EFFICACY OF TARSAL IMMOBILIZATION TO ALLEVIATE ACHILLES TENDON STRAIN IN VIVO – DIRECT MEASUREMENTS VIA A DIFFERENTIAL VARIABLE RELUCTANCE TRANSDUCER™ (DVRT) STRAIN GAUGE IN A CANINE MODEL

by

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Abstract

Objective: To measure strain in vivo in the calcanean tendon during trotting in canines, and to compare to strain present after tibiotarsal immobilization.

Animals: 6 canines

Procedures: A Differential Variable Reluctance Transducer^{TM} (DVRT®) strain gauge was surgically implanted on the common gastrocnemius tendon. Surface EMG, % strain, and ground reaction forces were measured prior to intervention and after immobilization. Peak vertical force (Fz), vertical impulse, initial, maximum and final strain, and peak-to-peak EMG amplitude were recorded. Data was analyzed using repeated measures analysis of variance and paired t-tests ($p \leq 0.05$).

Results: Timing of strain data correlated closely to the hind limb footstrike and EMG activity in all dogs. Maximum tendon strain occurred simultaneous with peak Fz. Continued muscle contraction was evident after immobilization. There was no statistical difference in maximum strain after immobilization compared to normal motion. Minimum strain, both at the beginning and end of the strain curve, was significantly decreased with the immobilized state compared to non-immobilized joints.

Conclusions and Clinical Relevance: Tibiotarsal immobilization did not eliminate calcaneal tendon strain during weight bearing. Decreased isometric muscle contraction during swing phase of the gait would account for smaller minimum strain in immobilized joints. Immobilization is frequently applied after Achilles tendon rupture to alleviate strain and force on the sutured repair, with possible complications due to the immobilization method. Direct correlation of strain with

tendon force was not made in this study. This would be an important factor before adjusting current treatment recommendations.

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CHAPTER 1 - Literature Review

Tendons

Tendon Anatomy and Function

Tendons are anatomic structures connecting muscle and bone that serve to transmit the forces created by muscle to bone and transfer muscle energy into action.[1] Elastic energy is also stored in tendons under stress, and then used in subsequent strides to reduce energy requirements and increase efficiency of movement.[2] Because of their micro and macroanatomic structure, tendons are considered viscoelastic structures, whereby the strength depends upon the rate at which load is applied, and strength is strain-dependent, where a tendon's resistance to tension is greater at higher strain rates or displacement rates.[3]

The gross structure and size, as well as type of tendon sheath, varies with the size and function of the associated muscle. Overall, tendons are complex structures, composed of a series of progressively smaller subunits. Larger fascicles are composed of fibrils which are made up of subfibrils, and then microfibrils.[2] On a microanatomic level, tendons are composed of a combination of parallel collagen fibres and elastin, with sparse tenocytes located between the fibres, and an extracellular matrix made up of proteins and proteoglycans.[1,2] The function of the cells is to produce and maintain the extracellular matrix.[2] Tendons largely contain Type I collagen (65-75% dry mass of the tendon). Smaller amounts of type III and V collagen (5% dry mass) are also present.[1,2] Multiple lysine-derived intermolecular cross-links are present between the collagen molecules.[2] In the unstretched form, the collagen fibres give tendons a characteristic wavy appearance under light microscopy.[1] Some tendons are enclosed by a synovial sheath with an outer fibrous layer, then 2 inner layers of synovial sheaths, which secrete peritendinous fluid and act to decrease friction. Other tendons do not have synovial sheaths, and are simply enclosed in a loose capsule of loose areolar connective tissue (paratenon) that functions as an elastic sleeve (less effectively then a true tendon sheath) and permits free movement of the tendon against the surrounding tissues. The paratenon is composed of type I and III collagen fibrils, elastic fibrils and a layer of synovial cells that line the inner surface of the paratenon. Under the paratenon, the entire tendon is surrounded by another layer of dense connective tissue,

the epitenon, which is contiguous with the paratenon on the outer surface and the endotenon on the inner surface. The endotenon encloses each tendon fibre within the tendon and binds individual fibres and fibre bundles together, as well as carries blood vessels, nerves and lymphatics to the deeper portion of the tendon, and allows the fibre groups to glide on each other.[1]

The ultrastructure of tendons is very closely organized, with fibres arranged in parallel bundles oriented along the long axis, and cells in parallel rows between fibres. This orientation is determined by several factors. Cell orientation has an influence on the assembly and alignment of matrix macromolecules.[2] The matrix also exerts a reciprocal influence on the cells, so that feedback can alter matrix synthesis.[2] There are several hypotheses to explain the cell alignment. Mechanical deformation of the cell membrane may activate ion channels, leading to cell alignment along lines of stress.[4] Cell slope and pressure may also mediate the mechanical control of matrix composition, leading to direct mechanical effects on cell metabolism.[5,6] It is also thought that mechanical forces can influence the blood supply, which indirectly could alter tendon cell metabolism due to associated changes in oxygen tension and nutrient flow.[2]

Tendon Injury

There are multiple mechanisms for tendon injury. Tendons typically fail by either acute mechanical overload, or by accumulated damage from repeated subclinical injury.[1,2] With acute overload there is a characteristic pattern of failure, and in post-mortem trials when tendons are loaded to failure, the central fibres typically fail first.[2]

Some implicated factors for repetitive damage include exercise induced hyperthermia, hypoxic injury, reperfusion injury and mechanical strain/stretching. Exercise induced hyperthermia has been speculated to be a possible mechanism for cell damage. Measurement of the core temperature of equine superficial digital flexor tendons during exercise revealed that the core temperature can reach 45 degrees Celsius during relatively short exercise periods.[8] Temperatures above 42.5 degrees Celsius are known to result in fibroblast death.[8,9] However, in vitro studies have also demonstrated that core tenocytes are relatively resistant to heat stress. The mechanism of action for this heat tolerance is unknown, although it is speculated that tenocytes possibly have higher levels of heat shock proteins, in particular HSP70, allowing them more tolerance to the effects of high temperature.[9] Birch et al in 1997 demonstrated that after 10

minutes at 45 degrees, 91% of core tenocytes cells remain viable. After 10 minutes at 48 degrees, only 22% survival was present.[9]

Hypoxic injury and reperfusion injury are other speculated causes of repetitive injury. Reperfusion injury can occur secondary to free radical formation after ischemic periods during exercise.[2] Tendons in general have a poor central blood supply. Cyclical loading is thought to result in an associated ischemia during the period of maximum tensile strain.[2] Support for this theory is found in the presence of hypoxic changes on scanning electron microscopy of Achilles tendons of humans. Degenerate tendon tissue contains hypoxic changes in tenocytes, with alterations in mitochondria, endoplasmic reticulum, ribosomes, and number of lysosomes.[10] However, tendon fibroblasts are also less sensitive to reduced oxygen tension then fibroblasts from different tissues.[11]

Mechanical strain can also lead to repetitive breakdown of the tendons. When strain is initially applied to tendons, individual collagen fibres undergo elastic deformation and straighten out, losing their wavy appearance. When this strain is relieved, they resume their normal unstressed shape. Individual fibres demonstrate a linear, elastic response to 2-5% elongation, and then resume a normal wavy structure once the strain is removed, with resumption of normal length. Microfailure of fibres starts to occur at the end of this loading region with occurrence of plastic deformation. When tendons and ligaments are loaded as a whole unit, they demonstrate a linear response to a much larger amount of strain, as high as 20-50%. This is thought to be due to the three-dimensional organization of fibre bundles within the whole tissue.[1]

Medical conditions that affect collagen such as hyperadrenocorticism and steroid use can promote tendon rupture.[12-15] Steroid injections into or around tendons substantially weaken the tendon within 2 weeks with progressive thinning and reduction of collagen, and effects persist for up to 1 year.[1] Injection directly into tendons results in necrosis due to pressure increase and hypoxia within the tendon, and also have substantial effects on tensile strength, modulus of elasticity and healing.[1,12,13,16] Other mechanisms for injury include trauma via laceration or tenotomy, and parasitic migration within tendons.[17]

Tendon Healing

Tendons heal via three phases. The first phase involves hemorrhage and hematoma formation, followed by inflammation. This phase begins immediately after injury. Fibrin and fibronectin can be seen in the area within 1 hour after injury, and form cross-links with collagen.

Polymorphonuclear leukocytes and monocytic cells are attracted by the production of chemotactic agents including histamine, fibronectin, bradykinin, and prostaglandin E_2. This migration of cells into the injured area is seen within several hours, and continues for several days. The inflammation phase can last from 4-7 days in controlled injuries. The inflammation and disorganization of collagen fibres progress from the gap or surgical repair to the adjacent normal tendon, until the junction between normal and healing tendon is very indistinct. This is followed by a second proliferative phase, with attraction of undifferentiated mesenchymal cells and production of matrix and unorganized collagen fibres. This phase begins during the inflammatory phase, and continues until approximately the 21st day. Initially, the collagen is mostly immature type III, with small diameter fibrils arranged in haphazard fashion. By the 12-14th day, the type III collagen will begin to be replaced by type I collagen. The final phase involves maturation and remodeling, and begins by 3 weeks post-injury. Over time, type I collagen begins to predominate again, with increases in fibril diameters, and formation of stable cross-links. Reorganization of collagen fibres parallel to lines of stress is first seen by 5-6 months after the initial injury. Decreases are also seen in the number of macrophages, fibroblasts, myofibroblasts, and capillaries, as well as decreased amounts of glycosaminoglycans. The collagen becomes much more densely packed, and becomes almost exclusively type I. This reorganization can take up to a year, and is associated with increasing strength.[1,2,18, 19]

The healing tendon undergoes a process that has been described as a "wave of injury." Healing initially begins at the site of the surgical repair or injury. The inflammatory process and disorganization progresses towards the origins, then regresses towards original wound as healing progresses.[19] By 3 weeks post injury or repair, the border between the undamaged tendon and the area of the initial injury is indistinguishable, with hypercellularity and disorganized matrix extending towards the origins of the tendon. Scar tissue then progressively recedes over time towards the site of the initial injury.[14]

Healing tendons are slow to regain strength. Dueland and Quenin in 1976 investigated the healing of canine triceps tendons after tenotomy and suture repair. In the first three weeks after repair, no substantial increases in strength were noted. During the 3-6 week period after repair, a steady gain in strength occurred, until by 6 weeks the tendons had regained 56% of their normal tensile strength, with rigidity increasing during the entire 6 week period. Further gains occured much more slowly, after 1 year tendons had only regained 79% of their original tensile

strength.[18] However, under normal use, tendons are subjected to only 25-33% of their maximum strength, and the degree of healing present by six weeks is sufficient for limited activity.[18]

Tendon healing is influenced by many factors. This includes healing across a gap versus appositional healing, healing during immobilization versus non-immobilization, the cause of the injury (acute trauma versus corticosteroid-induced), location of the injury, and severity of trauma/type of injury, as well as the presence of infection, foreign bodies or ischemia.[14] Injuries resulting from tenotomy or lacerations have the best healing capacity, whereas more severe injuries with substantial loss of parenchyma are more problematic. When injury results from a traumatic or degenerative etiology, it is recommended to sharply excise all abnormal tissue, then reappose normal tissue. When tendon injury has resulted from an underlying medical condition (i.e. hyperadrenocorticism), there is potentially poor healing capacity, unless the underlying cause can be reversed.[12-15]

The type of tendon can also play a role in the healing process. Tendons generally have a limited internal vascular supply, and are classified based on their ability to recruit vasculature during the healing process. Avascular tendons include those tendons with a tendon sheath, (e.g. the digital flexors). These tendons are considered to have a poor blood supply and resulting poor healing ability, with increased risk of adhesions between the tendon and the sheath. Vascular tendons include those tendons without a tendon sheath, that are surrounded by muscle and soft tissue, for example the Achilles tendon or the triceps tendon.[20]

It is vitally important not to overstress a tendon anastamosis in the early post-operative period before adequate healing has occurred, as this leads to gap formation and/or repair failure. If no gap occurs, then tendons heal without interposed scar tissue, and once healed, appear histologically normal. Tendons healing with a 1mm gap will always have a small width of scar tissue that can be distinguished histologically, even two and a half years later.[19] Tendons undergoing a gap greater than 3mm during the repair process heal at a slower rate, with decreased ultimate tensile resistance at 6 weeks compared to tendons healing with no gap or less than 3mm gap.[21,22] Gelbermann et al, in 1999, investigated the effects of gap healing after repair of canine digital flexor tenotomies. Tendons undergoing gaps at the repair >3mm, demonstrated no appreciable gain in strength from 10-42 days post-operative, where tendons with gaps less then 3mm demonstrated increasing rigidity and ultimate tensile strength during that same time period.[21] Boyer et al, in 2001, also investigated the effects of gap healing and showed lower ultimate tensile strain at 6 weeks in tendons undergoing a gap greater than 3mm during healing,

with 35% lower ultimate force at failure compared to tendons healing with no gap or a gap less than 3mm.[22]

Immobilization is typically enforced after tendon repair in an attempt to diminish strain on the repair and prevent gap formation or repair failure. However, some tension is beneficial during tendon healing, and limited physical therapy has been shown to result in stronger tendon repair, compared to patients treated with immobilization. Starting three weeks post-operative, as the final phase of healing begins, tension is thought to help orient the healing collagen fibres parallel to the lines of tension, resulting in stronger healing. Without any stress, there is no stimulus for re-organization of collagen.[18] In rabbits and mice, faster maturation of collagen fibres is noted in non-immobilized groups, as well as more rapid and continuous restoration of load to failure, compared to immobilized groups.[23-25] In mice, 4 months after sutured repair of calcaneal tenotomies, the group treated with immediate mobilization had regained their original tendon stiffness, whereas the group treated with immobilization had only regained 50% of the original stiffness.[24]

It seems that no additional benefit is obtained above a certain level of force. Boyer et al in 2001 compared the effects of low-force mobilization versus high force mobilization on the healing of canine digital flexor tendons. Patients underwent physical therapy for 5 minutes, twice daily at two different levels of force. In between sessions, they were completely immobilized. No difference were found in the ultimate tensile strength or force at six weeks between the low-force and the high-force groups.[22]

There are several adverse effects of immobilization on joints, tendons, and muscle. Immobilization leads to cartilage atrophy, muscle atrophy, and decreased range of motion. After only a few weeks in a non-weightbearing cast, cartilage thickness will be reduced by as much as 50%, with concurrent reduction in concentration and content of proteoglycans by as much as 50%.[26] Profound disuse atrophy also rapidly occurs in the periarticular muscles and subchondral bone when weight bearing is eliminated.[27] Immobilization of normal limbs results in decreased GAG content and collagen cross-links in periarticular tissue and tendons in dogs, rats and rabbits.[28-30] In rabbits, the ultimate tensile strength of the cranial tibial tendon has been shown to decrease following a period of immobilization.[31] Collagen and proteoglycan concentrations have also been shown to decrease in tendons after immbolization.[32] The effects are considered largely reversible after 6 weeks of immobilization, provided the joint is protected from vigorous use

once it is remobilized.[26,27] Some of the results may be permanent, and in horses, proteoglycan content may never return to normal.[33,34]

Tendon Repair Strategies

When dealing with tendon injuries, there are some confounding factors that affect the ease of repair. It is important to prevent gap formation, to optimize healing by promoting primary healing, maintaining vascularity, and decrease adhesion formation. Most tendons are under high tensile load, for example the Achilles tendon, patellar tendon, deep gluteal tendon, and triceps tendon. Weight bearing and muscle contraction put immediate stress on the repair, so the strength of the sutured repair must be sufficient to prevent failure. The use of strong, inelastic suture is of primary importance. Augmentation of sutured repairs with synthetic substances such as porcine small intestinal submucosa, fascia lata, polypropylene mesh, or bioabsorbable tendon plates made of poly-L lactic acid can provide additional support.[35-39] Protection of the repair with external support (immobilization) is widely employed.[14,40] In human medicine, early physical therapy through protected weight bearing with hinged splints allowing limited range of motion, as well as various orthotic such as heel lifts, combined with exercise restrictions and the use of crutches and canes is commonly employed. Non-weight bearing exercises for range of motion, as well as isometric exercises with the use of light weights are also encorporated into physical therapy regimens.[1,23,41-43]

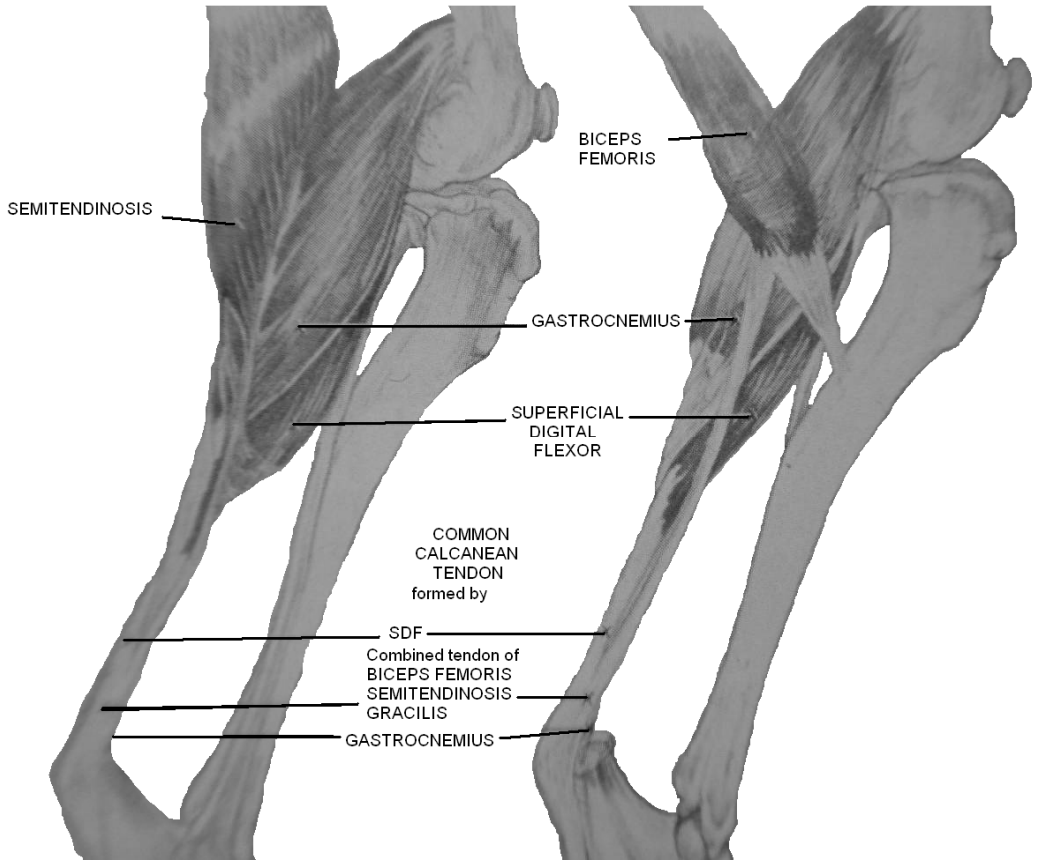
The anatomic arrangement of parallel fibres in tendons with few cross-linking fibres provides little to prevent suture from slipping and loosening towards the tenotomy when load is applied. There are specific suture patterns that are designed to hold in tendons and prevent gap formation under load: the 3-loop pulley,[44-47] Kessler locking loop, [46,49] and Krackow[49-51] suture patterns being three examples. The Kessler locking loop and Krackow sutures are considered for use more in flat tendons. The 3-loop pulley is considered better used in round tendons. Moores et al in 2004 compared the 3-loop pulley to 2 locking loop sutures, and found that the 3-loop pulley was more resistant to gap formation, although both had similar forces at ultimate failure. A 1mm gap formed at 44N with the 3-loop pulley, and at 18.4N with the locking loops. A 3mm gap formed at 56.3N with the 3-loop pulley and at 34.7N with the locking loops.[46] The 3-loop pulley has also been found to be almost twice as strong as a single locking loop suture in preventing both 1 and 3mm gap formation in Achilles tendon repairs under tension.[45]

Canine Achilles Tendons

Anatomy

The canine Achilles tendon is composed of the common calcaneal tendon and the superficial digital flexor tendon. The common calcaneal tendon is composed of the tendons of the gastrocnemius muscles, and the combined tendons of the gracilis, semitendinosus, and biceps femoris muscles. The gastrocnemius muscles are paired, but unite in the upper limb to form a single tendon which makes up the majority of the common calcaneal tendon. The superficial digital flexor initially runs deeply, between the two parts of the gastrocnemius, however the tendon later winds around the medial border of the gastrocnemius tendon into a more superficial location, forming a broad cap over the point of the hock. The calcaneal tendon inserts on the tuber calcani of the talus, while the superficial digital flexor tendon continues distally to divide and insert on the flexor surface of each digit.[52,53]

Figure 1-1: Anatomy of the canine calcaneal tendon



Modified from: Reinke and Kus, Compendium, 1982

Pathophysiology of Injury and Classification

Calcaneal tendon injuries are classified based on several factors, including age of the injury, degree of tendon involvement, location of the injury and etiology. Injuries occurring within less than 48 hours are classified as acute, while those occurring between 2 and 21 days are considered subacute, and injuries older than 21 days as chronic injuries.[54,55] Damage to a tendon caused by an external force are also termed strains. Degree of tendon involvement can vary from partial to complete. First-degree strains or mild strains result from short-lived application of moderate force, with relatively few damaged fibres, and minimal functional effects. Second-degree or moderate strains are characterized by increased numbers of damaged collagen fibres and marked functional deficit, although the tendon is grossly intact. Third-degree or severe strains have actual interstitial disruption (partial or complete) or avulsion of the ligament from bone, with complete loss of function.[56] In 2006, Nielson et al looked at 28 dogs with naturally occurring calcaneal tendon injuries, and found that 43% had complete rupture of all components, and 57% had only partial rupture, with one or more components still intact.[55]

Etiology, as previously discussed, varies from having a traumatic origin, with tendon lacerations, to being idiopathic or unknown. Steroid use or hyperadrenocorticism has been considered a potential pre-disposing cause.[12,15,16] Parasitic migration has also been linked to Achilles tendon rupture; Parker and Cardinet reported on a case of rupture associated with *Trichinella spiralis* infection.[17] The majority of cases are still considered idiopathic – either occurring acutely during vigorous exercise, or gradually with chronic tendinosis and strains. Chronic strain injuries due to overuse, hypoxic injury, reperfusion injury, and heat stress have been considered as potential causes.[2,8,10,52,54,57] There is also speculation about a potential genetic link in Doberman pinschers, with an increased incidence of Doberman pinschers with chronic calcaneal injuries reported in the literature, and many dogs developing bilateral injuries.[58-60]

Achilles mechanism injury is a broad term that encompasses many different subsets of injuries. The category includes musculotendinous separation or gastrocnemius muscle rupture, tendinous laceration or transection, chronic tendinosis and strains, tendinous avulsion from the tuber calcanei, avulsion from the supracondylar tuberosities of the femur, calcaneal physeal avulsion, and calcaneal fracture. Calcaneal physeal avulsions typically occur in immature animals less than 2 years of age, before complete closure of the calcaneal growth plate. Tendinous avulsion from the tuber calcanei and chronic tendinosis or strain injuries are the most

common presentation in animals older than 2 years of age.[55] All of these problems can result in a similar presentation, and they should all be considered as differential diagnoses. Other differential diagnoses for a plantigrade stance must include nerve injury, as a tibial nerve injury will result in a very similar stance.[52,54,55]

Clinical Presentation and Diagnosis

Diagnosis of complete Achilles tendon injuries is typically established through physical examination. After complete rupture, animals typically present either nonweight-bearing or with a plantigrade stance. A plantigrade stance is characterized by tarsal hyperflexion. On palpation, the tarsus can be abnormally flexed with the stifle extended. In a normal animal, minimal tarsal flexion is possible during complete stifle extension.[35,52,54] If the superficial digital flexor tendon is intact, hyperflexion of the digits is noted, with 'claw-like' foot position – this is the most common form of presentation.[54,58] If complete disruption of all components, including the superficial digital flexor tendon, is present, the paw will be in a plantigrade position, with loss of normal digital flexion.[54,58] A palpable discontinuity may be present in the muscle or tendon. If the tendon is partially ruptured, animals may present with hindlimb lameness and concurrent swelling of the calcaneal tendon, but without a plantigrade stance.[36,54,58,59]

Radiographs of the tarsus typically reveal soft tissue swelling in the region of the injury. Enthesiophytosis or roughening is often evident on the proximal surface of the tuber calcanei. Depending on the etiology, fracture of the calcaneous or physeal avulsion and proximal distraction of the fractured or avulsed segment may be evident.[52,54,61-64] If the gastrocnemius muscles have undergone avulsion from the supracondylar tuberosities of the femur, distal displacement of the popliteal sesamoids may be evident.[65]

Ultrasonography can also be used as a diagnostic tool. It is especially useful to differentiate partial and complete ruptures, as well as for monitoring during the course of tendon healing. Normal tendons have very characteristic architecture, with the mid-portion of the tendon appearing as an echogenic structure with parallel, hyperechoic lines, surrounded by a hyperechoic, thick, smooth band (peritenon). Abnormal findings in partial tendon ruptures include thickening of the calcaneal tendon, with loss of the normal linear pattern. The echogenicity is typically inhomogenous. With complete ruptures, complete interruptions in the tendon structure can be noted, with loss of the normal tendonous echo-structure. Hematoma formation can be evident, with an anechoic, inhomogenous, irregularly delineated area present

within the gap.[35,54,62] During the healing process, inhomogeneity decreases, and the typical fibrillar structure of the tendon reappears.[62]

Treatment of canine Achilles injuries

Preferred treatment methods of Achilles mechanism injuries is largely dependent on the type of injury present. With calcaneal fractures and calcaneal physeal avulsion, the preferred method of repair is reduction of the fracture and rigid repair, either with pins and a tension band, lag screws, bone plate, or partial tarsal arthrodesis, depending on the severity of the fracture. Conservative therapy with a cast is not recommended due to the pull of the gastrocnemius muscles causing fracture distraction.[52,56] After avulsion of the gastrocnemius muscle from its origin on the medial and lateral supracondylar tuberosities of the femur, repair relies on reattachment, either via cerclage wires incorporating the fabella, or through the use of suture anchors.[52,65] After musculotendinous ruptures, or mid-belly muscular ruptures, repair is generally via sutured methods, with the use of buttons or stents reported to improve suture holding power.[52] Spontaneous repair of mid-belly muscular ruptures has also been reported.[66]

The three most common presentations for Achilles mechanism injuries in dogs include severe strains and chronic tendinosis without rupture, tendinous laceration or rupture, and avulsion of the gastrocnemius tendon from the insertion on the calcaneous. Severe strains and chronic tendinosis are commonly treated with immobilization for 6-8 weeks and rest. Some of these will heal with this treatment, others may go on to completely rupture.[56] Gastrocnemius avulsions from the calcaneous are commonly repaired using suture methods, attached to the calcaneous via bone tunnels. The preferred treatment for acute tendinous lacerations and ruptures involves suture repair of each individual component of the calcaneal tendon. With chronic lacerations and ruptures, this distinction may not be possible, as the tendon ends retract, and a gap filled with fibrous granulation tissue and connective tissue is formed. In this situation, the tendon is treated as a single unit, with debridement of the ends and granulation tissue, and reapposition with suture techniques.[52,54,56-58]

Augmentation of sutured repairs via various methods has been reported, either as a means of avoiding post-operative immobilization and allowing early weight bearing, or to reinforce questionable repairs when the length or integrity of available tendon is insufficient for a standard sutured repair. A wide variety of materials have been used, including autologous tissue,

allografts, and synthetics. Types of autologous tissues include fascia lata grafts [35-38], and transposition of tendons (deep digital flexor tendon, peroneous brevis & peroneous longus).[59] These materials have been used to both reinforce sutured repairs, or to replace or fill gaps in repairs when the available tendon length is insufficient. Tendon lengthening techniques such as V-Y plasties at the musculotendinous junction, or sliding z-plasty in the mid-section of the muscles have been reported. The Z-plasty technique provides more lengthening than the V-Y plasty, but both techniques also narrow and weaken the remaining tissue.[59]

Under research conditions, cadaveric tendon allografts have been successfully implanted in defects in canine Achilles tendons.[67] In clinical practice, products such as porcine small intestinal submucosa would be more likely integrated into the sutured repair. Reported synthetics include the use of polypropylene mesh[35] and polyester tape[68], both of which allowed early return to weight bearing without ancillary immobilization.

Ancillary treatments to accelerate healing in chronic strains or after sutured repair are under investigation. Both stem cells and platelet rich plasma are rich sources of multiple growth factors, and have been used to accelerate wound healing pathways. Acellular bone marrow is another potential source of growth factors.[69,70] Smith et al in 2006 investigated the effects of acellular bone marrow and platelet rich plasma, and found that both demonstrate anabolic effects in vitro on equine suspensory ligament fibroblasts.[69] Haupt et al in 2006 investigated the effects of platelet-derived growth factors (from α granules in platelets) which are found in rich supply in platelet rich plasma and acellular bone marrow. They demonstrated induction of type I collagen gene expression on equine superficial digital flexor tendon cell culture within 48 hours of addition of platelet-derived growth factors.[70]

Various methods of tarsal immobilization and exercise restrictions are used to attempt to limit tendon strain after sutured repairs or during conservative therapy of severe strains, with mixed results. Every technique has potential complications, and the exact amount of strain present in the tendon prior to and after tarsal immobilization has never been measured.[48,57,58,71-73] Some methods of immobilization that have been used include transarticular external skeletal fixation[71,73-75], casts and splints[57], a tibiocalcaneal screw[76], and ring fixators.[77]

Immobilization at a normal standing angle or in mild extension is typically maintained for 6-8 weeks, before changing to a padded bandage for several additional weeks. Strict rest is

typically enforced for an additional month after immobilization removal, before gradual return to normal activity.

In human medicine, calcaneal tendon injuries are currently treated post-operative with partial immobilization via hinged splints with restricted ranges of motion, allowing protected stress of the tendon unit. This also allows physical therapy throughout the healing period. Studies have demonstrated faster return to normal function, with less muscle disuse atrophy in these cases.[41-43, 74] Mortensen et al in 1999 reported on 71 patients with acute Achilles tendon ruptures, managed with either a conventional cast for 8 weeks or use of a modified brace allowing a limited range of motion, with an incorporated heel lift. Patients randomized to receive the brace developed fewer adhesions between the healing tendon and skin, and had smaller initial losses in range of motion. They were able to return to work and sports activities sooner, and had greater patient satisfaction, without any increase in tendon lengthening or the ultimate strength of the healed tendon compared to patients treated with a cast.[42] However, in comparison to dogs' upright stance, humans have a plantigrade stance, leading to potentially different stresses on the calcaneal tendon. Also, post-operative activity can be more easily controlled in humans. For these reasons, use of partial immobilization is still in its infancy in dogs.

Prognosis and Complications

After surgical repair of calcaneal fractures and physeal avulsions, the prognosis for return to normal activity level is excellent, as long as the remainder of the tarsus including the articular surfaces remain intact.[56] If other injuries are present, there is a higher likelihood for development of osteoarthritis.[14] After repair of tendinous injuries – either midsubstance ruptures or tendo-osseous avulsions, the prognosis is still good but takes a longer period of time, due to the nature of tendinous healing. Reinke et al in 1993 reported on 11 dogs with avulsion of the gastrocnemius tendon from the calcaneous, which were repaired with locking loop sutures and bone-tunnels. All 11 dogs did well, with no major complications reported. More recently, Nielsen and Pluhar in 2006 reported on 28 dogs with mid-substance ruptures. 78% returned to an acceptable level of function following surgical repair & temporary immobilization, and complications were seen in 46%, with 17% having major complications. Complications were generally related to the methods of immobilization used.[55]

Strain Gauges

The force or strain that is present within tendons and ligaments in vivo can be measured directly through placement of a strain gauge or force transducer on the ligament or tendon, or estimated indirectly using inverse dynamic calculations.[78] Direct measurement of strain or force is considered essential for accurate and precise information, as well as to validate analytical indirect methods. Indirect estimation using inverse dynamic calculations involves measurement of ground reaction forces, and measurement of joint moments from film analysis. The joint moment includes all the forces exerted by ligaments and muscles, the reaction force to this resultant force, and the effects of gravity and friction. From measuring the joint moment, and taking into account the lever arm, attempts have been made to inversely estimate tendon forces.[79,80]

Types of transducers that can be used to measure strain or force include three broad categories, based on the physical principles upon which they operate: electrical resistance, for liquid metal strain gauges and other extensometry gauge transducers (buckle transducers, implantable force transducers); variation of magnetic field, for Hall effect transducers; and variation of light flow for optical strain-measuring systems.[78,81]

The Differential Variable Reluctance Transducer™ (DVRT) (Microstrain, Burlington, VT) is a strain gauge that is directly attached to the surface of the tendon of interest. This strain gauge is a small, highly compliant displacement sensor that correlates voltage output with displacement of a freely moving core within a sensor cylinder.[81] It is secured to the tendon using barbed attachments, one on the core and one on the cylinder of the sensor. To ensure firm attachment to the tendon/ligament, use of additional sutures around the sensor/barbed attachments is recommended.[82] The DVRT was selected for use in this project due to its small size, minimal associated morbidity, and biocompatibility. Also, the DVRT is easily calibrated, and not dependent upon the tissue being measured.[81-85] Previous studies have used the DVRT sensor for *in vivo* measurement of anterior cruciate ligament strain in humans.[82-87]

Figure 1-2: Strain curve from the calcaneal tendon of a trotting dog using a DVRT

Biomechanical studies investigating tendon strain and forces have been performed in many species. Strain has been directly measured in vivo, and has been shown to vary depending on the implanted tendon and the position of the limb and joints, as well as the activity in which the animal is engaged. As an example, maximum tendon strain has been measured at 2.6% in the lateral digital extensor tendon of sheep at a trot.[88] Maximum strain in the superficial digital flexor tendon of walking horses has been measured between 3.1%-7.6%, and 6.5-10.1% at a trot, and 11.5-16.6% at a gallop.[89] Another study recorded 7.1% strain in the superficial digital flexor tendon of horses while standing, and 10.8% while walking.[90] No prior measurements of in vivo calcaneal tendon strain in the dog have been reported.

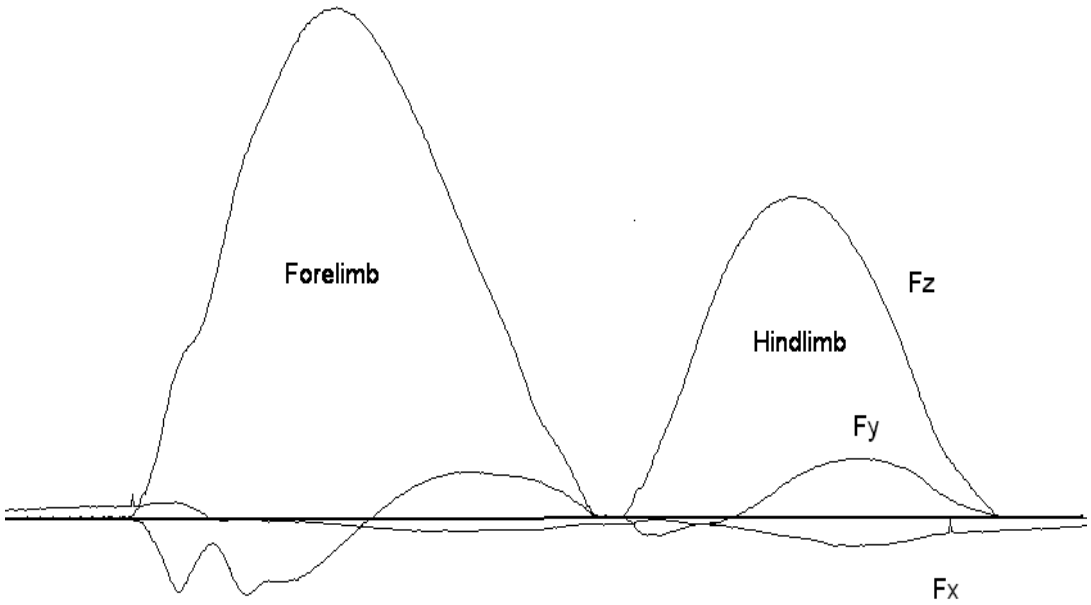
Previous studies have also investigated forces normally present in the calcaneal tendon. Tendon force has been measured in vivo in humans, rabbits and cats.[79, 91-95] Peak forces in feline gastrocnemius tendons have been shown to increase with increasing movement intensity, either through increasing speed or external resistance, ranging from 14.2-27N at a slow walk to 72.9-89.1N at a trot.[94] Calcaneal tendon forces in humans range from 489-661N during cycling[93], and from 1896-3787N during vertical jumping, which is 1.8-2.5 times the measured corresponding ground reaction forces.[79, 92] In vivo calcaneal tendon forces have not been reported in the canine.

Although tarsal immobilization is frequently used to limit calcaneal tendon strain, the associated muscles may still undergo contraction during weightbearing, resulting in continued tendon stress despite the elimination of movement at the joint (isometric contraction). By direct measurement of tendon strain, the efficacy of immobilization can be determined.

Ground Reaction Forces

Ground reaction forces are objective measures of lameness. They are obtained by converting the amount of force applied to the ground by the foot into electric signals. Measurements are then normalized for weight, to allow direct comparison between different animals, and between different times, as the weight of a single animal can vary. A force platform is most commonly used to obtain these measurements, although other instruments include pressure sensing mats or platforms. Ground reaction forces are three-dimensional vectors, and there are three vectors of forces that can be measured: vertical forces (Z), propulsive and braking forces (Y), and medial and lateral forces (X).[98,99] Ground reaction forces have been shown to be affected by velocity, gait, acceleration, morphological parameters, intra and inter-individual variation, the handler, training and pathological status.[100-104] During data collection, speed and acceleration are typically controlled to within pre-set parameters. It is also important to obtain a clean foot-strike, wherein only one foot contacts the force plate at a given time. Valid trials in canines typically are collected at a trotting gait, as peak vertical forces obtained at a walk can be less reliable in distinguishing between lame and non-lame dogs.[105,106]

Figure 1-3: Ground Reaction Forces from a trotting Canine



Vertical forces have been shown to be a more reliable and consistent criterion than any horizontal force components.[101, 107,108] Data that can be measured from ground reaction forces

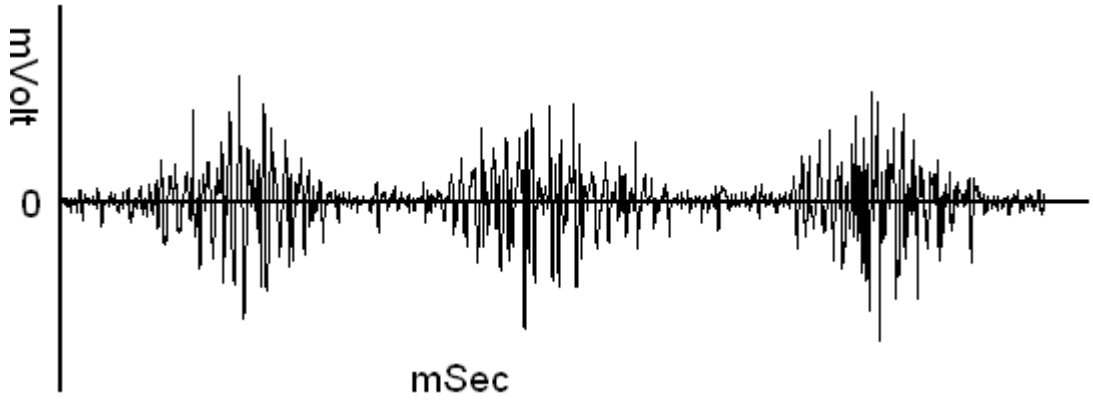
include the percentage of body weight placed during the footstrike, correlating to peak vertical force (Fz). Vertical impulse is also another commonly used measurement, and reflects the total force applied during the stance phase, or the integral of the area under the vertical force/time curve. Other data that is commonly collected are stance times, and loading and unloading rates, which can be obtained from the slope of the force curves.[98-100,107,108]

Since ground reaction forces are objective measures of lameness, they can be used to compare weight-bearing between limbs, or to assess for improvement in lameness after an interventional treatment or medication.

Surface Electromyography

Normal muscles are quiet at rest, without any electrical activity.[109] The source of the surface electromyographic (EMG) signal is the motor unit action potential. A motor unit action potential represents the summated electrical activity of the muscle fibres innervated by a single motor unit that are within the range of the recording electrode. Motor unit action potentials are characterized by their amplitude, phase and duration.[110] Compound muscle action potentials are the sum of the action potentials of the individual muscle fibres, and are the signal most commonly recorded using surface EMGs.[110]

Figure 1-4: Compound muscle action potentials recorded from surface EMGs



Needle electrodes provide exact and detailed information regarding individual motor unit action potentials, and the functional state of the lower motor neurons. They are mostly applied for differentiation of myopathic and neurogenic disorders in diagnostic electromyography.[111]

Surface electrodes are used to register the total activity of a muscle, and give a general impression of the electrical activity of the muscles over which they are placed.[111] The advantages of surface electromyography are that they provide a safe, easy and non-invasive method to allow objective quantification of the activity of a muscle. When placed over different muscle groups simultaneously, this allows the observer to see the muscle activity at rest, and the associated change during the course of a movement, as well as to differentiate how different muscles and muscle groups perform various movements.[112]

The measured signal undergoes differential amplification during recording. This is accomplished by using sensors with paired recording electrodes, as well as a ground reference electrode. The ground reference electrode is placed over a bony prominence with no muscle underneath. The energy reaching both recording electrodes is compared to the reference electrode, and only the signal that is unique to the recording electrodes is passed on for further signal conditioning.[112]

Limitations of surface EMGs include the possibility of "cross-talk", where electric signals from one muscle group travels over into the recording field of another muscle group, potentially making it difficult to isolate the surface EMGs from an specific muscle.[112] Also, variation in sensor placement, or movement or loss of contact of the electrodes during recording, will affect signal strength. The amplitude of surface EMGs is affected by the size of the muscle mass, the amount of energy exerted by the muscle, the overlying tissues between the electrode and the muscle (i.e. fat reduces signal) and skin impedance.[112] Skin impedance can vary depending on the moisture of the skin, superficial oil content, and the density of keratinized layer. It is important to keep the impedance of the skin as low as possible during surface electromyography – this is accomplished by abrading the skin vigourously with an alcohol pad prior to electrode placement.[112]

When applying surface EMG electrodes, it is also crucial to place the electrodes in areas with a minimum of tissue between the muscle and the skin surface. Electrodes should be placed with the dual sensors parallel to the muscle fibres to maximize sensitivity and selectivity. Electrodes should also be placed over sites with good anatomical landmarks, to facilitate reliable placement during later recording sessions. The electrode size should be appropriate to the muscle size so as to minimize "cross-talk". Direct contact or dry electrodes function best during quiet muscle contraction, as there is some risk in slippage of the electrodes during activity.

"Floating" or wet electrodes are designed to float 1mm above the skin surface, with a gel layer between the electrode and the skin. "Floating" electrodes are designed to minimize artifact from slippage during active motion.[112]

When evaluating the surface EMG signal, timing of the muscle activity can be correlated to the activity performed, or the phase of the gait cycle. Many attempts have been made to correlate strength of EMG signal to strength of contraction and force output. Correlation of EMG signal to force output is affected by many variables, including type of muscle fiber, range of force, and 'time-constant' of integration. Force output from a muscle has been correlated to the strength of the associated myoelectric signal.[113-116] In 1997, Savelberg and Herzog measured concurrent EMGs and tendon forces in cats, then devised an artificial neural network, which was successfully used to predict dynamic tendon forces in another cat based on EMG signal.[114] In 1990, van Ruijven and Weijs reported on the correlation of EMG strength to the bite force of the jaw muscles in rabbits, and showed a statistically significant correlation.[115]

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CHAPTER 2 - Efficacy of Tarsal immobilization to alleviate Achilles tendon strain in vivo – direct measurements via a DVRT strain gauge in a canine model

This portion of the thesis contains a manuscript submitted to *The American Journal of Veterinary Research* for publication detailing the results of the project.

Efficacy of Tarsal immobilization to alleviate calcaneal tendon strain in vivo

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Abstract

Objective: To measure in vivo strain in the calcanean tendon during trotting in canines, and to compare to strain present after tibiotarsal immobilization.

Animals: 6 canines

Procedures: A DVRT® strain gauge was surgically implanted on the common gastrocnemius tendon. Surface EMG, % strain, and ground reaction forces were measured prior to intervention and after immobilization. Peak vertical force (Fz), vertical impulse, initial, maximum and final

strain, and peak-to-peak EMG amplitude were recorded. Data was analyzed using repeated measures analysis of variance and paired t-tests ($p \leq 0.05$).

Results: Timing of the strain data correlated closely to the hind limb footstrike and EMG activity in all dogs. Maximum tendon strain occurred simultaneous with peak Fz. Continued muscle contraction was evident after immobilization. There was no statistical difference in maximum strain after immobilization compared to normal motion. Minimum strain, both at the beginning and end of the strain curve, was significantly decreased with the immobilized state compared to non-immobilized joints.

Conclusions and Clinical Relevance: Tibiotarsal immobilization did not eliminate calcaneal tendon strain during weight bearing. Decreased isometric muscle contraction during swing phase of the gait would account for smaller minimum strain in immobilized joints. Immobilization is frequently applied after Achilles tendon rupture to alleviate strain and force on the sutured repair, with possible complications due to the immobilization method. Direct correlation of strain with tendon force was not made in this study. This would be an important factor before adjusting current treatment recommendations.

Abbreviations:

Fz: Peak vertical force

VI: Vertical impulse

EMG: Electromyograph

DVRT: Differential Variable Reluctance Transducer™

Introduction

Calcaneal tendon injuries in dogs range from tendon lacerations and acute ruptures to chronic strain injuries. Severe damage to the calcaneal muscle-tendon unit results in a plantigrade stance in the affected hindlimb and marked mechanical lameness. Healing of tendon injuries is problematic, as controlled weight bearing and exercise restrictions can be difficult to achieve. Injuries of the tendon are typically repaired by debridement and reattachment via various suture techniques.

Optimum tendon healing requires some degree of strain along the length of the tendon to stimulate proper recovery, as the strength of collagenous tissue is positively affected by exercise, and lessened with immobilization.[1-3] Several studies have investigated the effects of immobilization on tendon healing. In rabbits and mice, faster maturation of collagen tissue is noted in non-immobilized groups, as well as more rapid and continuous restoration of load to failure, compared to immobilized groups.[4-6] Four months after sutured repair of calcaneal tenotomies, mice treated with immediate mobilization had regained their original tendon stiffness, whereas only 50% stiffness was regained in mice treated with immobilization.[5]

It is also important not to overstress a tendon anastamosis in the early post-operative period, as this leads to gap formation and/or repair failure. Tendons undergoing a gap greater than 3mm during the repair process heal at a slower rate, with decreased ultimate tensile resistance at 6 weeks compared to tendons healing with no gap or less than 3mm gap.[7,8] For this reason, the repair is typically protected for 6-12 weeks. Breaking tendon strength is slow to be regained after sutured repairs, regaining only 56% by 6 weeks, and 79% by 1 year post-operative.[9]

Various methods of tarsal immobilization and exercise restrictions are used to attempt to limit calcaneal tendon strain, with mixed results, as every technique has potential complications.[10-12] Associated muscles may still undergo contraction during weightbearing, resulting in continued tendon stress despite the elimination of movement at the joint (isometric contraction). By direct measurement of tendon strain, the value of immobilization can be determined.

In human medicine, calcaneal tendon injuries are treated post-operative with partial immobilization via hinged splints, allowing protected stress of the tendon unit and physical therapy throughout the healing period. Studies have demonstrated faster return to normal function, with less muscle disuse atrophy in these cases.[13,14] In comparison to dogs' upright stance, humans have a plantigrade stance, leading to different stresses on the calcaneal tendon. Also, post-operative activity can be more easily controlled in humans. For these reasons, use of partial immobilization is still in its infancy in dogs.

Biomechanical studies investigating tendon strain and forces have been performed in other species. Strain has been directly measured in vivo, and has been shown to vary depending on the implanted tendon, as well as the activity in which the animal is engaged.[15-17] Calcaneal tendon force has been measured in vivo in humans, rabbits, horses and cats.[17-22] Peak forces in feline gastrocnemius tendons have been shown to increase with increasing movement intensity, either through increasing speed or external resistance.[19] No prior measurements of in vivo calcaneal tendon strain or force in the dog have been reported.

The hypothesis tested was that tendon strain in the calcaneal tendon is partially but not completely eliminated by tarsal immobilization in animals with an upright stance, and that active muscle contraction continues to significantly contribute to tendon strain. The hypotheses were

tested by in vivo direct tendon strain measurement with an implanted strain gauge as well as surface electromyographic electrodes and measurement of ground reaction forces using a forceplate during ambulation, both before and after tarsal immobilization.

Materials and Methods

This project was approved by the Institutional Animal Care and Use Committee at Kansas State University.

Six purpose bred hound dogs were acclimatized with daily training for two weeks prior to beginning the study to accustom them to walking on a leash wearing a backpack, as well as to the force plate and the associated electromyographic leads and cables.

A single force plate[a] built into a 25-foot walkway was used in this study, operated with custom Acquire software (Version 7.5),[b] which allowed simultaneous integration of the electromyography and strain gauge data with the ground reaction force data. An 8-channel analog/digital laboratory acquisition system (DataLINK)[c] was used for the additional patient data acquisition. Multi-use surface electromyographic (EMG) sensors with built-in pre-amplifiers[d] were used to collect EMG data. Microminiature Differential Variable Reluctance Transducer™ (DVRT) strain gauges[e] and a DEMOD-DC™ signal conditioner[f] were obtained and modified for operation with the DataLINK system.

(Insert Figure 1)

The DVRT strain gauges and signal conditioner were factory-calibrated to correlate voltage with displacement, calculating a slope (millimeters / volt) and offset for each DVRT, establishing the linear range of the device. This process has been previously reported elsewhere.[23] The DVRTs used in this project had a functional stroke length of 3mm, over which the voltage output depended on the core position within that stroke length. The mid-point of the

voltage range could also be obtained with the core completely removed. To allow operation with the DataLINK system, the signal conditioner was modified by the manufacturer to bypass the internal voltage regulator and the electronics were upgraded, eliminating the output RC filter. Further modification was performed on site by incorporating a resistor network into the sensor. This attenuated the voltage signal by half, resulting in an operating range of 0-2.3970V, within the range of the DataLINK system (+/- 3V).

The reference length to be used in calculating strain was determined based on 2 measurements. First, an initial reference length was determined prior to implantation of the DVRT. The DVRT core was fully inserted to obtain the maximal voltage, and the distance between the insertion barbs was measured using digital calipers accurate to $1/100^{th}$ of a millimeter. Voltage at that length was recorded, providing the initial reference length. A second measurement was obtained after implantation of the strain gauge in each animal, and the difference between the two voltages was used to calculate the actual reference length.

Prior to surgical implantation of the strain gauge, an initial data set of ground reaction forces and surface electromyography was obtained from each patient. Trials were considered valid when patient speed fell within the parameters of 1.6 – 2.3 m/s, and both the ipsilateral forelimb and hind limb struck the force plate. Acceleration was kept between -0.5 to +0.5m/s/s. Results of valid trials were averaged for each measurement time period.

Animals were pre-medicated with acepromazine (0.02mg/kg IM) and morphine (0.5mg/kg IM), induced with thiopental (10-20mg/kg IV), then maintained under general anesthesia using isofluorane. After standard surgical preparation, an approach to the lateral aspect of the calcaneal tendon was performed, with incision of the tendon sheath. The DVRT was implanted onto the craniolateral aspect of the common gastrocnemius tendon, using the 2

barbed attachments – one proximal on the sensor, and one distal on the core. The DVRT was implanted with the calcaneal tendon in a zero-strain position under general anesthesia, with the tarsus maximally extended and stifle flexed. During implantation, voltage readings were obtained, and the barbs were fixed at a distance approximating the mid-portion of the functional range of the DVRT. Care was taken to ensure secure fixation of the DVRT into the tendon substance, and to avoid the superficial digital flexor tendon. The DVRT was further fixed in position to the tendon with 2 cruciate sutures of 3-0 polypropylene, one around each barbed attachment. The cable was tunneled proximally in the subcutaneous space, exiting just distal to the stifle, then secured in place using friction sutures. The incision was closed in routine fashion in two layers. Smooth 3/32" intramedullary pins were placed in the distal tibia and tuber calcani. Holes were pre-drilled with a 2mm drill bit, before placing the pins at low speed. The tarsus was immobilized by placement of a type II fixator frame using 2 carbon fiber rods and 4 large S-K clamps[g] at a normal standing angle.

When not in use, the DVRT cable and fixator were kept covered with a soft padded bandage. Post-operative, deracoxib (1-2mg/kg PO) was given every 24 hours for 5 days, and acetaminophen with codeine (1-2mg/kg PO) was given for 24-48 hours as needed for pain control. Animals were evaluated via complete physical examination and pain scoring twice in the first 24 hours after both placement and removal of the implants, then once daily afterwards for an additional 4 days. General attitude and degree of weight bearing were also evaluated 3-4 times daily for evidence of discomfort, while the fixator and strain gauge were in place. All animals appeared comfortable post-operative, and would almost fully weight bear while standing or at a slow walk. Due to the physical impediment of the fixator, some of the dogs would

initially lift the limb off the ground while at a fast walk or trot, yet when the fixator bars were removed, would almost fully weight bear.

After implantation of the strain gauge in each patient, voltage readings were obtained with the tarsus in a zero-strain position. Zero-strain was considered to occur with the patient under general anesthesia, eliminating active muscle contraction, and with the tarsus fully extended. Five voltage readings were averaged, to obtain the zero-strain reference. The difference between the zero-strain voltage and the pre-implantation voltage / reference length allowed computer calculation of the actual implanted reference length for each patient using the slope (mm/Volt) of the strain gauge curve. Tendon strain was then calculated using the equation in figure 2. *(Insert Figure 2)*

After implantation of the strain gauge, the initial data sets were collected. Data sets included ground reaction forces, surface electromyography and strain gauge data. Two data sets were collected - the first data set was collected with the external fixator frame in place, immobilizing the tarsus at a normal standing angle. The second data set was obtained after removal of the fixator bars, allowing free movement. Care was taken to ensure adequate weight-bearing at a trot based on comparison to pre-implantation peak vertical forces. If adequate weight-bearing was not present, repeat attempts were made twice daily until data could be obtained (ranged from 1 to 3 days). Non-immobilized and fixator-immobilized data were collected on the same day, to minimize the amount of variability related to possible dysfunction of the strain gauge or variation in the degree of weight-bearing.

Three surface EMG sensors were used in collection of EMG data. Sensors were placed over the lateral and medial gastrocnemius, and the cranial tibial muscle. The placement sites were prepared by clipping the hair and cleaning with an alcohol pad to remove surface oils and

keratin to decrease the surface impedance of the skin, and ensure good adherence of the electrodes to the site. Care was taken that EMG sensors were placed over the mid-portion of the muscle belly, with the two electrodes parallel to the muscle fibres. A ground reference electrode was placed over the olecranon, which was similarly prepared.

The dogs were sedated with acepromazine (0.02mg/kg IV) and morphine (0.25mg/kg IV). The pins were removed and a cast was placed from mid-tibia to below the tarsus, at a similar angle as previously immobilized. Angles were measured with a goniometer to ensure consistency. Measurements were attempted 6 hours later, however if adequate-weight bearing at a trot was not occurring due to the physical impediment of the cast, further measurements were attempted on subsequent days (up to 3 days after placement). The cast and strain gauge were removed under general anesthesia. After recovery, the dogs were eligible to be adopted.

For each scenario (non-immobilized, fixator immobilized, and cast immobilization), peak vertical force and impulse data were collected. After DVRT implantation, EMG data and strain gauge data were evaluated in reference to the hindlimb strike. Initial strain, maximum strain, and final tendon strain for each gait cycle were determined from the strain curve. Peak-to-peak amplitude was collected from the EMG data. Timing of EMG impulses and tendon strain were evaluated in reference to the hindlimb strike. Using tendon strain and EMG data, comparisons were made between the non-immobilized state and the two methods of immobilization.

Mean peak vertical force and impulse were calculated for each hindlimb pre-implant, post-implant, and post-immobilization for the fixator and for the cast. Mean start and stop strain, as well as peak strain were calculated for each patient in the implanted limb at each time-point. Repeated measures analysis of variance and Newman-Keuls multiple comparisons tests were run to evaluate for significant differences in peak vertical force and impulse between the 4 data time

points. Paired t-tests were run to compare post-implant and post-immobilization strain gauge data. Cast data could only be obtained from 2 dogs, due to failure of dogs to adequately weight bear at a trot. Due to this lack of adequate numbers, the cast data was not included in the statistical analysis. Differences were considered statistically significant for $p<0.05$.

Results

Six purpose-bred female hounds, ranging in size from 20.6-31.7kg, were used in this study. *(Insert Figure 3)*

After triggering the initial light sensor, a cyclical / waveform pattern corresponding to footstrikes was obtained from the strain gauges in all dogs during the 4 second recording period. This waveform was symmetrical, and consistent between trials. Occurrence of the waveform was closely correlated to the hindlimb footstrike. In some dogs, an initial rise in strain was evident at the initiation of the footstrike, followed by a further elevation in strain correlated to a burst of muscle activity detected by surface EMGs placed over the gastrocnemius muscles. Peak strain occurred simultaneously with peak vertical force, then strain decreased towards the end of the footstrike. A wide variation was present between dogs in measured percent strain, but within each dog, values were very consistent among all trials.

EMG data was variable between trials among dogs. No valid quantitative comparisons could be made concerning peak-to-peak amplitude of muscle contractions occurring in the immobilized and non-immobilized state. Occurrence of bursts of muscle electrical activity correlated well with the strain curve and hindlimb foot strike, with contraction of the gastrocnemius muscles consistently occurring at the initial moment of ground contact for the hindlimb. A second burst from the gastrocnemius was seen occurring at the end of the footstrike.

No significant difference in peak vertical force was present between pre-implantation and post-implantation, or between post-implantation and fixator immobilization. A significant difference was present between pre-implantation data and post-fixator immobilization. No significant difference in vertical impulse was present between the 3 time points. *(Insert Table 1)*

After implantation of the strain gauge, average maximum strain in the gastrocnemius tendon ranged from 2.31 to 12.99%. After immobilization of the tibio-calcaneal joint with an external fixator, average maximum strain in the gastrocnemius tendon ranged from to 2.34 to 12.55%. For the overall group, there was no statistical difference in average maximum strain between non-immobilized and fixator immobilized states. ($p=0.3174$)

After implantation of the strain gauge, average minimum initial strain in the gastrocnemius tendon ranged from 0.94 to 8.58%. Average minimum final strain in the curve ranged from 0.92 to 8.62%. After immobilization of the tibio-calcaneal joint with a fixator, average minimum initial strain ranged from -1.17 to 6.97%. Average minimum final strain ranged from -1.04 to 7.45%. Average minimum initial ($p=0.009$) and final ($p=0.010$) strain were significantly less in the fixator group compared to the non-immobilized group.

Discussion

No significant difference in maximum tendon strain was found between fixator immobilized and non-immobilized tarsi. Contraction of the gastrocnemius muscles was still evident during trotting, as evidenced by the EMG activity. These findings support the original hypothesis that calcaneal tendon strain persists post-tarsal immobilization due to the effects of isometric muscle contraction during weightbearing.

The percentage of weightbearing and type of activity would be expected to affect the forces and strain in a tendon.[16,17,19,20] From measured ground reaction forces, dogs were

significantly lame after implantation of the strain gauge. However, as no significant difference in ground reaction forces was present between the immobilized and non-immobilized scenarios, this should not affect our comparisons.

Although no difference in maximum strain was present, minimum strain at the beginning and end of the curves was significantly lower in the fixator immobilized tendons. These findings could be explained by a decreased need for muscle contraction to hold the tarsus fixed during the swing phase of the gait, as the fixator takes over this function. Decreased muscle contraction would lead to decreased magnitude of strain in the tendon during this time.[24-27]

Wide variability in magnitude of surface electromyograph curves was present in our study. This was likely related to difficulty in maintaining good adhesion of the EMG sensors between trials. Quantitative analysis of EMG size, therefore, was not possible. Electromyographs were useful to visualize the timing of muscle bursts in correlation to the occurrence of the hindlimb footstrike and rise in tendon strain. Previous research regarding timing of the EMG burst in the medial gastrocnemius muscle in cats, has shown that the primary burst of activity occurs before foot contact. During stance, a second burst of activity occurs, and is responsible for the residual tension in the muscle.[27] Similar timing of EMG activity was also seen in our study, with an initial burst of activity in the gastrocnemius muscles occurring simultaneous to the initial footstrike, and a second burst visualized later. This demonstrated that muscle contraction persisted in the immobilized limbs.

The original goal was to obtain data sets after immobilization both with a cast and with a fixator. We experienced difficulty in obtaining full data sets for the cast-immobilized tarsi. Many of the dogs would not adequately bear weight in a cast at the appropriate speed and gait for

the force platform analysis. Use of pressure platform for analysis might have allowed analysis of data at a slower speed.[28]

The DVRT was selected due to its small size, minimal morbidity, and biocompatibility. Previous studies have used the DVRT sensor for in vivo measurement of anterior cruciate ligament strain in humans.[23,29,30] A zero-strain reference needed to be established for each animal to allow accurate strain calculations. The optimal zero-strain reference would be based on the length of the ligament just as it begins to bear load (slack-taut transition). However, it is difficult to determine the true neutral length without destructive sectioning.[31] We chose to establish our zero strain reference with the animal under anesthesia, and the tarsus fully extended, as this would simulate zero strain and was a reproducible position. Many other investigators have used arbitrary references based on particular joint position.[32,33] These references provide a value of relative strain or percent elongation, and are considered useful to compare peak strains of a ligament under different conditions in a particular study.[31] Since the zero-strain reference was re-established for each dog, and comparisons were made between the two scenarios based off the same reference measurement, this was considered valid.

A wide range in measured tendon strain was obtained between different animals, with maximum measured tendon strain ranging from 2.31-12.99%. This variability between animals can be explained by several factors. Individual dogs varied in size and degree of weightbearing, which could be expected to have some effect on tendon force and strain.[16,17,19,20] Also, strain distribution within a tendon is not consistent along the length of the tendon, as strain increases near the tendinous insertion.[34] Care was taken to place the strain gauge in the mid-point of the tendon, to limit this effect, but some variability due to position is likely.

When working with animals, exposed equipment remains vulnerable to damage. A new strain gauge was not used for every dog, and one strain gauge was used several times before being damaged prior to removal. Minor fatiguing of the cable could have affected the voltage obtained prior to final destruction leading to variability in maximum strain between dogs. To minimize this possible effect on our results, efforts were made to collect data for the immobilized and non-immobilized state on the same day. As trials for individual dogs were very consistent in the level of measured strain and each dog acted as its own control, the variability between dogs was not considered important.

The main limitation of this study was the lack of correlation of measured strain to force within the tendon. Tendon strain is directly related to the cross-sectional area of the tendon, the tendon stiffness (modulus of elasticity) and the force applied. However, direct calculation of tendon force from strain is inaccurate. As tendons are viscoelastic structures, the modulus of elasticity will be strain dependent. As higher strain is applied, there is greater resistance to tension.[35] Strain is standardly calibrated to tendon force using post-mortem methods, by comparing measured strain with known weights.[17,30,31] Knowledge of the amount of force present within the tendon during ambulation would allow direct correlation to force withstood by a sutured repair. Direct correlation of strain with tendon force would be an important factor before adjusting current treatment recommendations for immobilization after tendon repair.

Conclusion

Tibiotarsal immobilization is frequently applied after Achilles tendon rupture to alleviate strain and force on the sutured repair, with possible complications due to the immobilization method. As demonstrated in this study, immobilization has no effect on maximum strain in a weight-bearing situation. Clinical benefits may result more from decreased weight-bearing due

to the method of immobilization, as well as exercise restrictions. Direct correlation of strain with force within the tendon was not made in this study. Future research investigating the corresponding forces present in the calcaneal tendon prior to and post-immobilization, as well as the degree of force and strain that can be withstood by a sutured repair would be important information, before adjusting current treatment recommendations.

Footnotes

a. Kistler force plate, Kistler Instrument Corp., Amherst, NY
b. Acquire, Version 7.5, Sharon Software, DeWitt, MI
c. DataLINK DLK900, Biometrics, Cwmfelinfach, Gwent, UK
d. Surface electromyographic sensors, SX230, Biometrics, Cwmfelinfach, Gwent, UK
e. Differential Variable Reluctance Transducer™ strain gauge, Microstrain, Burlington VT
f. DEMOD-DC™ signal conditioner, Microstrain, Burlington VT
g. S-K clamps, Imex™ Veterinary, Inc., Longview TX

Figures and Tables

Table 2-1 Averaged data for all animals and trials. Values denoted with the same letter are not significantly different. Denoted with consecutive letters are significantly different

Time	pFz	VI	Initial strain	Peak strain	Final strain
Pre-implant	67.88±10.19 [a]	9.56±1.96 [a]			
Post-implant	58.18±3.32 [ab]	7.94±0.91 [a]	4.57±3.18 [a]	7.69±4.61 [a]	4.59±3.16 [a]
Fixator	52.95±12.98 [b]	7.76±3.11 [a]	3.34±3.60 [b]	6.70±4.37 [a]	3.31±3.55 [b]

Figure 2-1: DVRT strain gauge

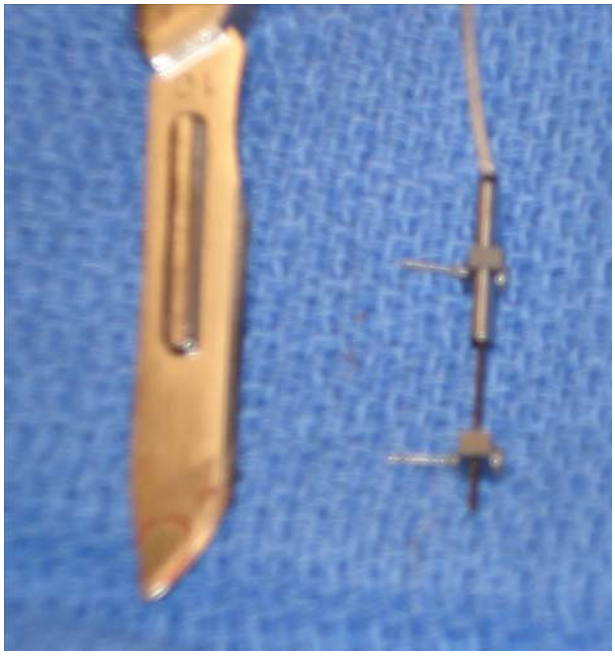
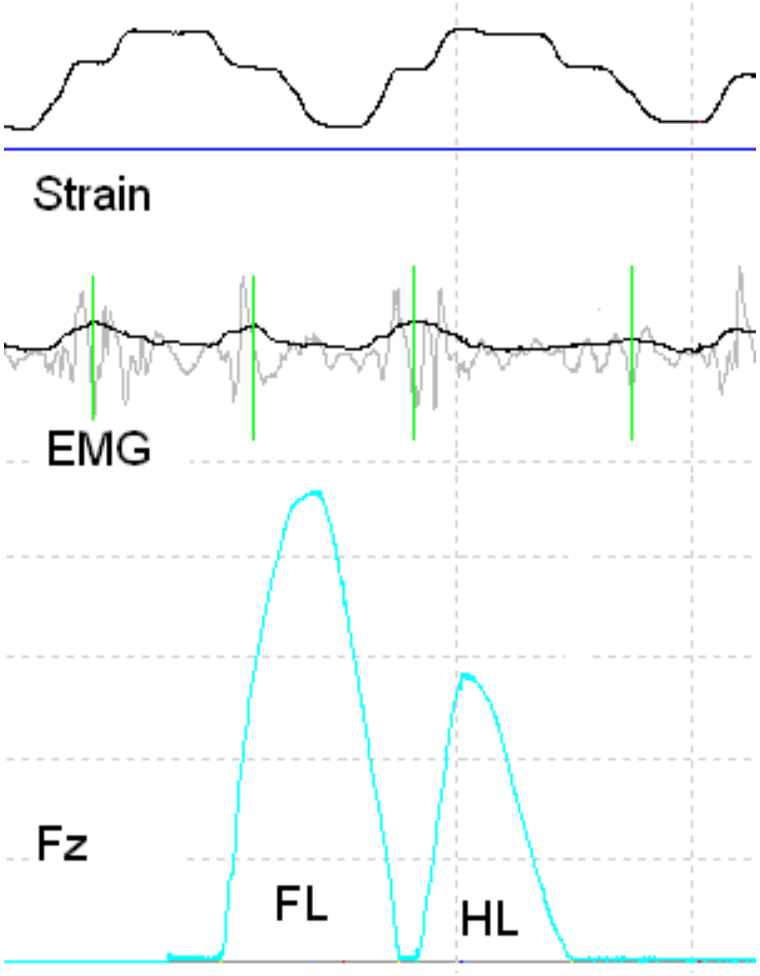


Figure 2-2: Calculation for determining % strain

$$\% \text{ strain} = \frac{\Delta \text{ length}}{\text{Reference length}} = \frac{L - L_o}{L_o} \times 100$$

Figure 2-3: Output from a single trial demonstrating timing of curves. EMG data is from the lateral gastrocnemius muscle



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