consisting of four regions (Figure 13). First, there is a toe region resulting from tendon strain less than 2%. This region is a result of

stretching-out of crimped tendon fibrils as a result of mechanical loading of the tendon. Second, there is a linear region resulting from strain less than 4%, marking the physiological upper limit of strain in tendons. Collagen fibrils orient themselves in the direction of the tensile mechanical load. The slope of the linear region is called the Young's modulus of the tendon and represent the tendon stiffness. Yield and failure regions follow when the tendon stretches beyond its physiological limit, and intramolecular cross-links between collagen fibers fail. If micro-failure continues to accumulate, stiffness is reduced and the tendon begins to fail, resulting in irreversible plastic deformation. If the tendon stretches beyond 8-10% of its original length, macroscopic failure soon follows.[59] A stress-strain curve is useful in evaluating tensile deformation of a tendon. It is different from a load-displacement curve due to the fact that it accounts for the cross-sectional area of the tendon.

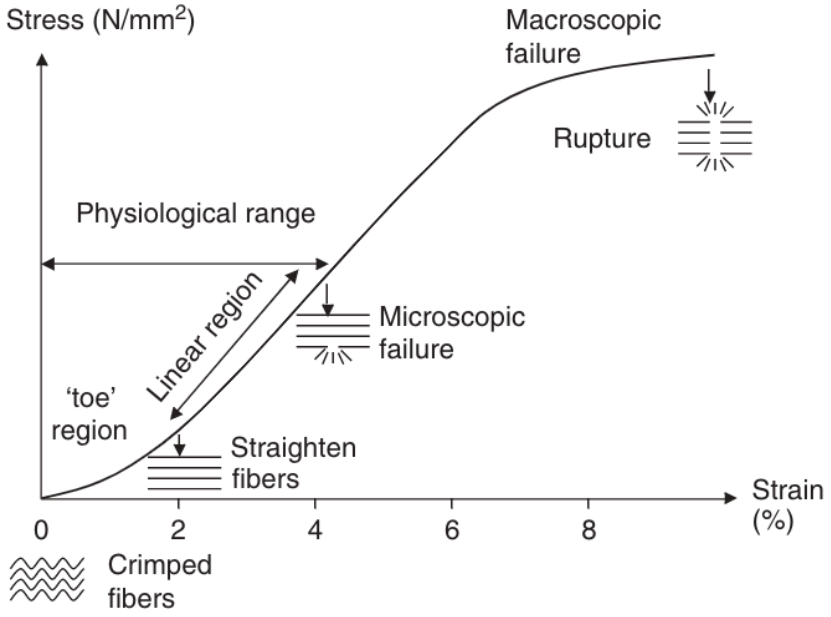


Figure 13. Tendon stress-strain curve, taken from Wang 2006.

The mechanical properties of tendons have been shown to be relatively uniform across a range of vertebrate animals.[60,61] A range of ex vivo mechanical tests have been carried out to determine tendons ultimate tensile strength and how repetitive loading affects their tensile strength.[60,62-64]

i. Ex vivo testing

The majority of our knowledge of biomechanical tendon properties result from isolated material testing. Traditionally, two methods have been used for biomechanical testing of tendons including a free-vibration method and tensile testing methodologies. The free-vibration method measures decay in oscillation amplitude after a specimen has been loaded.[65] Tensile testing methodologies are based on stretching a specimen using an external force while recording both specimen deformation and the applied force.[24] This latter methodology is generally preferable as it more accurately reflects in vivo loading of tendons.[60,66,67]

A tensile testing machine consists of an actuator and a load cell (Figure 14). The tendon is secured between a static (load cell) and a moving (actuator) clamp. The load cell measures the tension when the actuator is set in motion. The tensile lengthening is measured from displacement of the actuator (whole specimen) or by use of an extensometer (region of specimen).[68]

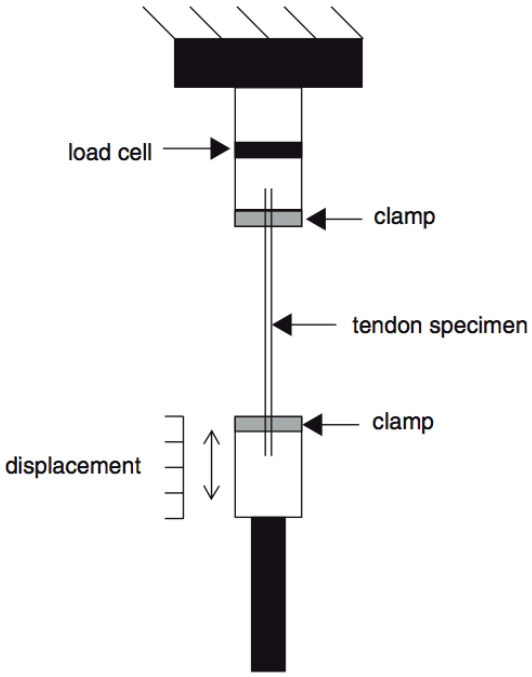


Figure 14. Tensile testing machine, taken from Tendon injuries,1st edition.

When elongation-to-failure is tested, the resulting stress-strain curve will vary with different cross-sectional areas and tendon lengths. If the testing is performed to measure intrinsic material properties rather than structural properties of the specimen, tendon force can be reduced to stress and tendon deformation to strain by normalization to the tendon's cross-sectional area and the tendon's original length, respectively, to account for these inter-specimen differences.[68]

ii. Biomechanical testing in horses

Mathematical models assuming biomechanical tendon properties related to elasticity, viscosity, plasticity, internal friction and strain history are typically employed for in vitro testing. It is generally assumed that the stress–strain curve represents the instantaneous response of tendon. Despite inherited flaws, in vitro testing methods have been shown to result in objective and repeatable data on the biomechanical properties of equine tendons.[69]

In vivo biomechanical testing of equine tendons has been performed using strain gauges, ground reaction force (kinetic), and motion analysis (kinematic).[24,70] Typically, strain and force transducers have been used for direct or indirect measurements of strain.[24,71,72]

g. Biomechanical properties of the equine DDFT

It has been demonstrated that the increase in DDFT load in a horse in motion occurs at a slower rate compared to the superficial digital flexor tendon (SDFT).[73] Normal DDFT and SDFT strains were evaluated *in vivo* in ponies using force plate analysis and kinematic motion analysis systems revealing a normal mean strain of 1.15% and 2-4% at a walk and 1.7% and 4-6% at a trot, respectively.[70] In an average-sized, approximately 500 kg horse, the tensile load on the DDFT at a walk is slightly over 400 kg. This exceeds the loads on the SDFT (363 kg) and the suspensory ligament (172 kg) at the walk. Loadbearing in the DDFT increases with speed (Table 1).[74]

TABLE 1: Tendon loading parameters measured over 4 consecutive contacts per *n* trials at each gait for 3 Thoroughbred horses

Tendon	*n*	Gait	Speed (m/s)	Strain (%)	Stress (MPa)	Force (N)
DDF	9	Walk	1.7	0.8 ± 0.2	10.9 ± 0.23	1058 ± 22.7
	6	Trot	4.1	1.2 ± 0.3	15.5 ± 0.44	1521 ± 39.7
	3	Canter	7.0	2.0 ± 0.1	25.1 ± 0.99	2446 ± 95.9
SDF	10	Walk	1.7	3.6 ± 0.1	45.7 ± 0.98	3802 ± 81.6
	7	Trot	4.1	5.6 ± 0.2	71.8 ± 1.96	5971 ± 163
	3	Canter	7.0	4.8 ± 0.2	61.7 ± 2.81	5136 ± 233

Values are grand mean ± s.e. and represent average, not peak, loading parameters; DDF = deep digital flexor tendon; SDF = superficial digital flexor tendon; MPa = megapascals; N = Newtons.

Taken from Butcher et al. 2007.

Lochner et al. (1980) reported that a DDFT can be loaded with 1020 kg without failing[24] and it has been demonstrated that equine flexor tendons can extend 10- 12% of their original length before failure occurs.[69]

The DDFT load can be modified by veterinary or farrier interventions. Buchner at al. (1996) demonstrated that desmotomy of the ALDDFT results in increased load onto the SDFT and the DDFT.[41] On the contrary, application of a shoe with a heel wedge or an egg bar shoe, results in reduced strain in the DDFT and ALDDFT.[70] Because of the ability to modify the DDFT load and the potential for redistribution of loads with veterinary intervention to other components of the equine distal limb, in situ testing of limbs that preserve muscular attachments of the DDFT may help improve our understanding.[75]

II. Objective

Currently, there is no universaly accepted surgical therapy for horses with chronic, refractory laminitis. Approximately 20% of horses with lamintits suffer morbidity affecting the horse's use and function.[5] Deep digital flexor tenotomy provides a fast way to counteract rotational forces on P3, restoring perfusion and potentially lamellar tissue to the dorsal hoof.[76] Deep digital flexor tenotomy is considered the current standard surgical treatment for horses with chronic laminitis, however, horses undergoing this treatment have a poor prognosis for return to work leading investigations into alternative surgical treatment methods.[28,30] However, none have gained widespread industry acceptance.

A hemitenotomy lengthening technique has been used in the human orthopedic field for several decades,[50] but there are no studies investigating such technique in the horse to date.

The objective of this study was to develop a double hemitenotomy (DHT) technique to lengthen the equine DDFT. Models were used to; 1) evaluate the DDFT tensile strength following DHT; 2) evaluate the DDFT lengthening following DHT compared to complete tenotomy (CT) and; 3) compare the reduction in DDFT load following DHT to following CT.

a. Hypothesis

We hypothesized that DHT would result in less than 3 mm lengthening of the DDFT and at least half the load reduction of CT.

III. Methods and Materials

a. Preliminary study

A preliminary study was performed by the present research group to assess the effects of differences in hemitenotomies. Equine cadaver limbs were severed at mid tibia (part 1) or distal humerus (part 2) and freshly frozen at -16° C. Prior to testing, the limbs were thawed at room temperature overnight, and prepared and tested the following day. The plantar or palmar distal limb was evaluated using ultrasound imaging. The distal limb was clipped and alcohol applied. A linear array transducer (Canon Aplio i700 Ultrasound Machine, 7-18 MHz) was used to confirm that the DDFT was structurally normal.

Part 1: Nine DDFTs were isolated via transection at the level of the tarsometatarsal joint proximally and pastern distally. Hemitenotomies were performed at mid-cannon bone level in various combinations (mediolateral, lateromedial, dorsoplantar and plantarodorsal) with the tendons in an Instron® materials testing machine. They differed by distances apart (1-3 cm), widths of the DDFTs (25-50%) and number of hemitenotomies (2-4). The Instron® software program measured the reduction in load (28.5-59%), the load to tendon failure (116-476 kg) and the DDFT lengthening (3-3.5 mm). A larger distance between, and fewer hemitenotomies appeared to reduce the risk of tendon failure and result in similar load reduction compared to using >2 hemitenotomies, supporting use of 2 hemitenotomies with a 3 cm distance for future investigation.

Part 2: Sixteen forelimbs, 6 including mid-radius and 10 complete radius, were placed vertically in an Instron® materials testing machine and double hemitenotomies (3 cm distance) resulted in a 22.4% to 36.4% reduction in load (Figure 15) and a 5 to 10 mm tendon lengthening

compared to pre-procedure in limbs with intact muscular attachments (including complete radius). Complete DDF tenotomy performed in the contralateral limbs resulted in a 65.7% to 80% reduction in load compared to pre-procedure. Hemitenotomies resulted in 47.6% to 71% less load reduction than complete DDF tenotomies. In conclusion, the developed double hemitenotomy technique appeared clinically applicable and the research team believed that the resulting reduction could be clinically significant in affected horses where distal phalanx rotation has resulted in an already significantly reduced DDFT force.[77]

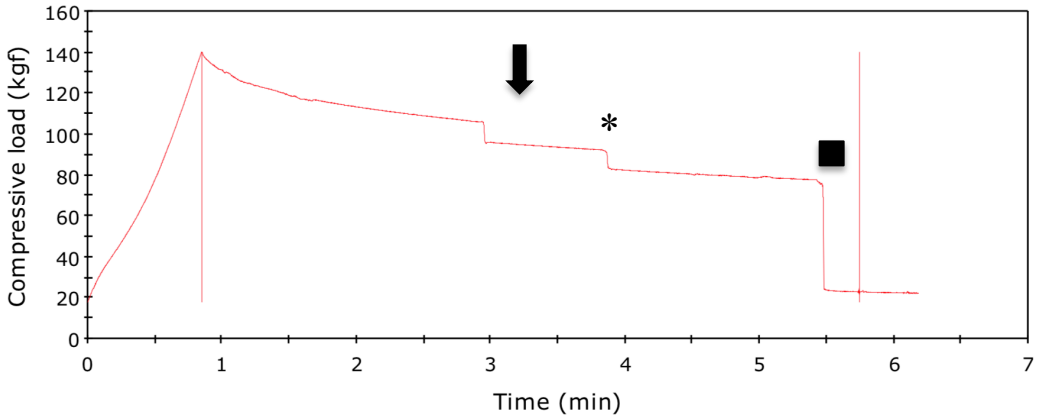


Figure 15: Illustration of load reduction (31%) in one forelimb with each of the two hemi-tenotomies. Test started at 140kg compressive load (500kg body weight x 0.28). The first (arrow) and second (asterix) hemitenotomies were performed following relaxation of the tendon fibers. A complete tenotomy was finally performed (square) in order to document the load of the musculoskeletal structures themselves.

b. Horses

The study was divided into two parts using cadaveric models. A power analysis based on a pilot study by the authors indicated that a minimum of 30 isolated tendons and 16 intact cadaveric forelimbs were needed to demonstrate a statistically significant difference in load reduction

between treatment and control groups, assuming a standard deviation of 3 mm tendon lengthening and 50% load reduction with a significance level of 5% and power set at 80%. All horses were humanely euthanized for another study in accordance with the Auburn University´s Institutional Animal Care and Use Committee Protocol no. 2017-3170 for reasons other than laminitis or DDF tendonitis. Age (year), sex, breed, and weight (kg) were recorded for each horse.

c. Specimen preparation

Paired limbs from the same horse were severed at mid radius (part 1) or distal humerus (part 2) and freshly frozen at -16°C. Prior to testing, the limbs were thawed at room temperature overnight, prepared and tested the following day. The limbs (part 1) were randomly assigned to either treatment (DHT) or reference (intact tendons) groups.

In preparation for ultrasound evaluation, the distal limb was clipped and alcohol applied. A linear array transducer (Canon Aplio i700 Ultrasound Machine, 7-18 MHz) was used to confirm that the DDFT was structurally normal as assessed by ultrasonography.

For part 1, the DDFT (n=30) was dissected from each limb and removed by transecting at the level of the carpus proximally and pastern distally. The tendons were rinsed with 0.9% NaCl, sealed in plastic and immediately transported for testing.

For part 2, cadaveric limbs (n=16) were disarticulated at the cubital joint to preserve the radial and ulnar muscular attachments of the DDFT. The skin was removed and the limbs sealed in plastic bags and immediately transported for testing.

In a pilot study performed by the authors prior to the experimental study, palmarodorsal and dorsopalmar hemitenotomies 3 cm apart resulted in less tendon failure and more consistent tendon lengthening and load reduction compared to other orientations and distances between

hemitenotomy placement. Therefore, a proximal palmarodorsal and distal dorsopalmar hemitenotomy 3 cm apart was used for this study (Figure 16).

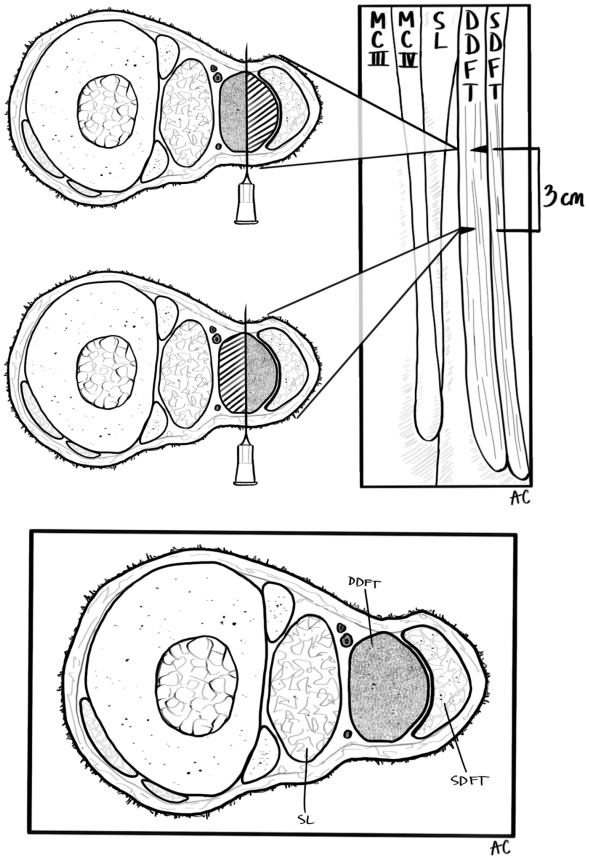


Figure 16. Lateral and cross-sectional illustrations of needle placement at 50% DDFT thickness at the intended hemitenotomy sites (3 cm apart) and subsequent gap formation following hemitenotomies at the level of mid-metacarpus. Image provided by A. Caldwell.

d. Part 1: Evaluation of hemitenotomy in isolated DDFTs under tensile load

Thirty DDFTs were collected from 15 horses (12 mares and 3 geldings). There were 4 American Quarter horses, 3 Thoroughbreds, 2 Tennessee Walking horses, 1 American Paint horse, 1 Haflinger, 1 Shetland Pony, 1 Paso Fino, and 1 Draft horse.

Biomechanical testing was performed using the Instron® 5566 machine (Norwood, MA). To ensure exact measurement and to prevent tendon slippage, tendons were fixed within a cryo-clamp device system for testing as described by Domnick et al. (2016).[80] The tendons were conditioned for testing by applying a 90 kg load followed by relaxing the tendon. The testing was started and the DDFT was again loaded with 90 kg, corresponding to the approximate tendon load in a standard 450-500 kg horse during normal stance.[25] 20 gauge, 1.5 inch needles were placed into all tendons (n=30) to mark a 5 cm distance at mid cannon bone level. These needles served as distance markers and the length between them was measured continuously during testing. Both hemitenotomies were performed between these needles in the treatment group (n=15). Tendons for the reference group (n=15) were left intact. In tendons for hemitenotomy, a digital slide caliper was used to measure the DDFT diameter and 50% of the diameter was designated using two 20 gauge, 1.5 inch needles 3 cm apart, at the level of the intended hemitenotomies. Level with these needles, one proximal palmar and one distal dorsal hemitenotomy incorporating 50% of the tendon thickness were horizontally created using a Lichty teat knife (Jorgensen labs, US, Figure 17) within each DDFT just distal to the insertion of the accessory ligament of the DDFT (palmar hemitenotomy incision) and 3 cm further distal (dorsal hemitenotomy incision). Half thickness tenotomies were ensured by transecting the tendon until the needles were contacted (Figure 18). Tendon lengthening was measured between the two outermost needles using a digital slide caliper. Testing was performed at a speed of 10

mm/min and the load was increased until tendon failure or a maximum load of 500 kg was reached following both hemitenotomies in DHT tendons, or needle placement in reference tendons. Elongation and load were continuously recorded by the Instron Universal Testing Machine during testing and following the first and second hemitenotomy. Data recording was performed using the material testing software Blue Hill® Universal (Version 4.13, Instron®, USA). Tendon failure was defined as a marked, sudden drop in load and the value at failure was recorded. If failure was not achieved, a value of 500 kg was used.

Figure 17. Lichty teat knife used for hemitenotomies (part 1 and 2) and complete tenotomies (part 2).

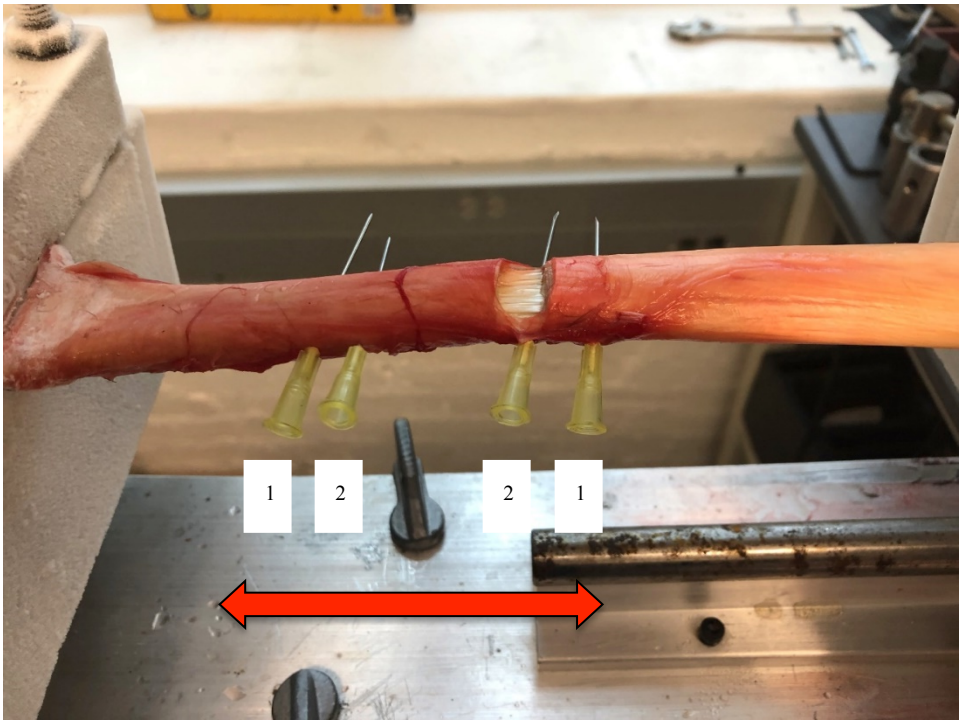


Figure 18. Isolated DDFT mounted in a material testing apparatus using custom-engineered cryoclamps to prevent tendon slippage. The 2 outermost needles (1) were used for measuring lengthening following hemitenotomy incisions. The 2 innermost needles (2) were placed 3 cm apart at a 50% tendon thickness at the hemitenotomy sites. A tensile (arrow) load was applied until tendon failure or a maximum of 500 kg load was reached.

e. Part 2: Evaluation of hemitenotomy in DDFs in situ under compressive load

Sixteen complete forelimbs were collected from 8 horses (3 mares and 5 geldings). There were 3 American Quarter horses, 3 Thoroughbreds, 1 Tennessee Walking horse, and 1 mixed-breed pony.

Biomechanical testing was performed using the Instron® 5566 machine (Norwood, MA).

Limbs were placed vertically with the carpus in extension in the Instron® (Figure 19). A bolt was placed in the Instron in front of the hoof to maintain carpal extension and prevent the hoof from slipping with increased loads or tenotomies. The limbs were conditioned for testing by applying a 500 kg load followed by relaxing the limb. Needle placement and double hemitenotomies were performed through the paratenon (n=16) in a manner similar to that described for part 1. The SDFT, suspensory ligament and extensor tendons were left intact. The limb was loaded to 28% of the horses' weight to approximate the load of a single forelimb in stance.[25] Elongation and load were recorded by the Instron Universal Testing Machine and a digital slide caliper before and following the first and second hemitenotomy. Data recording was performed using the material testing software Blue Hill® Universal (Version 4.13, Instron®, USA).

Figure 19. Complete forelimb mounted in an Instron Testing Machine. A compressive load (arrow) corresponding to in vivo conditions was applied. Hemitenotomies were created and measurements recorded. A complete tenotomy was then performed, and length and load reduction recorded.

f. Statistical analyses

Statistical analysis was performed with commercial software to collate (Excel; Microsoft Corporation, Redmond, Washington) and analyze (SPSS 26.0; IBM Corporation, Armonk, New York) data. Descriptive data were generated for both parts. Cohen's d was calculated for paired data by dividing the average difference by the standard deviation of the difference.

Data normality was determined by visual inspection of the Q-Q plots. Relationships between age or weight and load or length were tested using Spearman's correlation.

For part 1, length (millimeters) and load (kilograms) changes were compared between baseline (intact tendons) and after hemitenotomy using the Wilcoxon Signed Ranks test. The

Wilcoxon Signed Ranks test was used to compare weight (kilograms) at failure between the reference (intact tendons) and treatment (hemitonotomised tendons) groups.

For part 2, data from each limb was averaged. The Friedman test with Dunn-Bonferroni test for post-hoc testing was performed to analyze tendon lengthening, load, and change in load between baseline, first hemitenotomy, second hemitenotomy, and CT. Statistical significance was set at $P<0.05$.

IV. Results

a. Part 1

Age and weight were normally distributed. All other variables were non-normally distributed. The median and mean age were 11.1 and 11.8±7.5 yrs, respectively (range, 1 to 32 yrs). The mean body weight was 442 kg (range, 177 to 548 kg) (Table 2).

Age was not significantly associated with length or load changes (p=0.27 and p=0.5, respectively). There was no significant influence of body weight on length or load changes (P=0.1 and P=0.8, respectively).

Twelve of 15 (80%) isolated DHT tendons failed whereas no intact tendons failed at 500 kg load (P= 0.0001). The twelve DHT tendons failed at a median tensile load of 196 kg (interquartile range 56.5-274) and four of the intact tendons slipped at a median tensile load of 403 kg (interquartile range 360-451; P=0.0024). The effect size of tenotomy for tensile load at failure or slip was 1.26. Total tendon lengthening was significant after DHT (1.9 mm, inter-quartile range 1.25-2.1 mm) compared to the length of intact DDFTs (P=0.001). The effect size of tenotomy was 2.35 for length.

Table 2. Case details for study subjects of part 1.

Specimen	Weight (kg)	Age (yrs)	Breed	Sex	Hemitenotomy	Failure (kg)
1	500	12	TB	G	1	340
2	500	12	TB	G	0	-
3	513	12	TB	M	1	215
4	513	12	TB	M	0	-
5	454	11	APH	M	1	283
6	454	11	APH	M	0	-
7	500	16	AQH	M	1	265
8	500	16	AQH	M	0	-
9	431	32	WB	M	1	374
10	431	32	WB	M	0	472
11	500	14	TWH	M	1	55
12	500	14	TWH	M	0	-
13	454	14	AQH	G	1	-
14	454	14	AQH	G	0	-
15	520	11	Haflinger	M	1	-
16	520	11	Haflinger	M	0	430
17	510	10	TB	M	1	300
18	510	10	TB	M	0	344
19	548	10	AQH	M	1	223
20	548	10	AQH	M	0	-
21	520	7	TWH	G	1	58
22	520	7	TWH	G	0	-
23	375	19	AQH	M	1	-
24	375	19	AQH	M	0	-
25	177	2	Shetland	M	1	155
26	177	2	Shetland	M	0	375
27	380	1	Draft	M	1	144
28	380	1	Draft	M	0	-
29	250	28	PF	M	1	195
30	250	28	PF	M	0	-

M, mare; G, gelding; APH, American Paint Horse; AQH, American Quarter Horse; Draft, Draft Horse; PF, Paso Fino; Shetland, Shetland pony; TB, Thoroughbred; TWH, Tennessee Walking Horse; WB, Warmblood yr, year; kg, kilograms. The mean± SD weight, age and load at failure was 442±104.7 kg, 11.8±7.5 yrs and 173.8± 127.6 kg, respectively.

b. Part 2

All variables were non-normally distributed. The median age was 13.8 yrs (range, 5 to 18 yrs). The median body weight was 498 kg (range, 363 to 850 kg) (Table 3).

Age was not significantly associated with length or load changes (P>0.39, P>0.47, respectively). Body weight was significantly associated with load at baseline (P=0.003), but not load after any tenotomies. Body weight was not associated with tendon lengthening (P>0.49).

There was a statistically significant difference in tendon length (P=0.0001). There was a significant difference between baseline and the second hemitenotomy, baseline and CT, and the first hemitenotomy and CT (P=0.002, P=0.0001, and P=0.002 respectively). The effect sizes were 2.0, 3.1, and 3.4, respectively. Tendon lengthening after CT (9.95 mm, inter-quartile range 10.125-13.975 mm) was greater than lengthening recorded after both the first (0.65 mm, inter-quartile range 0.3-1.08 mm, P=0.008) and second (3.05 mm,inter-quartile range 0.78-1.7 mm, P=0.008) hemitenotomy (Figure 20A). DDFT failure was not observed (i.e. there was no sudden reduction in load on the Instron) after DHT in any of the limbs prior to CT.

There was a statistically significant difference in load (P=0.001). There was a significant difference between baseline and the second hemitenotomy, and between the first and second hemitenotomy (P=0.001 and P=0.022, respectively). The effect sizes were 1.8 and 2.3, respectively. The differences in load were statistically significant (P=0.008). The change in load load between baseline and the first hemitenotomy was smaller than the change load between the second hemitenotomy and the CT (P=0.012). There was no significant difference in load reduction following DHT and CT (P=1.0) (Figure 20B).

Table 3. Case details for study subjects of part 2. All had double DDF hemitenotomy followed by complete tenotomy.

Specimen	Weight (kg)	Age (yrs)	Breed	Sex
1	416	11	TB	G
2	416	11	TB	G
3	480	5	TB	G
4	480	5	TB	G
5	850	18	TWH	M
6	850	18	TWH	M
7	503	21	AQH	M
8	503	21	AQH	M
9	363	15	Mix pony	M
10	363	15	Mix pony	M
11	461	11	AQH	G
12	461	11	AQH	G
13	454	13	AQH	G
14	454	13	AQH	G
15	454	14	TB	G
16	454	14	TB	G

M, mare; G, gelding; AQH, American Quarter Horse; TB, Thoroughbred; TWH, Tennessee Walking Horse; yr, year; kg, kilograms. The mean± SD weight and age was 498±143.5 kg, 13.8± 4.6 yrs, respectively.

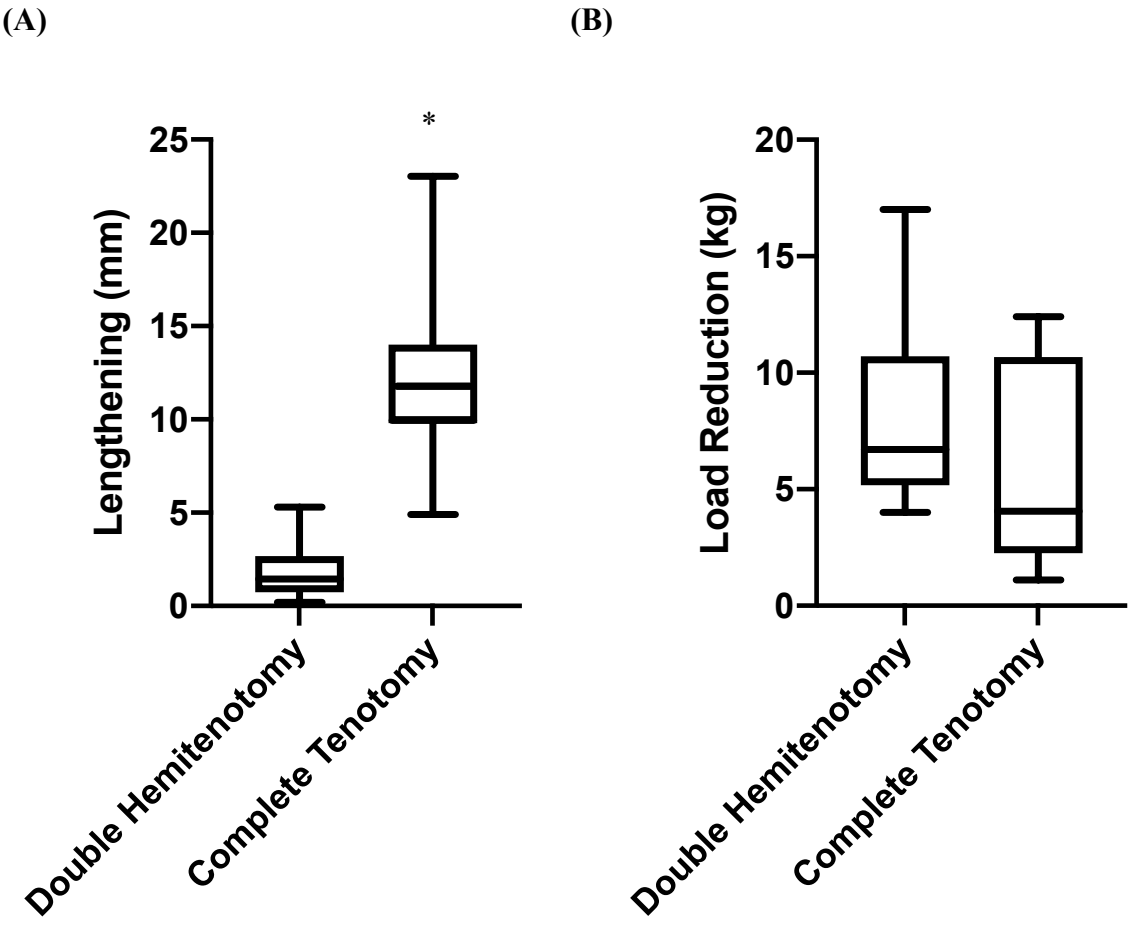


Figure 20. Lengthening of tendon (A) and load reduction (B) in the deep digital flexor tendon, stratified by tenotomy method (double hemitenotomy (n=16) versus complete tenotomy (n=16), part 2). The asterisk (*) indicates a statistically significant difference (P=0.008). There was no difference in load reduction between the DHT and the CT (P=1.0).

V. Discussion

In the present report we describe a novel DHT technique for lengthening and load reduction of the DDFT. This technique could be considered an alternative treatment to CT in cases of refractory laminitis, but in vivo testing is still needed. Unlike CT, the longitudinal integrity of the DDFT is preserved instead of transected with this technique, thereby maintaining part of the tendons function.

The research results found that our hypotheses could be accepted. The increases in tendon lengthening were found to be significant and overall the changes were less than 3 mm per hemitenotomy. More importantly, the results of this cadaveric study indicated that the reduction in load of the DDFT following DHT was comparable to the traditional method of DDFT force elimination, CT.

Decreases in load of the DDFT were only comparable to CT after the second hemitenotomy, supporting use of a DHT technique. Lengthening of the tendon occurs when discontinuous fibrils slide past one another throughout the length of the tendon.[47,78] A gap (partial thickness defect) can be observed in longitudinal direction.[79] For this mechanism to occur, interfibrillar bonds need to weaken by transection of neighboring tendon fibers parallel in the sagittal axis.[47] Previous studies have emphasized the importance of transection of 50% of the tendon diameter for the sliding mechanism to be successful.[47,52] Using needles to mark 50% of the tendon thickness, resulted in grossly accurate half-thickness hemitenotomies in this study.

In humans, the fibers of the Achilles tendon twist 11 to 65 degrees, therefore, off-set hemitenotomies are more accurately performed through an open approach to the tendon to ensure that the twisting of the tendon fibers does not prevent appropriate placement of the

hemitenotomy.[47,53] In horses, however, the DDFT has a uniform linear fiber alignment that runs parallel to the long axis of the metacarpus.[80] In a pilot study performed by the authors prior to the experimental study, palmarodorsal and dorsopalmar hemitenotomies 3 cm apart resulted in less tendon failure and more consistent tendon lengthening and load reduction compared to other orientations and distances between hemitenotomy placement. Therefore, a proximal palmarodorsal and distal dorsopalmar hemitenotomy 3 cm apart was used for this study.

The ALDDFT and digital flexor tendon sheath were avoided in all limbs using a 3 cm distance between the proximal and distal hemitenotomy at the mid-level of the metacarpus. The recommended distance between hemitenotomies in humans, varies according to the severity of the equinus deformity, but is typically between 3-5 cm.[45] Generally, the greater the distance between hemitenotomies, the greater the lengthening of the tendon.[81] Additionally, the risk for total rupture of the hemitenotomized Achilles tendon decreases with increased distances between hemitenotomies.[45] DHT reduced tensile strength of the equine DDFT, but failure of the hemitenotomized tendon was not observed in the intact limbs. We speculate that this is due to re-distribution of load onto the remaining tendons and ligaments within the limb. ALDDFT desmotomy has previously been reported to result in increased loading of the SDFT and DDFT during stance.[41]

A blunt-pointed bistoury knife was used for hemitenotomies instead of a scalpel for clinical applicability. Advantages include a protected blade to protect surrounding tissues. The instrument is feasible and common to large animal surgeons.

After each hemitenotomy, a gap of 2 mm or less was observed between incised tendon ends. We speculate that the resulting small gap may compare favorable to the significantly larger CT gap for subsequent tendon healing and function. Flexor tendon gap sizes as small as 3 mm have

been associated with adhesion formation and a poor functional outcome in dogs and humans.[40,82] Recently, one-year post-operative magnetic resonance imaging (MRI) revealed complete Achilles tendon tissue repair following triple hemitenotomy for tendon contracture in humans.[52]

a. Limitations

Limitations of this study include use of cadaveric models that were modified for mechanical testing. The experimental models were, however, successfully used to obtain preliminary and experimental data for the present study. Additionally, load contributions of the DDFT, SDFT and suspensory ligament measured using these models in a preliminary study correlated with previous studies.[24] The loading conditions may not accurately reflect dynamic forces placed on the DDFT during in vivo conditions (stance and locomotion). Finally, reports defining the necessary size of load reduction on the DDFT to eliminate its rotational force on P3 are lacking. A study of the strength of the laminar junction of horses with chronic laminitis refractory to treatment was only 42% compared to that of normal horses.[15] However, the difference was not statistically significant. Accordingly, this study compared the results from the hemitenotomy to the standard CT technique.

VI. Conclusions

This research aimed to evaluate double hemitenotomy of the DDFT as an alternative surgical treatment method for horses with refractory laminitis. This ex vivo study identified a similar load reduction after DHT and CT. Therefore, results of this study support further investigation into the use of double hemitenotomy and it's effects on tendon healing in the clinical setting. Experimental in vivo studies are needed prior to using this technique to treat clinical cases.

Based on a 2-part biomechanical ex vivo study, it can be concluded that double hemitenotomy results in smaller lengthening, but comparable load reduction of the DDFT to complete tenotomy. It can therefore be concluded that hemitenotomy may be useful as an alternative technique for surgical management of horses with laminitis. However, further evaluation of the double DDF hemitenotomy technique in vivo, such as in a randomized, prospective clinical trial in patients with refractory laminitis is warranted. Such investigation could include measuring load reduction with strain gauges in the DDFT comparing the results of the DHT technique to complete tenotomy in healthy and laminitic horses.

VII. References

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VIII. Appendix

a. Forces on P3

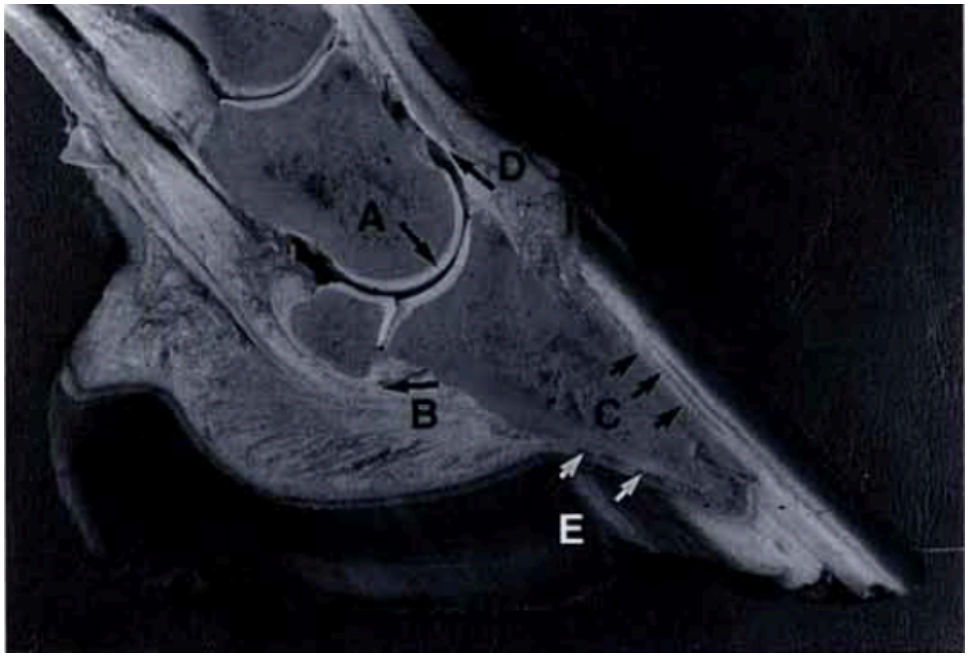


Figure 1. The five main forces acting on a normal P3 include compressive load as a result of the weight of the horse (A), tensile load as a result of the deep digital flexor tendon attaching on the palmar surface of the P3 (B), tensile loads generated by the laminar interface (C), tensile loads as a result of the common digital extensor tendon attaching on the extensor process of the P3 (D), and compressive loads against the sole (E), taken from Hood 1999.

b. Hoof wall

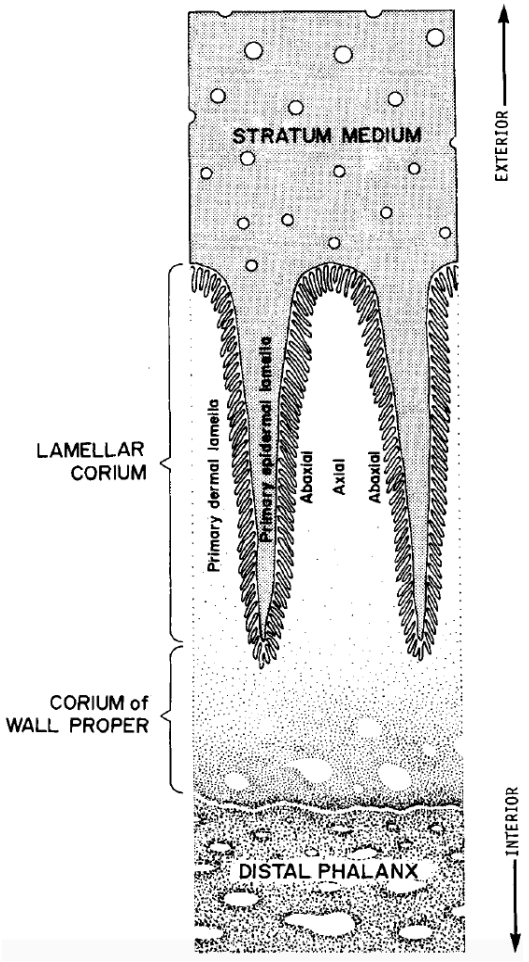
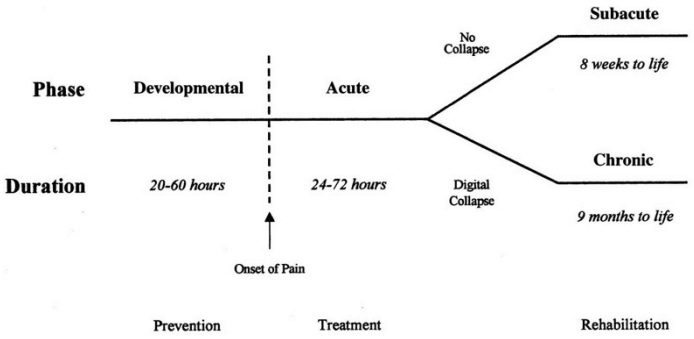


Figure 2. Schematic illustration of a cross section through the hoof wall showing primary and secondary lamellae. The lamellar corium forms the deepest layer of the hoof wall, also called stratum internum, taken from Mishra and Leach 1983.

c. Laminitis phases and associated changes

(A)



(B)

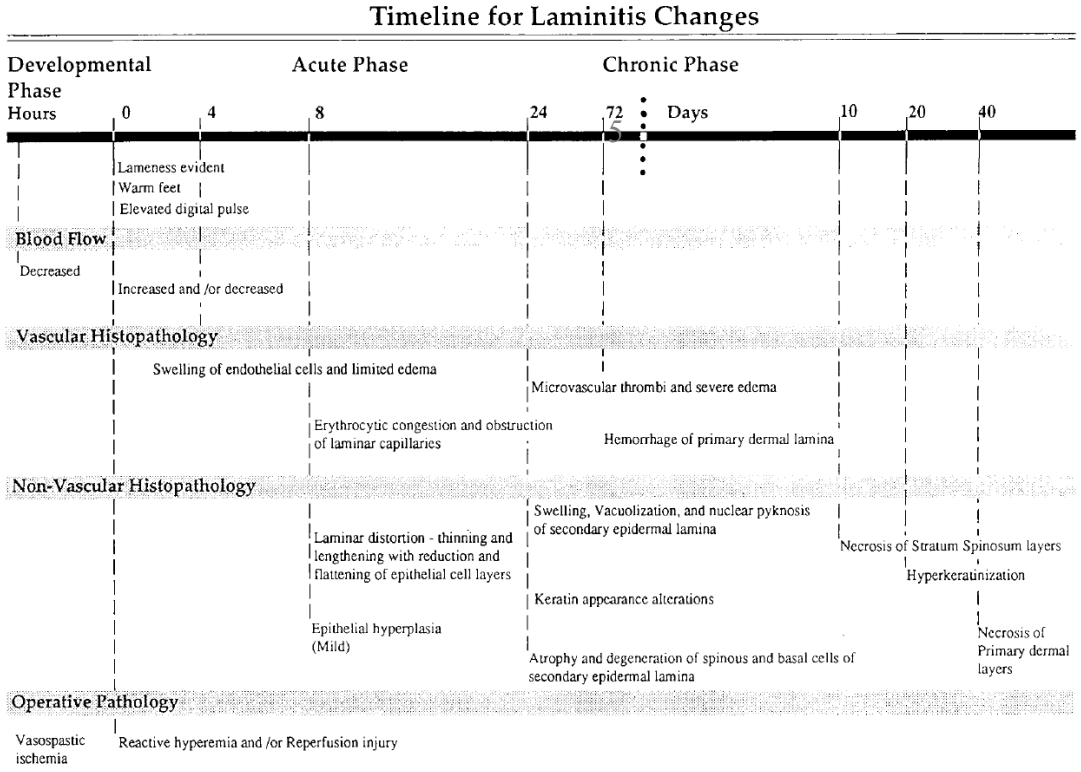


Figure 3. Laminitic phases. A, Duration and therapeutic goals, taken from Hood 1999. B, Clinical, hemodynamic, and histopathologic changes occurring in horses with laminitis, taken from Hood 1993.

d. P3 displacement

(A) **(B)**

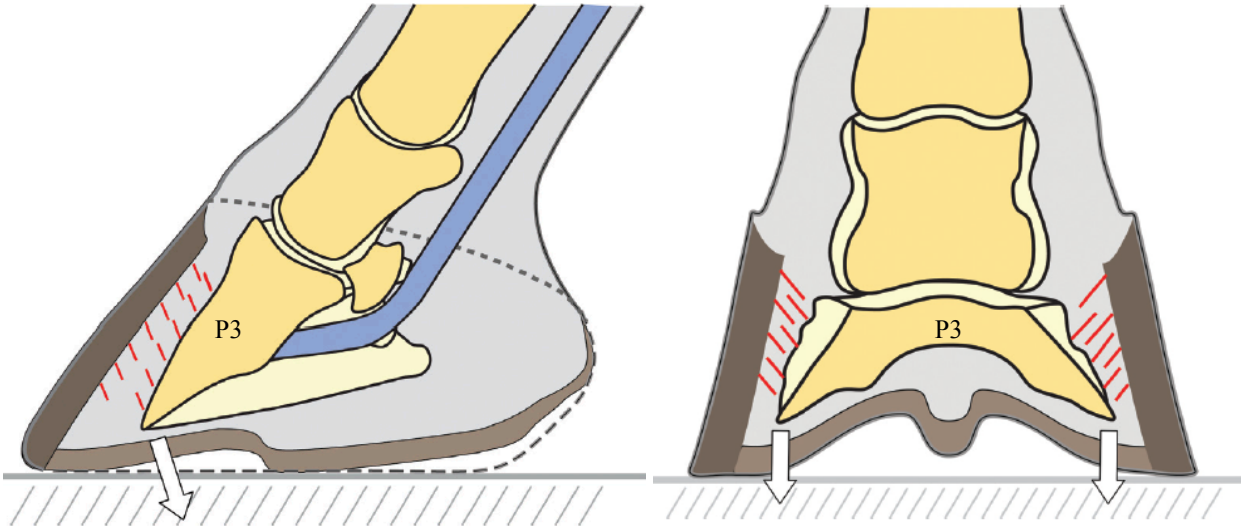


Figure 4. Displacement of the third phalanx (P3) within the hoof capsule. A, In horses with rotational displacement failure of the lamellae supporting the P3 is greatest dorsally, while the heels may be relatively unaffected B, In horses with vertical displacement disruption of the lamellae supporting the distal phalanx appears to occur around the circumference of the foot. The deep digital flexor tendon is outlined in blue and the hoof capsule in brown, taken from Equine Laminitis, 1st edition.

e. Forelimb flexor tendon anatomy

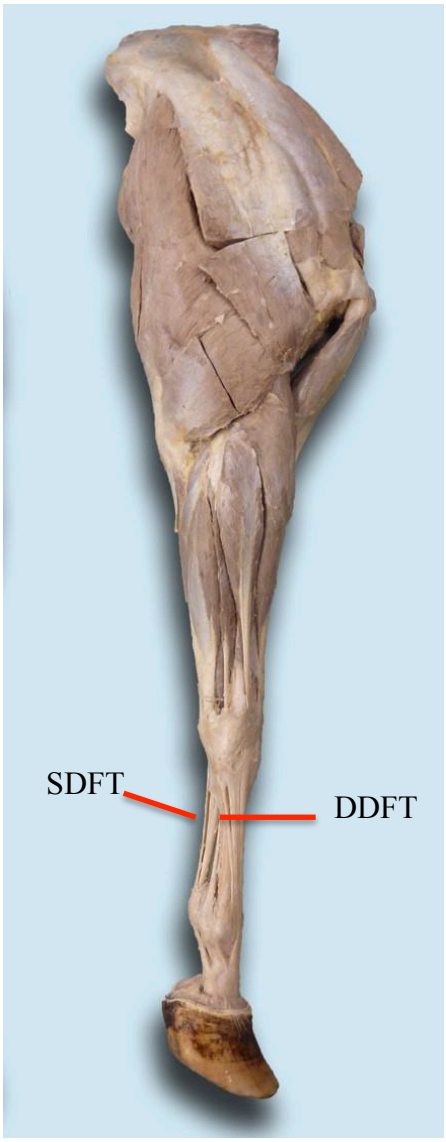


Figure 5. Schematic of the muscles and tendons of the forelimb of the horse. Image provided by R. Wilhite.

f. Current surgical treatments of laminitis

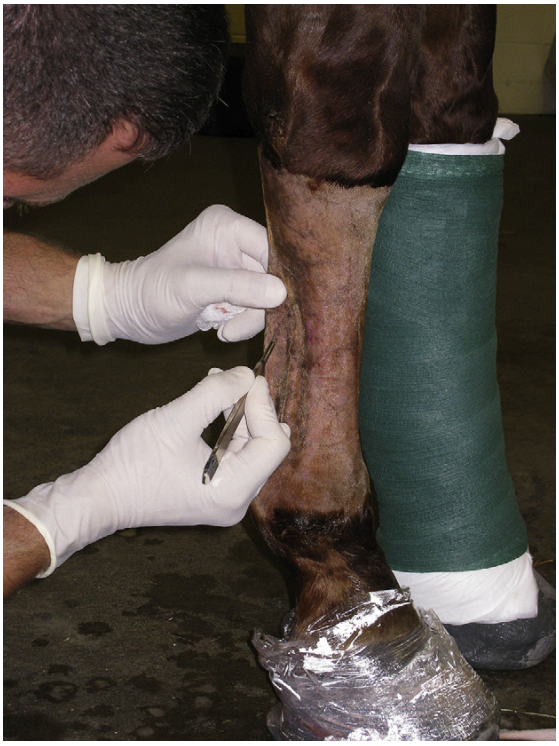


Figure 6. DDFT tenotomy performed at the mid metacarpal region in the standing and sedated horse following regional anesthesia and realignment shoeing, taken from Morrison 2011.

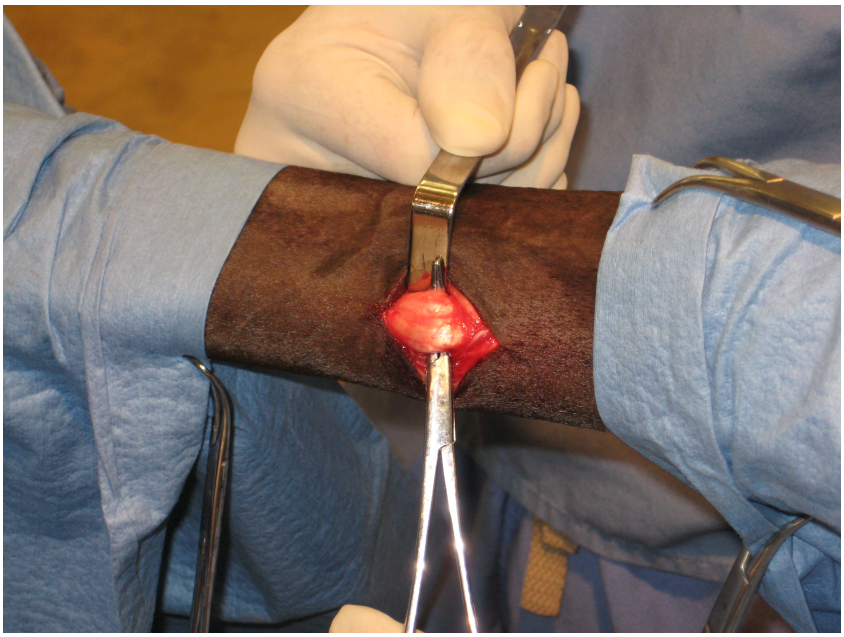


Figure 7. The ALDDFT is isolated and exteriorised above artery forceps prior to sharp complete horizontal transection of the ligament. Image provided by F. Caldwell.

Figure 8. Photograph showing a 5.5-mm-diameter cortical bone screw with a washer aseptically placed through the midline of the dorsal hoof wall into P3 two cm distal to the coronary band, taken from Carmalt et al. 2019.

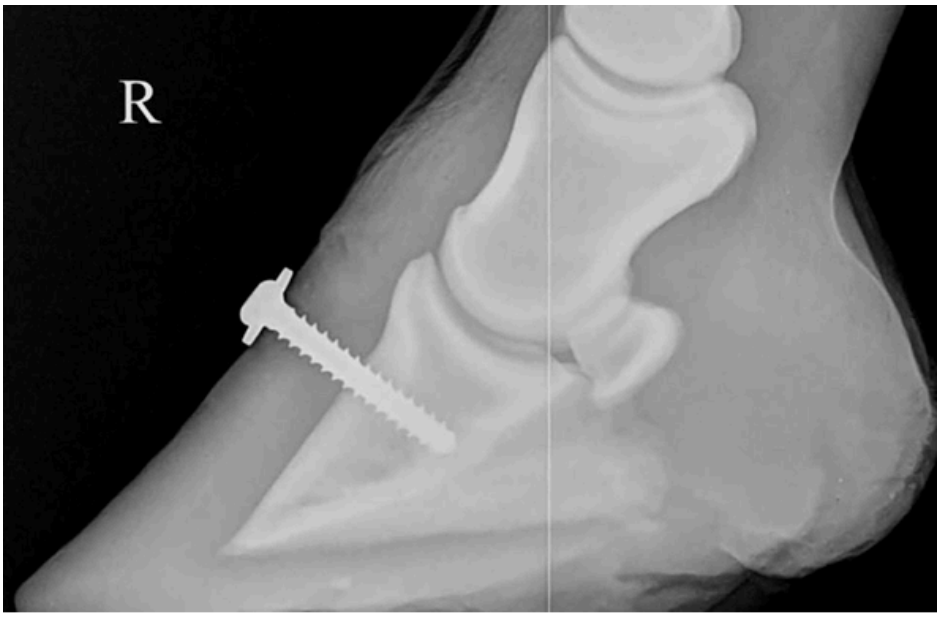
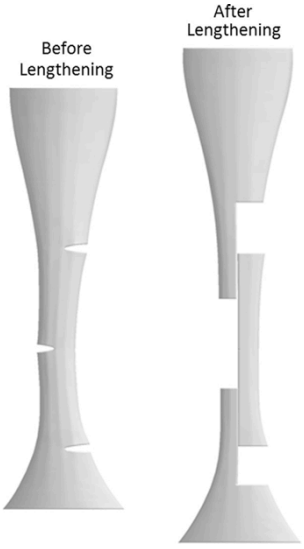


Figure 9. Lateromedial radiograph illustrating a right front unicortically placed screw with a washer through the dorsal hoof wall and P3, taken from Carmalt et al. 2019.

g. Hemitenotomy

(A) **(B)**



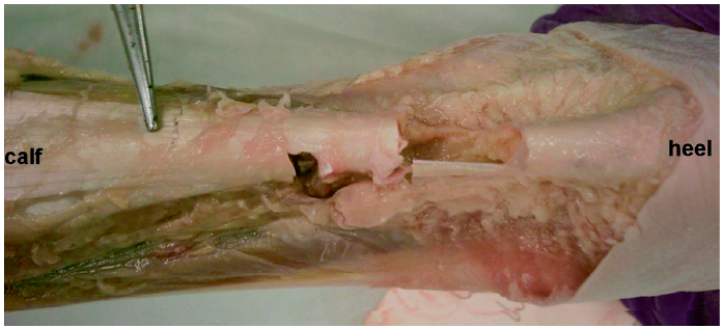


Figure 10. Triple hemitenotomy for Achilles lengthening. A, Illustration of the tendon lengthening process before and after dorsiflexion for slide lengthening, taken from Von Forell

and Bowden 2014. B, Failure of tendon lengthening. The proximal cut (distal to scissor) did not slide, and the distal cut ruptured during the sliding procedure, taken from Hoefnagels et al. 2007.

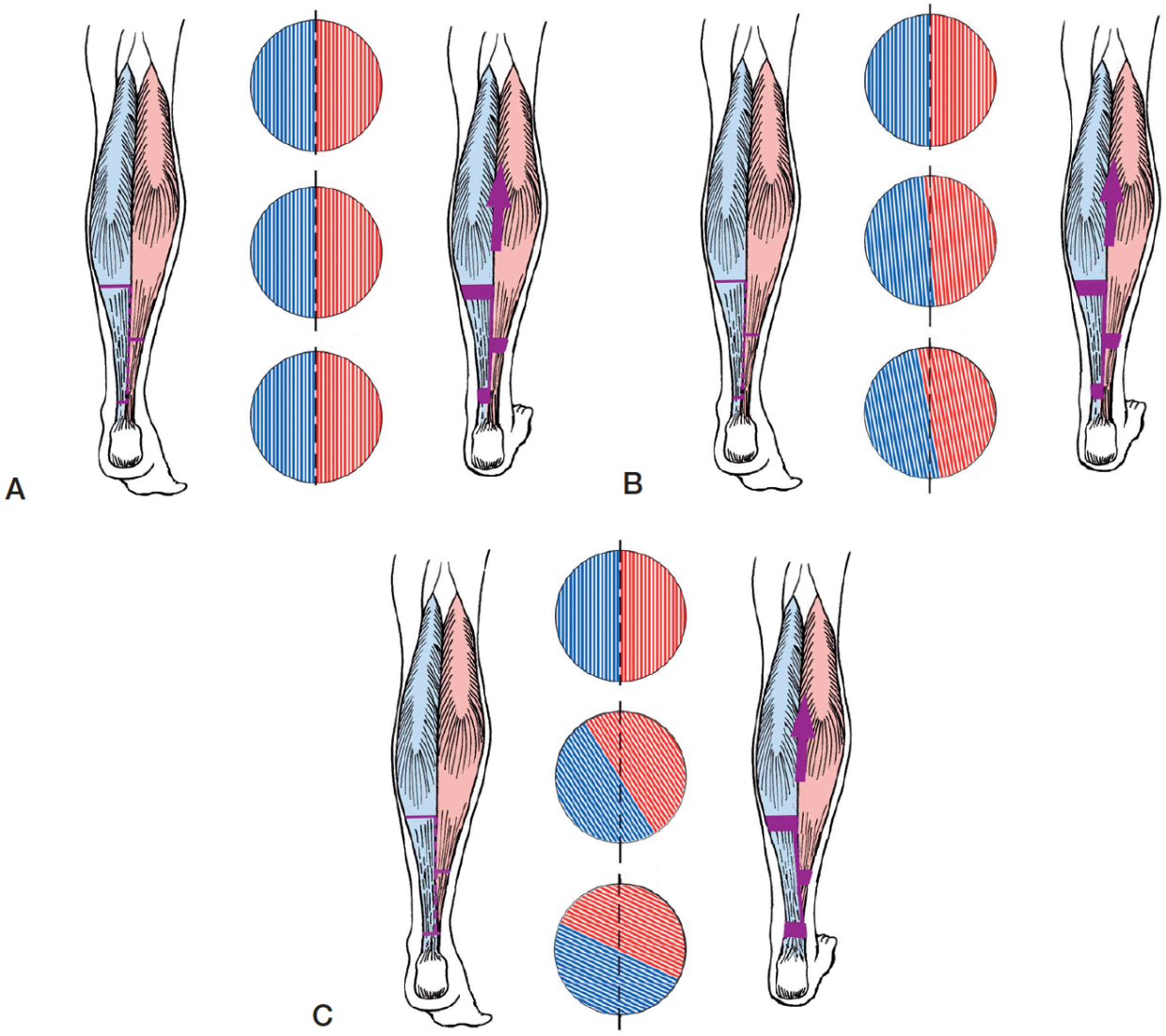


Figure 11. Illustration of triple-hemitenotomy Achilles tendon lengthening with cross sections. A, 0° torsion. Three hemisections will sever neighboring fibers and the fiber bundles will glide along each other. B, 11° torsion. The tendon fibers are still almost parallel and the fibers will still glide along each other after 3 hemisections. C, 65° torsion. The three hemisections do not sever

neighboring fibers, and the gliding mechanism will fail or remain incomplete, taken from Hoefnagels et al. 2007.

h. Normal tendon

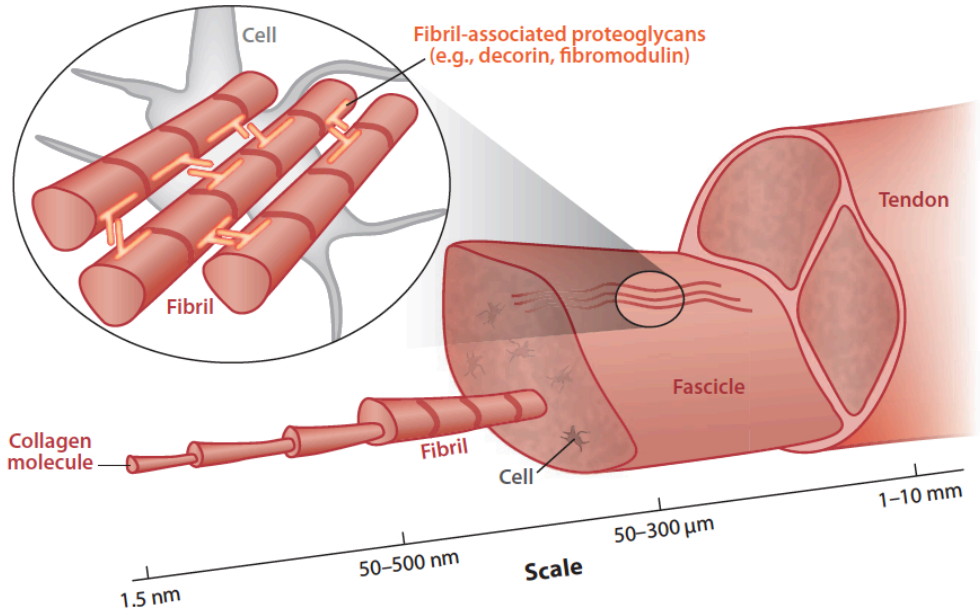


Figure 12. Hierarchial structure of a tendon, taken from Voleti et al. 2012.

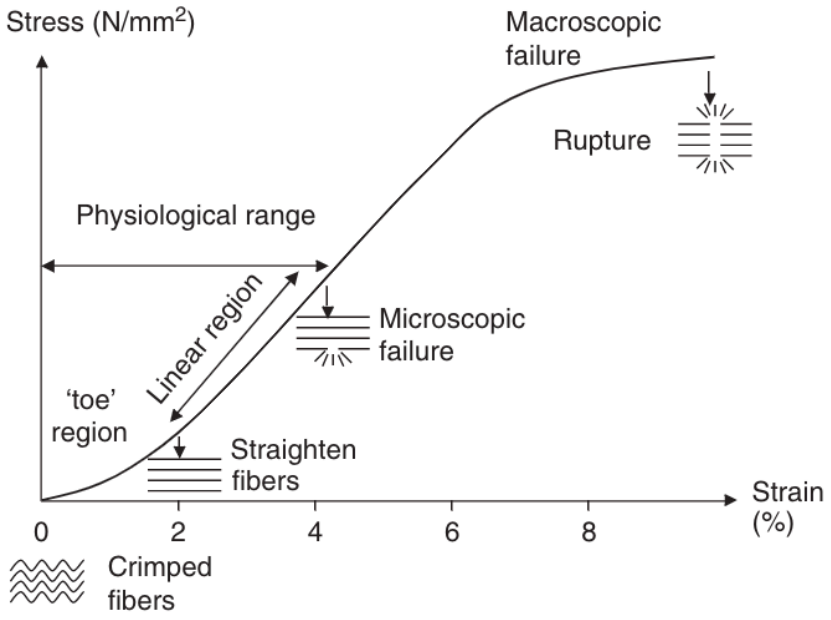


Figure 13. Tendon stress-strain curve, taken from Wang 2006.

i. Biomechanical testing of tendons

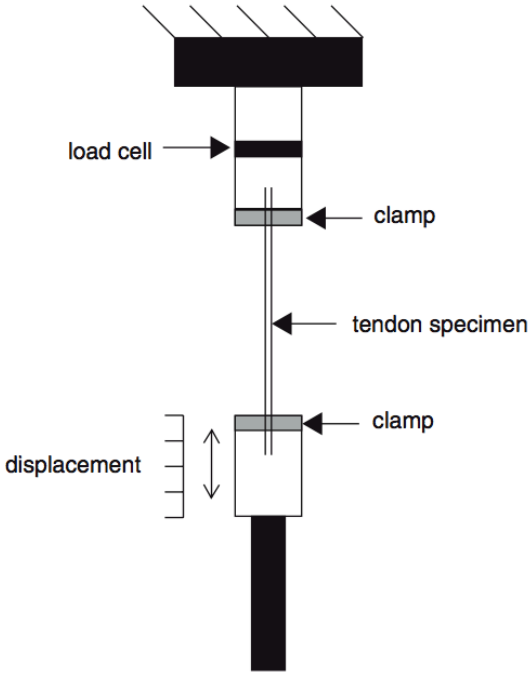


Figure 14. Tensile testing machine, taken from Tendon injuries,1st edition.

TABLE 1: Tendon loading parameters measured over 4 consecutive contacts per *n* trials at each gait for 3 Thoroughbred horses

Tendon	*n*	Gait	Speed (m/s)	Strain (%)	Stress (MPa)	Force (N)
DDF	9	Walk	1.7	0.8 ± 0.2	10.9 ± 0.23	1058 ± 22.7
	6	Trot	4.1	1.2 ± 0.3	15.5 ± 0.44	1521 ± 39.7
	3	Canter	7.0	2.0 ± 0.1	25.1 ± 0.99	2446 ± 95.9
SDF	10	Walk	1.7	3.6 ± 0.1	45.7 ± 0.98	3802 ± 81.6
	7	Trot	4.1	5.6 ± 0.2	71.8 ± 1.96	5971 ± 163
	3	Canter	7.0	4.8 ± 0.2	61.7 ± 2.81	5136 ± 233

Values are grand mean ± s.e. and represent average, not peak, loading parameters; DDF = deep digital flexor tendon; SDF = superficial digital flexor tendon; MPa = megapascals; N = Newtons.

Taken from Butcher et al. 2007.

j. Preliminary data

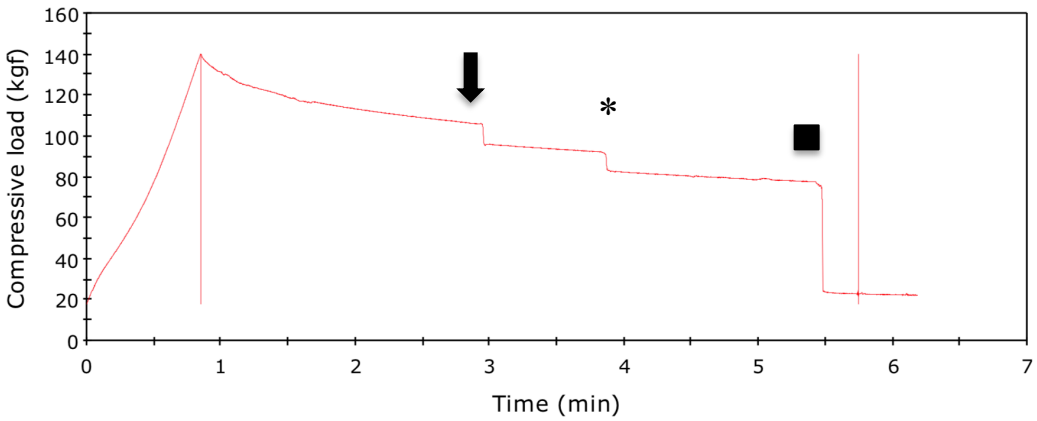


Figure 15: Illustration of load reduction (31%) in one forelimb with each of the two hemi-tenotomies. Test started at 140kg compressive load (500kg body weight x 0.28). The first (arrow) and second (asterix) hemitenotomies were performed following relaxation of the tendon fibers. A complete tenotomy was finally performed (square) in order to document the load of the musculoskeletal structures themselves.

k. Figures from Methods and Materials

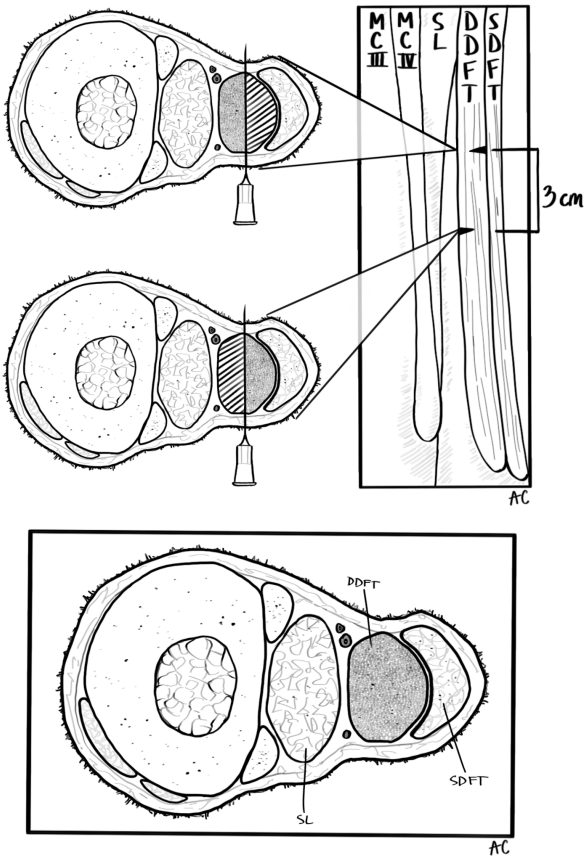


Figure 16. Lateral and cross-sectional illustrations of needle placement at 50% DDFT thickness at the intended hemitenotomy sites (3 cm apart) and subsequent gap formation following hemitenotomies at the level of mid-metacarpus. Image provided by A. Caldwell.

Figure 17. Lichty teat knife used for hemitenotomies (part 1 and 2) and complete tenotomies (part 2).

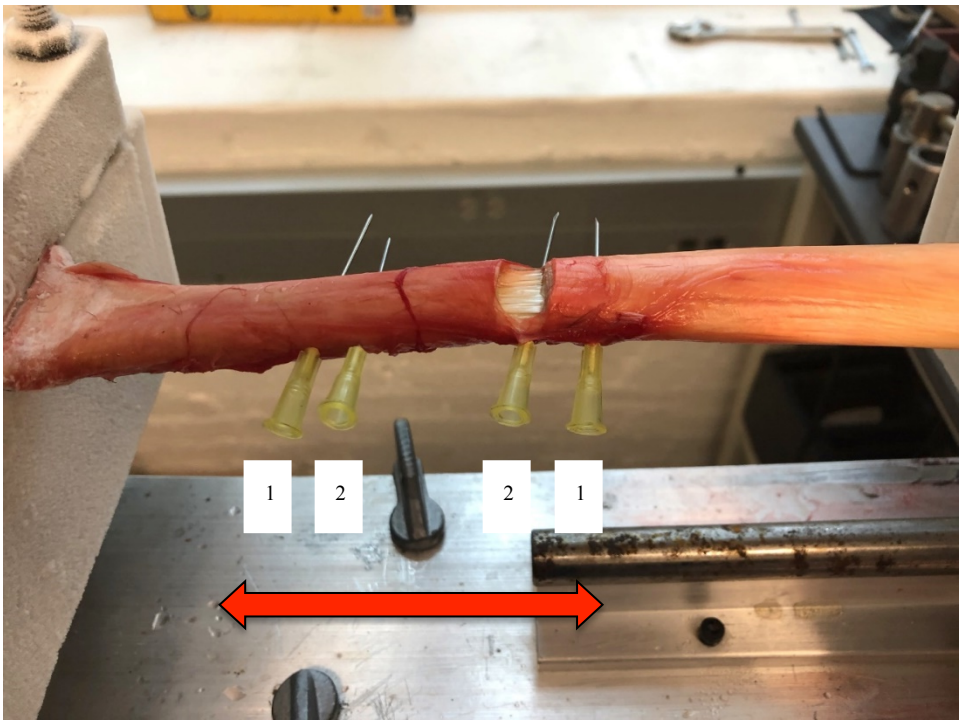


Figure 18. Isolated DDFT mounted in a material testing apparatus using custom-engineered cryoclamps to prevent tendon slippage. The 2 outermost needles (1) were used for measuring lengtening following hemitenotomy incisions. The 2 innermost needles (2) were placed 3 cm apart at a 50% tendon thickness at the hemitenotomy sites. A tensile (arrow) load was applied until tendon failure or a maximum of 500 kg.

Figure 19. Complete forelimb mounted in an Instron Testing Machine. A compressive load (arrow) corresponding to in vivo conditions was applied. Hemitenotomies were created and measurements recorded. A complete tenotomy was then performed and length and load reduction recorded.

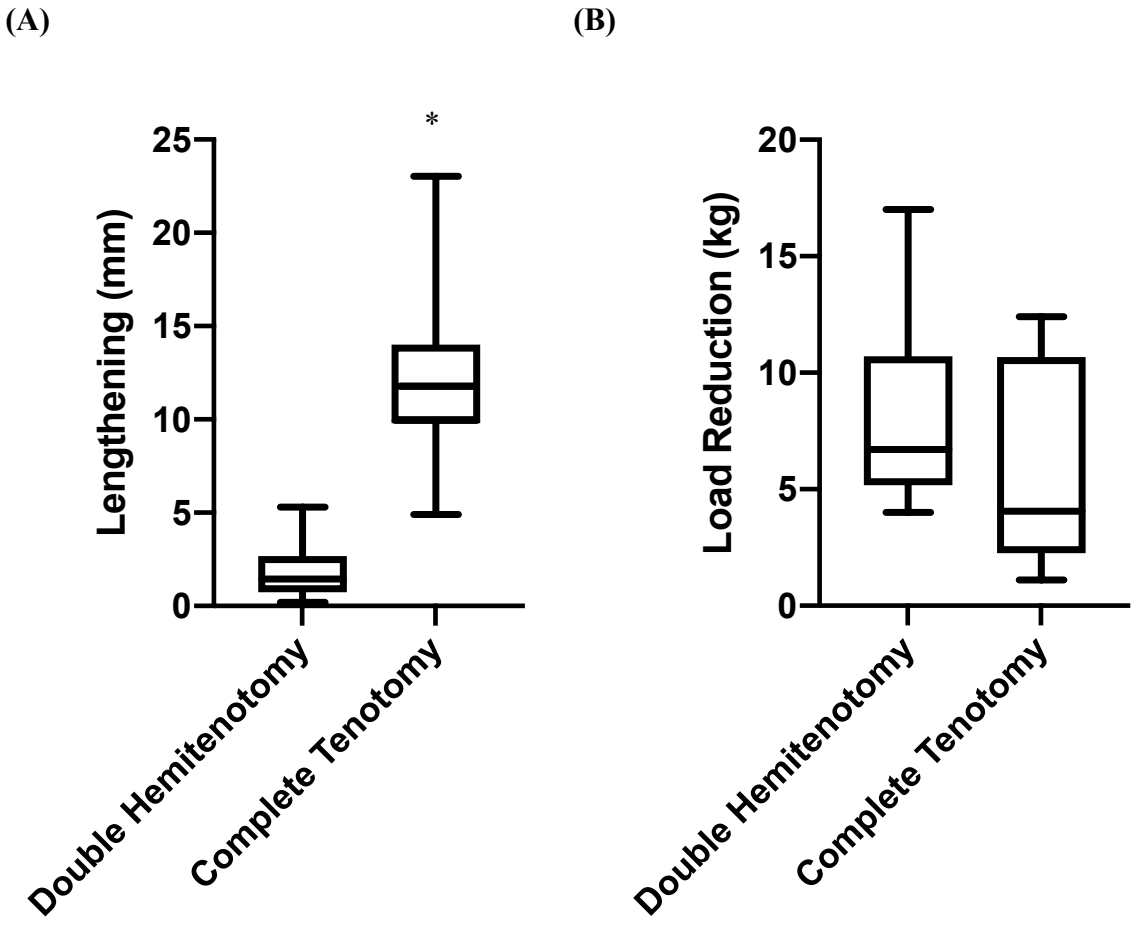


Figure 20. Lengthening of tendon (A) and load reduction (B) in the deep digital flexor tendon, stratified by tenotomy method (double hemitenotomy (n=16) versus complete tenotomy (n=16), part 2). The asterisk (*) indicates a statistically significant difference (P=0.008). There was no difference in load reduction between the DHT and the CT (P=1.0).

l. Tables and from results

Table 2. Case details for study subjects of part 1.

Specimen	Weight (kg)	Age (yrs)	Breed	Sex	Hemitenotomy	Failure (kg)
1	500	12	TB	G	1	340
2	500	12	TB	G	0	-
3	513	12	TB	M	1	215
4	513	12	TB	M	0	-
5	454	11	APH	M	1	283
6	454	11	APH	M	0	-
7	500	16	AQH	M	1	265
8	500	16	AQH	M	0	-
9	431	32	WB	M	1	374
10	431	32	WB	M	0	472
11	500	14	TWH	M	1	55
12	500	14	TWH	M	0	-
13	454	14	AQH	G	1	-
14	454	14	AQH	G	0	-
15	520	11	Haflinger	M	1	-
16	520	11	Haflinger	M	0	430
17	510	10	TB	M	1	300
18	510	10	TB	M	0	344
19	548	10	AQH	M	1	223
20	548	10	AQH	M	0	-
21	520	7	TWH	G	1	58
22	520	7	TWH	G	0	-
23	375	19	AQH	M	1	-
24	375	19	AQH	M	0	-
25	177	2	Shetland	M	1	155
26	177	2	Shetland	M	0	375
27	380	1	Draft	M	1	144
28	380	1	Draft	M	0	-
29	250	28	PF	M	1	195
30	250	28	PF	M	0	-

M, mare; G, gelding; APH, American Paint Horse; AQH, American Quarter Horse; Draft, Draft Horse; PF, Paso Fino; Shetland, Shetland pony; TB, Thoroughbred; TWH, Tennessee Walking Horse; WB, Warmblood yr, year; kg, kilograms. The mean± SD weight, age and load at failure was 442±104.7 kg, 11.8±7.5 yrs and 173.8± 127.6 kg, respectively.

Table 3. Case details for study subjects of part 2. All had double DDF hemitenotomy followed by complete tenotomy.

Specimen	Weight (kg)	Age (yrs)	Breed	Sex
1	416	11	TB	G
2	416	11	TB	G
3	480	5	TB	G
4	480	5	TB	G
5	850	18	TWH	M
6	850	18	TWH	M
7	503	21	AQH	M
8	503	21	AQH	M
9	363	15	Mix pony	M
10	363	15	Mix pony	M
11	461	11	AQH	G
12	461	11	AQH	G
13	454	13	AQH	G
14	454	13	AQH	G
15	454	14	TB	G
16	454	14	TB	G

M, mare; G, gelding; AQH, American Quarter Horse; TB, Thoroughbred; TWH, Tennessee Walking Horse; yr, year; kg, kilograms. The mean± SD weight and age was 498±143.5 kg, 13.8± 4.6 yrs, respectively.