The Equine Tendon in Health and Disease

Henry Jann
Editor

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nova publishers
New York

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Library of Congress Cataloging-in-Publication Data

The equine tendon in health and disease / editor, Henry Jann (College of Veterinary Medicine, Oklahoma State University, Stillwater, OK, USA).
pages cm
Includes index.
ISBN: 978-1-62808-060-5 (eBook)
1. Tendons. 2. Tendons--Wounds and injuries. 3. Horses--Wounds and injuries. 4. Horses--Health. I. Jann, Hank W., editor of compilation.
SF959.T47E68 2013
636.1'0897474044--dc23
2013018499

Published by Nova Science Publishers, Inc. † New York

To my father: A far better scholar than myself. He taught me how to hunt and how to work but never how to quit.

To Donna: Horsewoman, and healer of countless creatures both great and small. She personifies everything that is good about the veterinary profession.

The Village Blacksmith, Henry Wadsworth Longfellow:

Toiling, rejoicing, sorrowing,
Onward through life he goes;
Each morning sees some task begun,
Each evening sees it close;
Something attempted, something done,
Has earned a night's repose;
Thanks, thanks to thee, my worthy friend,
For the lesson thou hast taught!
Thus at the flaming forge of life
Our fortunes must be wrought;
Thus on its sounding anvil shaped
Each burning deed and thought.

Hank Jann

CONTENTS

PREFACE

This book is designed to provide information on all aspects of clinical management of tendon problems in horses. The chapters cover the spectrum of understanding structure and function, diagnostic modalities, treatment options, rehabilitation strategies, etiology of injury, and prevention of tendon injury. All authors have vast clinical experience and are among the most qualified in their respective areas of expertise. It is the editor's goal to provide the reader with a state of the art resource that will aid in dealing with tendon problems. This includes diagnosing and treating the acute injury to assessment of healing and formulating a rehabilitation strategy. All of these aspects are important if an optimal outcome is to be achieved because treating tendon injuries is not a simple exercise. Each injury is unique and each horse is unique. No one treatment or rehabilitation strategy can be universally applied. All injuries must be carefully diagnosed and monitored throughout the rehabilitation process. This book is a guide for the prevention, diagnosis, and treatment of tendon injuries that is practical, reliable, and easily understood. It will hopefully allow veterinarians to deal with tendon injuries with more confidence and a higher quality of post injury return to function.

Chapter 1 – Tendons are a specialized form of dense fibrous connective tissue composed of fibers and extracellular matrix having a parallel fiber arrangement. Tendon tissue is highly organized. Tendon has great tensile strength as a result of a parallel fiber arrangement; not only within the tendon but to the muscle associated with it. This specialized form of connective tissue unites bone to muscle, unites muscle to muscle, and covers tissue. The parallel fiber arrangement allows for great tensile strength in the tendon due to it being able to resist stretching in one direction. The ability to withstand tensile forces allows the tendon to transmit forces generated by a contracting muscle to the bone which allows for movement and stability in the joint.

Chapter 2 – The vast majority of tendon and ligament pathologies to date are predominantly evaluated with ultrasound and magnetic resonance imaging. Ultrasound offers widespread availability, exquisite spatial resolution, cost-effectiveness, logistic ease of imaging, guided needle placement, immediate visualization, visualization of proximal limb structures, and is usually performed standing. The technology continues to advance, providing improved abilities to diagnose injuries and follow the repair process. Techniques to improve diagnosis and interpretive skills are discussed, as well as notable limitations. Magnetic resonance imaging (MRI) is a vastly different imaging technique, and considered the gold standard for musculoskeletal imaging in some circles. Although expensive, with limited access and sometimes requiring general anesthesia, ligaments and tendons are very well assessed on MRI, and many practitioners consider an evaluation critical for optimal treatment

and clinical outcome. Size, shape, architecture, and quality of collagen matrix are evaluated in all soft tissues included in the study, as well as any bone and joint pathologies. Many lesions seen on MRI cannot be visualized ultrasonographically or radiographically. Several common reasons to request an MRI exam include accurate and thorough diagnosis following an acute or chronic lameness, assess additional complicating lesions following a known soft tissue or bony injury, and assess the presence and quality of repair after injuries. Multiple lesions are frequently found on MRI, and some equine practitioners experienced with MRI evaluations report much improved return to function after addressing all significant lesions.

Chapter 3 – While the treatment of acute soft tissue injury is common in equine sports medicine, many different opinions exist as to the best approach to optimize healing. The divergence of treatment methods is the result of variation of injury, individual experience and conflicting scientific literature. Previous attempts usually have focused on the merits of a specific modality. This chapter presents an outline of therapeutic approach that can be modified as future research illuminates merits and disproves assumptions of specific elements of the treatment program. Current germane literature was reviewed and presented to demonstrate the current understanding and rationale for use in the treatment of acute tendon injury. The chapter provides an open approach for the treatment of the acute tendon while allowing individual preference for specific injuries which can later be modified as newer information becomes available.

Chapter 4 – Horses have to be able to function at one hundred percent of their capabilities in order to perform in the highly competitive field of performance horses. When injuries occur, horses must be assisted back into health quickly and restored to normality. Regenerative medicine, new surgical techniques, new diagnostic imaging, therapeutic laser, acupuncture, shock wave therapy and a better understanding of rehabilitation has lessened convalescent time and improved the quality of healing. No longer are stall rest and anti-inflammatory agents the only treatment options. By no means, do the new therapies replace surgical intervention or all of the traditional therapies; many times the new therapies are used simultaneously or as a supplemental therapy.

Chapter 5 – Given the general anatomy and athletic ability of horses, conditions involving the soft tissue structures of the limb are commonly seen in equine practice. These soft tissue injuries can be potentially devastating to the athletic career of the horse, in addition to being financially burdensome to the client. Currently there are multiple treatment regimens available to attempt to help resolve these conditions and help achieve the ultimate goal of getting the horse back to work. They include but are not limited to cold therapies, compression therapy, extracorporeal shockwave therapy, therapeutic ultrasound, low level laser therapy, topical counter-irritation, systemic anti-inflammatory drugs, intralesional injections, controlled exercise programs and surgical interventions. This chapter will focus on some common surgical treatments for equine tendons and ligaments.

Chapter 6 – The importance to the horseracing world of Superficial Digital Flexor (SDF) Tendonitis was recently made very clear when I'll Have Another was forced to scratch from the 2012 Belmont Stakes. This important tactical decision not only cost the horse and his connections a chance of winning the prestigious Grade 1 Stakes, but forfeited I'll Have Another's probable potential for capturing the elusive Triple Crown. That feat hasn't been repeated since Affirmed won it in 1978; some 34 years ago. However, trainer Doug O'Neill and owner J. Paul Redman are to be complimented on their difficult decision not to jeopardize their horse and rider, or the other contestants in the race. Especially with so much money and

prestige at stake, the decision to scratch such a horse from a race, or to retire a horse from any other form of competition due to a damaged tendon or suspensory isn't easily made; however, of even greater challenge is the course chosen for trying to rehabilitate that horse and bring it back to its previous level of performance.

Chapter 7 – Preventing tendon injuries is a goal that is critical to any conditioning strategy. This chapter discusses the common etiologic factors associated with tendon injuries. Unfortunately some of these factors are difficult to avoid in the context of a rigorous competitive career. The more we are aware of these predisposing factors; hopefully the better we will be able to avoid them. The longevity of an athlete's career is directly related to the ability of avoiding injury. This chapter discusses the factors that cause tendon injury and how to avoid them.

Chapter 8 – Rehabilitation is crucial to an athlete's recovery from injury or illness. In humans, doctors prescribe physical therapy as an essential component of the patient's recovery after surgery or illness. Without adequate rehabilitation, science has shown that outcomes can be disappointing. In equine veterinary medicine, rehabilitation plays a vital role in returning a horse to its occupation or, in extreme cases, to restore its quality of life after a career-ending injury. Recently, the philosophy of rehabilitation has evolved to emphasize "prehabilitation," a protocol that addresses potential problems before they compound to diminish performance.

ACKNOWLEDGMENT

Special thanks to John Bennett for technical and literary assistance with this work.

In: The Equine Tendon in Health and Disease
Editor: Henry Jann
ISBN: 978-1-62808-048-3

Chapter 1

TENDON STRUCTURE AND FUNCTION

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ABSTRACT

Tendons are a specialized form of dense fibrous connective tissue composed of fibers and extracellular matrix having a parallel fiber arrangement (Weintraub 1999; Whiting and Zernicke 2008). Tendon tissue is highly organized (Elliott 1965; Whiting and Zernicke 2008).Tendon has great tensile strength as a result of a parallel fiber arrangement; not only within the tendon but to the muscle associated with it (Whiting and Zernicke 2008). This specialized form of connective tissue unites bone to muscle, unites muscle to muscle, and covers tissue (Lin, Cardenas et al. 2004; Levangie and Norkin 2005; Sharma and Maffulli 2006; Benjamin, Kaiser et al. 2008; Whiting and Zernicke 2008). The parallel fiber arrangement allows for great tensile strength in the tendon due to it being able to resist stretching in one direction (Whiting and Zernicke 2008). The ability to withstand tensile forces allows the tendon to transmit forces generated by a contracting muscle to the bone which allows for movement and stability in the joint(Marlin and Nankervis 2002).

STRUCTURE OF CONNECTIVE TISSUE

Tendon is a dense regular connective tissue with a white appearance (Weintraub 1999; Whiting and Zernicke 2008; Nunley 2009). The composition and organization lend to the tissue's ability to resist high loading of the unidirectional nature (Thorpe, Clegg et al. 2010). The dry mass of normal tendon is approximately 30% of the total mass (Sharma and Maffulli 2005) while water makes up the remaining 60-80% of the total mass (Weintraub 2003; Sharma and Maffulli 2005). Normal healthy tendons are mostly composed of parallel arrays of collagen fibers packed closely together in an extracellular matrix (Weintraub 2003). ECM

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supports most tissues and helps to give cells outer structure (Dowling, Dart et al. 2000). The ECM (extracellular matrix) is composed of collagen I fibers, elastic fibers, ground substance, and inorganic substances (Weintraub 1999; Kannus 2000; Whiting and Zernicke 2008; Nunley 2009). Collagen fibers, tough bundles of collagen, are the major element of the extracellular matrix (Dowling, Dart et al. 2000). The composition of tendon is of about 85% collagen, 2% elastin, 1% proteoglycans, and 0.2% inorganic components such as copper, manganese, and calcium (Kannus 2000; Vogel 2003; Levangie and Norkin 2005; Sharma and Maffulli 2006). Ground substance links to collagen to form the connective tissue, helps facilitate metabolism, provides support, decreases friction, and attracts water (Kannus 2000; Benjamin, Kaiser et al. 2008; Thorpe, Clegg et al. 2010).

Collagen is the main protein of connective tissue in animals and the most abundant protein in mammals (Dowling, Dart et al. 2000). It can be found in loose connective tissue, dense connective tissue, reticular connective tissue, bone, and cartilage (Kjaer 2004; Zhang, Young et al. 2005). Production of collagen involves the sequencing of three polypeptide chains to form a triple helix (Kjaer 2004). The collagen portion of tendon is comprised of between 65-80% type I collagen with smaller amounts of collagens III, IV, and V (Levangie and Norkin 2005; Sharma and Maffulli 2006). The content and organization of collagen reflects the mechanical properties allowing it to resist mechanical loading (Rumian, Wallace et al. 2007). The triple helix coils tight, resisting stretching when it is placed under tension making collagen invaluable for structure and support (Kjaer 2004). The higher amounts of Type I collagen with the lesser amounts of Type III collagen suggests that tendon has the ability to adapt to large tensile forces (Levangie and Norkin 2005). The remaining component in the tendon is known as ground substance (Kannus 2000). Ground substance is composed of proteoglycan, glycosaminoglycan, and aggrecan (Scott, Orford et al. 1981; Kannus 2000; Raspanti, Congiu et al. 2002; Vogel 2003). It is non-fibrous (Weintraub 2003).

Proteoglycans are composed of a protein core with glycosaminoglycans attached (Kannus 2000; Rumian, Wallace et al. 2007).These structures are mainly responsible for the viscoelastic nature of tendon (Benjamin, Kaiser et al. 2008). Proteoglycans also help regulate fibrillogenesis and help to organize the matrix (Thorpe, Clegg et al. 2010). The proteoglycans are interwoven within the collagen fibrils (Scott, Orford et al. 1981; Rumian, Wallace et al. 2007). It is believed that the proteoglycan content allows for greater slippage between collagen fibrils and fascicles allowing for greater tissue deformation to occur without increasing stress-strain levels (Rumian, Wallace et al. 2007). While intimately involved with collagen, the proteoglycans do not make any contribution toward the tensile strength of the tendon (Benjamin, Kaiser et al. 2008) but do contribute to its viscoelastic properties (Rumian, Wallace et al. 2007) and the ability to resist compressive forces (Kjaer 2004). Two types of proteoglycans have been found in tendon: small and large (Yoon and Halper 2005). Small proteoglycans influence tenocyte function, collagen fibrillogenesis, and fiber organization (Dowling, Dart et al. 2000). Large proteoglycans allow for the rapid diffusion of water-soluble molecules and the migration of cells (Yoon and Halper 2005).

Glycosaminoglycans concentration in tendon is considerably less when compared to other tissue (Kannus 2000). Glycosaminoglycans are polysaccharides present on the cell surface, in the ECM, or in association with proteoglycans (Rumian, Wallace et al. 2007). The major glycosaminoglycan (GAG) components of the tendon, derman sulfate, is thought to be responsible for forming associations between fibrils, and chondroitin sulfate is thought to be involved with occupying volume between the fibrils to keep them separated and help

withstand deformation. It also associates with collagen and is involved in the fibril assembly process during tendon development (Scott 2003). When decorin molecules are bound to a collagen fibril, their dermatan sulfate chains may extend and associate with other dermatan sulfate chains on decorin that is bound to separate fibrils, therefore creating interfibrillar bridges and eventually causing parallel alignment of the fibrils (McNeilly, Banes et al. 1996). Within the ECM, side chains of glycosaminoglycans have multiple interactions with the surface of the fibrils, showing that the proteoglycans are important structurally to the interconnection of the fibrils (Scott, Orford et al. 1981).

Tendon is also composed tenocytes and tenoblasts along with the extracellular matrix (Sharma and Maffulli 2006). The tenocytes and tenoblasts are known as tendon cells. Together, tendon cells encompass 90-95% of the cellular elements of the tendon (Kannus 2000). Tenocytes are specialized fibroblasts that can be found within the fascicles of tendon collagen(Weintraub 2003). Tenocytes are mature tendon cells. They are a low metabolic rate aged version of the tenoblast (Sharma and Maffulli 2006). Low metabolic rate within the tenocytes enhances the ability to carry load and maintain tension for sustained periods (Sharma and Maffulli 2005). Tenocytes are responsible for the secretion of the ECM and for collagen assembly and turnover (Benjamin, Kaiser et al. 2008). By contrast, tenoblasts come in different shapes and sizes with a high metabolic (Kannus 2000; Sharma and Maffulli 2006). Tenoblasts are immature tendon cells (Sharma and Maffulli 2005). As the tenoblasts mature, they will become spindle-shaped like tenocytes (Kannus 2000).

Blood Supply

The presence of blood supply is extremely important to the normal function of tendon cells and to the tendon repair process (Benjamin, Kaiser et al. 2008). The amount of blood available to the tendon is considerably less than the high metabolic rate muscles associated with that particular tendon (Benjamin, Kaiser et al. 2008). Tissues adjacent to tendon are more vascularized when compared to tendon (Benjamin, Kaiser et al. 2008). Vessels need to be long and winding to accommodate the great length changes that occur during movement (Kjaer 2004). Reduced blood supply can lead to tendon degeneration (Benjamin, Kaiser et al. 2008).

Blood supply to the tendon is through either an intrinsic or extrinsic system (Sharma and Maffulli 2005). In sheathed tendons, the mesotendon carries the extrinsic blood supply to the tendons. Major vessels at the sheath of the tendon pass through vincula to reach visceral sheet of synovial sheath where they form a plexus to supply superficial tendon areas (Sharma and Maffulli 2005). Blood vessels within the tendon are small and thin walled and may lie within the inter-fascicular grooves and run longitudinally (Benjamin, Kaiser et al. 2008). Vessels mainly originate from the epitendon and run longitudinally into endotendon penetrating the epitendon to form a connection between the tendinous vascular networks (Kjaer 2004). Blood vessels run parallel to the fibers within the endotendon with occasional branching transverse blood vessel connectors (Maffulli and Almekinders 2007). Non-sheathed tendons are covered by paratendon allowing the tendon to glide and supplies the extrinsic blood supply.

In order to provide optimal levels of blood, the endotendon relies on three possible sources for blood: musculotendenous insertion, osteotendenous insertion, and through the paratendon/synovial sheath (Sharma and Maffulli 2005). Musculotendenous and

osteotendenous insertion sites supply arterial blood supply to the tendon (Kjaer 2004). But, vascularity can be compromised at the junction zone insertion sites as well as at torsion, frictional, and compressive sites that the tendon encounters (Sharma and Maffulli 2005). Long tendons are supplied by several vessels running their length (Kjaer 2004). In the case of the Achilles tendon of the human, blood is supplied to the superior aspect is via the gastrocsoleus muscle complex and inferiorly by the bone tendon insertion into the calcaneous (Maffulli and Almekinders 2007). The blood vessels from the muscle-tendon insertion continue to 1/3 the length of the tendon.

Innervation

The internal tendon bulk is thought to contain no nerve fibers, but the epi- and peritendon contain nerve endings, while Golgi tendon organs are present at the junction between tendon and muscle. Nerves originate from cutaneous, muscular, and peritendinous nerve trunks (Sharma and Maffulli 2005). Nerve fibers cross over thru the musculotendinous junction and enter the endotendon, form plexues in the paratendon, and branches in the epitendon (Sharma and Maffulli 2006). Most nerves end on the surface of the tendon as nerve endings rather than penetrating the tendon proper (Sharma and Maffulli 2005). Both sympathetic and parasympathetic fibers are present in tendon (Sharma and Maffulli 2006). Nerve endings are the golgi tendon organs (GTO) (Sharma and Maffulli 2005). Nerve endings are myelinated and unmyelinated. Myelinated nerve endings form the golgi tendon organs (Sharma and Maffulli 2006). Nerve supply of tendon has nocioceptive and proprioceptive qualities (Benjamin, Kaiser et al. 2008). GTO detect changes in tension and pressure within the tendon (Sharma and Maffulli 2006). Unmyelinated nerve endings sense and transmit pain through nocioception (Sharma and Maffulli 2005).

TENDON ARCHITECTURE

The tendon itself is a collection of collagen arrangements that follow an increasing hierarchy of complexity (Sharma and Maffulli 2005). The collagen arrangements organize to form fibers. The collagen fibers aggregate end-to-end and side-to-side to produce collagen fibrils known as tropocollagen (Kannus 2000; Sharma and Maffulli 2006) The soluable tropocollagen converts to insoluble tropocollagen by forming cross-links (Sharma 2006). The insoluble tropocollagen is now ready to construct the necessary levels to form tendon. The beginning level, tropocollagen, align in parallel rows to form a microfibril bundles (Benjamin, Kaiser et al. 2008; Whiting and Zernicke 2008). Five tropocollagen bundles form the microfibril (Benjamin, Kaiser et al. 2008). Microfibrils aggregate into parallel bundles to form fibrils (Benjamin, Kaiser et al. 2008; Whiting and Zernicke 2008). The fibrils first form subfibrils and then fibrils (Whiting and Zernicke 2008). The fibrils take on a very complex triple helix formation where the fibrils are oriented longitudinally, transversely, and horizontally (Kannus 2000). The fibrils are grouped and form a subfascicle (Elliott 1965). Each subfascicle ranges from 15m to 400m in diameter (Kjaer 2004). Endotendon also binds subfasicles together creating the fascicle (Elliott 1965). The small bundle of fibers grouped

together form a fascile (Vogel 2003; Levangie and Norkin 2005). Fasicles are associated with discrete groups of muscle fibers or motor units (Levangie and Norkin 2005). The diameter of each fascicle varies from 150µm to 1000µm. Fascicles are bound together by the endotendon (Levangie and Norkin 2005; Whiting and Zernicke 2008). Proteoglycans lie between the endotendon and the fascicle to improve binding of these structures together (Kannus 2000).

Endotendon is a thin sheath of loose connective tissue (Whiting and Zernicke 2008) and ranges in diameter from 1mm to 3mm (Kjaer 2004). This loose connective sheath aids in the innervation and vascularization of the deeper tendon regions (Elliott 1965). Endotendon-bound fascicles form tertiary fiber bundles (Sharma and Maffulli 2006). The fascicle bundles slide independently from each allowing for better force transmission through joint angle changes and shape change with muscle contraction (Benjamin, Kaiser et al. 2008). Finally, a dense layer of loose connective tissue called the epitendon surrounds the tertiary fiber bundles to form an individual tendon (Sharma and Maffulli 2006). Epitenon is the white fibrous sheath surrounding a tendon (Levangie and Norkin 2005; Whiting and Zernicke 2008). The epitendon contains the vascular, lymphatic, and innervations for the tendon (Sharma and Maffulli 2006).

Paratenon is the connective tissue structure attached to and surrounding a tendon. It is a type of loose connective tissue that fills the spaces of the fascial compartment in which a tendon is situated (Levangie and Norkin 2005; Whiting and Zernicke 2008). The format of this structure allows the tendon to move freely. The paratendon is not organized into discrete tendon sheaths. The paratendon protects the tendon, enhances tendon movement, and its cells are used during the healing process. The paratendon may become a synovial filled sheath (Levangie and Norkin 2005). The synovial sheath develops in areas of increased stress that need efficient lubrication of the tendon for movement (Sharma and Maffulli 2006). The synovial sheath comes in two parts. An outer sheath which forms the pulley system acting as a fulcrum for movement and an inner sheath that produces synovial fluid (Sharma and Maffulli 2006). Tendons on occasion form "intermediate tendons" to connect muscle to muscle, "intramuscular tendons" to allow for the pennate arrangement of muscle, or 'flattened tendons", aponeuroses, as either fibrous sheets on the surface or within a muscle to assist with force transmission (Benjamin, Kaiser et al. 2008).

ENTHESES - OSTEOTENDINOUS JUNCTION AND MUSCULOTENDINOUS JUNCTION

A unique characteristic of tendon is the manner in which it inserts into muscle and bone. Enthesis is the specific term for the area of actual attachment or insertion site (Levangie and Norkin 2005; Sharma and Maffulli 2006). The specific name of the enthesis relates back to the tissue types being "joined" together. The osteotendinous junction (OTJ) creates a transition from tendon to bone or periosteum and the musculotendinous junction (MTJ) creates the transition from tendon to muscle (Rufai, Ralphs et al. 1995; Levangie and Norkin 2005; Whiting and Zernicke 2008). Entheses come in direct and an indirect forms (Doschak and Zernicke 2005; Sharma and Maffulli 2006; Whiting and Zernicke 2008). The direct, or fibrous, form is usually associated with the OTJ while the indirect, or fibrocartilaginous, form is associated with the MTJ (Sharma and Maffulli 2006). These entheses provide a gradual

transition from tendon to bone or muscle (Rufai, Ralphs et al. 1995; Levangie and Norkin 2005). The approach of the tendon into the insertion site is at an oblique angle (Sharma and Maffulli 2006) helping with tendon function. The oblique approach along with the gradual change across the enthesis aids in load transfer (Levangie and Norkin 2005).

OTJ is a direct form of insertion as bone and/or perisosteum is present (Sharma and Maffulli 2006). The OTJ enthesis has two distinct categories of insertion based upon tissue type present at the attachment site: fibrous and fibrocartilaginous (Levangie and Norkin 2005; Sharma and Maffulli 2006). The fibrous category directly attaches the tendon to the bone or indirectly to it via the periosteum (Sharma and Maffulli 2006). These two attachment means are known as bony and periseal, respectively (Levangie and Norkin 2005). Early contact of tendon with the bone prior to insertion on the bone helps decrease the load stress at the insertion(Sharma and Maffulli 2006). The transition from tendon to bone occurs over four distinct zones with no sharp boundaries (Rufai, Ralphs et al. 1995; Doschak and Zernicke 2005; Levangie and Norkin 2005; Sharma and Maffulli 2006; Whiting and Zernicke 2008). The first zone is still tendon proper and is similar to mid-tendon substance (Rufai, Ralphs et al. 1995; Levangie and Norkin 2005). This zone has a parallel fiber arrangement (Doschak and Zernicke 2005) and is populated with fibroblasts (Whiting and Zernicke 2008). It is interesting to note that Zones 1 and 4 signify the difficulty defining with any precision where the insertion begins or ends (Sharma and Maffulli 2006). The second zone is fibrocartilage (Benjamin, Toumi et al. 2006). This zone is the actual beginning of the transition to bone (Levangie and Norkin 2005). This zone is characterized by fibrocartilage that is populated by chondrocytes within lacunae (Whiting and Zernicke 2008). The tissue retains the arrangement of collagen fiber bundles occurring in pairs or rows (Doschak and Zernicke 2005). Zone 3 finds the fibrocartilage taking on a mineralized form (Rufai, Ralphs et al. 1995; Levangie and Norkin 2005; Sharma and Maffulli 2006; Whiting and Zernicke 2008). The calcified fibrocartilage is contiguous with the surrounding mature bone and forms a tidemark within this zone setting a mineralized matrix boundary (Doschak and Zernicke 2005). The fibers are also part of the outer fibrous layer of the periosteum, embedding in to the lamellae of bone (Doschak and Zernicke 2005). A tidemark is a boundary between hard and soft tissues where collagen fibers pass through (Levangie and Norkin 2005). The tidemark signifies the point at which the entheses is now predominately bony material. Sharpey's fibers are a matrix of connective tissues consisting of bundles of collagenous fibers connecting the periosteum to bone (Doschak and Zernicke 2005).The Sharpey's fibers are part of a 3 layer system also consisting of tendon and bone (Whiting and Zernicke 2008). Sharpey's fibers originate from the tendon and penetrate into the underlying bone through intramembranous ossification; thus cementing collagenous tendon into the bone. They are innervated with proprioceptive and pain receptors (Doschak and Zernicke 2005).The bone is the final and forth zone of the enthesis (Rufai, Ralphs et al. 1995; Doschak and Zernicke 2005; Levangie and Norkin 2005; Sharma and Maffulli 2006; Whiting and Zernicke 2008). Zone 4 is approximately 0.5 mm in length (Whiting and Zernicke 2008) and blends imperceptibly into the bone (Sharma and Maffulli 2006) completing the OTJ insertion.

The indirect form of enthesis serves to connect tendon to muscle. The indirect version is known as the musculotendinous junction (MTJ). It is fibrocartilaginous in nature and does not have periosteum or bone present (Sharma and Maffulli 2006). The role of the MTJ is to control movements and positions of joints by acting as prime movers, synergists, or antagonists, to stabilize joints and to store and release elastic energy (Trotter 2002).Indirect

entheses are comprised of two parts. Most fibrocartilaginous insertions (enthesis) consists of a fibrocartilaginous part and a fibrous part that is a more superficial or distal component of the tendon (Benjamin, Toumi et al. 2006) The interface between muscle and tendon is like an adhesive joint of folded tissue that increases the contact area between the two (Trotter 2002). The folded tissue which looks like finger-like processes is known as interdigitation (Levangie and Norkin 2005). The MTJ transfers compressive and tensile loads from the muscle to the tendon through the interdigitation (Trotter 2002; Levangie and Norkin 2005; Whiting and Zernicke 2008). The ability to transfer load through interdigitation that occurs between the tendon and the muscle is critical in protecting the tissue (Levangie and Norkin 2005; Whiting and Zernicke 2008). Interdigitation ensures the only load applied at the interface is shear (Trotter 2002). This area is sensitive to decreased fiber loading conditions causing a flatter region with less infolding which in turn causes a weakness at the junction and increases the injury risk (Levangie and Norkin 2005). But, overall, failure at this at area is less likely and is actually greater within muscle belly (Trotter 2002).

BIOMECHANICS

Function

Tendon length varies between muscle and between individuals (Elliott 1965). Tendons vary markedly in design and these differences are related to the specific function of that tendon (Kjaer 2004). Tendon length is a discerning factor where muscle size and potential muscle size is concerned (Zhang, Young et al. 2005). Tendon length is determined by genetic predisposition (Elliott 1965). Tendon thickness varies along the length of the tendon (Kjaer 2004). The dimensions influence the ability to stretch, store and release energy (Kjaer 2004). Tendon has not been shown to either increase or decrease in response to environment, unlike muscles which can be shortened by trauma, use imbalances, lack of recovery, and stretching (Elliott 1965; Kjaer 2004; Sharma and Maffulli 2006; Benjamin, Kaiser et al. 2008).

Tendon function is essential to normal function of joints; without them, the various lever systems operating throughout the body would not work properly (Elliott 1965). Tendon traditionally was considered a mechanism by which muscles connect to bone and functioned solely to transmit force (Elliott 1965; Whiting and Zernicke 2008; Jung, Fisher et al. 2009). In reality, tendon primarily functions in three main ways. The first function allows distance between the muscles and joints they activate (Weintraub 2003; Currey 2006; Whiting and Zernicke 2008). The second function of tendon is to transmit forces around a corner using a lever and/or pulley system to accomplish joint movement (Levangie and Norkin 2005; Currey 2006; Whiting and Zernicke 2008). In horses, tendons help accurately position the limb during motion (Thorpe, Clegg et al. 2010). The last function is to function in a spring-like manner to provide for passive modulation of force (Kjaer 2004; Currey 2006; Thorpe, Clegg et al. 2010).

The mechanical properties of the tendon tissue allow for the spring-like mechanism (Thorpe, Clegg et al. 2010). Elastic properties allow tendon to modulate forces passively during locomotion while providing additional stability without active work (Kjaer 2004; Benjamin, Kaiser et al. 2008). The transmission of force from muscle to bone helps to

stabilize the joint through the movement (Levangie and Norkin 2005). Lastly, the ability of tendon to store energy is a very important component of its function (Weintraub 1999; Kannus 2000; Benjamin, Kumai et al. 2002; Weintraub 2003; Currey 2006). For example, during a human stride, the Achilles tendon stretches while the ankle joint dorsiflexes as the foot goes through heel contact towards toe-off. During the toe-off portion of the stride, as the foot plantar-flexes, the stored elastic energy is released. Furthermore, muscle is able to function with less or even no change in length as the tendon stretches allowing the muscle to generate greater force (Kjaer 2004; Benjamin, Kaiser et al. 2008). The orientation of fibrous tissue provides a mechanical influence upon tendon growth, but there is no strict relationship between muscle strength and tendon thickness and various muscles differ in the ratio of their total fascicular cross-sectional area to tendon thickness (Benjamin, Kaiser et al. 2008; Franchi, Quaranta et al. 2009). It is suggested that the duration as well as the level of transmitted tension influences the growth of collagen (Kjaer, Langberg et al. 2009). Tendon thickness may increase proportionately with muscle cross-sectional area under conditions that cause the muscle to hypertrophy (Kjaer 2004; Kjaer, Langberg et al. 2005; Kjaer, Langberg et al. 2009).

The mechanical properties of tendon are dependent on the collagen fiber diameter and orientation (Dowling, Dart et al. 2000; Kjaer 2004; Benjamin, Kaiser et al. 2008). The elongation and the strain of the collagen fibrils alone have been shown to be much lower than the total elongation and strain of the entire tendon under the same amount of stress, demonstrating that the proteoglycan-rich matrix must undergo deformation, and stiffening of the matrix occurs at high strain rates (Kjaer 2004). The collagen fibrils are parallel to each other and closely packed, but show a wave-like appearance due to planar undulations, or crimps (Levangie and Norkin 2005). The bands of proteoglycans allow tendon fascicles to slide relative to each other which help protect sensitive structures from damage due to twisting and shear (Vogel 2004). A process allowing the fibril to elongate and decrease in diameter under tension causes bridges between the fibrils to be continuously broken and reformed (Kjaer 2004). The proteoglycan components of tendons are important to the mechanical properties of tendon (Vogel 2003; Kjaer 2004; Vogel 2004). Proteoglycans provide a viscous environment allowing collagen to stretch, dissipate forces that occur with sudden loads, lubricating vascular elements, and resisting compressive stiffness (Vogel 2003; Vogel 2004). Decorin, a leucine rich proteoglycan, allows tendons to resist tensile stress (Kjaer 2004). Other proteoglycans, specifically aggrecan, allow them to resist compressive stress (Kjaer 2004; Kjaer, Langberg et al. 2009). The tendon has a tensile strength, testing at four times greater than the maximal tension transmitted *in vivo.*

In tendons, collagen I fibers have some flexibility which allows the formation of other conformations such as bends or internal loops in the triple helix and results in the development of crimps (Kjaer 2004). Tendon is a multi-stranded structure made up of many partially independent fibrils and fascicles and this property also contributes to its flexibility (Kjaer 2004; Benjamin, Kaiser et al. 2008). Crimp is a phase of initial laxity occuring in the toe region of the tendon stain pattern (Dowling, Dart et al. 2000). The crimp in the collagen fibrils allows the tendons to have flexibility as well as a low compressive stiffness. The fibrils appear to have a sinusoidal pattern prior to any tension being placed upon them (Dowling, Dart et al. 2000). Change in shape of the tendon occurs as the tissue is deformed or strained by external forces (Doschak and Zernicke 2005). Tensile resistance occurs in the tendon once the crimp pattern is straightened out (Dowling, Dart et al. 2000). The tendon is within the

linear region of tendon extensibility; meaning that the collagen fibers are now able to respond to external forces (Doschak and Zernicke 2005). Although the waveform seen on the surface of a tendon at rest is eliminated by less than 10 % of the maximum tension the muscle is capable of transmitting. It is possible that the normal range of tensions transmitted *in vivo* might fall within that part of the stress-strain curve where the tendon is still easily extensible (Doschak and Zernicke 2005).

The relationship between the stress and strain that a tissue displays is the stress-strain curve. The slope of the stress-strain curve provides the modulus of the tissue. Modulus of elasticity is the ratio of the stress applied to a body to the strain that results in the deformation of the body (Levangie and Norkin 2005). The modulus is a measurement of the stiffness, which remains constant over a range of stress. Tendon tensile modulus is very high (Kjaer 2004).

Three types of the modulus of elasticity affect the behavior of tendon when it is loaded. Young's modulus, elastic modulus, describes the tensile elasticity of a tendon when force is applied along the axis. It is a ration of tensile stress versus tensile strain (Levangie and Norkin 2005). Shear modulus, modulus of rigidity, describes a tendons tendency to shear when opposing forces are applied. Tendon has a low resistance to shear forces due to the alignment of the fibers (Kirkendall and Garrett 1997) Lastly, bulk modulus, an extension of Young's modulus, describes tendon's ability to deform in all directions when uniformly loaded. It is the inverse of compressibility.

The stress-strain curve or load-displacement of tendons generate a characteristic curve with 3 distinct regions: toe, linear, and failure (Levangie and Norkin 2005; James, Kesturu et al. 2008). The toe region represents un-crimping of the wavy appearance of the collagen (Levangie and Norkin 2005). Crimped tissue has relatively low stiffness as the tissue stretches and straightens tissue stiffness is increased relative to the load encountered (Kjaer 2004).

The linear region allows the tissue to continue increase stiffness as it is loaded (Levangie and Norkin 2005). Collagen tissue has two primary features that occur in the linear region: stress-relaxation and creep (Kirkendall and Garrett 1997; James, Kesturu et al. 2008) and two other features: a hysteresis loop and viscoelastic properties (James, Kesturu et al. 2008). Stress-relaxation is a decreased stress over time with constant deformation (Kirkendall and Garrett 1997). This means that tissue stress will be reduced or relaxed when structures are under a constant deforming load (James, Kesturu et al. 2008). Creep is an increase in length over time with constant load (Kirkendall and Garrett 1997). These two behaviors are the result of the viscoelastic behavior of tendon. An important concept to remember is that the stress-strain relationship of tissue is not constant but actually based upon time of loading.

Hysteresis is also a property of viscoelastic tissue. It is a dissipation of energy with the reduction of a load (Kjaer, Langberg et al. 2009). When a viscoelastic material is loaded and unloaded, the unloading curve does not follow the loading curve (Levangie and Norkin 2005). The difference between the two curves is the amount of energy dissipated during loading (James, Kesturu et al. 2008). The loading occurring in the linear region does not cause permanent deformation as a part of these tissue behaviors (Levangie and Norkin 2005; James, Kesturu et al. 2008).

Permanent deformation occurs in the failure region (Levangie and Norkin 2005). The tendon either tears within the tendon mid-substance or at the tendon-bone interface (Kirkendall and Garrett 1997).

Healthy Tendon Response to Loading and Unloading

Tendon behavior is complex but is dependent upon age and activity(James, Kesturu et al. 2008). Tendon functions primarily to transfer high tensile loads, from muscle to bone, to provide movement (Kirkendall and Garrett 1997; Reeves 2006). Tendon is designed to transmit these forces with minimal deformation or energy loss (Kirkendall and Garrett 1997; Sharma and Maffulli 2006). Properties leading to the load-deformation relationship of tendon are dependent on prior activities (Kirkendall and Garrett 1997). Normal tendon does not fail in response to strain (Kirkendall and Garrett 1997). Tendon exhibits elastic properties allowing it to return to its original length when the load is removed (Elliott 1965). The parallel fiber arrangement allows for this to occur efficiently (Kirkendall and Garrett 1997; Kannus 2000) Loading of tendon creates a strain in the tissue (Levangie and Norkin 2005). The elastin component of the tendon provides the stiffness that occurs with the load (Kjaer 2004). Collagen is relatively inextensible and crimped, wavy, when unloaded (Levangie and Norkin 2005). Proteoglycans play a role in the mechanical behavior of tendon (Vogel 2003). Proteoglycans provide a viscous environment allowing for the stretch and dissipation of sudden loads (Vogel 2003). Loading causes the collagen to straighten (Kjaer, Langberg et al. 2009). Tissue deformation increases as collagen is stretched in the direction of the deformation (Vogel 2003). As a result, the collagen fibers are aligned in the direction of the load (James, Kesturu et al. 2008). The alignment helps to provide stiffness to the structure in order to transmit load and to create a stable environment for movement to occur. Fibrocartilage develops in tendon where needed to provide compressive stiffness adding to mechanical reinforcement (Vogel 2003; Vogel 2004). The ability of the tissue to handle loading is based upon the stress-strain relationship previously discussed (Levangie and Norkin 2005). Localized damages can occur to collagen fibers when tendons are exposed to high strain during high speed locomotion (Thorpe, Clegg et al. 2010). Tendon elongation regulation is based upon the amount of load that is transferred to it (James, Kesturu et al. 2008). Strain on individual collagen fibers is greater than on the entire tendon meaning other structures must help resist tension to prevent injury to tendon (Kjaer 2004). The ECM is responsible for linking tendon to other tissues which plays a key role in force transmission and tissue structure maintenance when loads are encountered (Kjaer, Magnusson et al. 2006). This linking allows for the sharing of tissue loading. Extremely high loads will result in failure of the fibers and the tendon will tear (Kirkendall and Garrett 1997).

A relationship exists between tendon size and muscle function (Elliott 1965). Tendons while seemingly inert are sensitive to changes in the loading levels they experience (Reeves 2006). Tendon remodels according to Wolff's Law as does all tissue (Levangie and Norkin 2005; Whiting and Zernicke 2008; Jung, Fisher et al. 2009). In humans, repetitive loading increased the cross sectional area of tendon (Kjaer, Langberg et al. 2009). Mechanical properties of the tendon fluctuate based upon the level of mechanical usage (Reeves 2006). Tendon is directionally dependent in the function of its mechanical properties which influences changes that occur (Jung, Fisher et al. 2009). The loading of tendon depends upon joint angle as the load is encountered (Vogel 2003). The joint angle influences the mechanical stress based on the fiber orientation of the tendon (Elliott 1965; Vogel 2003; James, Kesturu et al. 2008). Tendon growth is regulated by the differing growth rates of the muscle and the bone it lies between (Elliott 1965). Tendon loading is a direct response to the activity, size, and strength of the muscle attached to the tendon (Kjaer, Langberg et al. 2009). In 12-14 weeks of training, no changes in size of tendon were seen but the composition and the

modulus of elasticity changed (Kjaer 2004; Kjaer, Magnusson et al. 2006; Kjaer, Langberg et al. 2009). Training, or chronic loading, leads to both increase collagen turnover and net collagen synthesis (Kjaer, Magnusson et al. 2006). Collagen synthesis, ECM turnover and MMP activity increase with mechanical loading (Kjaer, Magnusson et al. 2006). Simultaneous synthesis and angiogenesis of collagen in tendon occurs and the goal is to put the rate toward an imbalance in favor of synthesis (Kjaer 2004). The ability to transform a net synthesis of collagen into an increase tendon size is difficult (Kjaer 2004). But, an increased turnover of type I collagen allows for a reorganization of tissue (Kjaer 2004; Kjaer, Magnusson et al. 2006; Kjaer, Langberg et al. 2009). In addition, local tissue expression and growth factor release, TGF-β –I, CTGF, and IGF-I, are enhanced with activity (Kjaer, Magnusson et al. 2006). Circulatory response improves in tendon with prolonged training which creates a better environment for the tendon tissue (Kjaer, Magnusson et al. 2006). Because of the overall improvement of the tissue properties, there is a possible improvement in load resistance (Kjaer, Magnusson et al. 2006). Cells respond to mechanical environment changes by altering synthesis and degradation of the matrix (Thorpe, Clegg et al. 2010).

Alterations in tendon mechanical properties have implications for speed of force transmission, muscle function, and injury potential (Reeves 2006). Typically, tendon hypertrophy occurs at the proximal and distal attachments (Kjaer, Langberg et al. 2009). These changes modify the mechanical and viscoelastic properties of tendon (Kjaer, Magnusson et al. 2006). Habitual loading causes a robust change in tendon size and mechanical properties (Kjaer, Langberg et al. 2009). No strict correlation between muscle cross sectional area and tendon thickness exists (Elliott 1965). Tendon cross sectional area might be determined by average tension and duration exerted on the muscle rather than by muscle size or maximum strength (Elliott 1965). However, prolonged training equals an increase in tendon tissue with an associated assumption of strength alteration (Kjaer 2004). Tensile strength and other mechanical properties are correlated to tendon thickness and not muscle strength (Elliott 1965).

INJURED TENDON

Tendons heal and recover from injuries in a process controlled by the tenocytes and the surrounding extracellular matrix. Tendon healing occurs intrinsically as well as extrinsically (Sharma and Maffulli 2005). Extrinsic healing is the result of the stimulation of the peritendinous tissue to proliferate and a well-established supply of the cells and capillaries needed for healing (James, Kesturu et al. 2008). Extrinsic healing involves the movement of fibroblasts and inflammatory cells from the outer tendon to the injury (James, Kesturu et al. 2008). These structures are instrumental in the repair and regeneration process. A natural consequence to the extrinsic healing process is the formation of adhesions (Sharma and Maffulli 2005; James, Kesturu et al. 2008) that decreases efficient tendon movement (Sharma and Maffulli 2005). The mechanism for intrinsic healing entails the migration and proliferation of cells from the endotendon and epitendon to the injury (James, Kesturu et al. 2008). Intrinsic healing is supported by a sparse intrinsic blood supply nourishing approximately one-fourth of the tendon volume. The intrinsic blood supply is not sufficient to support primary healing of the tendon in most cases. Tendon healing within a sheath is

prolonged compared to healing outside the sheath. However, intrinsic healing results in better conditions for maintaining normal tendon movement (Koob and Summers 2002; Sharma and Maffulli 2005). Both of these healing mechanisms are dependent on tendon location, extent of trauma, and post-injury motion (James, Kesturu et al. 2008). However, the healed tendon will never regain the mechanical properties prior to the injury (Sharma and Maffulli 2005).

The sequence of healing overall follows three main stages: inflammation, repair or proliferation, and remodeling (Sharma and Maffulli 2005; Sharma and Maffulli 2005; Sharma and Maffulli 2006; James, Kesturu et al. 2008). Remodeling can be further divided into consolidation and maturation stages (Sharma and Maffulli 2005). The stages of the healing process overlap each other. In the first stage of healing, the biological sequence is the same as with any healing wound, except in the case of tendon it is slower. Inflammatory cells such as neutrophils are recruited to the injury site, along with erythrocytes. Monocytes and macrophages are recruited within the first 24 hours, and phagocytosis of necrotic materials at the injury site occurs. After the release of vasoactive and chemotactic factors, angiogenesis and the proliferation of tenocytes are initiated. Tenocytes then move into the site and synthesize collagen III. At five to seven days post injury, the tendon is its weakest. Blood supply is crucial in injury and resolution (Doschak and Zernicke 2005). The inflammation stage usually lasts for a few days. The repair stage then begins. In this stage, lasting for about six weeks, the tenocytes are involved in the synthesis of large amounts of collagen and proteoglycans at the site of injury, and the levels of GAG and water are high. A fibrovascular callus forms surrounding the tendon and attaching all structures of the wound together. This part of the healing process is responsible for the formation of adhesions of the tendon to adjacent structures of the wound (James, Kesturu et al. 2008).

Tenocytes actively synthesize ECM components as well as enzymes such as matrix metalloproteinases (Kjaer 2004; Sharma and Maffulli 2005; Sharma and Maffulli 2006). Matrix metalloproteinases (MMP's) have an important role in the degradation and remodeling of the ECM during the healing process after a tendon injury (Koskinen, Heinemeier et al. 2004). Certain MMPs, including MMP-1, MMP-8, MMP-9, and MMP-13, have collagenase activity that makes them capable of degrading collagen I fibrils (Kjaer 2004; Sharma and Maffulli 2005). The degradation of the collagen fibrils by MMP-1 along with the presence of denatured collagen are two factors believed to cause weakening of the tendon ECM which increases the potential for another rupture to occur (Kjaer 2004). Repeated mechanical loading or injury causes cytokines to be released by tenocytes inducing the release of MMPs causing degradation of the ECM leading to injury (Sharma and Maffulli 2005; Sharma and Maffulli 2006).

The remodeling stage begins as the repair stage is reaching completion. The first part of the remodeling stage is consolidation and usually lasts from about six to ten weeks after the injury. Collagen and GAG synthesis decreases. Cellular proliferation is also decreased as the tissue becomes more fibrous as a result of increased production of collagen I. An initial period of increased collagen turnover occurs, suggesting the ability of tendon to restructure and adapt to increased loading (Benjamin, Kaiser et al. 2008). The final remodeling stage, maturation, occurs after ten weeks and is marked by an increase in cross-linking of the collagen fibrils. The increased cross-linking causes the tissue to become stiffer. A longitudinal orientation of fibroblasts and fibers begins. Fibrils follow Wolff's Law aligning in the direction of mechanical stress (Elliott 1965; Benjamin, Kaiser et al. 2008). At 45 days post-injury, collagen lysis and collagen formation reach a state of equilibrium. Early

collagen bundle formation is seen 90 days post-injury. The collagen bundles appear much like normal tendon at 120 days post-injury. Gradually, over the next year, the tissue will turn from fibrous to scar-like as a result of stresses placed upon the tendon (Elliott 1965).

Five growth factors are significantly active during tendon repair and regeneration: insulin-like growth factor 1 (IGF-I), platelet-derived growth factor (PDGF), vascular endothelial growth factor (VEGF), basic fibroblast growth factor (bFGF), and transforming growth factor beta (TGF-β) (Kjaer 2004). These growth factors play different roles during the healing process. IGF-1 increases collagen and proteoglycan production during the first stage of inflammation. PDGF is present during the early stages after injury promoting the synthesis of other growth factors along with DNA synthesis and tendon cell proliferation. VEGF promotes angiogenesis and induces endothelial cell proliferation and migration. VEGF mRNA has been shown to be expressed at the site of tendon injuries along with collagen I mRNA. TGF-β plays a role in wound healing and scar formation. Bone morphogenetic proteins (BMPs) are a subgroup of TGF-β family and induce bone and cartilage formation as well as tissue differentiation, and BMP-12 specifically has been shown to influence formation and differentiation of tendon tissue and to promote fibrogenesis.

Effects of Activity on Healing

Tendons recover from injuries in a process controlled by the tenocytes and the surrounding extracellular matrix. Local tissue expression and growth factor release, TGF-β – I, CTGF, and IGF-I, is enhanced with activity (Kjaer, Magnusson et al. 2006; Kjaer, Langberg et al. 2009). In animal models, extensive studies have been conducted to investigate the effects of mechanical strain in the form of activity level on tendon injury and healing. The medically advocated practice of rest may actually cause more harm than good. The complete unloading due to bed rest or injury reduces both stiffness and modulus of elasticity (Reeves 2006). The reduction in stiffness and elasticity does not provide tissue with proper loading capabilities. With injury, cell activity is compromised due to changes in the physiologic or mechanical environment (Thorpe, Clegg et al. 2010). The poor blood supply results in low oxygen levels compromising cell activity (Thorpe, Clegg et al. 2010). The use of active recovery is the best means of remodeling tissue appropriately. A benefit of exercise is an increase in circulation. The improved circulatory response improves the healing capacity of the tendon (Kjaer, Magnusson et al. 2006). By improving circulation, the overall environment of healing improves. Collagen synthesis and MMP activity increase with mechanical loading (Kjaer, Magnusson et al. 2006).

Prolonged loading results in a net synthesis of type I collagen (Kjaer 2004). ECM turnover is influenced by physical activity (Kjaer, Magnusson et al. 2006). Intensity and load pattern along with recovery play an important role in ECM adaptation to stress (Kjaer 2004). Collagen turnover increases (Kjaer, Magnusson et al. 2006). Increased turnover of type I collagen allows for a reorganization of tissue (Kjaer 2004).

Cell activity is compromised due to changes in the physiologic or mechanical environment (Thorpe, Clegg et al. 2010). Immobilization of tendon following injury often has a negative effect on healing. Only chronic unloading due to permanent injury seems to cause tendon atrophy (Reeves 2006). Immobilization causes an increase in production of matrix degrading enzymes (Thorpe, Clegg et al. 2010). In rabbits, collagen fascicles that were

immobilized show decreased tensile strength, and immobilization resulting in lower amounts of water, proteoglycans, and collagen cross links in the tendons (Dowling, Dart et al. 2000). Increased tendon thickness was noted in subjects immobilized in a stretched position (Elliott 1965). Cell response levels are lowered so that cells are unable to respond properly to mechanical stimuli resulting in the inability of microdamage repair. Also, degradation of the matrix occurs which may accelerate microdamage rate (Thorpe, Clegg et al. 2010). Stretching can also disrupt healing during the initial inflammatory phase. Controlled movement of tendon after about one week following an acute injury promotes the synthesis of collagen by the tenocytes. Simultaneous synthesis and degradation of collagen in tendon occurs and the goal is to put the rate toward an imbalance in favor of synthesis (Kjaer 2004). Circulatory response improves the synthesis of collagen (Kjaer, Magnusson et al. 2006). High strain tendons have a greater matrix capacity to repair that low strain tendons do not (Thorpe, Clegg et al. 2010). Specifically, equine tendon placed under a low load repetitive stress situation had higher collagen levels that tendon placed under higher loads (Kjaer 2004). Low strain tendons are protected from remodeling by the tenocytes making them transiently weaker (Thorpe, Clegg et al. 2010). Prolonged training equals an increase in tendon tissue and with that an associated assumption of strength alteration (Kjaer 2004). Collagen synthesis leads to increased tensile strength and diameter of the healed tendon. The ability to transform this net synthesis into an increase tendon size is difficult (Kjaer 2004). Fewer adhesions occur as a result of controlled movement when compared to tendons that are immobilized for prolonged periods. Mechanical loading stimulates fibroblast proliferation and collagen synthesis along with collagen realignment in chronic tendon injuries; all of which promote repair and remodeling of the tendon.

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In: The Equine Tendon in Health and Disease
Editor: Henry Jann
ISBN: 978-1-62808-048-3

Chapter 2

IMAGING

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ABSTRACT

The vast majority of tendon and ligament pathologies to date are predominantly evaluated with ultrasound and magnetic resonance imaging. Ultrasound offers widespread availability, exquisite spatial resolution, cost-effectiveness, logistic ease of imaging, guided needle placement, immediate visualization, visualization of proximal limb structures, and is usually performed standing. The technology continues to advance, providing improved abilities to diagnose injuries and follow the repair process. Techniques to improve diagnosis and interpretive skills are discussed, as well as notable limitations. Magnetic resonance imaging (MRI) is a vastly different imaging technique, and considered the gold standard for musculoskeletal imaging in some circles. Although expensive, with limited access and sometimes requiring general anesthesia, ligaments and tendons are very well assessed on MRI, and many practitioners consider an evaluation critical for optimal treatment and clinical outcome. Size, shape, architecture, and quality of collagen matrix are evaluated in all soft tissues included in the study, as well as any bone and joint pathologies. Many lesions seen on MRI cannot be visualized ultrasonographically or radiographically. Several common reasons to request an MRI exam include accurate and thorough diagnosis following an acute or chronic lameness, assess additional complicating lesions following a known soft tissue or bony injury, and assess the presence and quality of repair after injuries. Multiple lesions are frequently found on MRI, and some equine practitioners experienced with MRI evaluations report much improved return to function after addressing all significant lesions.

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INTRODUCTION

Advanced diagnostic procedures are riding the wave of technology, as are advanced treatment options for musculoskeletal disease in horses. This is especially true with magnetic resonance imaging and ultrasonography for tendon and ligament injury assessment. Other imaging techniques (such as radiography) are severely limited; and although contrast radiography, CT, and nuclear scintigraphy are all used for some soft tissue diagnostics, this chapter will focus on the more common use of ultrasound and magnetic resonance imaging for diagnosing and prognosticating tendon and ligament injuries.

ULTRASOUND

Since the 1980's diagnostic ultrasound imaging has grown to be an important part of equine veterinary medicine. Initially used for reproductive imaging, pioneering description regarding application as musculoskeletal imaging modality was published by Rantanan et al. in 1986. [1] Since that time, progressive advancement of equipment technology and technical skill sets has promoted the status of diagnostic ultrasound to be an essential modality for most clinicians.

The physics of diagnostic ultrasound imaging was presented in the equine veterinary literature by FR Pipers in 1982. [2] The constant development of wave forming and processing has created a moving target for understanding and is beyond the scope of this text. It suffices to say that each system manufacturer is striving to obtain the best quality image for their system's intended use.

Advantages of Ultrasound

The widespread acceptance and use of diagnostic ultrasound is the result several attributes; namely, diagnostic ultrasound improves quality of health care delivery, it is usually a profit center for the practice and it subjectively adds to the enjoyment of veterinary medicine by the practitioner. Each of these attributes is listed in Table 1 below with more specific examples of how each attribute is supported by the use of ultrasound in the realm of soft tissue musculoskeletal health care.

Disadvantages of Ultrasound

While the advantages of ultrasound use are plainly evident, the disadvantages must be clearly understood so as to minimize their effect. It should be noted that these same disadvantages can lend themselves to advanced abilities to aid in interpretation of sonograms. Disadvantages can be related to one of two causes: first, physical limitations regarding penetration and diffraction and secondly, by operator limitations.

Table 1. Attributes and examples of advantages of diagnostic ultrasound

I. Improved quality of health care delivery

Advantage:	Example:
Logistic ease of imaging	a) Able to perform imaging on location b) No anesthesia needed c) Non-invasive and well tolerated d) Images acquired relatively quickly
Guided needle placement	a) Allows accurate intralesional therapy b) Validates intra-thecal deposition of anesthesia c) Guides perineural anesthesia of non-palpable nerves
Immediate visualization	a) Imaging enhanced during acute swelling b) Improved management of healing progression c) Able to better determine best therapeutic course based on type of injury
Visualization of proximal limb structures	a) Allows imaging of musculoskeletal structures proximal to carpus and tarsus b) Able to verify needle placement for therapeutic and diagnostic injections

II. Profit center for the practice

Advantage:	Example:
Moderate equipment cost	a) Readily available from multiple vendors: competition reduces cost and increases features b) Minimal consumable costs c) Machines are relatively durable d) Manufactures usually support updates e) Most systems are able to return capitalization costs after one year.
Increasing demand and application of use	a) Able to be used in other aspects of practice: reproduction, abdominal scans, cardiac evaluation, wound management, etc. b) Need for multiple progress evaluations of healing for adequate case management c) “standard of care” may necessitate use to document adequacy of treatment
Training readily available	a) Facilitates development of new diagnostic and therapeutic techniques b) Develops future professional relationships

III. Subjectively adds to the enjoyment of veterinary medicine by the practitioner

Advantage:	Example:
Able to visualize soft tissue	a) Certainty of accurate needle placement b) Confidence of detailed injury assessment c) Assurance from more objective assessment of healing
Advancement of knowledge	a) Able to apply latest therapeutic techniques b) Able to apply latest diagnostic techniques

At its very essence, ultrasound is simply wave energy emitted from the probe, which then penetrates tissue and then after impact, a certain amount of energy is directed back to the probe whereby the computer assigns a certain amount of grey scale. This grey scale information is then reassembled into a picture for us to view. This process lends itself to very distinct physical limitations:

Limitations imposed by Physics:

1) *Sound waves are reflected by soft tissue interfaces with bone and gas.* Therefore, ultrasound is not effective in evaluation of interior bony structure. However, the surface architecture of bone is quite detailed. Ultrasound can be quite sensitive to changes to insertion/origin of soft tissue structures. See Figure 1a/b.

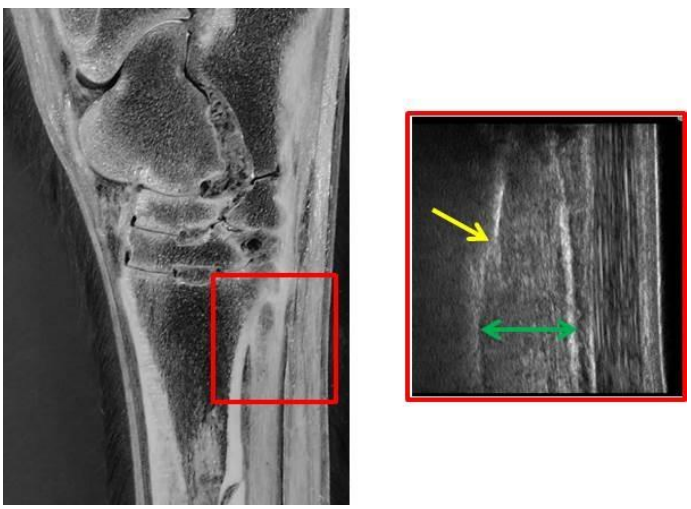


Figure 1a. Example of sensitivity of ultrasound to bone surface changes in the evaluation of subtle enthesopathy (yellow arrow) associated with thickened hind proximal suspensory ligament (green arrow).

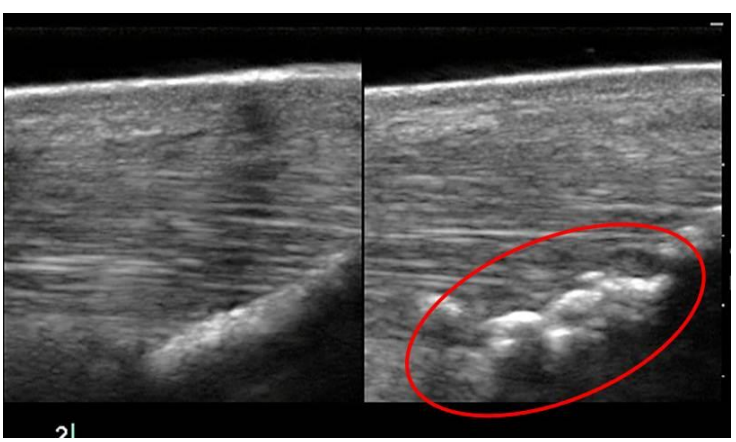


Figure 1b. Additional example of sensitivity of ultrasound to bone surface changes as evidenced by imaging of more obvious enthesopathy at insertion of suspensory ligament on abaxial sesamoid bone (red oval on right image) compared to normal insertion of left image.

Similarly, gas interfaces reflect most of the applied sound energy. This is particularly problematic when trying to ultrasound immediately after diagnostic anesthesia. However, it can be very useful in determining if traumatic wounds have penetrated vital structures in the frog by following the highly reflective gas. See Figure 2.

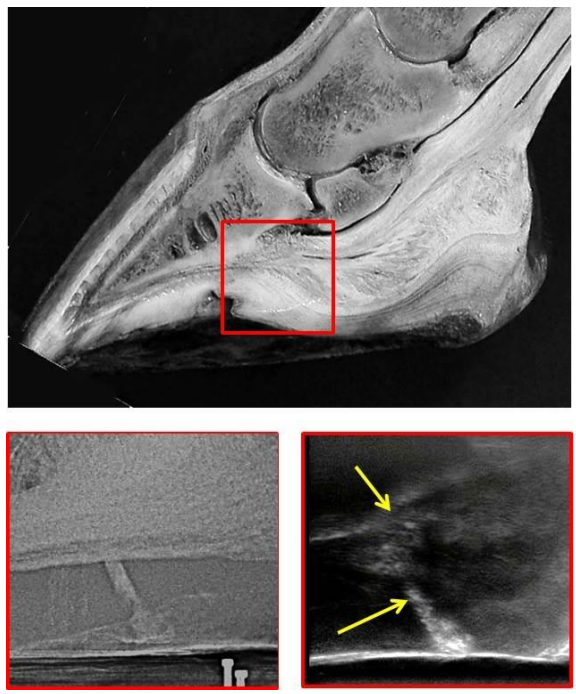


Figure 2. Example of usefulness of gas reflection using ultrasound: Significance of penetration of frog with foreign body is not fully demonstrated with contrast placement in tract with radiography (bottom left image). Ultrasonography demonstrates contamination of insertion of DDFT on solar surface of P3 by clearly identifying presence of highly reflective gas along penetration tract (yellow arrow, right image). Reference image provided above.

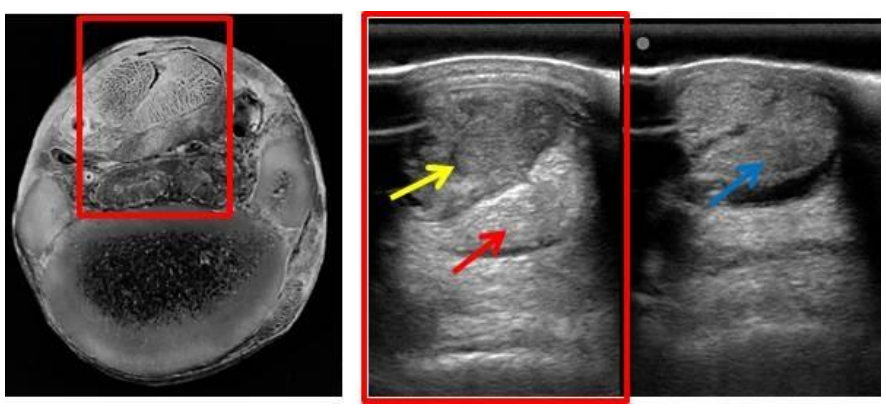


Figure 3. Example of enhanced echogenicity of deeper structures in presence of inflammation. Comparative images of BF limbs: severe acute tendinosis of LF (yellow arrow) facilitates penetration of acoustic wave energy to cause a hyperechoic DDFT (red arrow). The contralateral DDFT (blue arrow) shows normal echogenicity. Images captured at same machine settings and examination session.

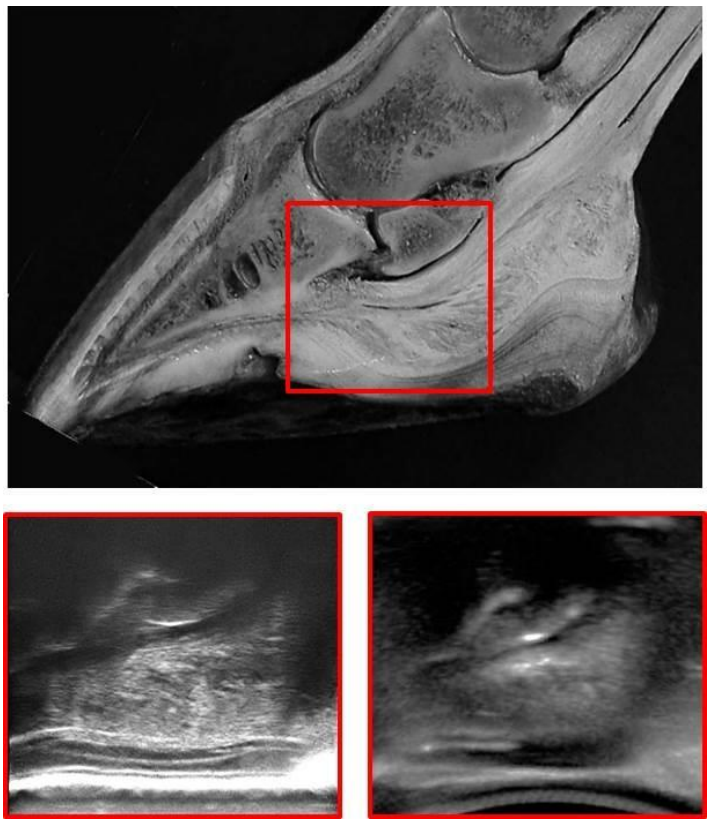


Figure 4. Comparison of image detail with high frequency probe image (lower left) and low frequency macro convex probe image (right lower) Reference image above.

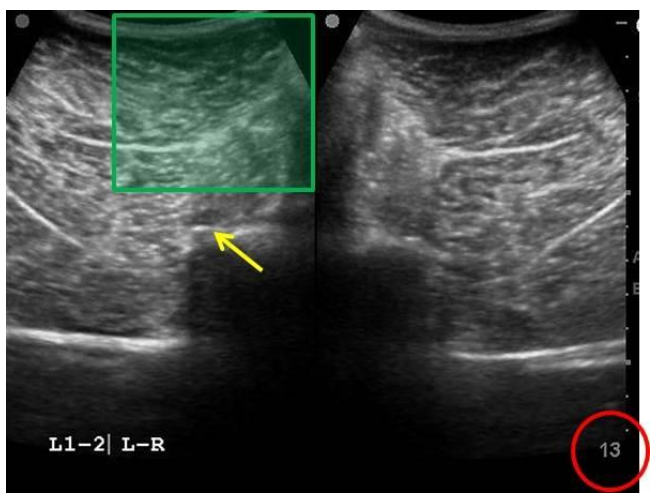


Figure 5. Transverse section of L1-2 facets (yellow arrow) with macro convex probe. Lower frequency of probe (2-5 MHz) allows imaging of deeper structures. In this case, imaging is acquired to a depth of 13 cm (red circle). Depths may be imaged to 30 cm. The green shaded area demonstrates depths that are accessible with a higher frequency (7-10 MHz) linear probe clearly demonstrating the need of a low frequency probe to image lumbar articular facets.

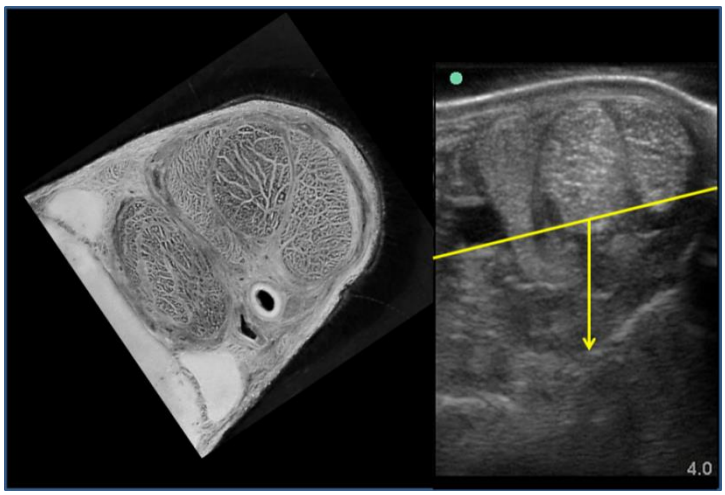


Figure 6. Lateral view of mid metacarpus demonstrating diffraction of sound waves in deeper depths (below yellow line) causing diffuse graying and loss of image detail. Reference image on left.

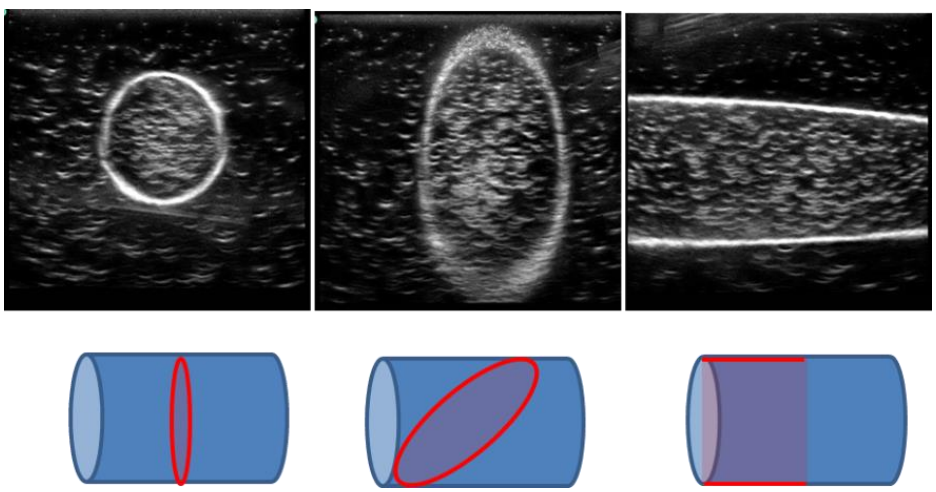


Figure 7. Examples of 2-D image variation dependent on probe orientation during image capture of 3-D object. Red represents approximate plane of image capture as represented in corresponding sonograms. Note how divergent the image appearances are in three images of the same object.

2) *Penetration of sound waves is highly dependent on the fluid content of the soft tissue*. Fluid filled vessels allow easy penetration of sound waves while edges of fibrous structures attenuate wave propagation. This may lead to areas of hyper (former) and hypo (later) linearly echogenic artifacts within the same image. In addition, acute inflammation will cause an overall more echogenic image because of the increased penetrability of the acoustic waves. See Figure 3 It should be noted that there is no need to wait for acute inflammation to subside before ultrasound imaging. The contrary, acute inflammation represents an ideal opportunity to use ultrasound imaging. Conversely, in cold climates, the dermis is often dryer with less superficial capillary blood flow causing poor acoustic imaging.

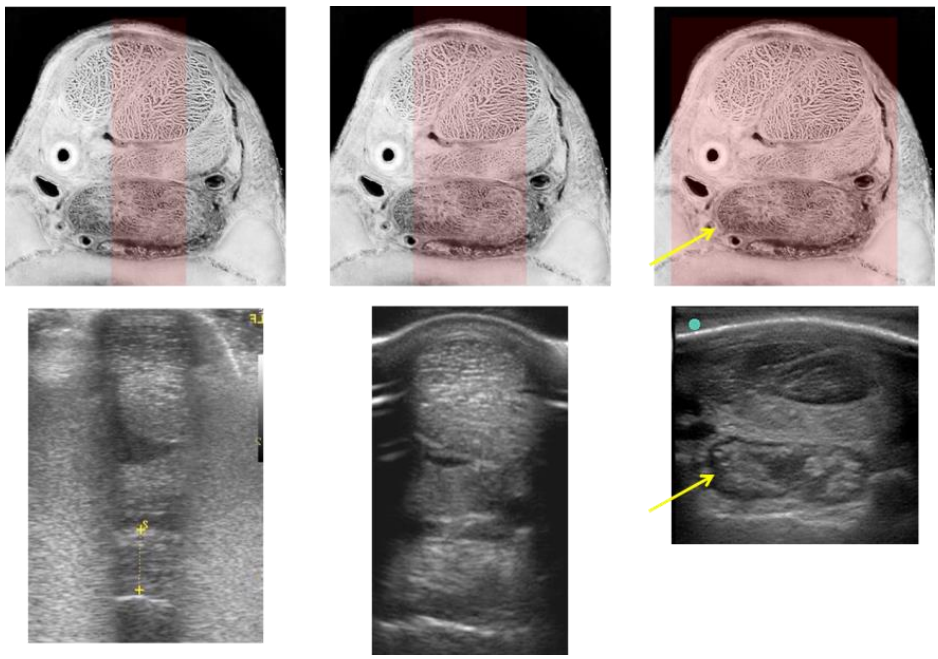


Figure 8. Attenuation of parisaggital soft tissue as a result of reduced acoustic contact area. Left images: weight bearing image without use of standoff pad between probe and limb. Red shaded area demonstrates acoustic window limited by narrow contact area. Center images: weight bearing image with use of standoff pad. Deformation of pad facilitates larger acoustic window with improved visualization of soft tissue structures. Right images: non-weight bearing limb with standoff pad (superficial structures become deformed). Deformation of superficial soft tissue structures and standoff pad create optimal acoustic window to fully visualize suspensory ligament (yellow arrows). Note that contrast enhancing technique used to produce image of suspensory ligament (technique described later in text).

3) *Sound waves that form high detail images have difficulty penetrating deeper than 6 cm*. Low frequency sound waves are able to penetrate more deeply (up to 30 cm) into soft tissue however provide less detail. Higher frequency waves are easily attenuated or diffracted and are unable to penetrate into deeper tissue. See Figure 4,5 In addition, often the deep waves are scattered and can form a artifactual gray haze which can be mistaken for actual tissue. See Figure 6.
4) *Ultrasound forms a two dimensional image of a three dimensional object*. Because the ultrasound beam forms "sliced images" of a three dimensional object, it is possible to capture very different image profiles of the same object. As an example below, a cylinder is imaged with three different orientations yielding three very different representations. See Figure 7 This characteristic can complicate ultrasonographic evaluation three ways:
    a. Orientation can be difficult when imaging complex or unfamiliar structures.
    b. Cross sectional area evaluations can be significantly affected by probe orientation.
    c. Because no standardization exists for probe orientation, it can be difficult to convey representative imaging to another veterinarian.

5) *Excellent acoustic contact along full length of probe is required to avert attenuation of image margins.* Although simple in concept, this can be particularly frustrating as the transverse palmar/plantar surfaces of commonly evaluated areas are most often very curved and need to have contact with a flat probe surface. Soft standoff pads are implemented to improve contact and optimize focal depth. In addition, legs can be held in a non-weight bearing or relaxed position to facilitate surface congruity. Water baths have been suggested; pragmatically they are too cumbersome for everyday use. In Figure 8 above, the effect of poor acoustic congruity is demonstrated by the poor demarcation of the margins of the structures. This poor delineation of the structure margins may lead to inaccurate cross sectional area measurements and inadequate assessment of the questioned structures.

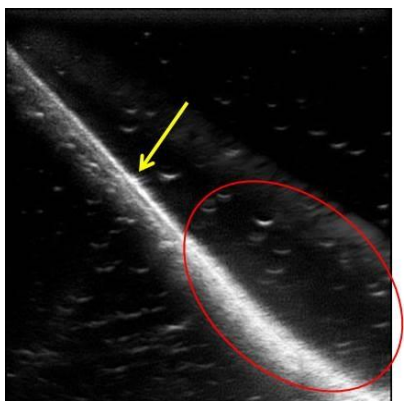


Figure 9. Sonogram of vertebral dorsal spinous process in water bath at 45° beam incidence. Note that beam diffraction causes progressively reduced surface definition (right of yellow arrow) of deeper areas and increased scattering (area within red oval) causing gray haze artifact.

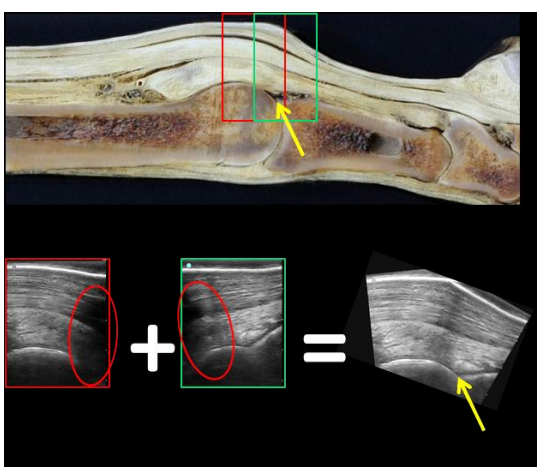


Figure 10. Demonstration of diffraction secondary to curvilinear fiber orientation. Areas designated by red ovals in both sonograms (red and green outline) demonstrate lack of tissue visualization as acoustic waves are diffracted and not directed back toward the probe. Imaging of such structures requires the mental assimilation of both orientations to yield a composite image similar to the digital composite to the right. Yellow arrows identify the palmar margin of the MCP joint.

6) Acoustic waves scatter secondary to diffraction upon impact with oblique surfaces. These surfaces may be oblique planar as is the case when the ultrasound beam impacts a bone at a 45° angle (see Figure 9) or upon contact with a curvilinear structure such as a tendon rounding a joint (see Figure 10). If either situation is present, it will difficult to produce a uniform sonogram. Usually, the specific structure of question must be imaged from a variety of angles to form a composite evaluation.

Once the physical limitations of ultrasonography are clearly understood, their effect can be minimized to decrease the impact on imaging quality. As suggested prior, some of these limitations can be used to an advantage to highlight ultra-architecture. This will be described later. The optimal application of technique lends itself to a discussion regarding the limitations imposed by the operator.

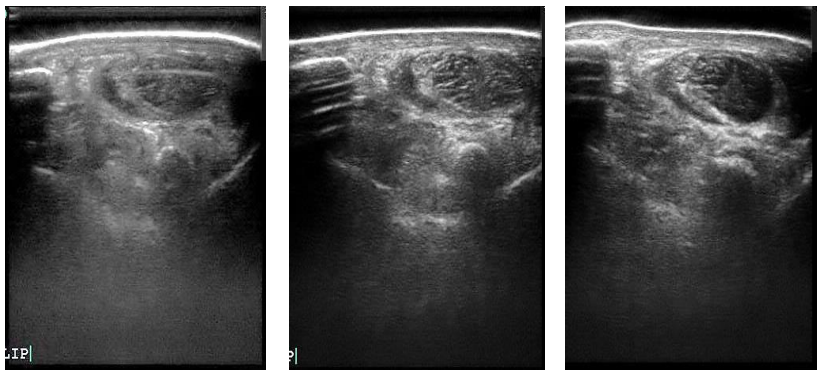
Limitations imposed by Operator:

1) *Machine operation:* The operation of a machine starts with its selection for the intended use. The veterinary market place has a variety of system vendors. Each of their ultrasound systems presents a value proposition regarding cost, form factor, image quality etc. for their machine. It might be extraordinarily durable and water proof but screen size is too small for adequate tendon evaluation. It might be simple to operate but lack post processing software. It might have optimal imaging but be fragile. Each machine has advantages and disadvantages in a given clinical setting. The improper selection of a system for its intended use will ultimately compromise image acquisition and/or quality.

   Once a system is selected it is incumbent that the operator fully understands and comprehends the operation of the machine. This has been termed: "knobology". Some systems are quite simple and require little knowledge while others are quite complex. More complex machines, while able to optimize their images, can be quite demanding with a narrow margin of optimal image rendition. Most operators significantly underestimate the impact of poor knobology on the quality of their image.

   Finally, the machine will only give optimal image acquisition when it has optimal acoustic coupling to the region of interest. The best machine with optimally set knobs will only produce a mediocre image if contact is poor. Simply put: the less hair and more water, the better the image. Usually, the restrictions regarding shaving of hair are placed on the operator by the owner. Most owners are sensitive to having clipped patches on their horse prior to competition or sale. In Figure 11 below is a comparison of various preparations on the same horse.
2) *Anatomic command:* The key to understanding ultrasound and the acquisition of diagnostic representative images lies in the command of anatomy of the operator. This implies that the operator has studied and is able to mentally transform the three dimensionality of anatomy into the two dimensional image present on the screen. Fortunately, the time and effort spent on anatomic study is seldom wasted as it is used regardless of imaging modality. Most manufacturers/distributors offer continuing education as do a variety of wet lab programs. At this time, the most comprehensive program is offered by the International Society of Equine Locomotor

Pathologists (http://www.iselp.org). In addition, various written materials are available on line to facilitate anatomic comprehension. Some selected reference examples are provided. [3, 4, 5, 6]



NO CLIPPING #10 CLIPPING #40 CLIPPING

Figure 11. Comparison of various skin preparations on same horse. Note that adequate imaging can be obtained with a #10 blade clipping with the use of copious water.

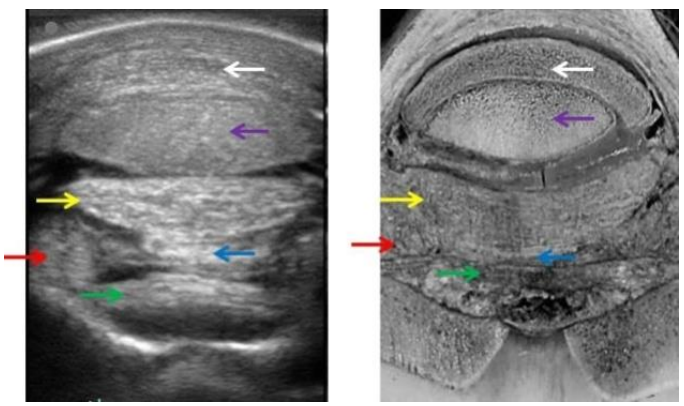


Figure 12. Example of complex anatomy: transverse section of proximal palmar pastern. Sonogram on left; correlative anatomic specimen on right: White arrow=SDFT, purple arrow=DDFT, yellow arrow=straight sesmoidian ligament, blue arrow=sagittal portion of oblique sesmoidian ligament, red arrow=oblique sesmoidian ligament, green arrow=cruciate sesmoidian ligament.

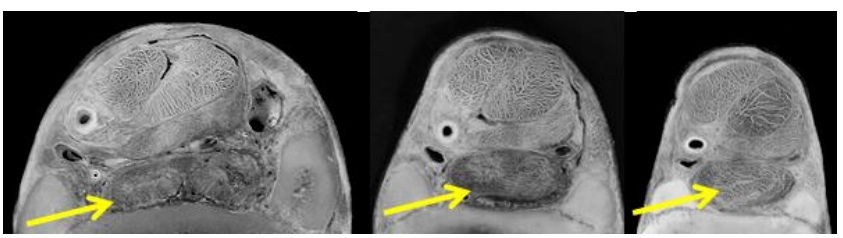


Figure 13. Example of simple anatomy with complex architecture taken from same limb on same speciman: transverse section of proximal metacarpus at 2 (left), 5 (center) and 10 cm (right) distal to carpal metacarpal joint. Note alteration of suspensory ligament ultra-architecture. Yellow arrow=suspensory ligament.

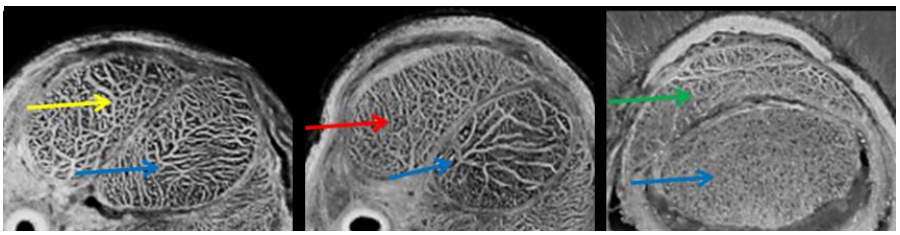


Figure 14. Example of simple anatomy with complex ultra-architecture taken from same limb on same specimen: transverse section of SDFT at 2 (left), 15 (center) and 25 cm distal to carpal metacarpal joint. Note that tendon fascicles are separated by prominent endotendon (fascicular membrane) in a somewhat random pattern (yellow arrow). At mid metacarpus, the endotendon becomes finer with a more dorsal palmar orientation (red arrow). At distal metacarpus, endotendon becomes progressively finer with a dominant medial lateral architecture (green arrow). Note the changes that occur in the DDFT in the same specimen group (blue arrow).

Anatomic fluency is required especially in complex structure evaluation. Two situations exist to add complexity: first, some areas are highly complex; i.e. the distal sesmoidian ligaments. See Figure 12. Secondly, some structures are simple but have a complex architecture; i.e. proximal suspensory ligament and superficial digital flexor tendon. See Figures 13, 14.

The operator is then responsible for appropriate representation of anatomy. This is difficult as there currently is no standardization regarding number, orientation or formatting of images. This complicates the interpretation by other observers, makes archiving difficult and decreases the likely hood that other operators will produce corresponding images unless the original is provided. See Figure 15.

3) Interpretive skills: Ultimately the operator is asked to provide an interpretation of the findings yielding the final limitation imposed by the operator. Comparison evaluations of lesions by MRI and ultrasound consistently determine that ultrasound commonly misses pathology or incorrectly identifies normal tissue as pathologic. Several factors are responsible:
   a. Ultrasound interpretations are commonly based on imaging only. This is problematic because tissues are biologic; they have structure variation between limbs and between other horses. In addition, often soft tissue structures are injured, healed and remodeled so that imaging later will demonstrate a variation that may not be clinically significant. If an operator looks hard enough there is always variation in tissue, therefore it is incumbent on the operator that a validating thorough clinical examination be performed prior to ultrasonography. This is especially mandatory in the absence of obvious heat pain or swelling. This author feels strongly that when possible diagnostic anesthesia should be used to assist in the localization of injury. It is somewhat bewildering how frequently diagnoses are formulated by clinicians after a cursory examination and "validated" by ultrasound imaging. This often leads to imaging a non-affected structure and worse…a frustrated client. See Figure 16 a/b.
   b. Ultrasound interpretations were usually based on the identification of hypoechoic foci. This simplistic approach was born from previous techniques/interpretation/experience with older machines that simply did not have the capability to image tissue to the degree of detail necessary to appreciate

subtle changes. See Figure 17. A new paradigm of assessment must include the nuances of size, ultra-architecture and contrast enhancement techniques (to be discussed later in chapter). At this time, technology and operator skill sets have somewhat outpaced the interpretive skill set.

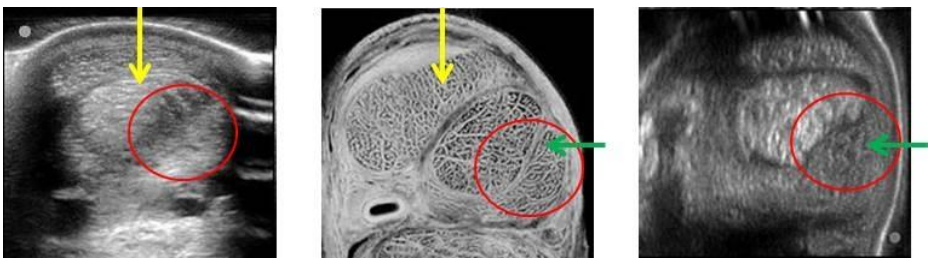


Figure 15. Comparison of traditional and nontraditional imaging orientation. Horse presented with enlargement and sensitivity to palpation of ALDDFT. Traditional image (left), viewed from palmar approach, yellow arrow, obfuscated the detail of pathology. Nontraditional image (right), viewed from lateral side of limb, green arrow, clearly demonstrates pathology of ALDDFT within red circle. Tissue enlargement with multifocal small hypoechoic foci along lateral palmar margin of ALDDFT adjacent to dorsal DDFT is present. Note that the right image was rotated 90° to right to afford correlative orientation with the specimen in center.

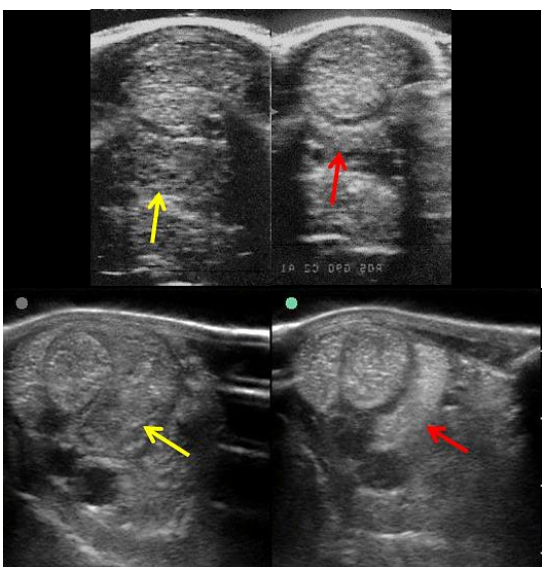


Figure 16 a. Comparison images of BF ALDDFT. Horse presented with diagnosis of ALDDFT desmitis of LF. Diagnosis based on clinical thickening of structure, sensitivity to palpation and ultrasound imaging (top images). Trainer requested second opinion after lack of improvement (images on bottom). Note improvement in image quality as a function of improved acoustic contact because of lateralized probe orientation. Interpretation: LF ALDDFT (yellow arrow) thickened compared to contralateral limb structure (red arrow). Mild heteroechogenicity with enlargement and minimal edema suggests remodeling. Lack of hyperechogenicity of deep structures under suspect area suggests that edema often present with acute injury, is not present. This sonogram is most consistent with a healed remodeled ALDDFT. This case is a good example of how valid ultrasonographic findings are not relevant to presenting clinical problem.

Figure 16. (Continued).

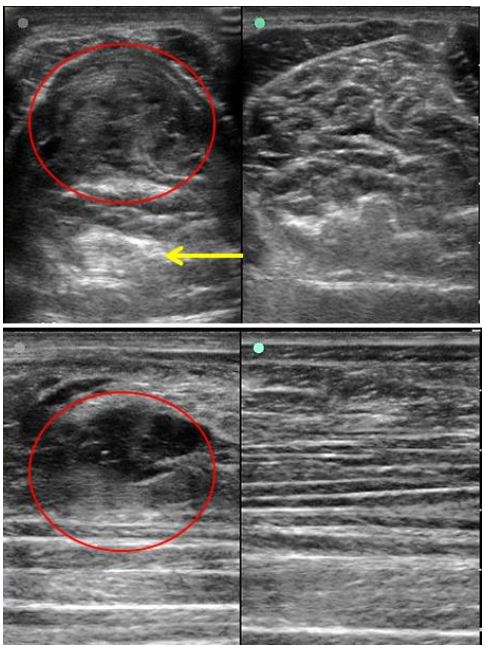


Figure 16 b. Comparison images of BF SDF musculotendinous junctions. Transverse images on top with longitudinal sections on bottom: Further investigation noted effusion of carpal canal with deformation of palmar surface of antebrachium. Imaging revealed disruption of the musculotendinous junction of the superficial flexor muscle and tendon. Note absence of muscle architecture on left images (red ovals) as compared to right images (unaffected limb). Also, in LF transverse image (top right) that a zone of hyperechogenicity exists deep to the injured structure (yellow arrow).

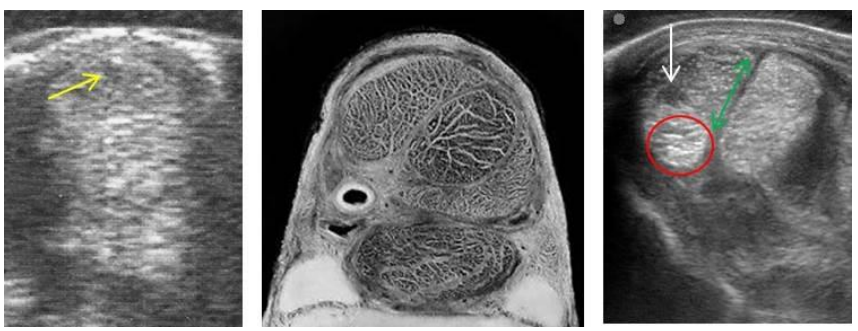


Figure 17. Image comparison of SDFT tendon pathology with older (left image and newer system (right image). Note sonogram on right demonstrates focal hypoechoic lesion in palmar SDFT (yellow arrow). Sonogram on right demonstrates a similar focal hypoechoic lesion (white arrow) with medial portion of tendon demonstrating hyperechogenicity (red circle) while lateral portion of tendon lacks fascicle architecture (green arrow) with overall enlargement and deformation. Reference anatomy in center image. Interpretation of right image: active tendonitis (white arrow) with chronic tendinosis (green arrow) as represented by reduction of endotendon architecture and overall increase in size and alteration of shape. Hyperechoic area could be secondary to fibrosis but most likely is artifactual because of formation of acoustic window in more superficial tissue as a result of active inflammation. This sonogram suggests that this injury is a recrudescence of a previous injury. Horses with similar sonograms are prone to repeated reinjury when maintained at the previous level of work. To keep serviceable, this horse will likely need to reduce or change his level of work.

c. The final limitation imposed by the operator is the lack of correlative studies between histology, magnetic resonance imaging and ultrasound imaging. This is clearly the missing link in the advancement of interpretation of ultrasound imaging.

Each horse that has had MRI performed should be looked at as an opportunity to sharpen ultrasound skills and interpretations. It is easy to underestimate how much structural detail is available through the use of ultrasound. The lesions of MR high signal are almost always visible with ultrasound although the changes can be quite subtle and can be overlooked. In addition, if such scrutiny is applied to all tissue it becomes easy to over interpret sonograms. Finally it is not practical to evaluate all tissue to the fineness of detail but when not performed the risk of overlooking the pathology is almost certain.

This highlights the importance to doing a thorough clinical examination prior to imaging so the attention of imaging can be focused to the areas of highest probability of injury. Other suggestions for improvement of ultrasound interpretation would include:

1) Each MRI case should be imaged with ultrasound to document ultrasonographic findings. These images then may serve as a comparison for the clinical management during the rehabilitation process. It is difficult to ultrasound a lesion six weeks after an MRI scan and objectively interpret a difference.
2) MRI centers should form maps of most common lesion sites of soft tissue structures. This would focus attention to most common sites of injury and not waste time on low risk areas.
3) When possible, histology should be performed so as to elevate understanding of the healing process and how such healing correlates to ultrasonographic imaging.

Image Quality Optimization

Image optimization is a multifaceted endeavor. Most practitioners are focused on the purchase of the “right” or “best” machine. As with our stereos in our homes, most people have much more capability in their existing systems than they are currently availing themselves of during imaging. Carrying the stereo metaphor further, we place speakers in corners so they are out of the way, we use connections because we have the cables immediately available, etc. These decisions are based on convenience not optimization of sound quality. Likewise with ultrasound imaging some operators will only image one limb or not clip the hair because of convenience. And so, decisions are made on convenience not image quality. To be fair, most operators desire optimal images but the constraints of time and cost become the limiting factors. That being said; the factors to consider when optimizing image quality are:

a. Machine selection: As alluded to in previous discussion, each machine has advantages and disadvantages. The selection criteria in order should be:

i Does the machine physically fit? Console units are very nice but they do not ship well in the back of an ambulatory vehicle although they typically give better images and have more advanced software than portable systems.

ii Is the machine appropriate? While durable hand carried bovine reproduction units are ideal for their intended purpose they typically are unable to produce desirable images for equine musculoskeletal studies.

iii Cost and brand. The danger here is purchasing a machine that is good but not quite good enough. The pitfall is that it imposes a limitation on the operator so that quality of image is not seen as desirable enough to justify ultrasound investigation of other than superficial areas so the machine is used less. Yet, the system cost was enough that owner is reluctant to replace the machine, trapping the owner in a cycle of mediocre imaging. Always purchase a machine that has excellent support.

b. Probe selection: No single probe can provide adequate imaging of the horse. Typically three probes are required:

i High definition linear probe: "tendon probe" with removable standoff. These will vary in frequency from 6 to 15 MHz. Most commonly 10 MHz provides the most useful penetration to detail quality. Width of the probe is also important. Some probes are 50 mm wide allow complete visualization of the origin of the suspensory ligament while others are 40 mm (or even narrower). The disadvantage is that the origin of the suspensory ligament will not be able to be captured in one frame and that longitudinal images include less length. However, a narrower probe allows more distal imaging of the DDFT because the probe is physically able to fit between the heel bulbs.

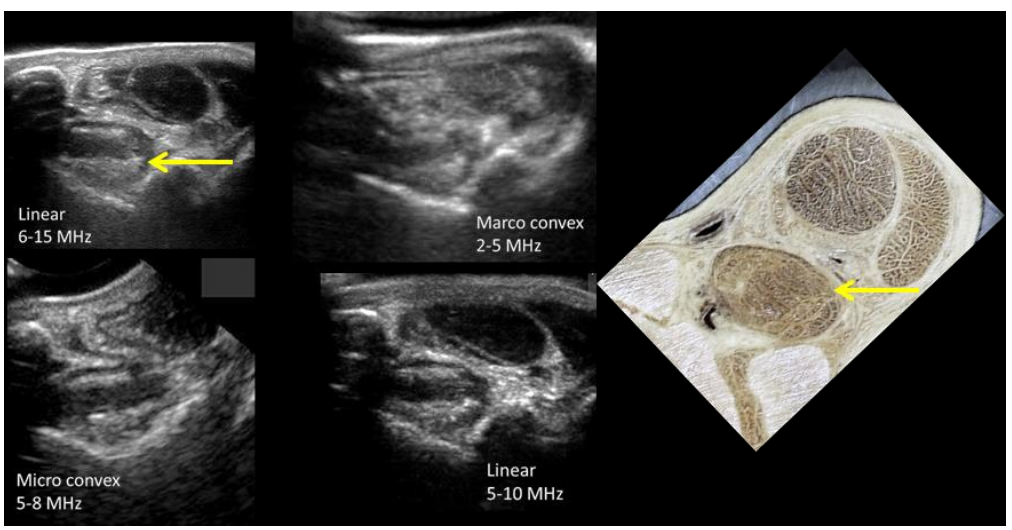


Figure 18. Comparison imaging using various probes on proximal hind suspensory ligament 5 cm distal to tarsal metatarsal joint with contrast enhancement technique. Yellow arrow demonstrates lateral plantar margin of proximal suspensory ligament. Reference image provided on right. Note that all probes are able to image the structure in question but that higher frequency probes provide better detail. Also, note that the linear 5-10 (rectal) probe can provide adequate imaging.

ii Macro convex probe: “big probe”. These will vary in frequency from 2-5 MHz. They are commonly used to gain access to deeper structures i.e. caudal ligaments of stifle, palmar heel structures and spine.
iii Micro convex probe: These will vary in frequency from 5-8 MHz. They are typically used in evaluation of the distal pastern between the heel bulbs and are essential in the procedure of ultrasound guided needle placement.
iv Rectal probe: These vary in frequency from 5-10 MHz. They are useful and essential for internal evaluation of the lumbar discs and ventral vertebral ligaments, SI joints and pelvis. The long length of the probe can facilitate external evaluation of longer structures, i.e. peroneus tertius injury.

While the cost of each probe is significant, each has a specific purpose and need. If a probe is not available, the image quality may be compromised or unusable. Above is a comparison of imaging quality using various probes on the same structure. As can be visualized, most of the probe can give significant structure information but certain compromises are made. See Figure 18.

a. Technique: All technique starts with site preparation. Before any imaging is attempted, it is recommended that the horse be moved to a quiet area of the barn with minimal distractions (especially during feeding or turn out). Routine sedation is recommended to that focus can be placed on image acquisition and not on horse behavior modification. 10 mg romifidine IV provides a nice low level sedation that clients do not find to be objectionable. Skin preparation of both limbs should include:
   i Remove as much hair as is allowed by owner/trainer. Keeping in mind that a #10 clip will usually provide adequate contact.
   ii Repeatedly soak skin with warm water while washing with gentle soap. In colder climates, scrubbing action and prolonged water contact help hydrate the dermis.
   iii Apply reference markings. Usually 5 cm markings from the nearest joint as verified by ultrasound. Typewriter correction fluid (White Out) works extremely well. This orientation technique is much more precise that the ambiguous designations by zone. In addition, it provides a very efficient and repeatable methodology for progress evaluation.
   iv Rinse soap and apply contact gel. If foam develops while scanning, alcohol may be applied to reduce acoustic interference.

After skin preparation, a suitable small stool or kneeling pad should be used to maximize comfort. Image quality is usually directly proportional to operatory comfort. Other items useful to have at hand are:

i Bucket with warm water
ii Towel
iii Contact gel
iv Spray bottle of alcohol
v Ruler
vi Marking fluid (White Out)

vii Mobile cart for ultrasound machine. It is easier to move a rolling cart next to a horse than to position a horse near a cart.

The next step includes a systemization for documentation. At this time there is no universally accepted formatting of documentation. This information represents the minimum needed for adequate case management. Additional information may be required to conform to standards for legal record keeping. At a minimum, each image should contain the following information:

i Date
ii Name of horse
iii Name of owner
iv Limb
v Structure of study focus
vi Probe
vii Depth
viii Orientation
ix Distance from nearest joint

Below is a sample screen at image capture. See Figure 19. As a reminder, all initial evaluations should include the contralateral limb as a comparison. In the author's opinion, this is best accomplished with the bilateral structure and orientation on the same split screen.

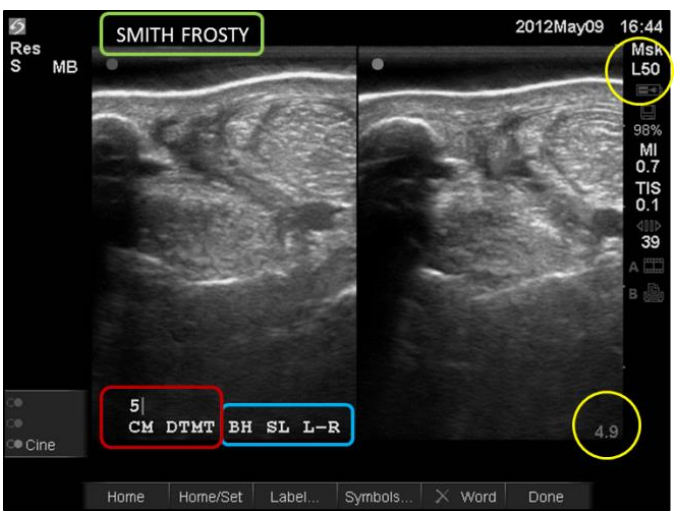


Figure 19. Sample of documentation protocol on ultrasound image capture screen:

- owner's last name and horse's name are highlighted in green.
- date in upper right.
- machine information including probe and depth circled in yellow.
- limb designation in blue indicating image of both hind suspensory ligaments; left limb to left, right limb to right.
- probe orientation in red indicating that this image set was taken at 5 cm distal to tarsal metatarsal joint.

This documentation protocol is quite reproducible and requires a minimum number of key strokes. In addition to this documentation information, it is essential that the orientation of the image be consistent and noted in medical records. Although not formally or universally adopted by the veterinary community, it is somewhat of a convention that ultrasound images are oriented so that:

i Medial is always to left of screen.
ii On split screens, both images have medial to left.
iii On split screens, left limb is on left, right on right
iv Proximal is oriented to the left. Distal is oriented to the right.

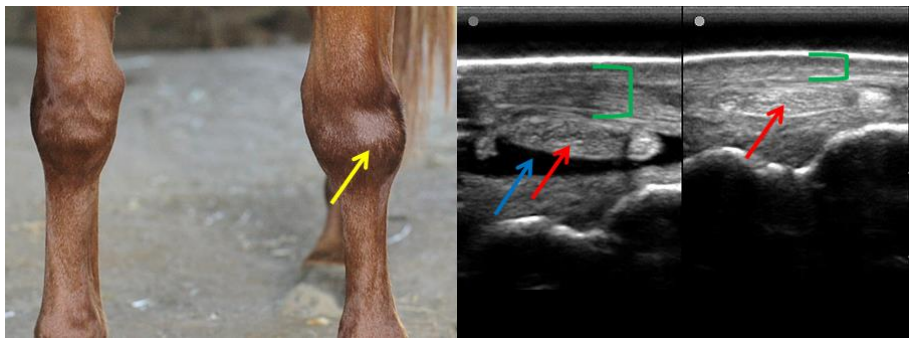
This convention may change with time. It would make sense if MR, CT and DUS would all follow a similar formatting. Because no standards are adopted, it becomes incumbent on the operator to declare his/her orientation when images are shared. Other helpful notes may be added to image to assist in interpretation i.e. lateral orientations, contrast enhancement techniques, non-weight bearing etc.

Using Ultrasound to Identify Tissue Pathology

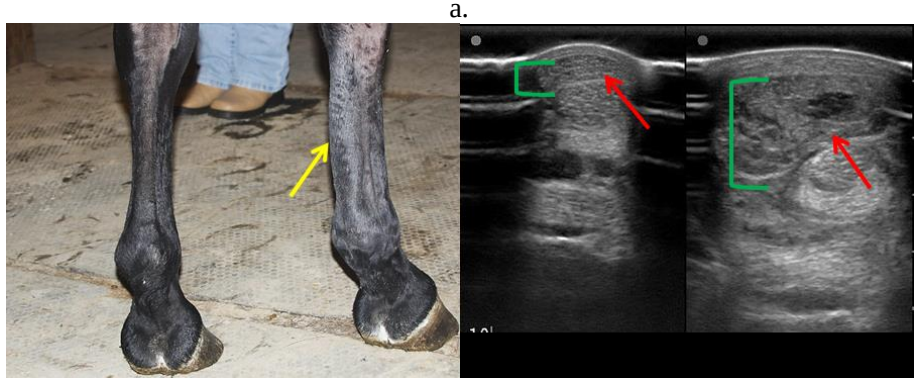
Interpretation of a sonogram is a subjective evaluation of a two dimensional image of a three dimensional structure based on the quality of image acquisition and the skill of the operator. It is no wonder that ultrasound diagnoses have been not well correlated to MRI, histologic or intra-operative findings. [7, 8, 9] This does not mean that ultrasound is not without merit as a diagnostic tool but rather it is an indictment for a more systematized and rational approach to interpretation of sonograms. Listed below are the criteria that are useful in the interpretation of ultrasonographic images:

1) *Validation* of origin of discomfort: if an area demonstrates *obvious* heat, pain and swelling coincident with onset of gait asymmetry then a reasonable assumption is that a structure within the inflamed area is related to the lameness. If no heat, pain or swelling is evident, then diagnostic anesthesia is essential to the localization of injury. Because soft tissue structures demonstrate natural variation (size and architecture) and remodel with injury, this criterion of validation of origin is the most important aspect of interpretative assessment. It is not difficult to determine if ultrasonographic structural asymmetry exists within a given soft tissue; however, it can be very difficult to determine if it is significant simply by ultrasonography. Ultrasonography is not a substitute for systematic gait evaluation followed by clinical examination and possibly diagnostic anesthesia. As a matter of interest, most artifacts induced by diagnostic anesthesia are resolved after 24 hours. [11]
2) *Sensitivity* to palpation: aside situations that include oblivious heat, pain and swelling, careful palpation of localized individual structures that are injured should repeatedly demonstrate sensitivity to palpation. Although the interpretation of sensitivity can be quite subjective depending on the technique of the examiner and the overall sensitivity of the horse, most horses typically demonstrate an increased sensitivity to palpation. Sensitivity to palpation is an important criterion to help

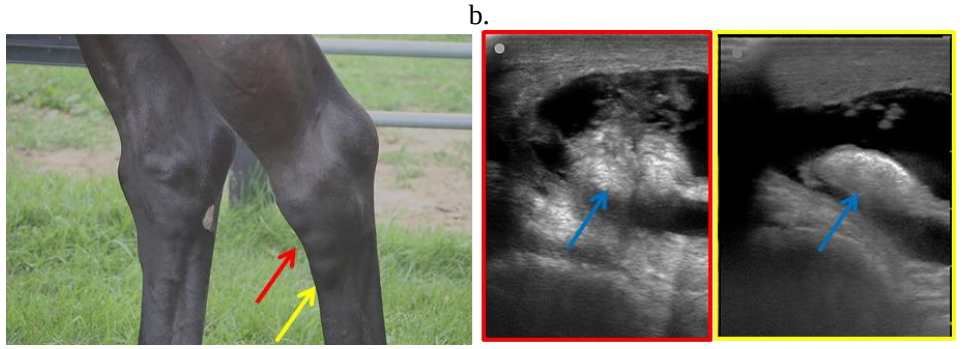
validate injury interpretation but it is the most subjective of the listed criterion and cannot be used as a sole validator of pathology.



a.



b.



c.

Figure 20. Examples of observable enlargement associated with soft tissue injury. a. Diffuse non-structure specific enlargement (yellow arrow on left) of LF dorsal lateral carpus. Ultrasound comparison of LF (on left) and RF (on right) demonstrates synovial effusion of common digital extensor tendon sheath (blue arrow), thickening of subcutaneous tissue (green bracket) and mild enlargement of CDE tendon (red arrow). b. Structure specific enlargement of RF superficial digital flexor tendon (yellow arrow). Ultrasound demonstrates injury to RF SDFT (red arrow in right sonogram) compared to contralateral limb (left sonogram). Note that majority of tissue enlargement is result of specific SDFT injury. Note comparison of RF to LF SDFT thickness (green brackets). c. Distension of extensor tendon sheath at dorsal tarsi. Sonogram to right (yellow box) is taken at level of greatest distension. Sonogram on left (red box) taken at mid-tendon sheath between tarsal and metatarsal retinaculi. Note that at the area of greatest effusion, the tendon (blue arrow) is intact but that more proximally, the tendon is fissured. These fissurations are common and associated with trauma secondary to contact with the retinaculi.

3) *Enlargement* of structure: enlargement may be identified by visual observation, palpation or ultrasonographic imaging. Visual observation of soft tissue injury can be associated with injury to a specific structure, diffuse non-specific inflammation/ fibrosis or effusion of a synovial structure. Specific enlargement of a single structure is very useful to localize injury. Diffuse enlargement may simply be caused by superficial trauma, localized or ascending cellulitis or a regional response to soft tissue injury. Therefore diffuse enlargement is most often an indicator of regional injury but not specific to the individual structure. Synovial structure enlargements, especially cases of acute tenosynovitis, clearly demonstrate visual enlargement. These enlargements are most often associated with injury to tissue within or adjacent to the sheath. The soft tissue injury induces an effusion with in the sheath. The sheath will enlarge at the point of least restriction and typically not at the point of injury. See Figure 20.

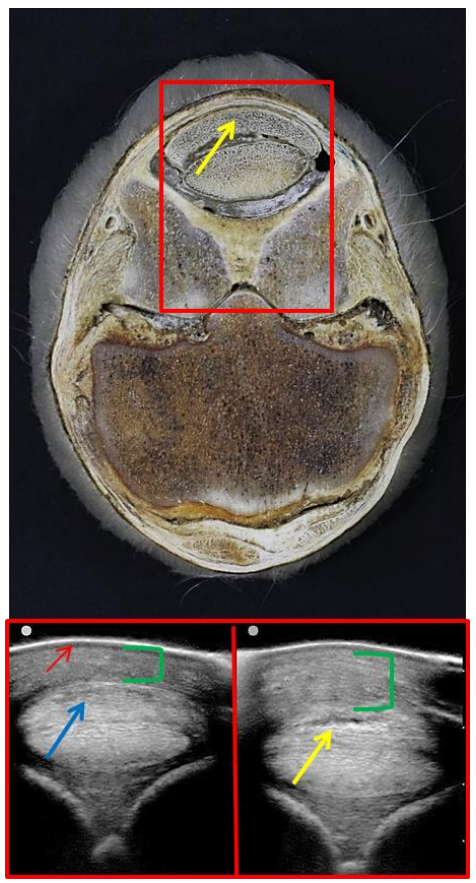


Figure 21. Example of complex bilateral enlargement of palmar distal metacarpus. Sonogram on right shows fissuration with linear hypoechoic lesion in mid SDFT tendon (yellow arrow). Green bracket demonstrates fibrotic remodeling of adjacent tissue. The contralateral limb (left sonogram) demonstrates the presence of tendinosis (blue arrow) of the SDFT with a lesser degree of fibrotic remodeling (green bracket). Note the deformation of palmar skin margin on medial aspect of limb (small red arrow). A reference image is provided above with yellow arrow identifying the corollary SDFT structure.

In addition to visualization, a good clinical examination includes palpation of all questioned structures. Palpation will identify diffuse and structure specific enlargements. As commonly interpreted, diffuse enlargement can be the result of many processes from orthostatic edema (stocking up) to phlebitis secondary from a hoof capsule abscess. Owners will not uncommonly call with concern regarding enlargement of the palmar metacarpus thinking an injury to the suspensory ligament has occurred when it is later identified to be an abscess of the foot. As can be surmised, diffuse enlargements must be corroborated with other evidence before a specific diagnosis is proposed. With skilled evaluation, specific structures maybe identified as being structurally enlarged. Because structure sizes vary from individual to individual, it is important to compare size to the contralateral limb, keeping in mind that bilateral structure pathology may exist. Specific enlargement without sensitivity to pressure is usually a sign of a more chronic or healed injury. However, individual variation in pain tolerance compromises the usefulness of interpretation. Generally speaking, palpable enlargement should serve as an indicator for further assessment, not as a specific criterion for diagnosis.

Aside from frank enlargement, the best assessment is with visualization using ultrasound. As demonstrated in Figure 21, it becomes quite straightforward to identify the presence, amount and cause of enlargement. Often ultrasound shows that enlargements are the composite effect of structure injury, inflammation and remodeling.

4) *Variation of architecture:* typically, ultrasonography is utilized to identify structure pathology by the identification of architecture variation. Most commonly, this is performed by the perpendicular placement of the probe to the questioned structure, alteration of the beam incidence to maximize the echogenicity of the structure and then determine if an area appears hypoechoic. While this approach may be useful as a starting point, it typically overlooks the most valuable and subtle changes that is essential for a proper interpretation. Images as shown below in Figure 22 are no longer adequate for the evaluation of soft tissue injury.

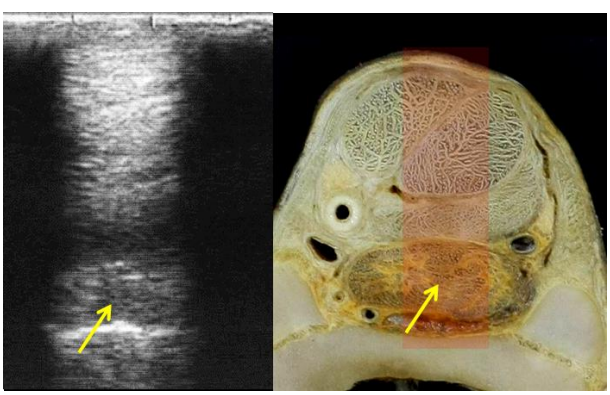


Figure 22. Example of an inadequate sonogram interpretation. The given example demonstrates a definitive hypoechoic focus (yellow arrow) in the body of the suspensory ligament (about 12 cm distal to CMC joint) which is wholly an artifact. The hypoechoic focus most likely is a result of an oblique incidence on the axial fibers of the suspensory ligament. The red shaded area on the anatomic specimen demonstrates the approximate imaging of the sonogram. The yellow arrow indicates the correlative imaging area. Misinterpretation of similar sonograms is fortunately becoming less common.

More recently, advanced interpretations have been described using contrast enhancement techniques. [4, 10] These techniques have been described as: “off incidence”, “off beam”, “oblique incidence” imaging. Other terms, coined by JM Denoix have been “contrast enhancement” and “angle contrast ultrasound” techniques. For simplicities sake, the term “contrast enhancement technique” (CET) will be used in this discussion. CET takes advantage of the anisotropic properties of linear connective tissue to facilitate evaluation of fiber linearity within the connective tissue.

Linear structures typically have directionality to their structure and are described as displaying “anisotropy”. Anisotropy is defined as a property of being directionally dependent. More to the point, the measure of echogenicity is dependent on the perpendicularity of the probe to the axis of the fibers. See figure 23.

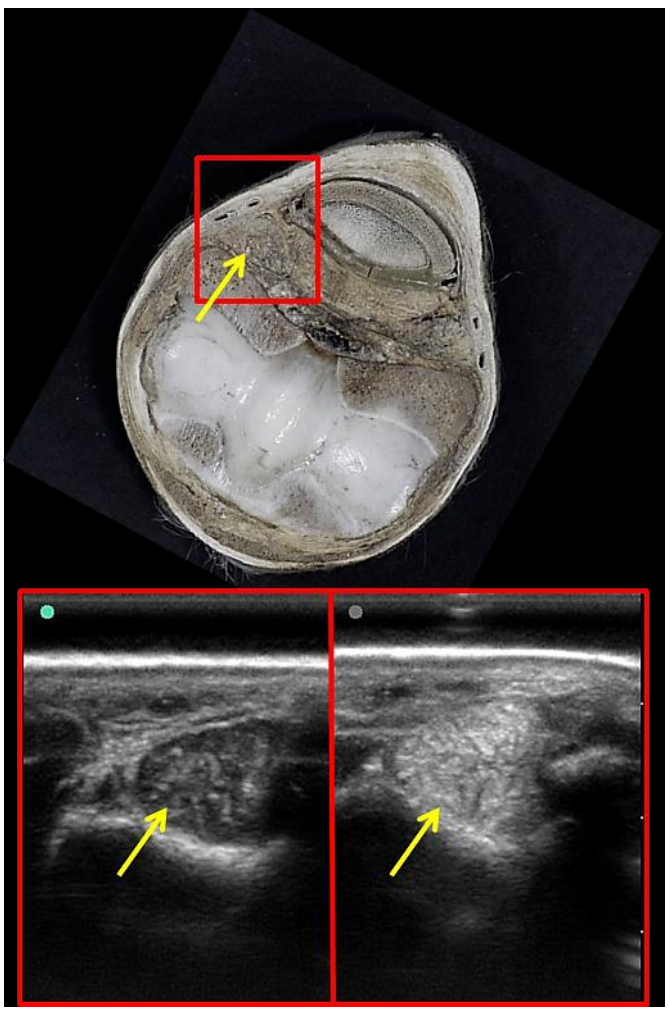


Figure 23. Example of anisotropy: Transverse images of lateral oblique sesmoidian ligament at level of MCP joint. Image on right demonstrates echogenicity associated with peripedicular beam incidence. Image on left demonstrates echogenicity associated with oblique beam incidence. Note echogenicity of LOSL structure (yellow arrow). Comparison anatomic image on top.

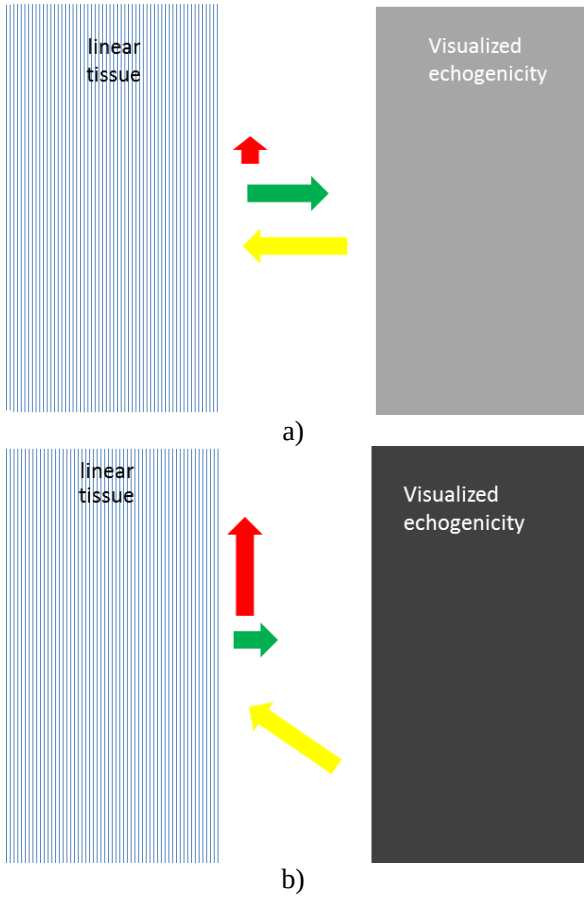


Figure 24. a) Yellow arrow represents acoustic energy from ultrasound probe. Upon impact with linear perpendicular fibers, the acoustic wave diffracts slightly with the majority of energy returning to the probe. The returned wave energy is represented by the green arrow. The diffracted energy is represented by the red arrow. The echogenicity is converted into a gray scale that is relatively bright. b) During CET, the oblique incidence of the acoustic wave causes much greater diffraction of wave energy away from the probe (larger red arrow). As a result, a smaller amount of energy is returned to the probe (green arrow). This yields a much darker gray scale.

To get a better understanding of this imaging and how it may provide additional interpretive value we should revisit the formation of ultrasound images. As shown in Figure 24a sound waves are produced by the ultrasound probe which are perpendicularly transferred into the soft tissue. Upon impact, the majority of wave energy is reflected back to the probe, where the amount of wave energy is given an amount of gray scale that we see on the screen: more energy=more white, less energy=more black. In Figure 24b, sound waves are obliquely transferred (CET) into the soft tissue. As a consequence of diffraction, most of the acoustic energy is diffracted away from the probe and less wave energy is returned.

Because most other connective tissues have less linearity to their structure, they have less anisotropy and are termed isotropic. In Figure 25 below, it is demonstrated that similar echogenicities are obtained even though different incidences of beam angle are used.

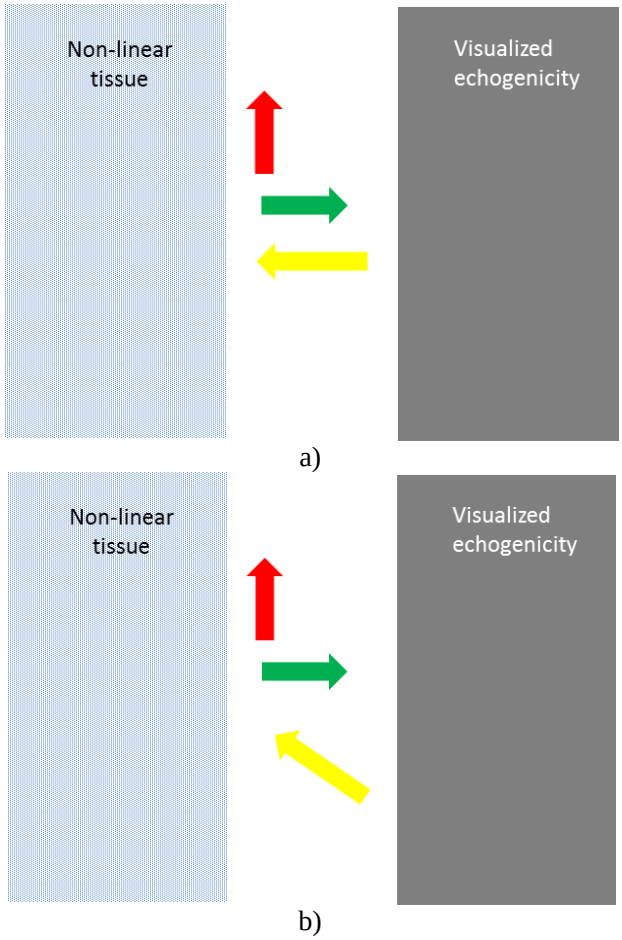


Figure 25. a) a non-linear tissue is perpendicularly imaged to produce a medium gray scale echogenicity. b) a non-linear tissue is obliquely imaged to produce a similar gray scale echogenicity. Note that the red and green arrows are the same size regardless of the orientation of the yellow arrow (incidence of acoustic wave transmission).

In the following Figure 26a, a composite tissue of linear and non-linear tissue is perpendicularly imaged where the top half of the object is linear and the bottom is non-linear. In Figure 26b, the same tissue is imaged with CET using an oblique incidence.

The images in Figure 26 are most similar to the tissue of the horse. There is always a composite of tissue types. When we scan with a perpendicular incidence only, we are typically gathering information and making assessments on echogenicity similar to Figure 26a below. If we avail ourselves of the anisotropic qualities and use CET, we are able to get much better contrast as exemplified in Figure 26b. As a more applicable example, the architecture of the proximal suspensory ligament in the hind limb is a composite of linear tendon like material with bundles of loose connective tissue, adipose and short muscle fibers in a non-linear orientation. Using traditional perpendicular technique, very little information can be gathered or interpreted regarding the structure. Using CET, the ultra-architecture becomes evident.

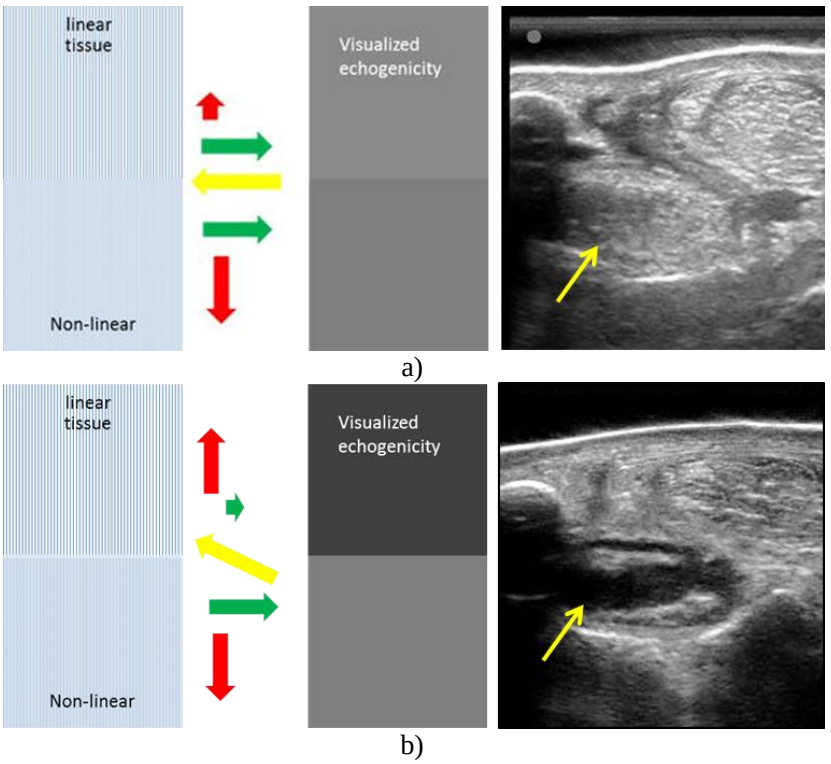


Figure 26. a) Normal perpendicular imaging of composite tissue. Note the minimal difference of the visualized echogenicity between the two tissue types. This lack of tissue differentiation is also evident in the transverse image of the hind proximal suspensory ligament (yellow arrow). b) Imaging of composite tissue using CET. Note the enhancement of the visualized echogenicity between the two tissue types. The corresponding sonogram demonstrates the differentiation between the tissue types within the proximal suspensory ligament.

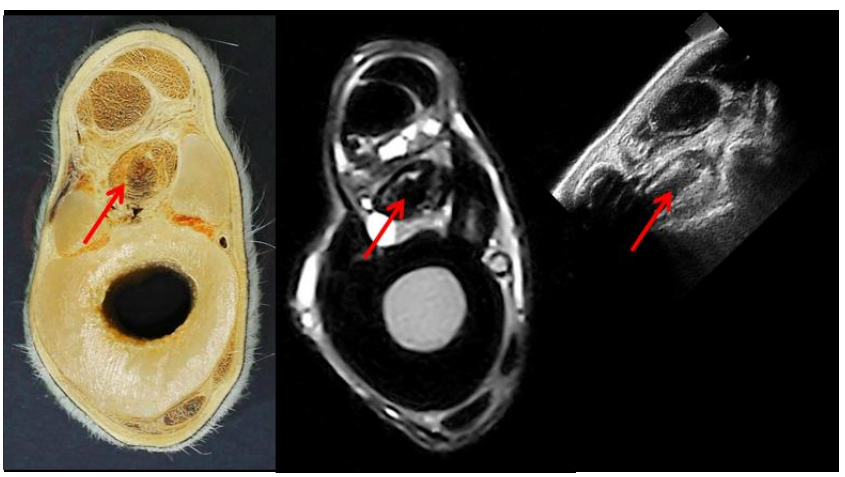


Figure 27. Imaging and anatomic correlation (transverse section at 5 cm distal to TMT joint) hind proximal suspensory ligament. Anatomic specimen on left, MRI center and CET ultrasound on right. Red arrow indicates sagittal part of proximal suspensory ligament. MRI image courtesy of Alexia McKnight DVM, DACVR.

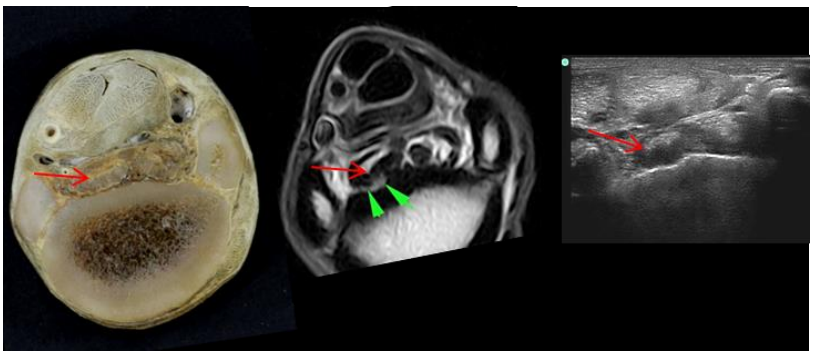


Figure 28. Comparison images of front proximal suspensory ligament. Left image=anatomic specimen, center image=MRI, right image=CET diagnostic ultrasound. Note increased signal at dorsal attachment of medial lobe of suspensory ligament (green arrow points) with increased thickness of medial lobe (between red arrow and green arrows. Although not the same horse, the CET ultrasound on the right demonstrates a similar increased thickness of the same portion of the medial lobe (note similar distance between red arrow and palmar surface of proximal metacarpus). The left anatomic specimen shows the typical thickness of the dorsal portion of the medial lobe. MRI image courtesy of Alexia McKnight DVM, DACVR.

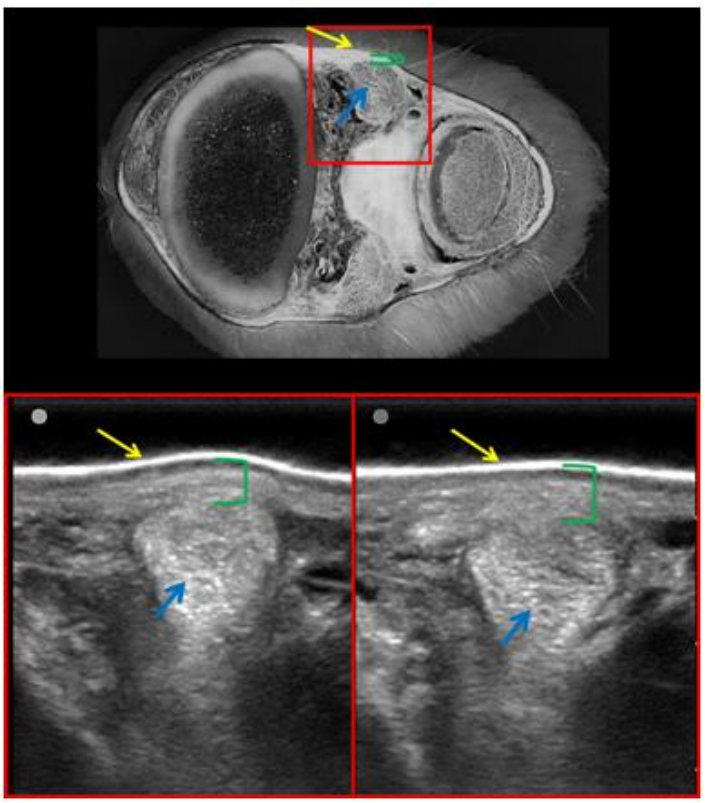


Figure 29. Example of skin deformation associated with desmitis of lateral branch of suspensory ligament (blue arrow) 5 cm proximal to MCP joint. Note flattening of skin profile (yellow arrow) on right image (SL LB desmitis) compared to left image (contralateral unaffected). Also, note increased thickness of soft tissue between triangular suspensory branch and dermis (green bracket). Anatomic reference provided on top of sonograms.

Using CET, better correlations can be made with anatomic specimens and MRI imaging. As an example, the proximal hind suspensory ligament is a simple structure with a complex ultrastructure. Traditional perpendicular probe incidence produces an image that might demonstrate gross structural enlargement but fails to give insight into tissue ultrastructure. See Figure 27.

Much more subtle interpretations are now possible because of the added information. We can evaluate:

- the boarder definition between the loose connective tissue bundles and the fibrous portions of the ligament.
- the compression of the loose connective tissue bundles secondary to edema.
- the enlargement of fibrous portions secondary to injury.

As an example, using CET, we are now able to appreciate the dorsal enlargement of the medial lobe of the suspensory ligament in the front limb. See Figure 28.

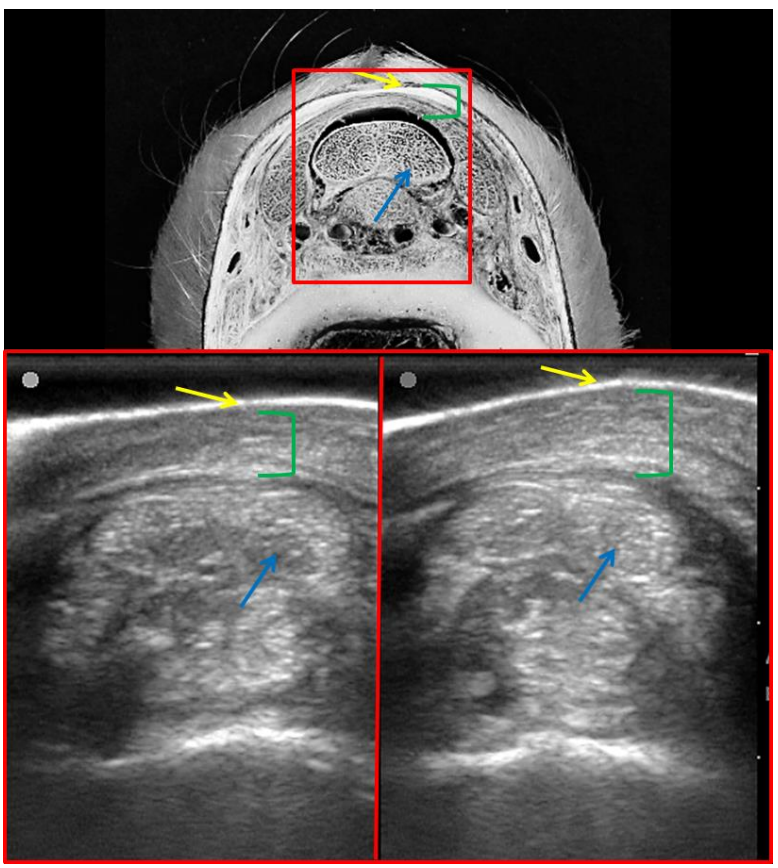


Figure 30. Example of skin deformation associated with fibrosis secondary to previous superficial injury at palmar pastern. This demonstrates that skin deformation may be secondary to processes other than musculoskeletal injury. Normal limb on left, affected limb on right, reference on top. Yellow arrow=skin contour, green bracket=sub dermal fibrosis, blue arrow=lateral lobe of DDFT.

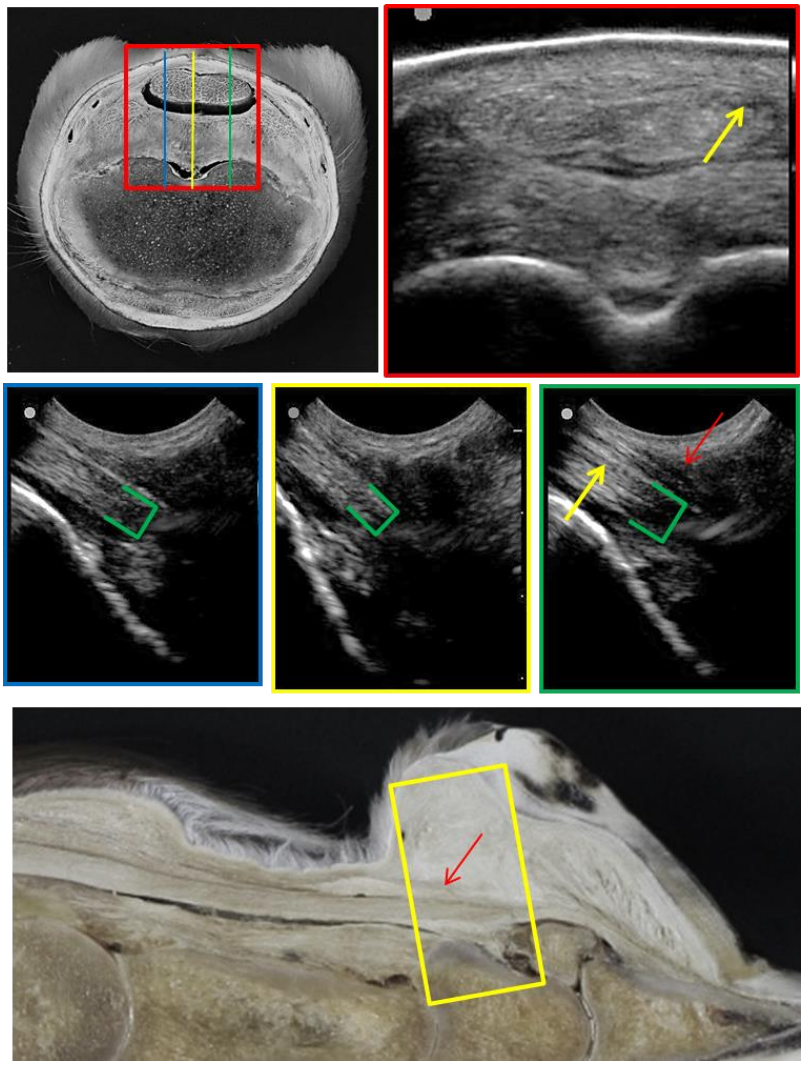


Figure 31. Transverse and longitudinal ultrasound images of DDFT at level of distal P1/proximal P2. Upper row: sonogram on right (red outline) demonstrates enlargement of lateral lobe of DDFT with irregular palmar border (yellow arrow) associated with adjacent hypoechoic defect. Note that the central and dorsal portion of the lateral lobe is hyperechoic consistent with fibrotic remodeling. A reference image is provided on the left. Second row: multiple longitudinal sections taken just distal to the transverse section. The yellow outlined image (center) is taken along the sagittal plane as approximated by the yellow line in the anatomic specimen in the first row. The blue outlined image (left) is taken along the medial lobe as approximated by the blue line in the anatomic specimen in the first row. The green outlined image (right) is taken along the lateral lobe as approximated by the blue line in the anatomic specimen in the first row. Note that variation in structure thickness can be a result of probe placement on a bilobular structure. As an example, compare the DDFT thickness (green bracket) in the blue and yellow outlined sonograms. This thickness in variation is not associated with injury. However, variation in DDFT thickness (green bracket) of the green and yellow outlined sonograms is the result of pathology (yellow arrow in green sonogram). As a matter of interest, the often overlooked distal annular ligament is visible and highlighted with the red arrow. Third row: sagittal anatomic image with highlighted distal annular ligament (red arrow). Yellow box approximates image projection of sonograms of second row.

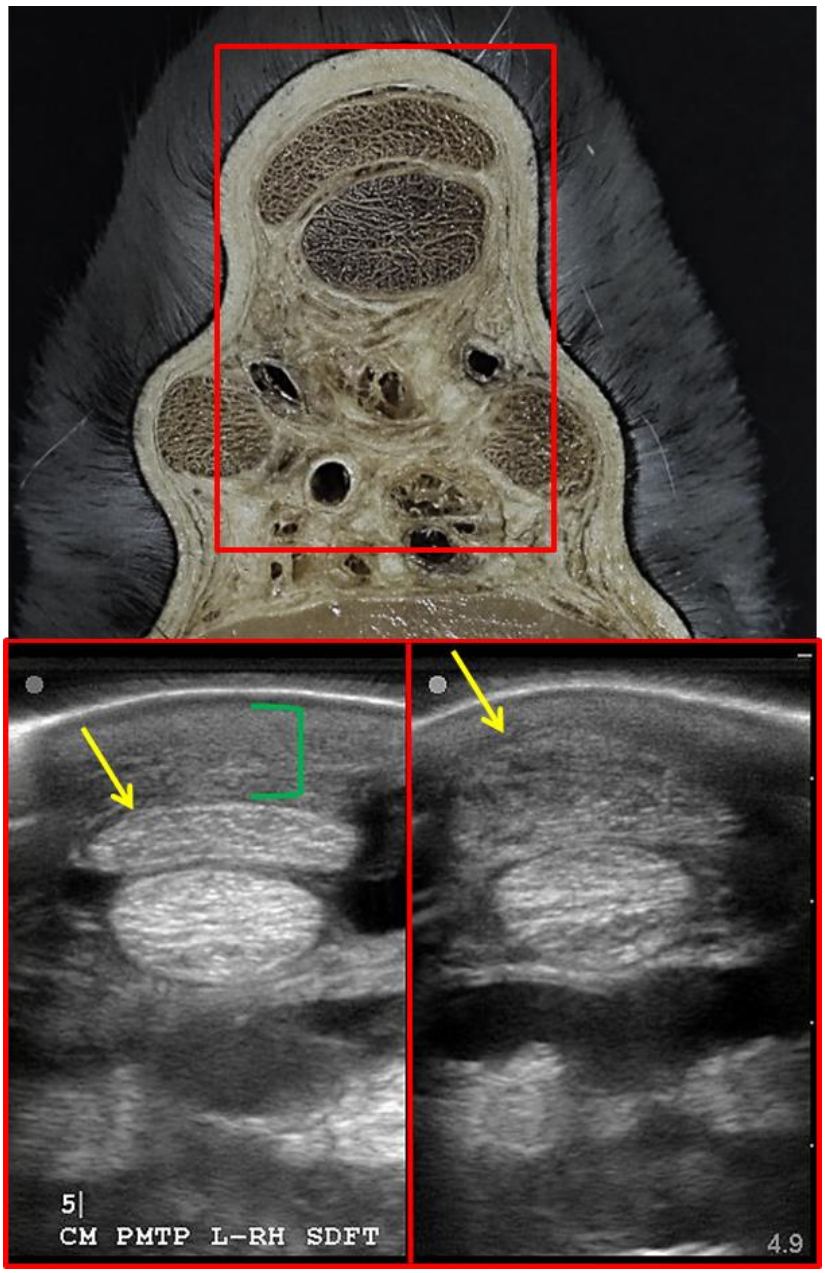


Figure 32. Transverse images of hind distal metatarsus. This horse presented with acute discomfort and enlargement of the RH distal plantar metatarsus. Comparison sonograms were acquired of both hind limbs. The right sonogram (affected limb) shows enlargement, deformation and echogenic changes of the SDFT. In addition the plantar border (yellow arrow) shows homogenization with the adjacent connective tissue. The left sonogram (contralateral) shows more normal SDFT size, shape and echogenicity although the adjacent connective tissue is thickened (green bracket). Interestingly, this horse recovered from the right hind tendonitis/tendonitis however developed acute tendonitis of the left hind limb with sonographic changes very similar to the right limb. Anatomic section provided for comparison.

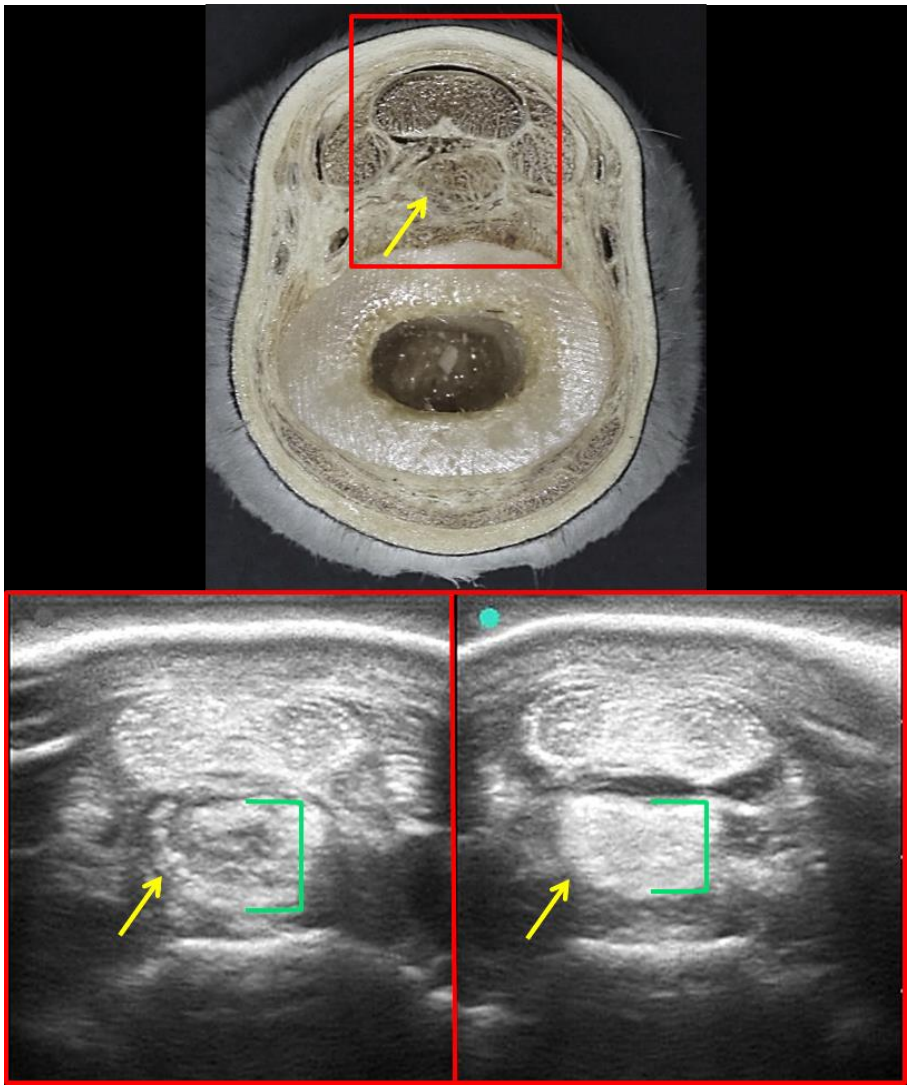


Figure 33. Transverse images of front pastern at mid P1. Horse presented with sudden onset lameness with diffuse enlargement of pastern. Sonogram of affected limb (LF) on left demonstrates: deformation of palmar skin surface, alteration of medial border of straight sesmoidian ligament (yellow arrow), enlargement of SSL (green bracket) with alteration of echogenicity. Sonogram on right is contralateral limb with markings for comparison. Reference anatomy provided above. Note that although the SSL typically has variation between and within individuals, the presence of multiple ultrasonographic findings supports validation of structure pathology.

The availability of advanced higher detail ultrasound systems, increased operator skill and emerging published comparative literature has begun a new era of ultra-architecture assessment. Several criteria are can be used to evaluate variation in soft tissue architecture:

(1) *Skin deformation:* often overlooked during ultrasound imaging, the contour of the overlying dermis typically demonstrates deformation when adjacent to soft tissue injury. A deformable standoff pad is essential in the evaluation of the overlying dermis. Comparisons should be made from images on the contra lateral limb. In

particular, it is not adequate to compare medial and lateral structures of the same limb as they often demonstrate a natural asymmetry. Keep in mind that excessive probe pressure can obfuscate dermal contour. See Figure 29 The presence of skin deformation is not specific to musculoskeletal injury but is usually present as either a consequence of acute inflammation or chronic fibrosis. Other causes of superficial skin trauma may easily mimic presentation of more serious injuries. See Figure 30.

(2) *Border definition and shape*: After evaluation of skin deformation, the border margin of the questioned structure should be identified. In acute cases, pathology will be associated with reduced echogenic clarity of boarder margin as the inflammatory process includes the structure and the adjacent tissue causing a homogenization of reduced echogenicity. In chronic active cases, homogenization of echogenicity is caused by fibrosis of structural and adjacent tissue. In addition, peripheral injury to structure will commonly cause an irregular border margin.

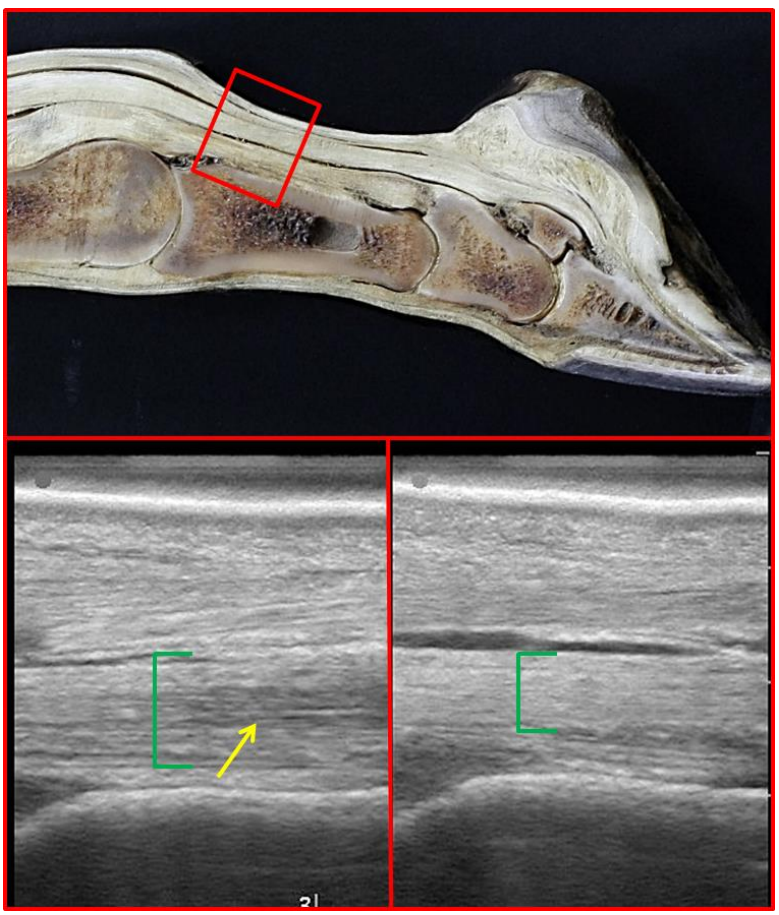


Figure 34. Longitudinal sections of mid pastern. The sonograms are of the same horse as in figure 33. The affected limb (LF-left image) shows sagittal thickening (green bracket) with diffuse hypoechoic density within SSL (yellow arrow). The contralateral limb is on the right with the SSL indentified with a green bracket. A reference anatomical image is provided above. Note that the linear density between the dorsal margin of the SSL (bottom of the green bracket) and the palmar margin of the pastern bone is the oblique sesmoidian ligament and not part of the SSL.

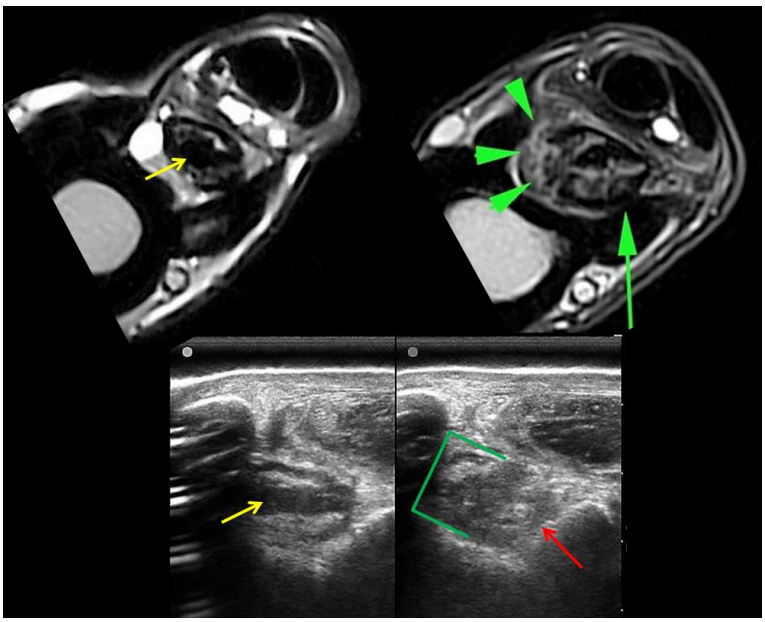


Figure 35. Transverse images of proximal metatarsus at 5 cm distal to TMT joint (MRI images of two different horses, sonograms of same horse). The right MRI image demonstrates increased signal uptake with architecture disruption (green arrows). The left image appears more "normal". The medial, sagittal (yellow arrow) and lateral portions of the proximal suspensory ligament are able to be identified as they are separated by a medial and lateral bundle of loose connective tissue made of adipose, muscle and loosely aligned connective tissue. Although the sonograms below are made of a different horse, they have similar appearance. The contralateral LH limb (left image) demonstrates the similar architecture provided by the MRI above. The yellow arrow also indicates the sagittal portion of the PSL. The affected RH limb (right image) demonstrates similar pathology as seen in the MRI above. In this image, there is enlargement (green bracket) with loss of border margin (red arrow). In addition, marked disruption of the architecture is demonstrated within the PSL. Note the usefulness of contrast enhancement technique to aid in the visualization of architecture in the sonograms. MRI images courtesy of Alexia McKnight DVM, DACVR

The shape of the structure is then evaluated using the contralateral limb as a reference. It is imperative to use the contralateral limb since much individual variation exists. Also, subtile size and shape changes are can be easily overlooked without a reference. The author suggests that after a survey scan of the affected limb, that images of the contralateral (unaffected) limb be scanned first and saved on half of the ultrasound system screen and then scans the affected limb be compared and saved on the second half of the screen. This allows the operator a real time comparison reference. See Figures 31, 32, 33, 34, 35.

Some ultrasonographers use cross sectional area measurements as an objective evaluation of injury and healing. Scientific literature is conflicting between two studies were one validated the objective use of CSA measurements while the other found that CSA measurements were not reliable. [12, 8] It is the experience of this author that objective interpretation of CSA measurements is difficult for a variety of reasons:

- dorsal margins of structures can be difficult to identify because of beam diffraction.
- it can be difficult to image the entire lateral/medial border margin because of inadequate acoustic contact with concave palmar/plantar surface.

- software guidance can be crude and awkward in field environment.
- subtile CSA changes are often within the realm of measurement error.
- measurements are sensitive to incidence of probe placement.

This is not to say that CSA measurement cannot be useful objective indicator of injury but rather, that CSA measurement is not simply acquired and the values may be subject to interpretation. Certainly, the use of CET may prove a very helpful technique to facilitate the accuracy of measurement however this has not been validated by study.

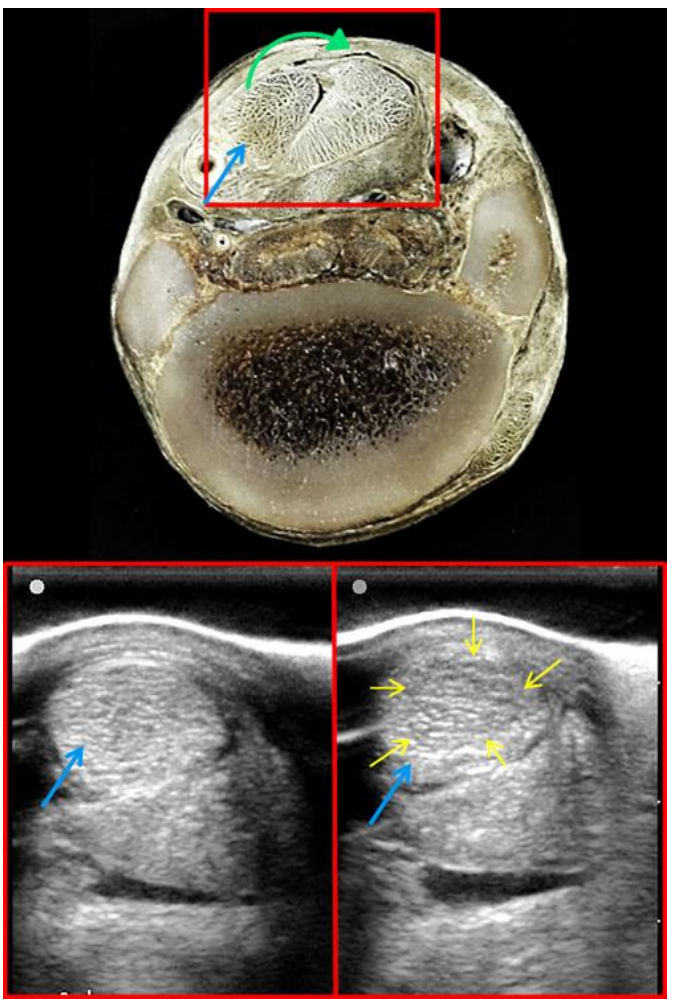


Figure 36. Example of tissue hypoechogenicity. Transverse sections taken of SDFT at carpal metacarpal joint. Blue arrows correlate medial margin of SDFT. Right front (right sonogram) demonstrates focal central area of hypoechogenicity (outlined by thin yellow arrows). Anatomic specimen provided above. [Green arrow demonstrates rotation deformation during specimen preparation] these signs are most consistent with a low graded tendonitis often seen with younger horses as they increase their athletic performance.

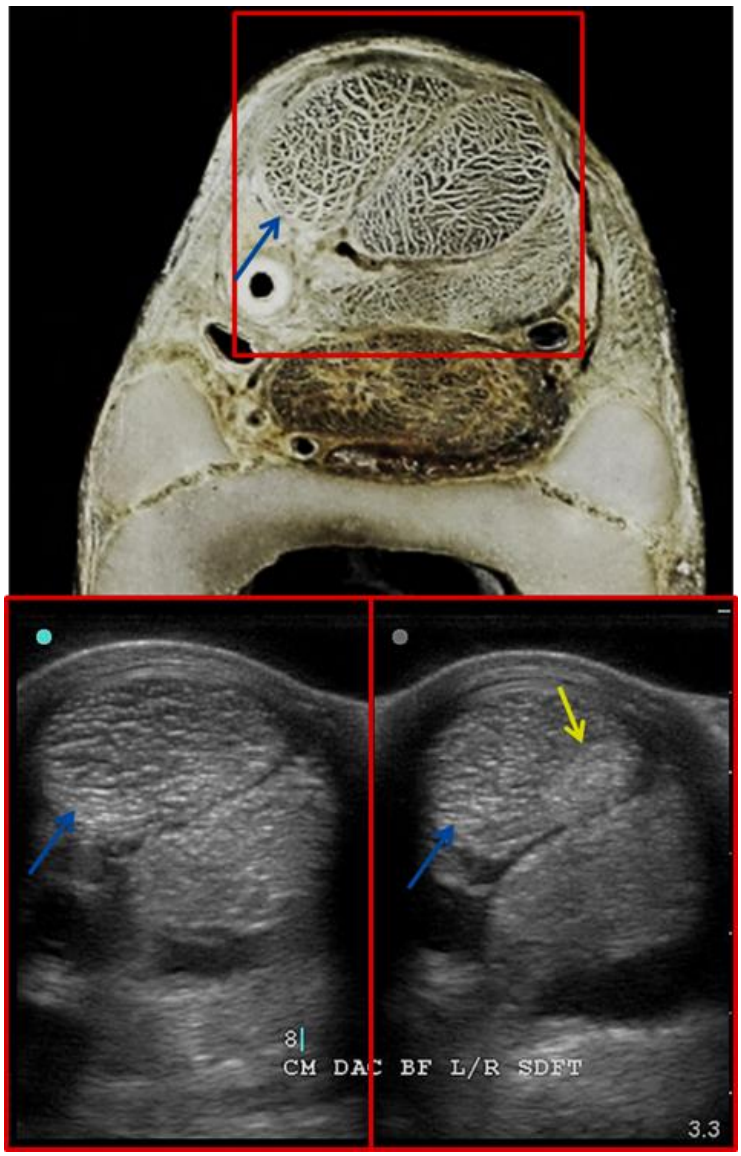


Figure 37. Example of tissue hyperechogenicity. Transverse sections taken of SDFT at 8 cm distal to carpal metacarpal joint. Blue arrows correlate medial margin of SDFT. Right front (right sonogram) demonstrates focal central area of hyperechogenicity (yellow arrow). Also note reduced prominence of endotendon architecture throughout RF SDFT. These signs are most consistent with increased fibrosis associated with healed remodeling of the tendon.

(3) *Echogenicity*: the perceived echogenicity of a tissue is simply an interpretation screen brightness of a structure based upon the amount of acoustic energy captured by the ultrasound probe. The structure brightness (echogenicity) is therefore dependent on multiple factors:
   (a) Probe selection
   (b) Probe incidence of contact
   (c) Acoustic contact
   (d) Skin hydration

(e) Superficial tissue density
(f) Tissue linearity
(g) Tissue anisotropy
(h) Tissue of interest density/architecture
(i) Tissue depth
(j) Machine algorithms
(k) Machine adjustment
(l) Ambient lighting at scanning site

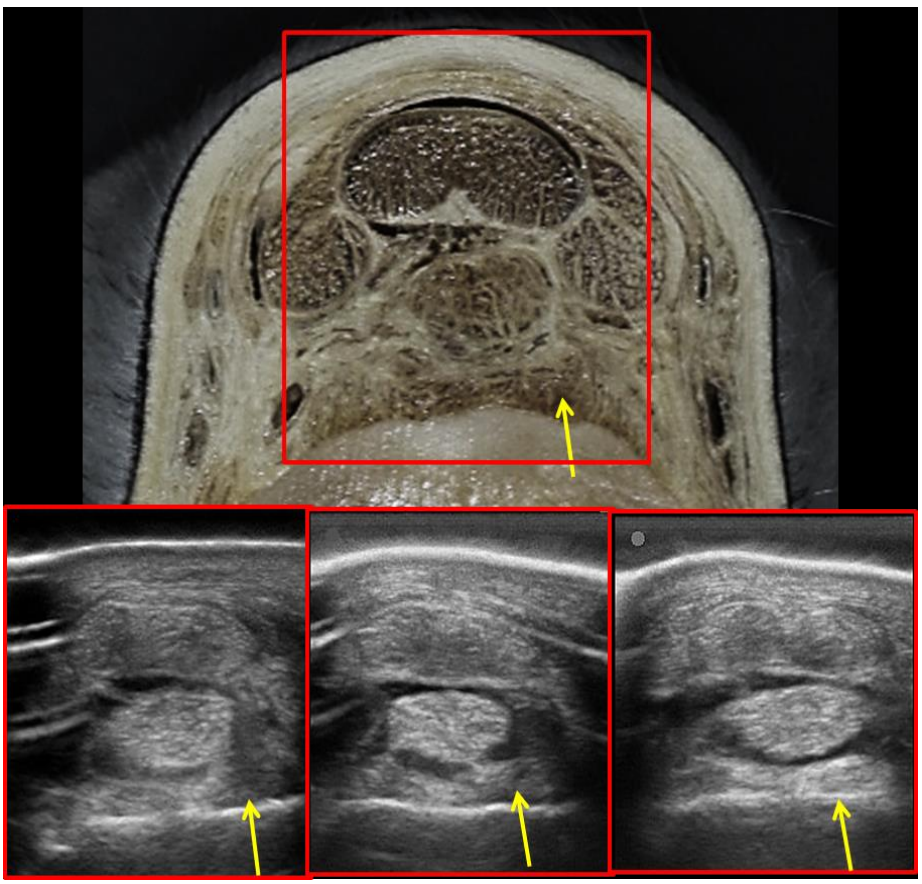


Figure 38. Example of echogenic changes associated with desmitis of lateral oblique sesmoidian ligament. Transverse section of mid P1 pastern. Yellow arrows correlate lateral oblique sesmoidian ligament just proximal to insertion on P1. Left sonogram shows skin deformation, enlargement of structure, reduced delineation of border margin and diffuse hypoechogenicity. Center image shows same horse and limb after twelve weeks of rehabilitation; a focal hypoechoic lesion is still evident but the border margins are much more evident and the ligament has increased echogenicity. This is most consistent with good progressive but incomplete healing. Right sonogram is the contralateral limb. Anatomic specimen provided above.

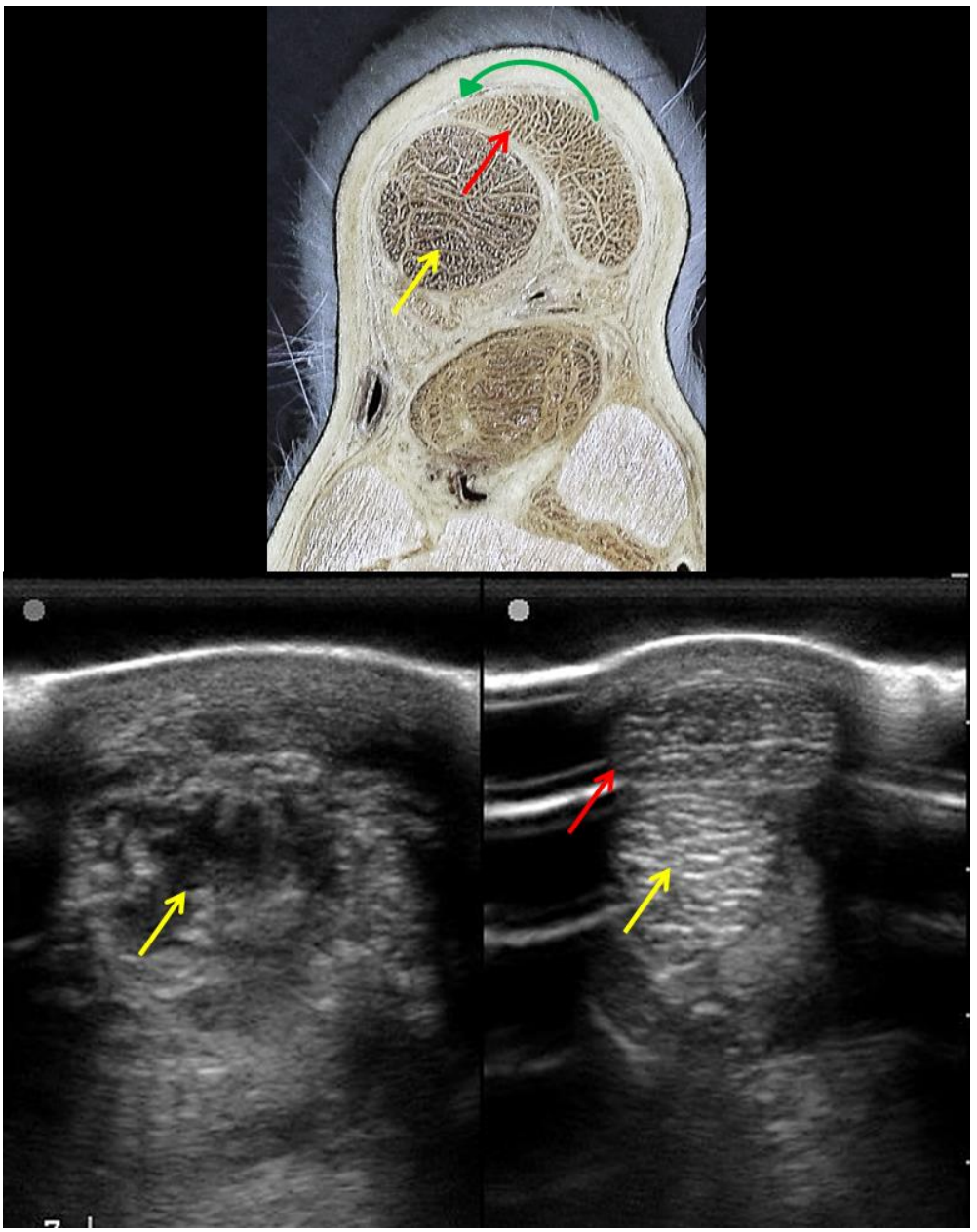


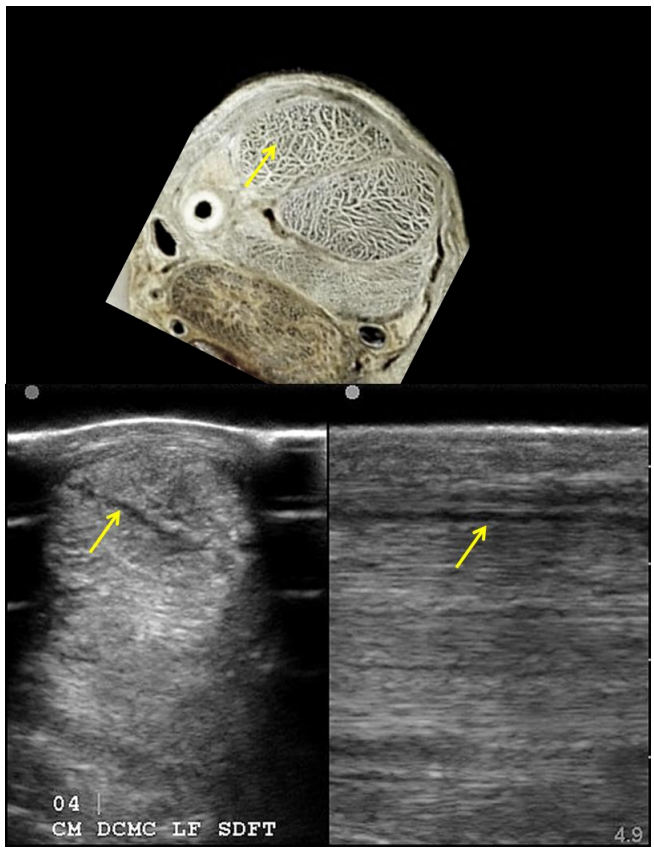
Figure 39. Example of echogenicity associated with severe tendinosis of hind DDFT. Transverse section of DDFT at 7 cm distal to tarsal metatarsal joint. Yellow arrow correlates position of DDFT. Left hind (left sonogram) demonstrates large hypoechoic foci with remarkable remodeling of remainder of DDFT. This is associated with long standing tendinosis with an acute rupture. Right hind sonogram on right. Note truncation of medial aspect of SDFT caused by reduced acoustic contact by probe (red arrow). Anatomic specimen provided on top as reference. [specimen deformation indicated by green arrow].

Assuming all of factors are accounted for and optimized, echogenicity is an important consideration to be used during ultrasonographic evaluation. On the most simplistic of terms, hypoechogenicity is related to a decrease of tissue density most often associated with

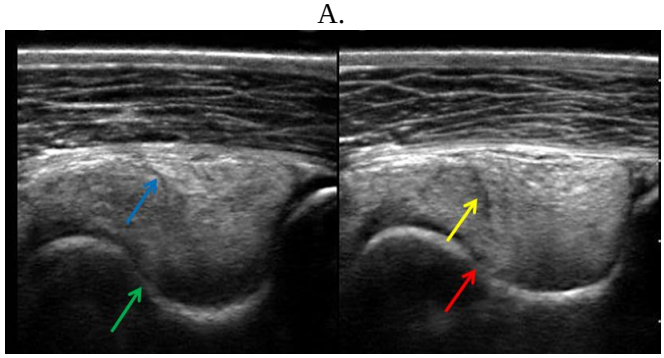
increased fluid content. As discussed above with regard to tissue anisotropy, hypoechogenicity should not always be associated with pathology but hypoechogenicity is often a sign to warrant further investigation. Below are several sonograms that illustrate the variation of echogenicity and its influence on interpretation. See Figures 34-40.

(4) *Fiber pattern*: Because echogenicity is affected by a variety of processes, it is important to add additional information regarding tendon architecture to validate suspected pathology. [13] Most frequently, the fiber structure can be used as somewhat subjective criteria to aid in tendon evaluation. The fiber information should be used as part of the global evaluation of the tendon not as a single criteria for the presence of pathology. Ultrasonographic fiber evaluation can add critical information in the following four situations.
   a. Lesion verification: As with radiology, all suspected lesions should be imaged in two planes to verify authenticity and extent of injury. Longitudinal sections with attention paid to fiber pattern gives very specific information that is not as prone to oblique incidence artifacts. Because many lesions are not centrally located in the structure, it is very important that the imaged section be acquired through the lesion which may necessitate a parasagittal image.
   b. Tissue edema: Evaluation of longitudinal fibers helps determine cause of hypoechogenicity of transverse images. Transverse imaging of structures with acute edema will show wider, more prominent linear fiber pattern. Alternatively, longitudinal sections will show smaller, less distinct fiber pattern that may be associated with tissue remodeling and deterioration.
   c. Strength: Typically, as healing progresses, injured tissue demonstrates more organized linear structure. As a pragmatic approach, workloads are kept to a minimum until the areas of repair start to develop linearization of fibers. Unfortunately, much of the repair process is beyond the resolution capabilities of current ultrasound technique/technology although newer technologies like computerized ultrasonographic tissue characterization (UTC) have demonstrated significant improvement in the evaluation of the healing process. [14] UTC uses comparisons between contiguous transverse images to identify structural vs non-structural soft tissue within the injured area. Until this technology develops further, we are still relegated to the subjective evaluation of fiber linearity.
   d. Prognosis: Fiber linearity may also aid in the formulation of a more accurate prognosis. In the presence of injury, more linearity is consistent with a less severe injury. Anecdotally, it seems that horses with soft tissue injuries that retain linearity of fiber pattern usually continue to perform longer at a higher level than horses with linear fiber ablation/remodeling.

In the figures below are several example with interpretation regarding linearity of fiber pattern. See Figures 41-4.



A.



B.

Figure 40. A, B Examples of linear hypoechogenicity. A) Transverse (left sonogram) and longitudinal (right sonogram) sections of SDFT at 4 cm distal to carpal metacarpal joint. Yellow arrows correlate fissuration of tendon. Anatomic specimen provided on top row. B) Transverse section of bicipital biceps tendon at proximal humerus. Pony presented with complaint of non-resolving gait asymmetry of right front. Asymmetry resolved with infiltration of bicipital bursa with anesthetic (ultrasound guided procedure). Ultrasound examination demonstrated fissuration of biceps tendon of RF (right sonogram). In addition, the fissuration was observed to be full thickness (yellow arrow) with a loss of hypoechoic margin adjacent to the bone (red arrow). The contralateral limb (left sonogram) demonstrated similar fissuration (blue arrow) but could not be observed to be full thickness and the hypoechoic margin (green arrow) between the tendon and bone remained intact. The changes in the RF tendon are most consistent with a degenerative tendinosis with fissuration in addition to a loss of fibrocartilage over the lateral proximal dorsal humerus.

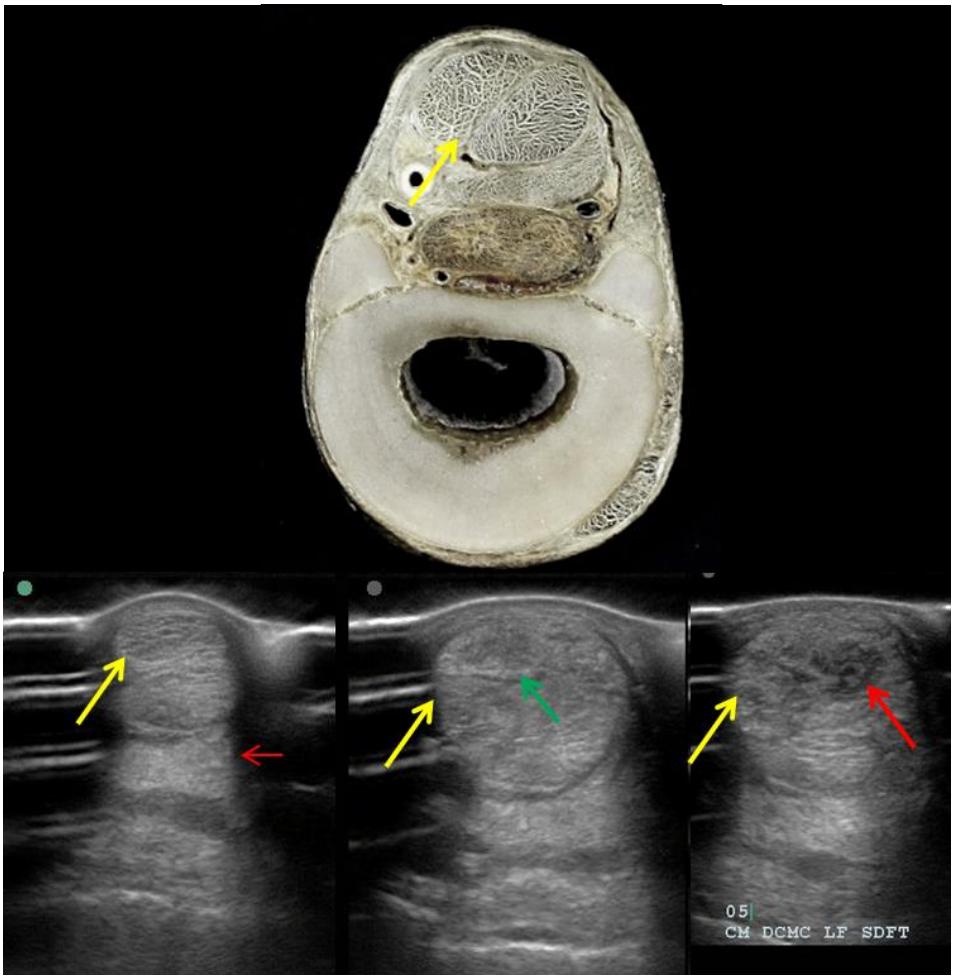


Figure 41. Example of complex echogenicities. Transverse images of SDFT 5 cm distal to carpal metacarpal joint. Yellow arrow correlates medial margin of SDFT. Center sonogram demonstrates skin deformation and SDFT enlargement with a linear hypoechoic lesion (green arrow). Right sonogram demonstrates healing after twelve weeks. Fissuration is still evident with development of a focal hypoechoic lesion surround by a zone of mild hypoechogenic tissue (long red arrow). Left sonogram of contralateral limb. Note attenuation of lateral margin of accessory ligament of DDFT (short red arrow) caused by reduced acoustic contact with the linear probe. This case demonstrates the benefit of sequential imaging required for optimal case management. It is not uncommon for lesions to become more focal in appearance and usually indicate the need for additional therapy and time for adequate healing.

Establishing Significance of Ultrasonographic Findings: The "Duck Test"

While ultrasound is widely used and relied upon by veterinarians, the interpretation of findings still varies significantly between practitioners. In addition, comparison with MRI and histology findings has shown a lack of sensitivity and specificity of ultrasound to pathology. Because of the inherent subjectivity of ultrasound interpetation, it is incumbent that we as veterinarians validate our findings. The adage: "if it walks, talks and looks like a duck then

let's do a duck test and if that's positive…then it might be a duck" could be applied. From a probability standpoint, the more characteristics a structure has associated with pathology, the more likely that the perceived pathology is real. From a pragmatic point, if a structure has enlargement, sensitivity, ultrasonographic anomalies and the area is validated by diagnostic anesthesia, then we can be relatively assured that the pathology is real. The lack of any of these conditions dramatically reduces the specificity of the ultrasonographic findings. See Figure 49.

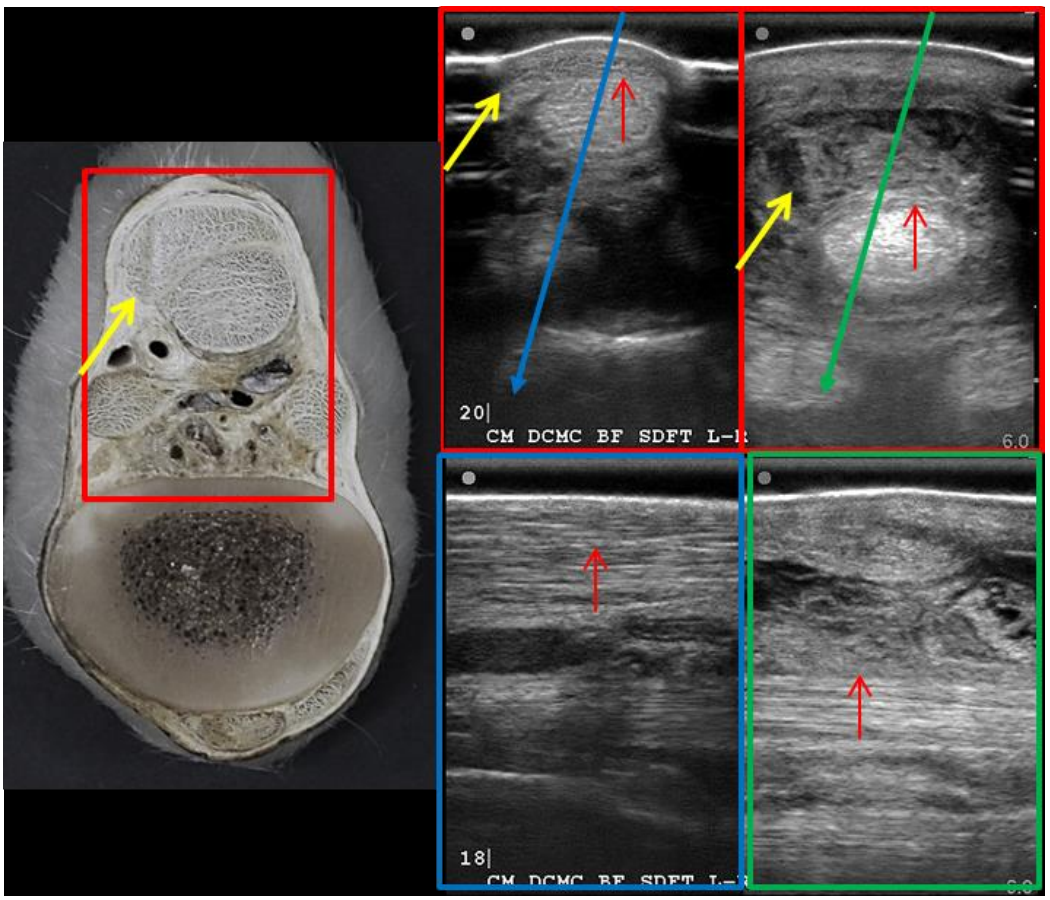


Figure 42. Example of complex echogenicities. Transverse and longitudinal images of SDFT 18-20 cm distal to carpal metacarpal joint. Yellow arrow correlates medial margin of SDFT. Red arrows correlate the palmar border of DDFT and dorsal SDFT. Top row of sonograms are transverse section of affected RF (right image) and of normal LF (left image). The right sonogram demonstrates skin deformation, adjacent connective tissue fibrosis, SDFT enlargement, non-discernible borders and heteroechogenic SDFT tissue. Longitudinal sections are shown below each transverse section. The approximate section path is indicated by a blue line on the left limb and a green line on the right limb. Note the mixed nature of echogenicity of the RF transverse section associated with a complete loss of linear fiber pattern on the longitudinal (green box) section. This presentation is most commonly associated with a chronic tendinosis associated with an acute exacerbation usually a tendon rupture. This syndrome is most commonly associated with aged geldings.

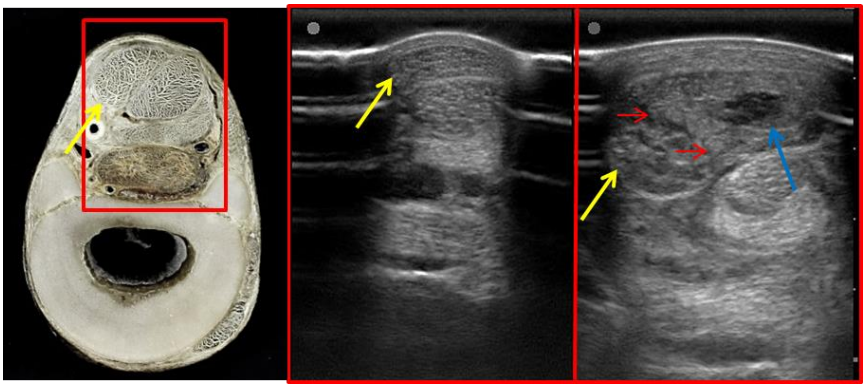


Figure 43. Example of complex echogenicities. Transverse images of SDFT 8 cm distal to carpal metacarpal joint. Yellow arrow correlates medial margin of SDFT. Affected RF limb (right sonogram) demonstrates skin deformation, adjacent fibrosis, structure enlargement and heteroechogenic tissue density. Red arrows highlight fissuration of medial portion of SDFT. Blue arrow highlights focal hypoechoic lesion. Unaffected LF limb sonogram on left. Anatomic specimen provided. Mixed echogenicity lesions such as this example are almost always associated with a long standing tendinosis associated with a perturbation leading to clinical presentation. It is important that the owners be advised of the almost certainty of recrudescence of additional tendinopathy.

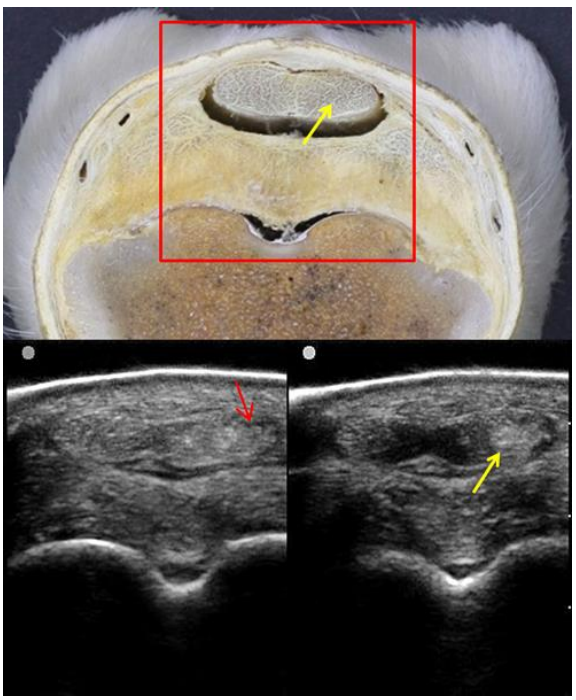


Figure 44. Example of changes of echogenicity associated with healing. Transverse sections of pastern at distal P1. Yellow arrows designate lateral lobe of DDFT. This horse presented with sudden onset gait asymmetry which improved with diagnostic anesthesia of the proximal digit. These images were acquired twelve weeks after initial imaging and diagnosis. The traditional sonogram on the left demonstrates an irregular palmar border (red arrow) with enlargement of the lateral DDFT lobe. Tissue disruption is still evident. A CET view is presented on the right. The oblique incidence demonstrates the non-linearity of the fibrous repair. This sonogram presentation is indicative of a fair but certainly incomplete repair process.

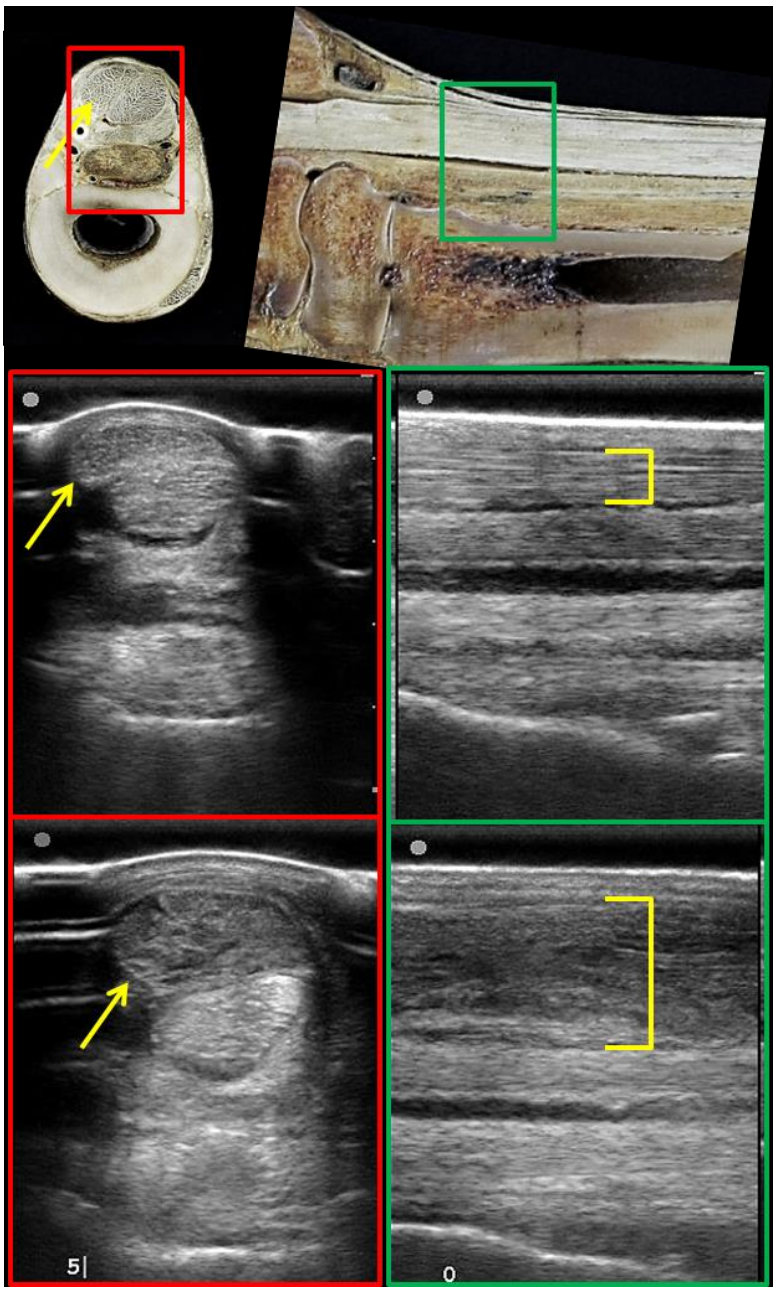


Figure 45. Example of sever fiber disruption. Transverse and longitudinal images of SDFT at 5 cm distal to carpal metacarpal joint. Yellow arrows correlate medial margin of SDFT. Affected limb (bottom row) sonograms demonstrate skin deformation, structure enlargement, hypoechogenicity and absence of linear fiber structure. Normal limb (middle row) provides a comparison of normal appearance. Note amount of linear fiber structure within normal tissue and absence in affected tissue as highlighted by the yellow brackets. Anatomic specimens provided above with red box indicating area of transverse sonogram and green box indicating area of longitudinal sonogram. This sonogram was taken 12 weeks after injury. While transverse section indicates significant echogenicity, the longitudinal sonogram demonstrates the incompleteness of healing. This horse will require significantly more time to provide a repair that will withstand return to work.

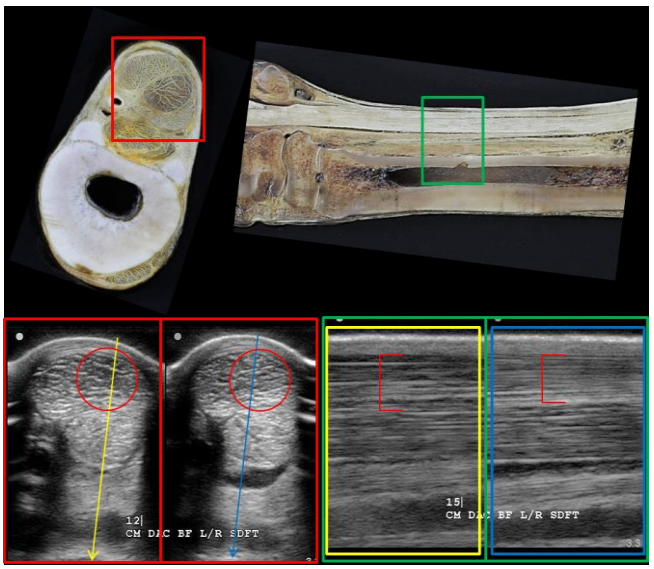


Figure 46. Example of mild fiber edema. Transverse and longitudinal images of SDFT at 12-15 cm distal to carpal metacarpal joint. Sonograms surrounded by red box are transverse images of SDFT with the LF (affected) on left and the RF (normal) on right. Red circles on left transverse image highlights hypoechogenicity as evidenced with comparison to tissue highlighted in right image. Longitudinal sections taken of each limb are to the right. The yellow box longitudinal image is of LF limb taken as approximated by the yellow vector indicated on the transverse (most left sonogram). The blue boxed sonogram is taken as approximated by the blue vector on the transverse image of the RF sonogram. Note the linearity as highlighted by the red brackets. The LF sonogram has a more distinct coarse linearity whereas the RF sonogram has a finer linearity with a more echoic appearance. This sonogram was taken of a 5 year old Warm blood gelding after trainer noted resistance to jumping. His jumping level had been recently raised to a higher level while simultaneously increasing the frequency of work. This sonogram pattern is most often associated with a low grade tendonitis that is usually transient in nature. The prognosis for continued work after resolution is usually quite good but suggests a need for more vigilant monitoring.

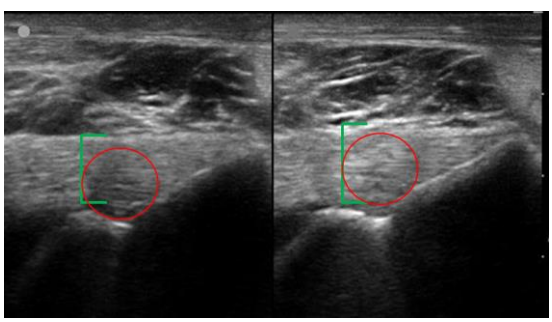


Figure 47. Example of subtle fiber pattern changes. Longitudinal images of insertion of medial cranial meniscal tibial ligament. Affected limb on right shows somewhat hyperechoic fiber pattern associated with coarseness as highlighted by the red circle. This is more evident when compared to the unaffected LH (left sonogram). Green brackets highlight increased thickness of RH ligament. This mare presented with chronic low grade RH asymmetry that definitively improved with intra articular anesthesia of the medial femoral tibial joint. The interpretation was low grade chronic active desmitis of the medial cranial meniscal tibial ligament. Because this fiber pattern suggests chronicity (hyperechoic tissue) and the gait asymmetry was long standing, rest was not included as part of management. This mare periodically imaged and has continued to perform at her desired level for five years with this injury.

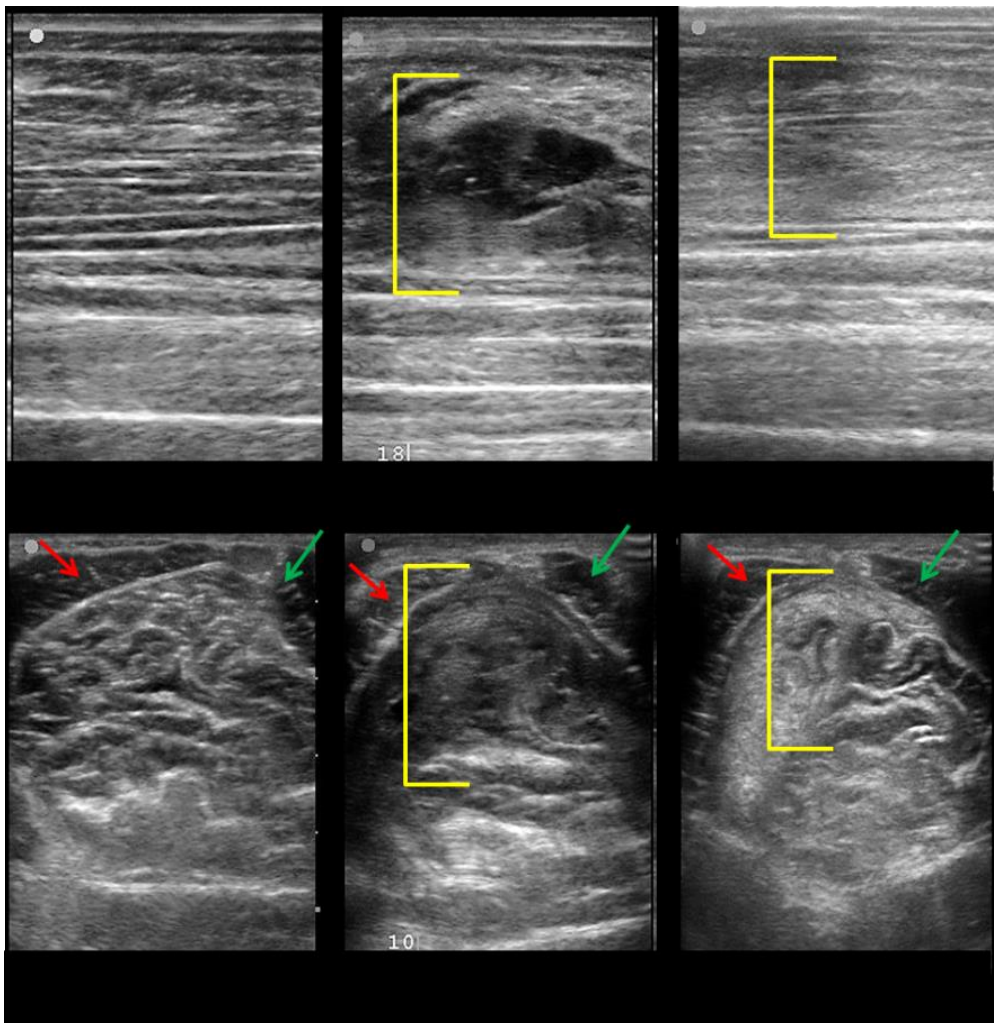


Figure 48. Example of monitoring healing with fiber pattern changes. Transverse and longitudinal sections of palmar antebrachium at 10 cm proximal to antebrachial carpal joint. Red arrow designates ulnaris lateralis while green arrow designates flexor carpi ulnaris muscles. The left sonograms are of the normal limb. Note the normal variegated architecture of the musculotendinous junction of the superficial flexor muscles and tendon. The longitudinal image above shows the multipennate arrangement of the musculotendinous unit. The central images show the injured limb at the time of presentation. Note the lack of organized architecture within the yellow bracket. The right sonograms show the same limb twenty weeks after injury. Not the diffuse echogenicity on the medial side of the muscle (left of bracket on bottom transverse image, within bracket on top image). The longitudinal image shows a reduction of size (compared to initial images), increased echogenicity and the beginning of linear fiber formation. This horse demonstrates adequate healing to start a slow progressive return to work program with the use of functional electrical stimulation.

If all the criteria are present then the probability of a "real" finding is greatly improved. However, it is important that these findings are supported by validation of localization. If the onset of gait asymmetry is not tightly associated with *obvious* heat pain and swelling, then it is imperative that diagnostic anesthesia be used to validate the location of injury. Only then can most assured that the ultrasonographic findings are valid.

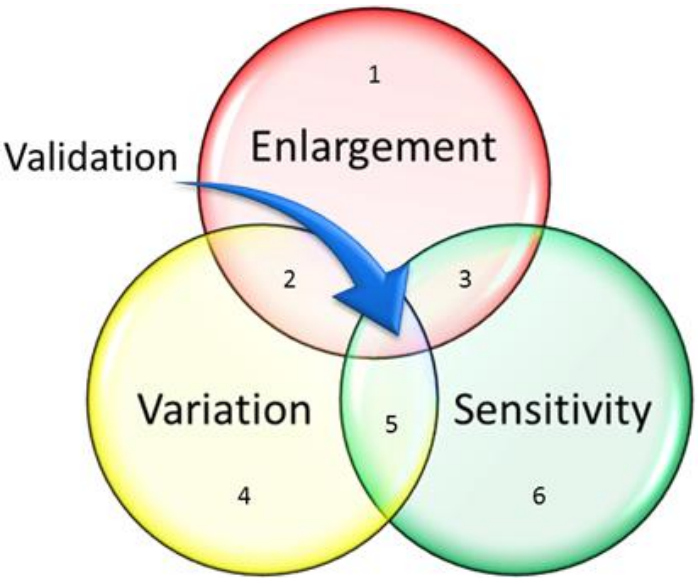


Figure 49. Venn diagram of "Duck Test". Each colored circle represents a major criteria group regarding the assessment of soft tissue injury as described in previous text. Numbered areas are described below:

1) Enlargement only: may be simply dependent swelling
2) Enlargement with ultrasonographic anomaly: may be old healed injury
3) Enlargement with sensitivity: may be cellulitis
4) Variation (ultrasonographic anomaly): non-clinical anatomic variation
5) Variation with sensitivity: sub clinical problem i.e. low grade suspensory branch desmitis but horse has fractured third trochanter of femur.
6) Sensitivity only: traumatic bruising.

MRI

Although MRI has been used as the gold standard for musculoskeletal (and neurological) imaging since the early 1980's in humans, the first live horse scan is reported to be in 1997 at Washington State University. Magnetic resonance imaging is not as widely available as ultrasound and is a much more expensive technology, but even so, it is increasingly popular for equine musculoskeletal evaluation. Best known for superior soft tissue contrast, MRI is also excellent for bone imaging. The use of MRI in horses has grown significantly in the past fifteen years, but compared to other imaging modalities, many equine practitioners consider MRI to be a relatively new imaging modality.

Although magnetic resonance imaging is a particularly expensive diagnostic modality, which is far less available than ultrasound, and, additionally, in numerous cases, its application requires general anesthesia, MRI offers significant clinical advantages. It provides superior contrast for soft tissue assessment as well as bony pathologies. Soft tissues that might be completely inaccessible to ultrasound are readily imaged with MRI. Tissues that are difficult or impossible to evaluate via ultrasound for the average practitioner can usually be well evaluated with MRI.

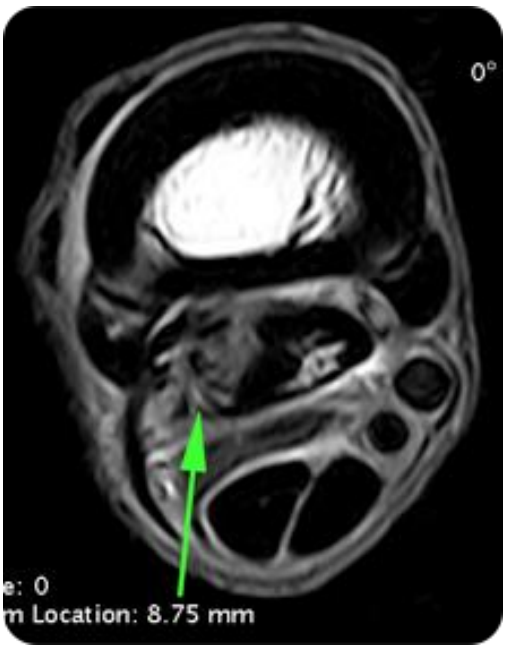


Figure 50a. Moderate to marked generalized PD signal increase in the lateral half of the proximal suspensory ligament body.

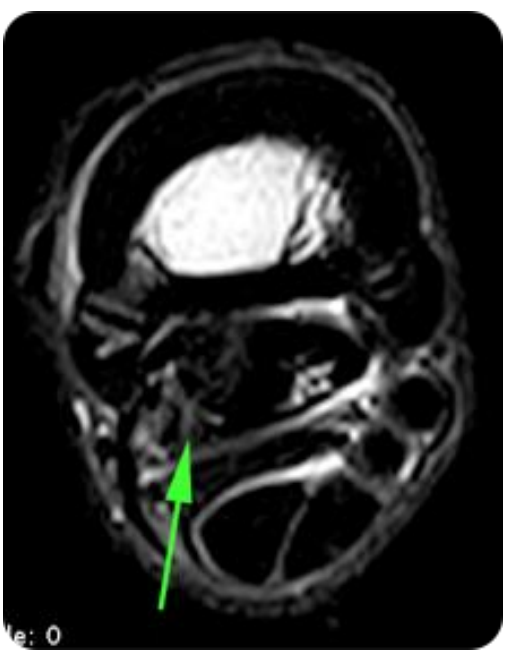


Figure 50b. Persistent T2 signal heterogeneity of the lateral aspect of the suspensory body, that is less pronounced than the PD signal increase, suggesting a chronic to chronic-active desmopathy, part of which has healed with some immature collagen, and other areas have not yet healed (namely the lateral corner).

Figure 50. (Continued).

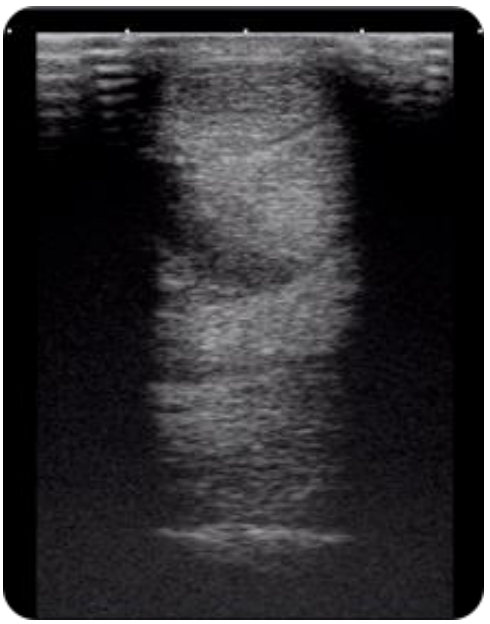


Image courtesy Dr Haynes Stevens.

Figure 50c. Ultrasound representative short axis ultrasound image of the proximal suspensory ligament, performed within one month of the MRI examination. The fiber architecture suggested somewhat underwhelming chronic changes that were not convincingly significant. Two separate and independent clinicians, both very experienced in equine ultrasound believed the appearance of the suspensory ligament on ultrasound was so underwhelming that they were considering the lameness was likely originating from the neck or shoulder. The horse continued to block however, to the region of the proximal suspensory ligament.

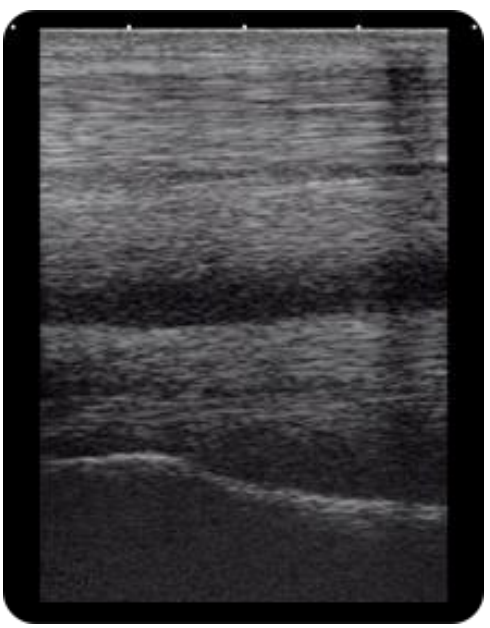


Image courtesy Dr. Haynes Stevens.

Figure 50d. Long axis view.

Technology

Magnetic resonance scanners are often categorized as "high field" or "low field" systems. Those scanning horses at high field are typically using systems with field strengths 1.5T or 1.0T. Low field systems are more typically in the 0.18 to 0.3T range. Higher signal to noise ratios can be obtained as field strength increases; higher signals are often used to generate images with improved spatial resolution and/or faster scan times--both are very beneficial advantages. Unfortunately, for many equine practices, high field MRI systems are usually cost-prohibitive to purchase and maintain. Alternatively, low field MRI systems offer the benefit of being more affordable with significantly lower maintenance costs as well as improved access, allowing much easier positioning of the horse into the scanner.

Both systems have been shown to provide extremely useful diagnostic information. Within the low-field equine market, there are a few different systems used that require general anesthesia and one existing system that can perform exams standing under heavy sedation (ref Hallmarq, Esaote, others).

Basic knowledge of MRI physics, technology, and artifacts is outside the scope of this chapter, but a working knowledge of the above is highly recommended to the potential user. Incomplete knowledge of these areas may prevent optimal use of any MRI system, no matter how sophisticated or basic the system is. In addition to the magnetic field strength of the system itself, settings and nuances in numerous other hardware, software, and imaging parameters have a significant effect on image quality. For example, surface coils, magnet homogeneity, gradient amplitude and strength, pulse sequences, imaging field of view, acquisition matrix, the number of acquisitions and slice thicknesses are some important examples of hardware and software parameters that a user needs to understand and master to optimize use of his/her MRI system.

A variety of pulse sequences are used during all MRI exams to create different tissue contrast in order to better characterize pathology. Although hundreds- if not thousands- of different pulse sequences exist, and are used for specific applications, some of the more basic and most commonly used sequences for musculoskeletal applications include T1, GE T1, T2, T2*, PD, and fat suppressed sequences, such as a STIR sequence. As an oversimplification, T1 and PD sequences are more frequently used for anatomical detail, and T2, T2*, and STIR sequences are helpful for determining presence of pathology. STIR or other fat suppressed sequences are critical for assessing bony pathologies where intramedullary signal from fat is suppressed, so that fluid containing pathologies can be visualized by contrasting pathology against a dark background. For example, pure water is darker (hypointense) on T1, and brighter (hyperintense) on T2 and STIR sequences, so fluid containing pathologies such as edema/inflammation, abscesses, infections, and neoplasias can be well seen compared to surrounding normal tissues. With a suspected osteomyelitis, intramedullary and surrounding soft tissue inflammation can be seen on MRI well before radiographic bony lysis occurs.

The combination of sequences also allow for tissue characterization -- for example, if the intramedullary cavity of the navicular bone lost all normal T1 signal and there was no corresponding abnormal STIR signal, then this is consistent with sclerosis. However, if the loss of T1 intramedullary signal corresponded with marked STIR signal, this would be more consistent with intramedullary edema/inflammation and osteonecrosis. Or if an old tendon core lesion was filled with tissue that was completely hypointense on T1, T2, PD, and STIR, that would indicate complete healing with excellent quality tissue, comparable to that of

normal tendon tissues. (It is very difficult to achieve this type of superior quality matrix following an injury.) If the old core contents were predominantly T2 hypointense, but corresponding T1, PD and STIR showed hyperintensity, then this would also indicate an inactive lesion, but with more typical scar tissue containing immature collagen.

In part, it is this tissue contrast obtained by using different pulse sequences that gives MRI such a diagnostic advantage over other imaging modalities. The other advantages are a result of combining this tissue contrast with very good spatial resolution and the multi-planar, multi-slice capability of MRI. Where signal to noise ratios are lower, as in most low field imaging, scan time is usually longer to retain clinically acceptable resolution. The benefit of avoiding general anesthesia is obvious with low field standing systems, though the scans must be performed quickly to minimize motion artifacts. In any system, compromised image quality results from either motion-corrupted images or pulse sequences that have an imbalanced exchange of speed for spatial resolution.

The cost-effectiveness of an MRI system depends strongly on which MRI system a practice decides to purchase or lease. The initial and ongoing maintenance costs of a high-field MRI system are often considered prohibitive for a private equine practice. Most practices deem these systems as an economic loss, even with extremely high equine caseloads. To date, the available MRI system used for low field standing equine MRI is typically on a lease contract-only basis, where the institution agrees to pay a monthly fee and a fixed amount for every horse scanned. Additional low field systems allow for purchase, and a typical break-even point (over five years) to purchase a dedicated, equine low-field MRI system which requires the subject to be examined under general anesthesia can range from as few as 2 or 3 horses a month, to 6 or 7 horses a month with a more expensive low-field MRI system. If a practice opts for a low-field, dedicated equine system and the practice is capable of treating a larger caseload than the above, the practice can potentially profit. Some equine practices have personnel and clientele to support a small-animal imaging service as well, and can more readily profit from their investment in MRI equipment.

Clinical Considerations

There are numerous reasons why an MRI exam is requested by a practitioner. The most common example is a lameness that is not responding to conventional therapy, such as intra-articular injections and/or rest. Another common scenario is a lameness that can be localized to a region, but without an identifiable cause -- usually using radiographs and ultrasound, and/or sometimes nuclear scintigraphy in addition to a good lameness workup. Often changes (or equivocal findings) may even be seen on these other imaging modalities, but are not consistent with the degree of lameness.

Interestingly, some practices that own MRI systems are developing distrust of their ultrasound findings following MRI examinations, although a better understanding of the pathology can ultimately improve ultrasound skills of those practitioners able to benefit from knowledge gained by MRI. These practitioners sometimes opt to skip ultrasound exams and request MRI instead given their prior experience. This is especially true where the pathology is likely in a location that is difficult (or impossible) to access with ultrasound. The proximal suspensory ligament region is a typical example of this. For instance, it is not uncommon for a presenting history to include a chronic proximal suspensory desmopathy (usually based on

ultrasonic assessment) where none is present on MRI, but rather MRI reveals other lesions instead. These other lesions typically include various abnormalities of the inferior check ligament, long plantar ligament, palmar carpal ligament, and bone injuries of the McIII/MtIII and carpal/tarsal joints. Conversely, where the proximal suspensory ligament is not believed to be an active pathology (usually based on ultrasonographic assessment), it often can be found to be quite abnormal on MRI. (Figure 50 a, b, c, d)

Another reason a practitioner may request an MRI exam is after a known lesion or injury is identified. Those having experience with equine MRI cases are fully aware that multiple lesions are frequently found (Figure 51a-f), thus the request is to more thoroughly examine additional injuries for a more detailed diagnosis prior to expensive therapy and a potentially long rehabilitation period. This includes non-invasive joint evaluations (assuming special pulse sequences are used to appropriately evaluate the articular structures) as well as improved bony and supporting soft tissue evaluations from the MRI study. Joint pathologies may significantly alter prognosis and/or treatment of other known supporting soft tissue lesions. Many of these lesions are completely missed or grossly underappreciated on ultrasound and radiographic exams. (One notable exception seems to be the mid metacarpus/tarsus, where ultrasound examination alone is considered excellent and MRI exam is much less frequently requested, though the quality of collagen matrix, including degenerative lesions is arguably better assessed on MRI).

In an otherwise obscure or perplexing lameness, MRI is often helpful, and is another common reason for MRI referral. However, it is very important to localize the lameness as well as reasonably possible, so the MR exam can be focused. The more body parts that are scanned, the longer the scan time required. Recognizing that perineural anesthesia is not a precise science, some practitioners are more comfortable with their diagnosis, by scanning both the foot and the pastern/fetlock that improved following a palmar digital nerve block, especially if the horse did not go lame in the opposite foot, as is often seen with navicular syndrome.

A follow-up examination to evaluate healing and/or to determine when it is safe to return to training is also a reason that MRI examinations are requested. The extent and quality of healing is usually well assessed. For example, upon MR exam, superior healing within the lesion intrasubstance may be identical to that of normal ligamentous and tendon tissue,.i.e., typically hypointense on all pulse sequences, with minimal to no surrounding scar tissue and adhesions to adjacent soft tissues, but this result is difficult to achieve with extensive injuries, without extended time in rehabilitation. Not uncommonly, MRI examination reveals a portion of the lesion, such as a DDFT tear, that appears to be moderately well healed, and another portion completely unhealed. (Figure 52a, 52b, 52c). In the author's experience, tendon and ligament tears are less likely to heal well upon rest alone. (Figure 53ab, 54ab, 55ab, 56, 57).

Much more so than ultrasound, it is also common practice to outsource the reading of MRI studies to radiologists or practitioners who are experienced with MRI interpretation. Like ultrasound, proper interpretation of the studies, as well as clinical correlation is paramount to successful treatment using MRI. When performed properly, the studies can be readily sent remotely and interpreted by those veterinarians with extensive MRI experience.

Disadvantages of MRI include the expense of the equipment, and therefore the corresponding expense of each study, and the lack of availability compared to ultrasound. An additional disadvantage is that MRI scans performed under general anesthesia are typically better quality, butome owners, understandably, are not comfortable with anesthesia for

elective purposes. Where standing exams avoid general anesthesia, these exams come with arguably notable limitations. Nonetheless, the clinical benefits of MRI are numerous, and this together with the subsequent focused treatment plan, are becoming the gold standard for analyzing and treating many musculoskeletal injuries.

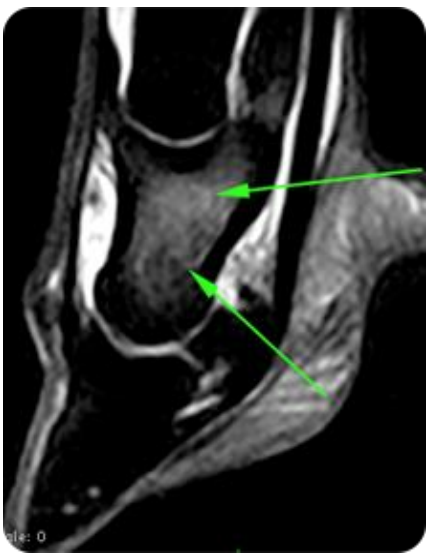


Figure 51a. 7yo American Saddlebred horse with a LF foot lameness that is not completely explained by a recent heel abscess. STIR sagittal foot--marked diffuse increase in signal throughout the intramedullary cavity of P2, corresponding with a network of intermingled linear T1 hypointensities suggesting a marked neovascularization/hyperemic bone response or stress injury.

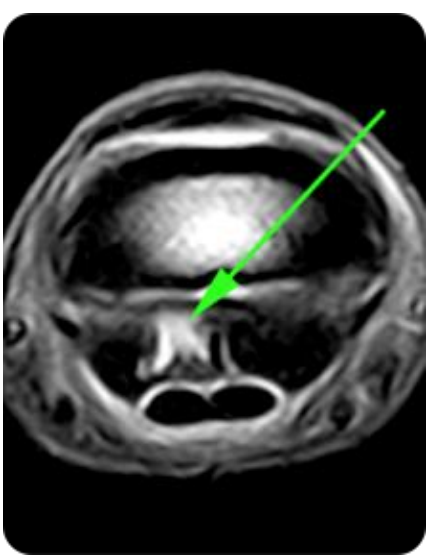


Figure 51b. PD axial, distal pastern--Large core lesion in the distal straight sesamoidean ligament, just proximal to the P2 site of insertion.

Figure 51. (Continued).

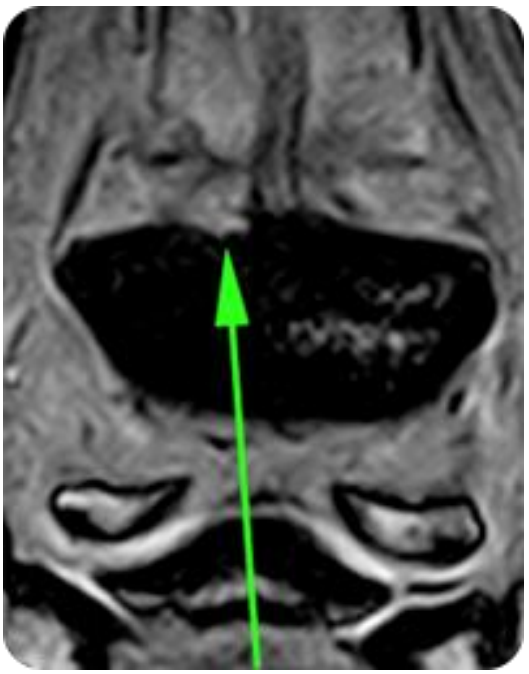


Figure 51c. 3D GE T1 dorsal plane foot--focal osteolysis just medial of midline of proximal P2 at the insertional site of the distal straight sesamoidean ligament.

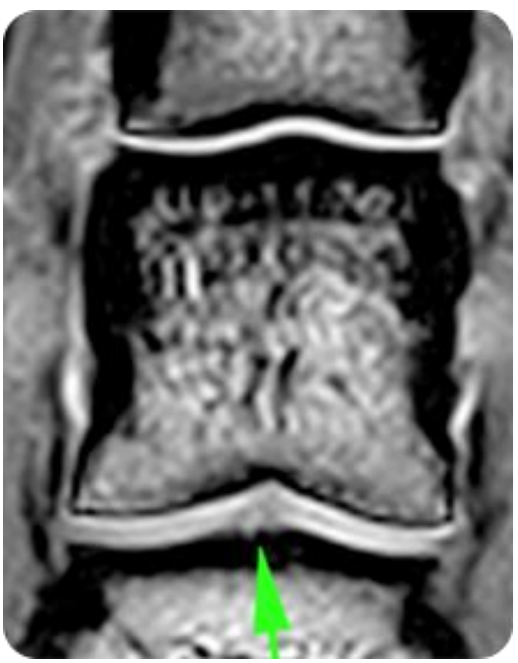


Figure 51d. 3D GE T1 dorsal plane foot--focal articular and subchondral defect in the coffin joint of proximal P3 on midline.

Figure 51. (Continued).

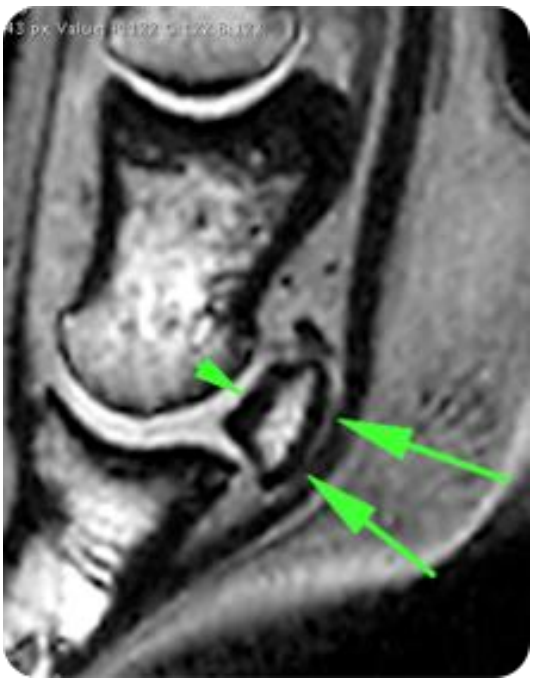


Figure 51e. 3D GE T1 sagittal plane foot--fibrocartilage degeneration along the flexor cortex of the navicular bone and a thickened dorsal cortex of the navicular bone.

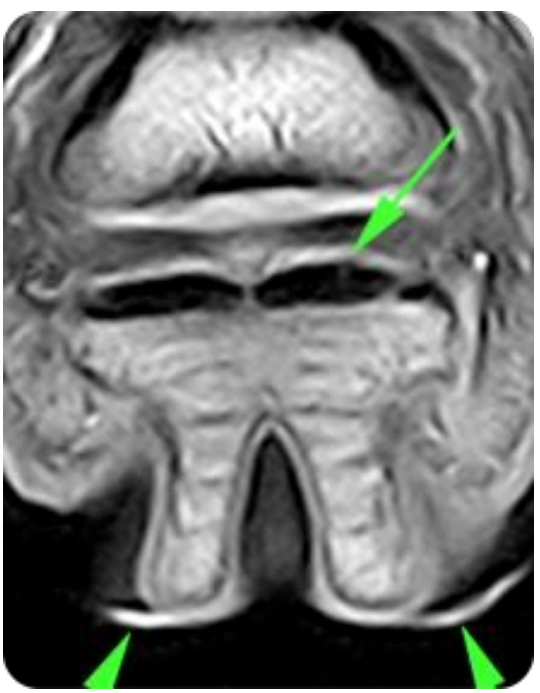


Figure 51f. axial foot, perpendicular to the DDFT-- old thin partial split of the lateral DDFT lobe, level of the proximal navicular bursa (upper arrow). Additional proteinaceous exudate emanating from the caudal heel. This case is an example of multiple significant lesions which is extremely common for the vast majority of MRI examinations.

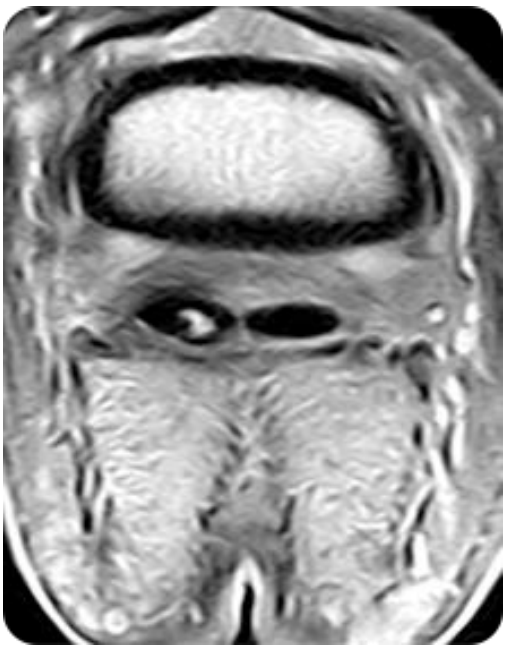


Figure 52a. 12yo WB horse with a LF lameness that blocks to a palmar digital nerve block. PD axial---Large, active core lesion breaking out the palmar aspect of the medial DDFT lobe at the level of the proximal navicular bursal junction with the distal digital sheath.

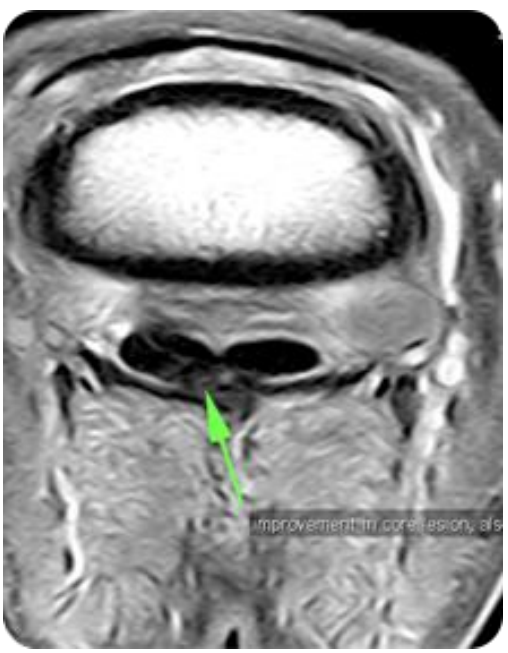


Figure 52b. PD axial--1 year follow-up after stem cell therapy and rest. The core lesion has filled in with scar tissue, and a moderate amount of scar tissue with adhesions between the palmar margin of both lobes and the distal digital annular ligament..

Figure 52. (Continued).

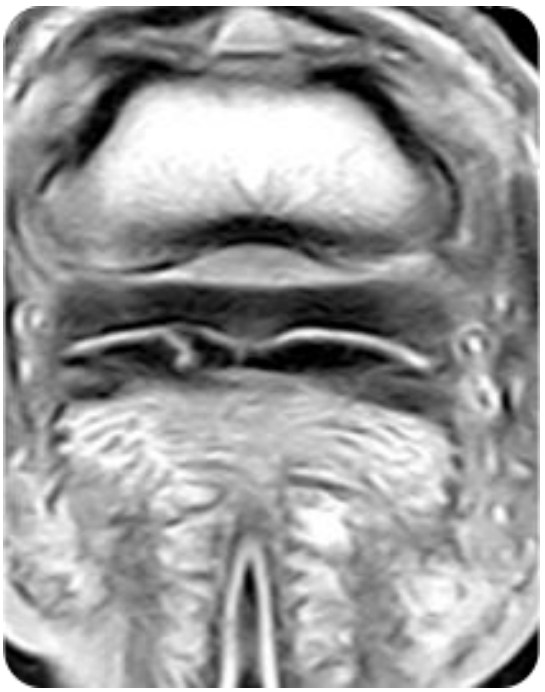


Figure 52c. Same horse as above at initial presentation. This portion of the DDFT tear is only 7mm distal to the core lesion shown above.

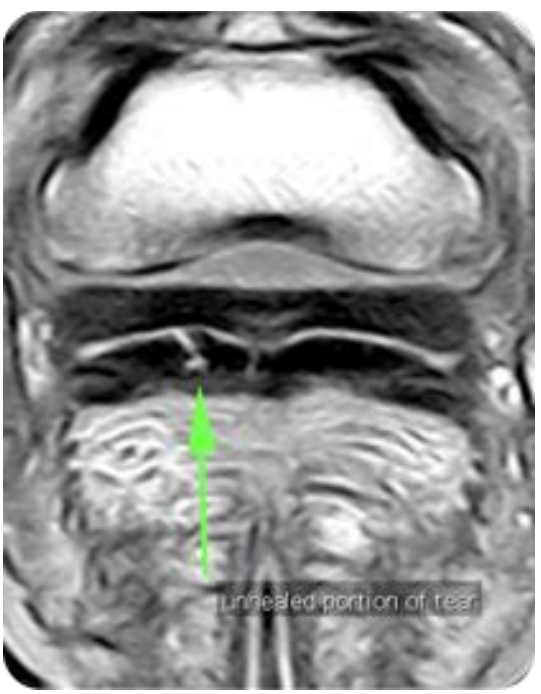


Figure52d. 1 year follow-up. Here, the tear appears to have changed very little (or retorn?)--whereas only mild narrowing of the tear gap is seen relative to the time of presentation.

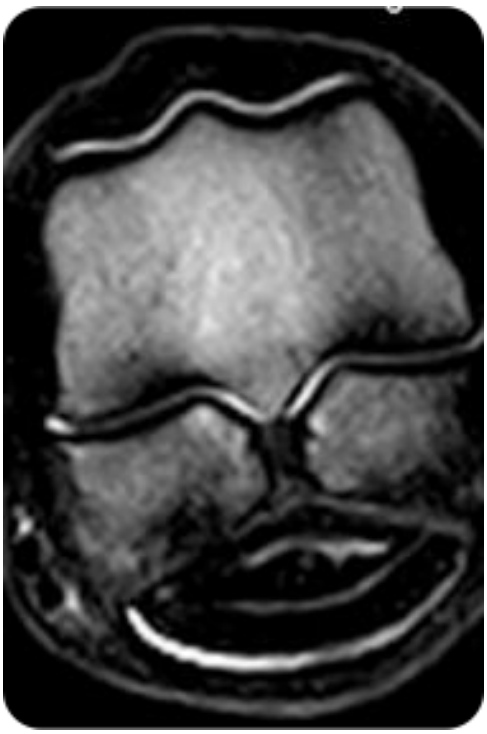


Figure 53a. T2 axial--Original horizontal tear through the DDFT at the level of the proximal sesamoid bones.

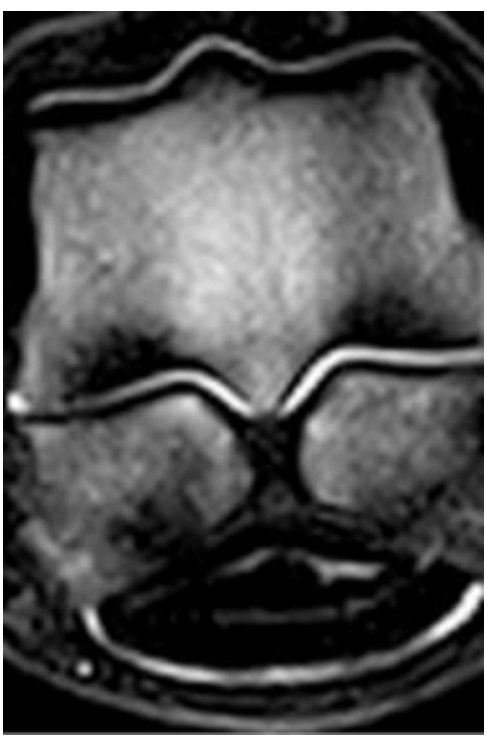


Figure 53b. T2 axial--Routine 1 year follow up. Horizontal DDFT tear essentially unchanged.

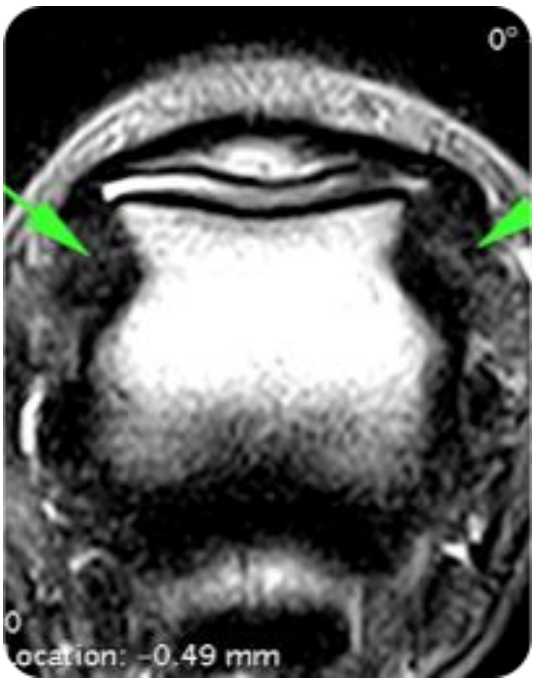


Figure 54a. T2 axial perpendicular to collateral ligaments of the coffin joint--Moderate to marked biaxial desmitis/desmopathy of the medial and lateral collateral ligaments of the coffin joint.

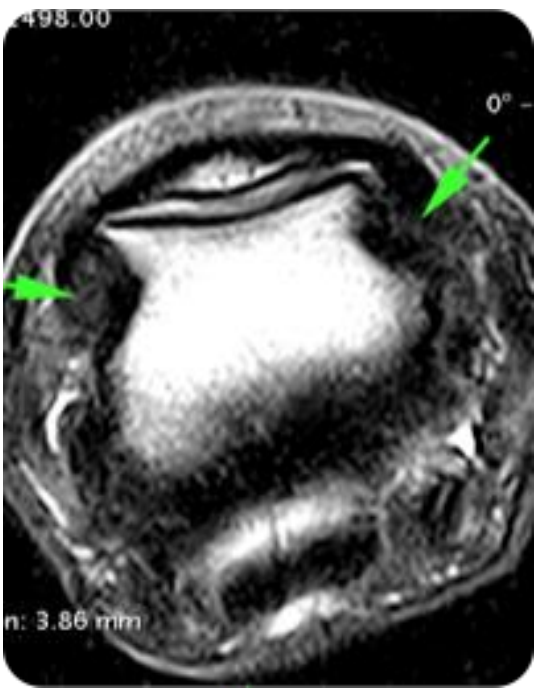


Figure 54b. 5mos. follow-up. No significant improvement. Subjective mild progression of the medial collateral ligament desmopathy (left arrow), and arguable mild improvement of the lateral side (right arrow).

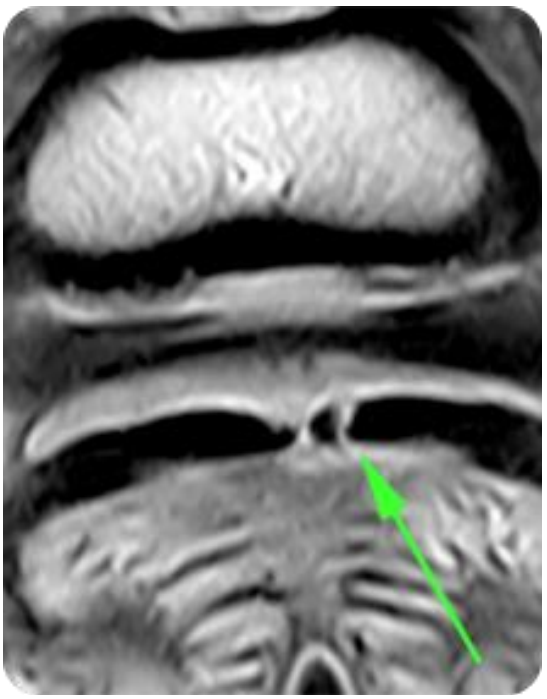


Figure 55a. PD axial--Dorsal to palmar margin DDFT tear upon presentation.

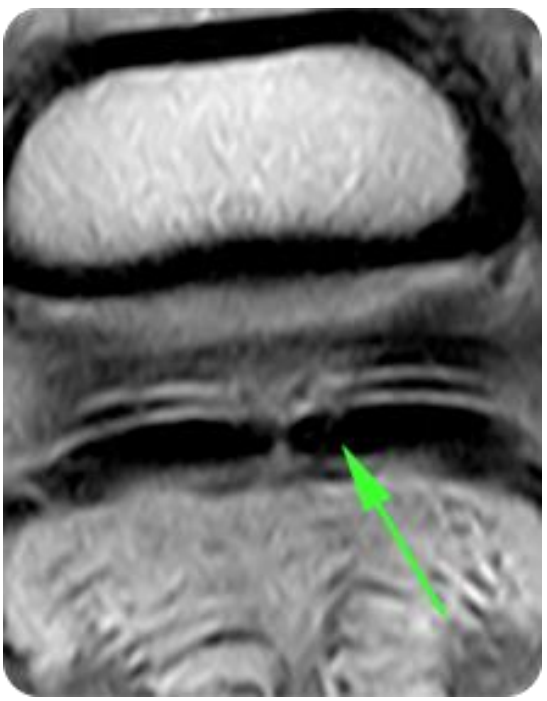


Figure 55b. 6mos follow-up. PD axial-The tear gap has narrowed significantly (but barely still visible) with minimal surrounding scar tissue. This suggests good healing.

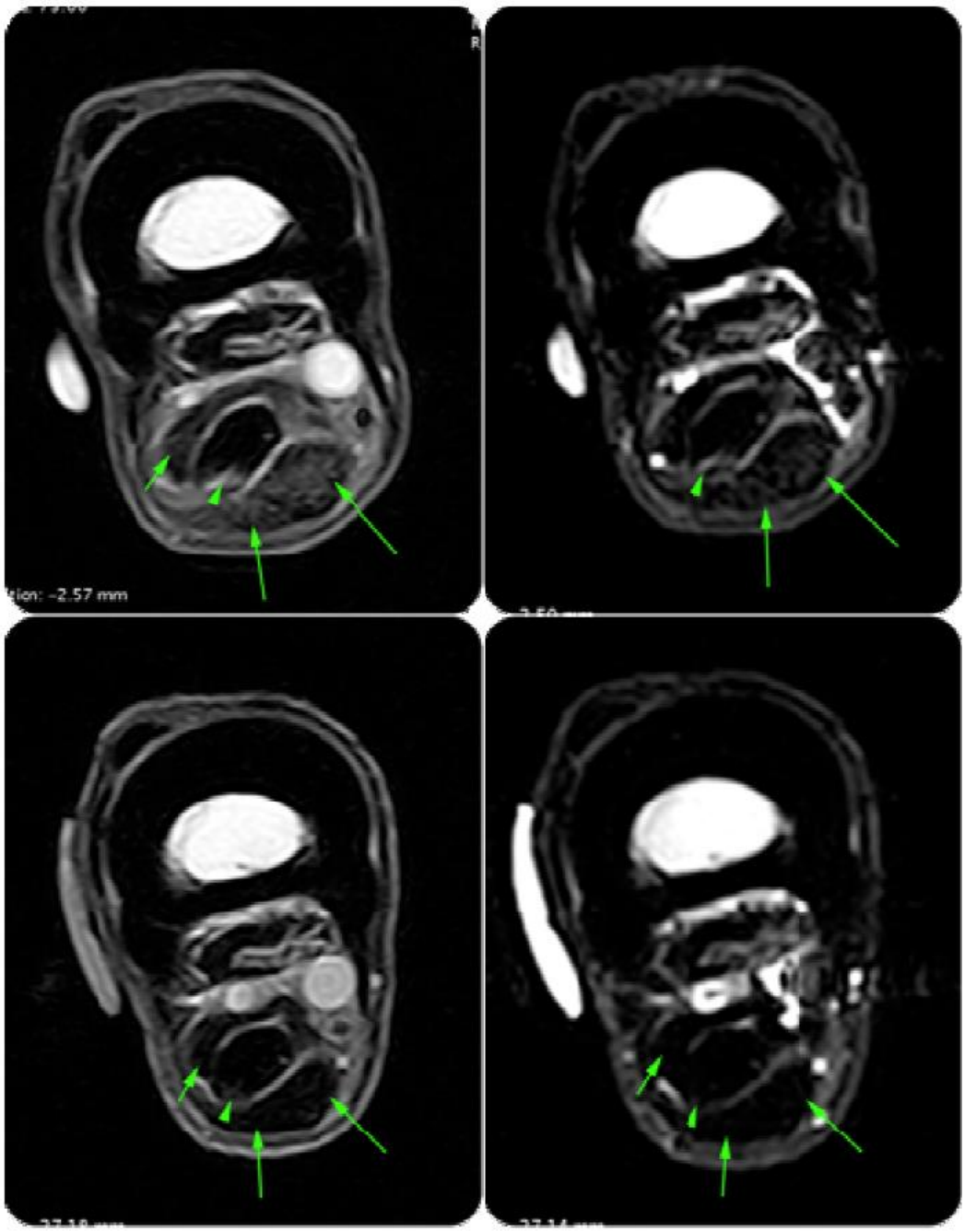


Figure 56a-56d. 4yo QH stallion used for reigning. PD axial top left, T2 axial top right- first MRI obtained 7 mos. after stem cell therapy due to SDFT, DDFT, and ICL injury. PD axial bottom left and T2 axial bottom right --5mos MRI follow-up (one year after stem cell therapy) with significant improvement in the PD and T2 signal of the ICL, DDFT, and SDFT. This is considered excellent quality collagen matrix comparable to that of normal tissue.

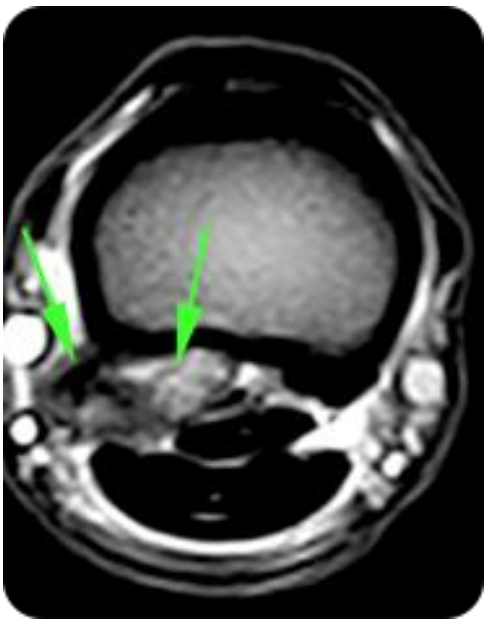


Figure 57a. T2 axial - 9yo hunter pony with a lameness localized to the coffin joint/pastern region. A severe desmitis of the medial ODSL (mid body) was diagnosed on MRI. After the examination, ultrasonic evaluation of the ligament was performed and reportedly unable to appreciate the severity of fiber tearing. The pony was placed in a cast.

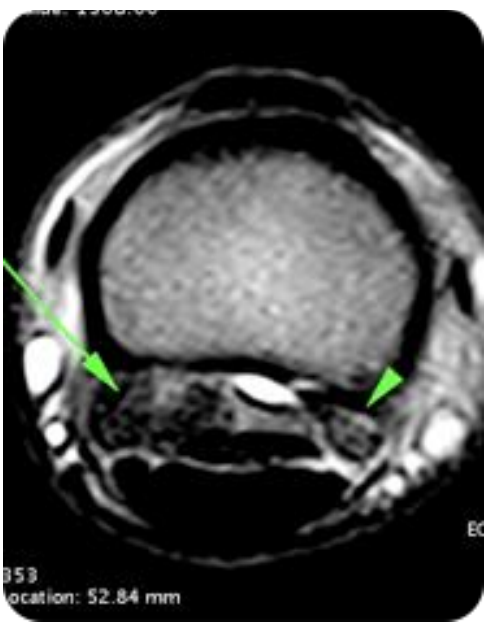


Figure 57b. 2 mos. follow-up -- T2 axial. The pony was sound when the cast was removed, but subluxated his pastern joint the next day and the lameness had progressed. This follow-up MRI revealed significant improvement in the signal and architecture of the medial ODSL (arrow), but progressive desmitis of the lateral ODSL (arrowhead).

Figure 57. (Continued).

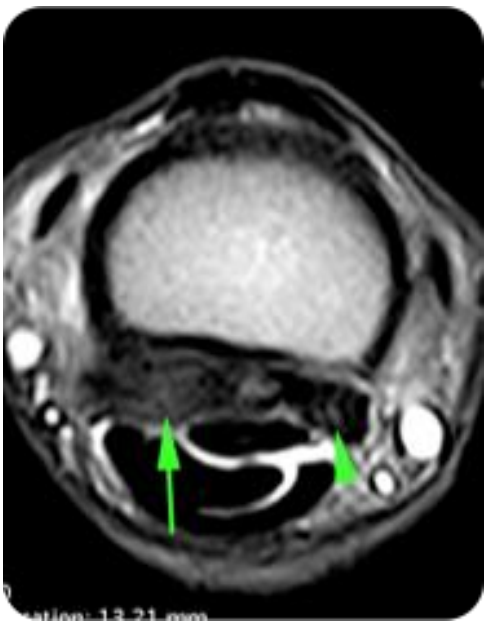


Figure 57c. 4 mos. follow-up. Progressive healing, though still chronic-active, of the medial and lateral ODSL branches (arrow and arrowheads). The MRI also revealed fiber tearing of the distal medial suspensory branch and the medial and lateral collateral ligaments of the fetlock joint. The pastern joint subluxation progressed and the pony was euthanized.

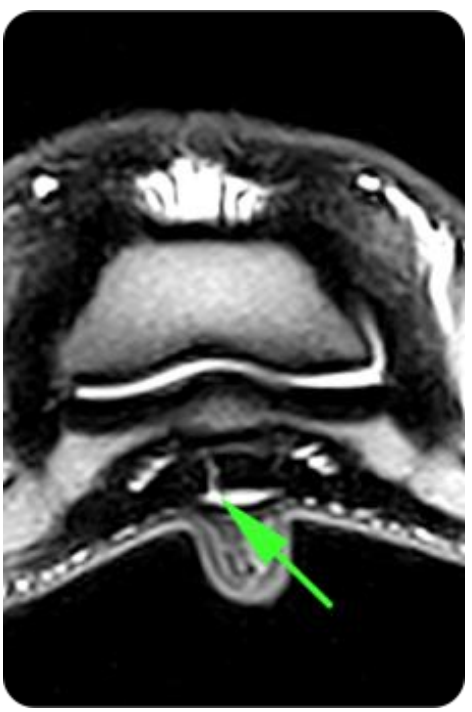


Figure 58. T2-weighted axial perpendicular to distal DDFT--Thin discrete split extending from the dorsal to the palmar margins of the distal DDFT, level of distal navicular bursa.

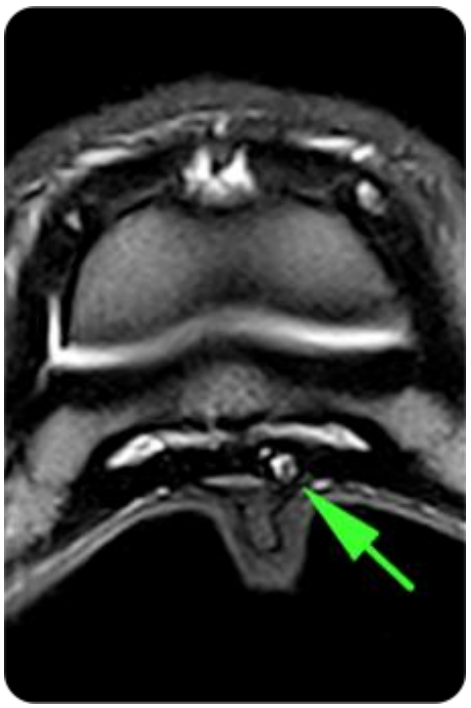


Figure 59. T2-weighted axial perpendicular to distal DDFT-- Active palmar margin DDFT core lesion distal DDFT, level of distal navicular bursa.

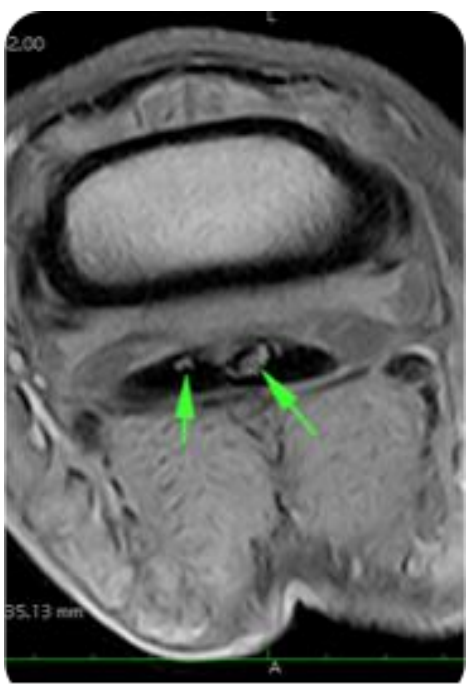


Figure 60. PD axial--large core lesion in the dorsal aspect of the medial DDFT lobe, and a much smaller core lesion in the lateral lobe at the level of the proximal navicular bursa.

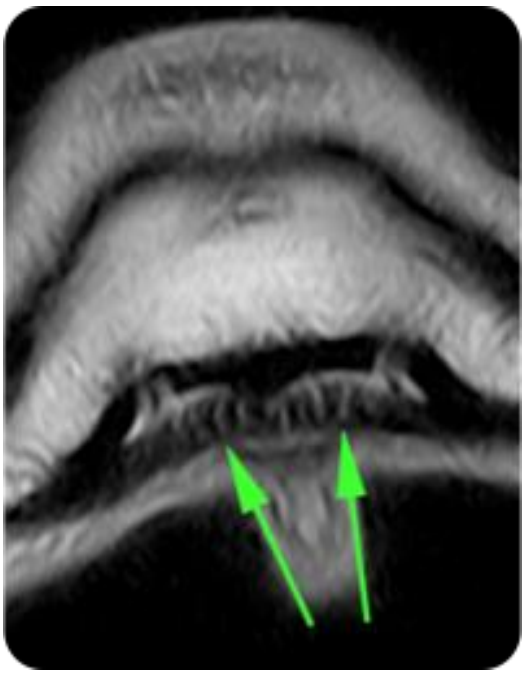


Figure 61. PD axial perpendicular to distal DDFT. Numerous prominent interfiber septations throughout the distal DDFT consistent with a generalized tendinopathy.

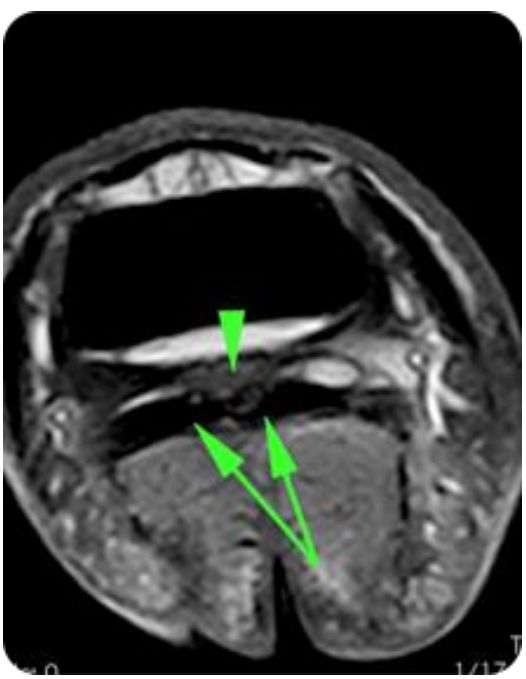


Figure 62. STIR axial--low signal tissue proliferation extending from the collateral sesamoidean ligament of the navicular bone to the DDFT typical of a bursitis with likely adhesions. Also associated dorsal margin and intrasubstance fiber lesions of the DDFT at this level.

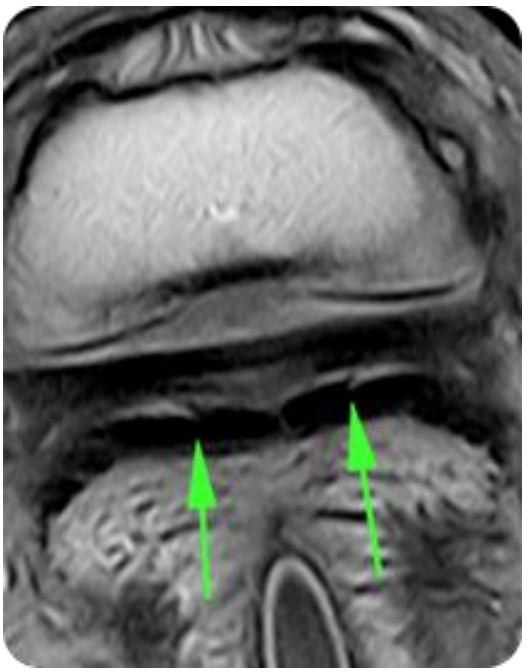


Figure 63a. PD axial perpendicular to the DDFT, level of proximal navicular bursa--small dorsal margins splits involving the medial and lateral DDFT lobes.

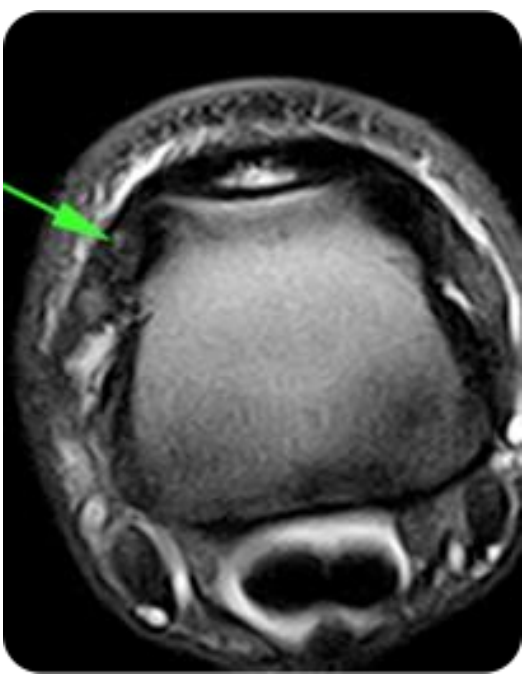


Figure 63b. T2 axial perpendicular to the collateral ligaments of the coffin joint. Well marginated increased T2 signal representing a core lesion in the origin of the lateral collateral ligament.

Figure 63. (Continued).

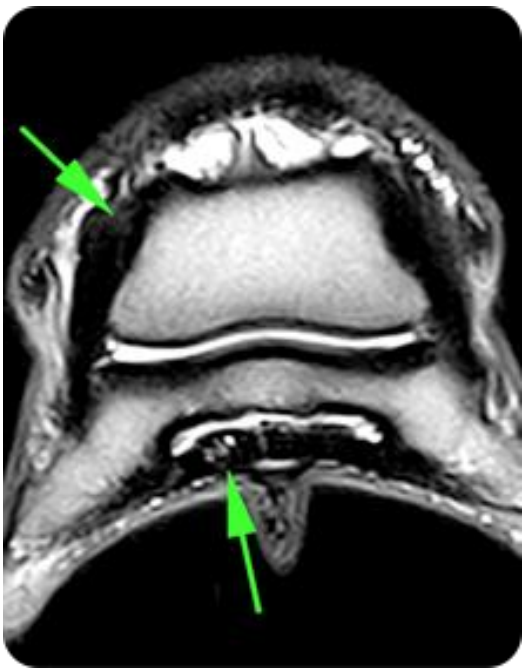


Figure 63c. T2 dorsal, perpendicular to the distal DDFT--active intrasubstance core lesion of the distal DDFT at the level of the distal navicular bursa. Also relatively mild increase in T2 signal seen within the origin of the medial collateral ligament of the coffin joint.

Perhaps it is unnecessary to emphasize the obvious, but localizing the lameness prior to MRI is paramount to a successful outcome. This is wrought with its own complications, given the variety of responses to intra-articular and perineural anesthesia and the complexities of an obscure lameness case. A horse is blocked, and often repeatedly re-blocked to be certain that the requested body part for MRI is the source of the lameness. Multiple sources of lamenesses are frequently reported (ex. foot and fetlock lesions), but some horses may also have unexpected results following perineural anesthesia--ex. blocking sound to a palmar digital nerve block, but having no identifiable lesions in the foot on MRI, but marked pathologies in the region of the fetlock. Some clinicians, to avoid this pitfall, may opt to scan both the foot and fetlock for a unilateral lameness that blocks to the foot region. It is particularly common for a horse to block sound in one foot, then go lame in the opposite foot, and in this case a bilateral foot exam is usually requested. Similarly with other body parts, a thorough lameness workup is exceptionally important prior to MRI exam.

Common Clinical Findings on MRI

Within the foot, several anatomic structures are very frequently found to be abnormal. These include the DDFT, the navicular bone, the navicular bursa, the distal impar ligament, the collateral sesamoidean ligament of the navicular bone, the coffin joint, and the collateral ligaments of the coffin joint.

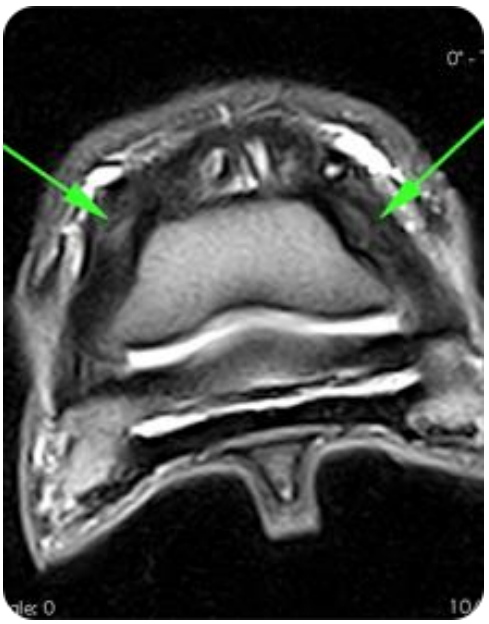


Figure 64a. 14 yo gelding Warmblood with a history of bilateral front limb lameness that is worse on the left front. T2 dorsal, parallel to the collateral ligaments of the coffin joint--increased heterogeneousT2 signal seen within the central and peripheral fibers of the medial and lateral collateral ligaments of the coffin joint representing a biaxial desmitis/desmopathies.

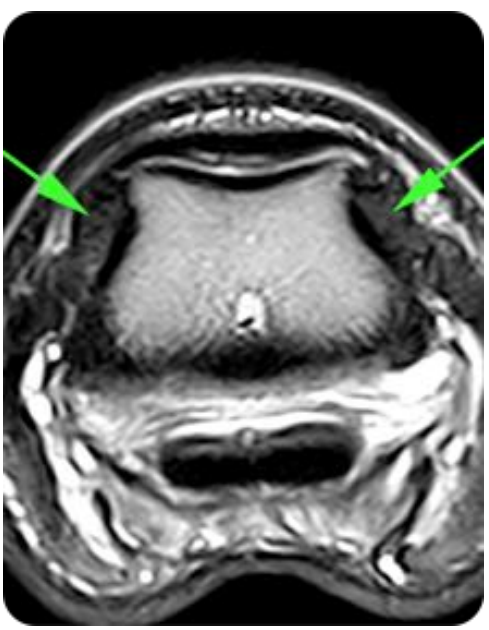


Figure 64b. T2 axial perpendicular to the collateral ligaments of the coffin joint that confirm a diffuse heterogeneous increase in T2 signal throughout the collateral ligaments of the coffin joint, typical of generalized desmopathies with numerous suspected fiber disruptions. This horse also had a residual abscess of the medial sole of the foot and some coffin joint pathologies (not shown).

Figure 64. (Continued).

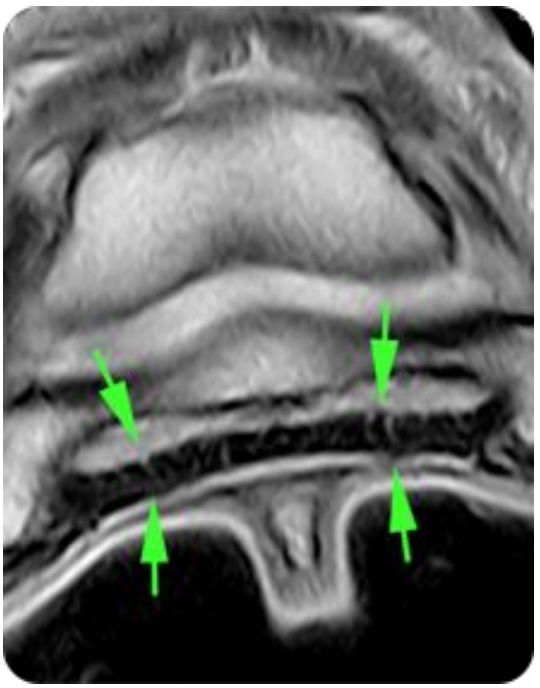


Figure 64c. T2 dorsal perpendicular to the distal DDFT shows numerous prominent inter-fibrous septations at the distal aspect of the DDFT consistent with a generalized tendinopathy.

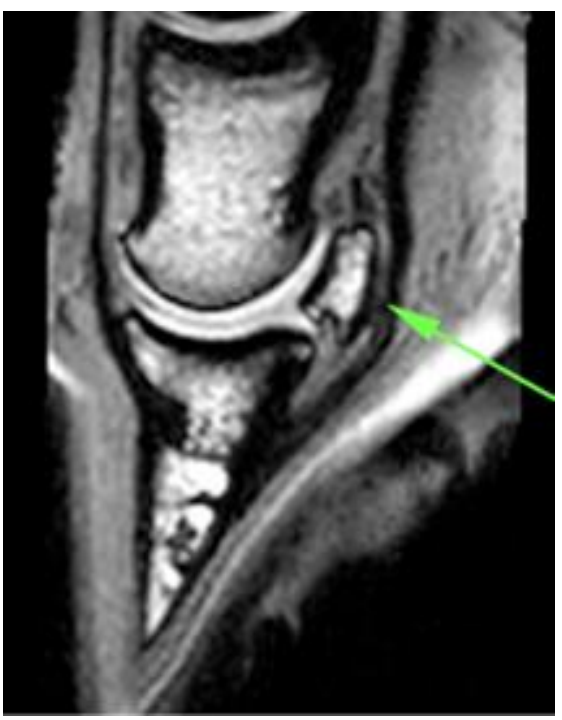


Figure 65. isotropic 3D GE T1 sagittal with focal decreased fibrocartilage signal suggesting degeneration/loss along the flexor surface of the navicular bone.

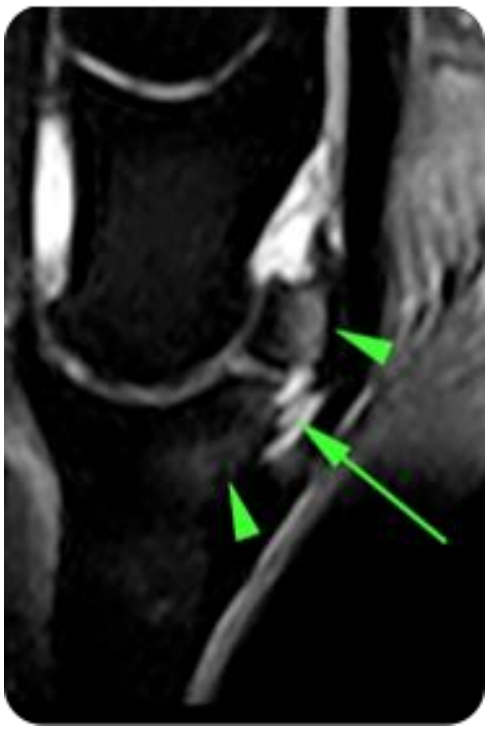


Figure 66. STIR sagittal--abnormal increased signal within the distal impar ligament (long arrow) suggesting an increase in size or number of synovial invaginations. Abnormal increase in STIR signal is also seen with the navicular bone and in palmar P3 (short arrows) where the distal impar ligament inserts, suggesting active intramedullary bone injuries.

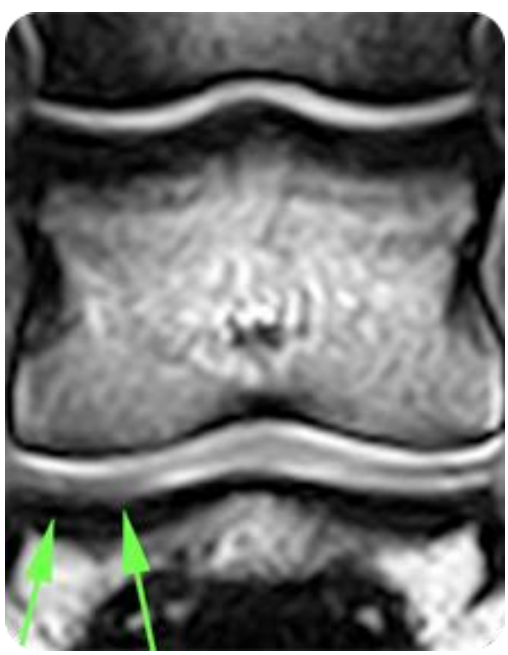


Figure 67. 3D GE T1 dorsal plane image shows an erosive articular and subchondral lesion in the coffin joint at the medial aspect of P3.

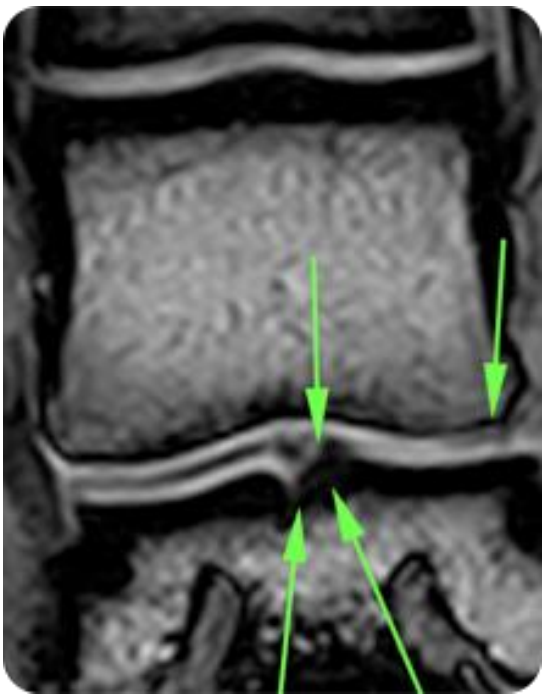


Figure 68a. 3D GE T1 dorsal--wedge-shaped defect present on midline in proximal P3 breaking out into the coffin joint. Associated articular cartilage signal decrease indicating degeneration/loss. Additional, cartilage signal decrease at the medial aspect of the joint in distal P2.

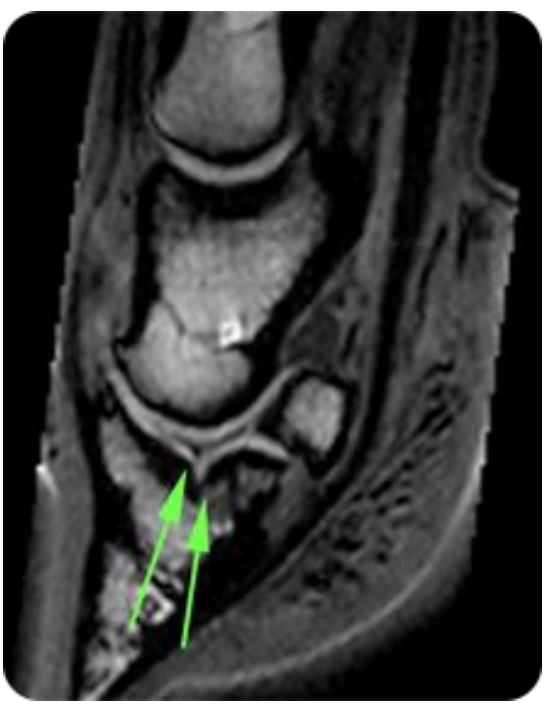


Figure 68b. The sagittal plane of the same horse showing the proximal P3 defect.

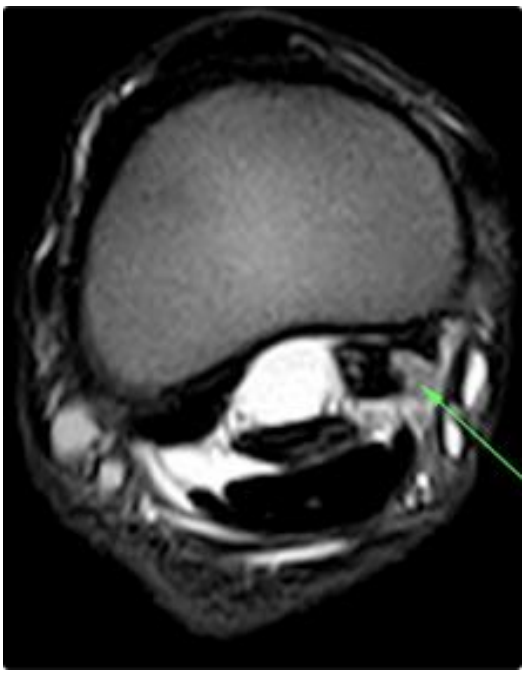


Figure 69a. T2 axial--Active core lesion breaking out the palmar margin of the medial oblique distal sesamoidean ligament.

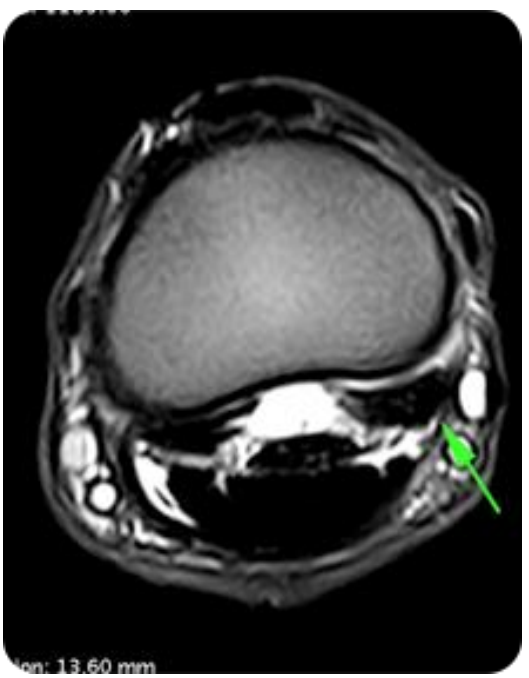


Figure 69b. 2 year follow-up. There is complete resolution of the fiber architecture, PD, and T2 signal (shown here) indicating very good quality healing in the previously seen core lesion of the medial oblique distal sesamoidean ligament. Additional lesions elsewhere in the limb were believed to be the cause of the recent lameness.

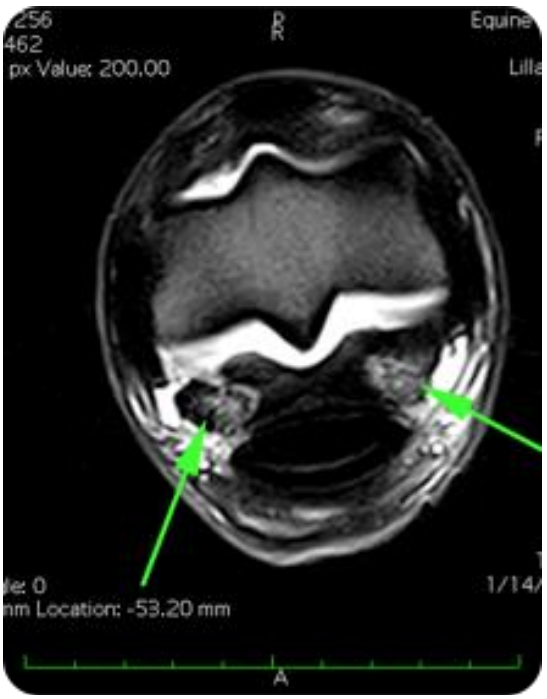


Figure 70. T2 axial - Generalized desmopathies of the medial and lateral oblque distal sesamoidean ligaments at their sites of origin.

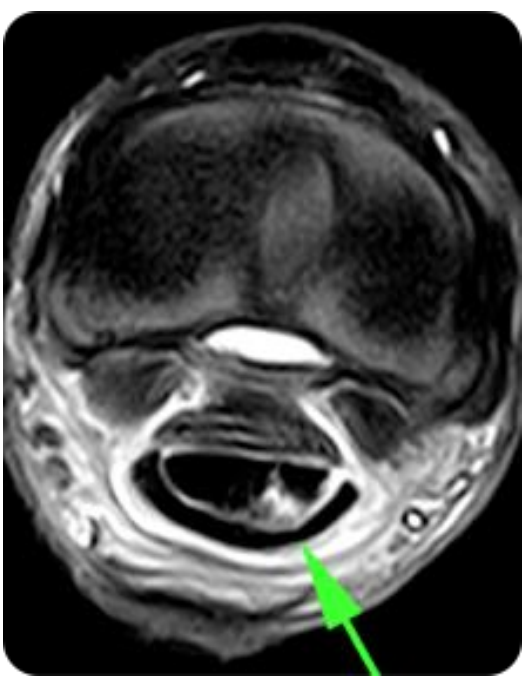


Figure 71. T2 axial -- large palmar margin crevice tear of the DDFT, level of the metacarposesamoidean joint.

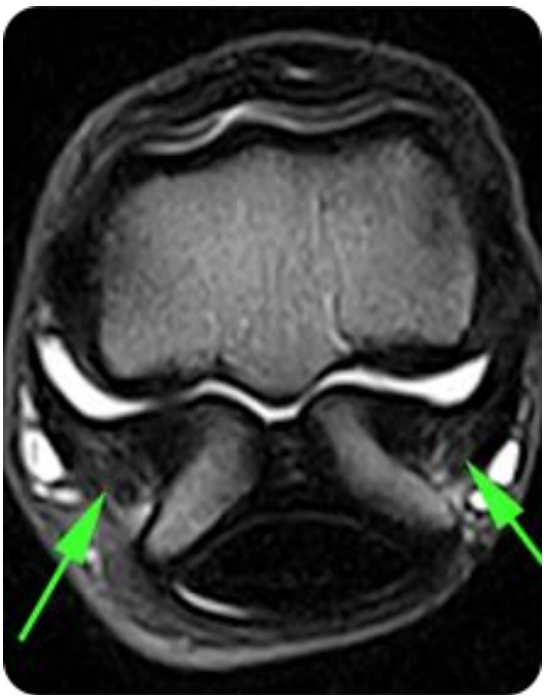


Figure 72. T2 axial -- typical mild generalized desmopathy at the distal insertional sites of the medial and lateral suspensory branches.

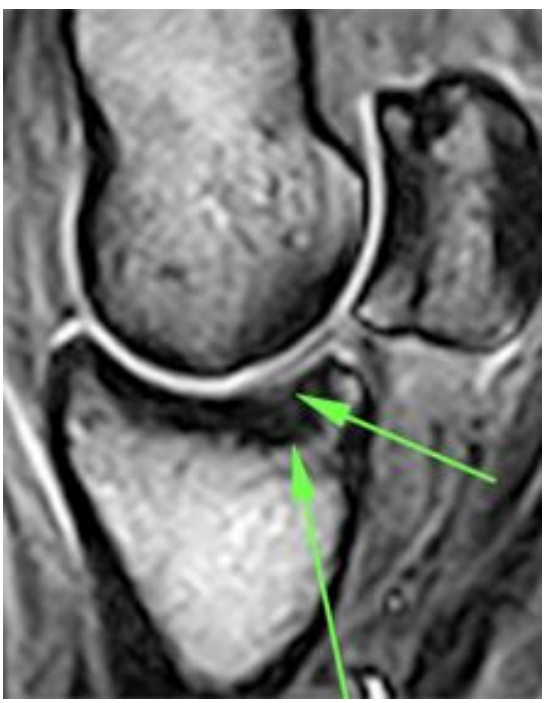


Figure 73. 3D GE T1 sagittal that shows an erosive lesion of proximal P1 at the metatarsophalangeal joint. Additional lesions were present of the supporting fetlock soft tissues.

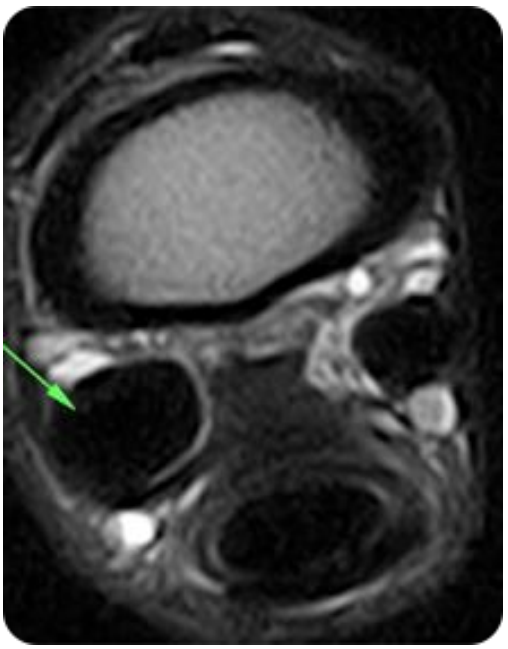


Figure 74a. T2 axial. 10yo Warmblood horse with a chronic lateral suspensory branch problem. The horse is lame again with sensitivity to palpation of the branch. The MRI shows completely quiescent and well healed distal suspensory branch desmopathy given the intrasubstance hypointensity on the PD and T2 sequences.

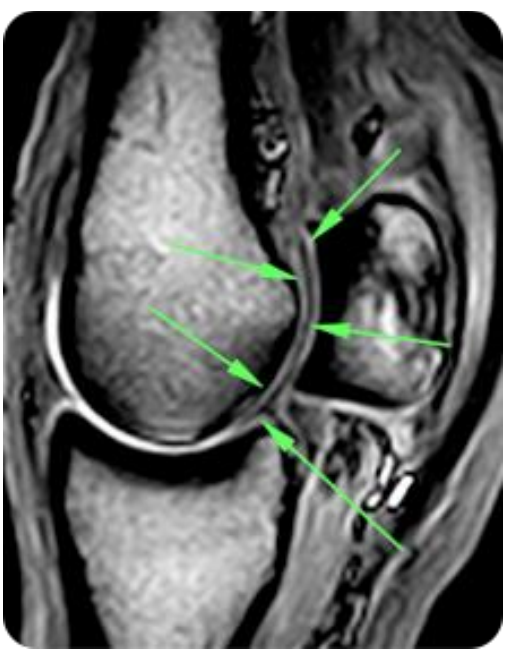


Figure 74b. 3D GE T1 sagittal sequence shows generalized decreased articular cartilage signal of the metacarposesamoidean joints suggesting degeneration.

Figure 74. (Continued).

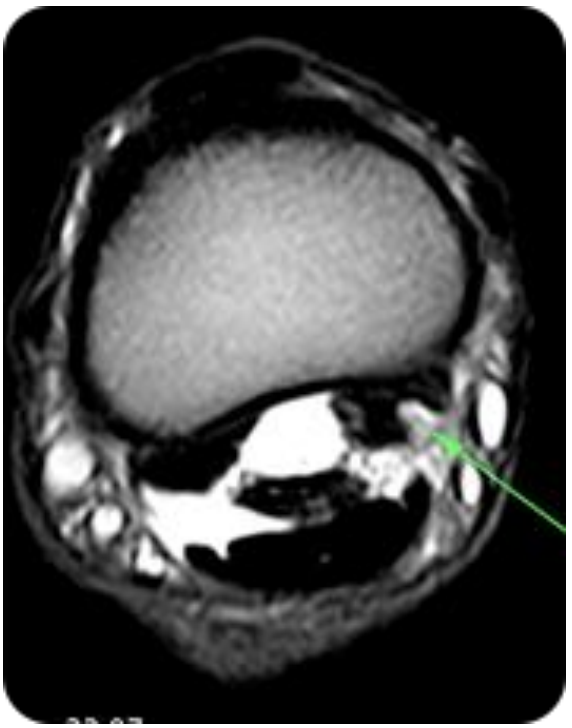


Figure 74c. T2 axial shows an active core lesion breaking out the palmar margin of the medial ODSL.

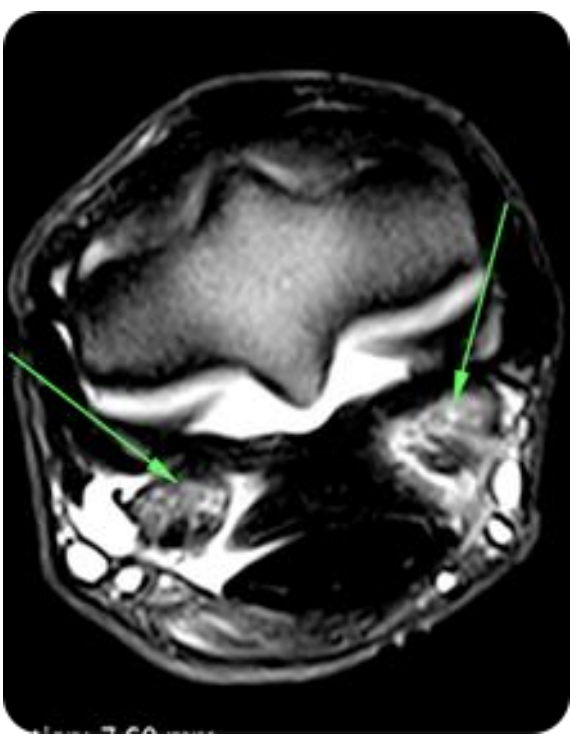


Figure 74d. T2 axial. Active desmopathy of the medial and lateral ODSLs.

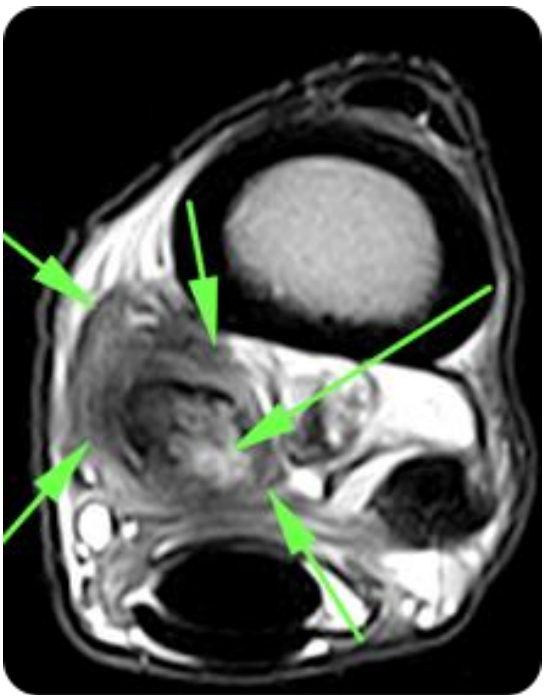


Figure 75. Marked to severe desmitis of the distal left hind medial suspensory branch. Active core lesion breaking out the plantar medial corner, generalized desmitis of the remainder cross-sectional area, all surrounded by a large ‘halo’ of connective tissue.

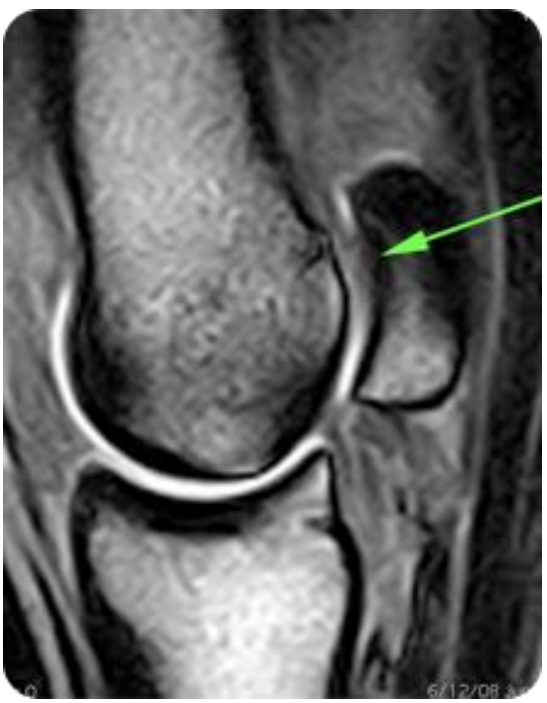


Figure 76. Isotropic 3D GE T1 sagittal showing an erosive lesion in the medial metacarposesamoidean joint. The horse also had numerous supporting soft tissue injuries.

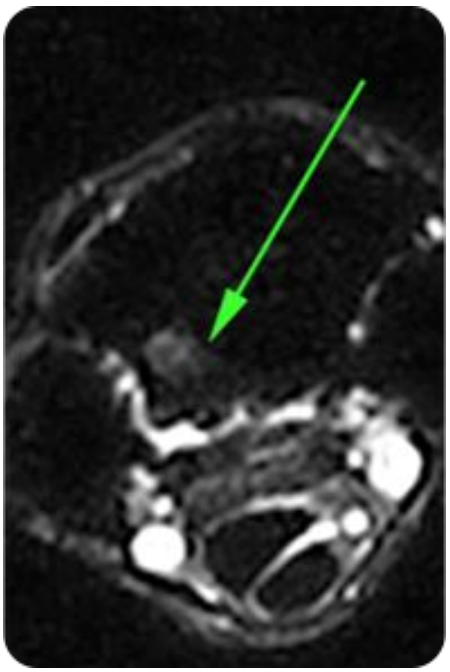


Figure 77a. 8 yo Warmblood gelding with a lameness localized to the carpus region. Axial STIR carpus, level of proximal metacarpus. Focal STIR signal seen at the lateral aspect of the palmar proximal metacarpus typical of an active bone injury (stress microfractures). The origin of the proximal suspensory ligament (not shown) was unremarkable.

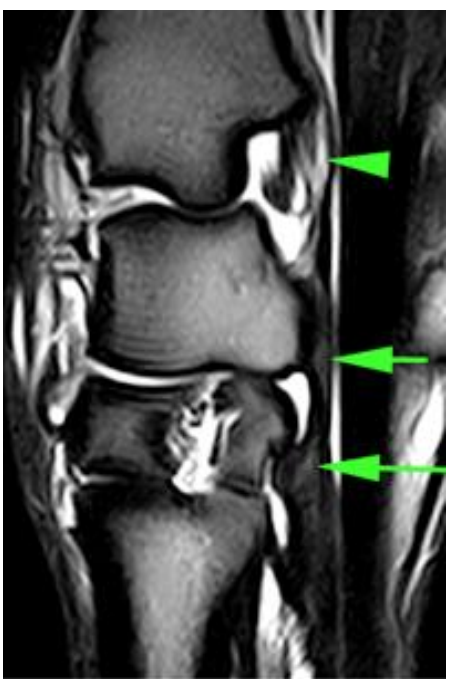


Figure 77b. Sagittal T2 carpus. Increased T2 signal seen at the origin and distal portions of the palmar carpal ligament and also at the origin of the inferior check ligament suggesting active desmitis/demopathies of all these structures.

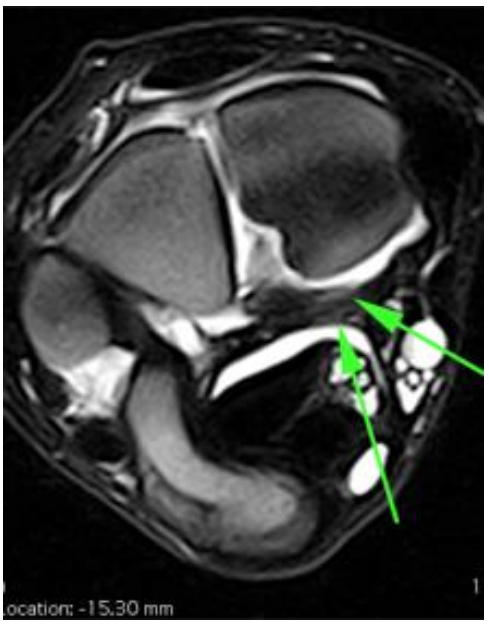


Figure 78a. 12yo WB horse with a long term RF lameness, localized to the subcarpal region. Does not improve with an intra-articular block to the joints of the carpus. Partial improvement with a block to the suspensory region but complete improvement following a carpal sheath block. No convincing ultrasonographic lesions. T2 axial carpus level of proximal row of carpal bones--mild heterogeneous increase in T2 signal seen within the medial portion of the palmar carpal ligament consistent with an active desmitis/desmopathy. An associated carpal sheath effusion is also seen.

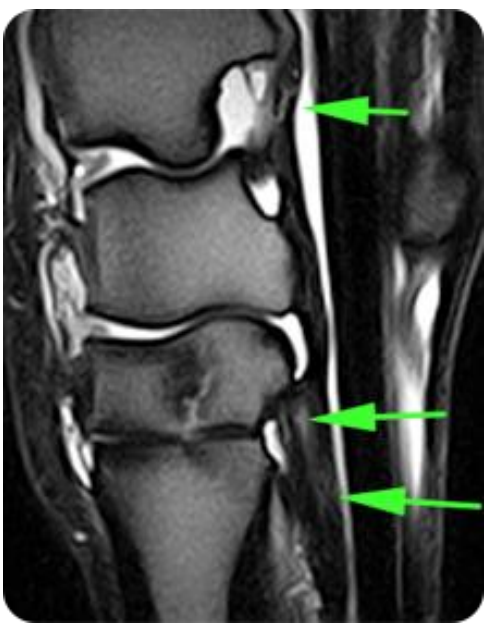


Figure 78b. corresponding T2 sagittal carpus, showing the heterogeneous increase in T2 signal of the proximal part of the palmar carpal ligament. Additional pathology within the origin of the inferior check ligament representing an active desmitis/desmopathy of this ligament as well.

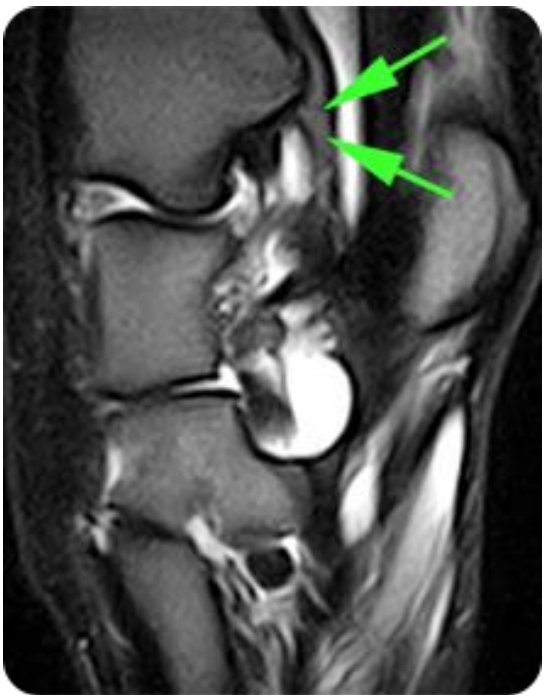


Figure 79. T2 sagittal carpus--Increased T2 signal seen at the origin of the palmar carpal ligament off the caudal distal radial physeal flare and surrounded by an abnormal amount of fluid in the carpal sheath consistent with an active desmitis/desmopathy.

Particularly common DDFT lesions include splits and crevices along the dorsal margin of the medial and lateral lobes at the level of the proximal navicular bursa. Larger core lesions may also occur at this level, and may extend (and originate) more proximally and distally as well. Prominent inter-fiber septations suggestive of a generalized desmopathy, discrete splits, dorsal margin crevices and additional core lesions may also be present at the level of the distal navicular bursa. (Figures 58, 59, 60, 61, 62, 63 a, b, c, 64 a, b, c)

Numerous supporting soft tissue lesions are seen in the navicular apparatus. The fibrocartilage layer along the flexor cortex of the navicular bone is frequently degenerative in performance horses and may also have adhesions to the adjacent DDFT. Navicular bursal pathology is also frequently seen with navicular syndromes and navicular diseases, and include soft tissue proliferation within the bursa, adhesions, and capsular thickening. The collateral sesamoidean ligament of the navicular bone is oftentimes thickened and with increased signal on all pulse sequences, indicating degeneration and micro-tearing. The distal impar ligament may show an increase in number and size of the synovial invaginations from the coffin joint. These pathologies are typical of ligamentous degeneration. Also frequently seen with this pathology, is a palmar P3 insertional enthesopathy where prominent vascular penetrations, increased STIR signal representing a bone reaction, and occasional discrete bone lysis are seen at the insertional site of the distal impar ligament. Similar pathology may also be present at the origin of the ligament on the distal navicular bone. (Figure 65, 66)

The articular cartilage of the coffin joint is also frequently abnormal. These pathologies may include evidence of cartilage degeneration, as well as discrete cartilage disruptions and an ill-defined subchondral plate, indicating subchondral erosions. (Figure 67, 68 a, b)

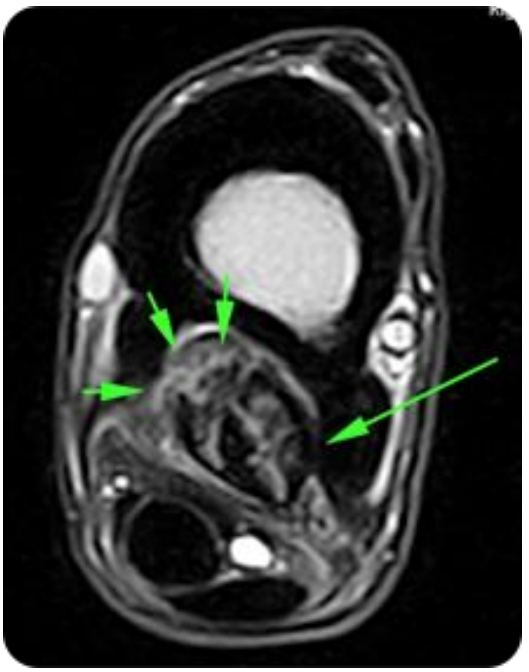


Figure 80a. 9yo Westphalian horse. T2 axial proximal RH metatarsus. Enlarged proximal suspensory ligament with moderate/markedly T2 hyperintense dorsal fibers of the medial lobe consistent with an active desmitis/desmopathy. More subtle/mild fiber tearing of the lateral fibers.

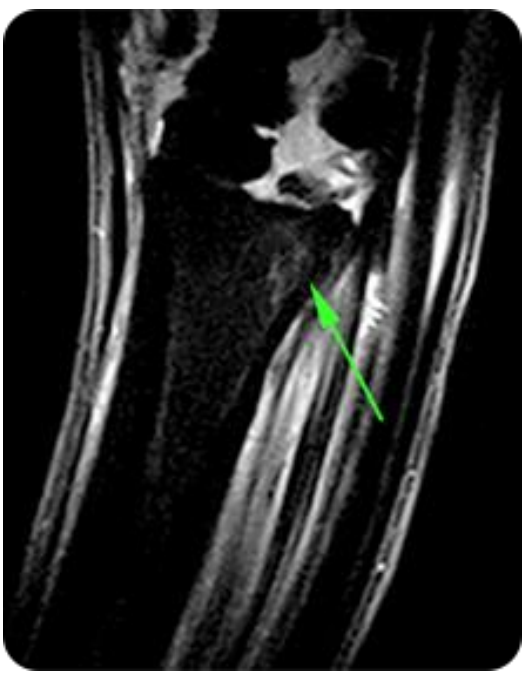


Figure 80b. STIR sagittal RH---additional bone pathology of the plantarolateral aspect of proximal MTIII suggesting trabecular microfractures.

Figure 80. (Continued).

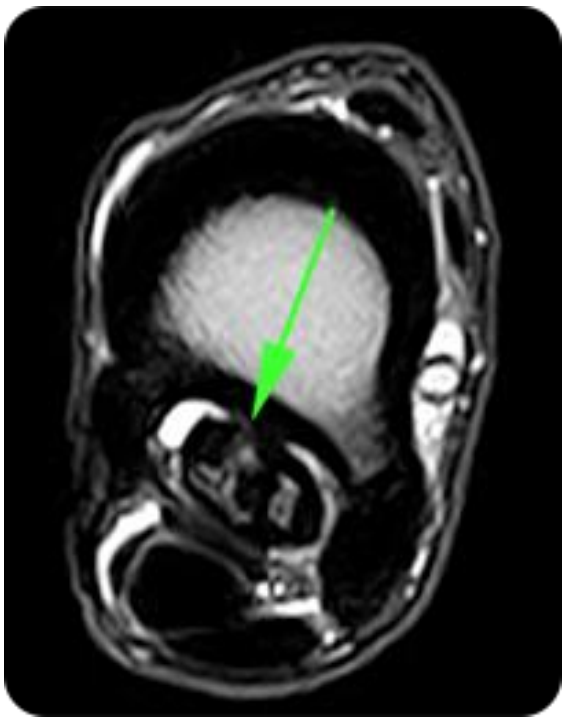


Figure 80c. T2 weighted axial proximal metatarsus LH--focal fiber tear breaking out the dorsal aspect of the medial lobe of the proximal suspensory ligament origin.

Both the medial and lateral collateral ligaments of the coffin joint are another anatomical structure that is frequently affected in a foot lameness. The medial collateral ligament is more commonly affected. Increased T2 and STIR signal can be seen within the origin and proximal portion of the ligament. Discrete splints, tears and core lesions are also seen, though less frequently, and even rarer are complete ruptures. Pathologies may also be present at the mid, distal and P3 insertional sites within the collateral fossae. Periligamentous enthesophytosis as well as osteolysis may be seen with distal collateral ligament desmitis/desmopathies. The course of both the medial and lateral collateral ligaments contain fibers that are invariably curved (angled) relative to the magnetic field, so interpretation of the MRI must take into consideration the magic angle artifact. This artifact is most prevalent on low TE sequences (T1 and PD weighted sequences) and, therefore, the T2 weighted sequences are considered much more reliable for accurate assessment of active pathologies, particularly in these troublesome areas. (Figure 63, 64)

Increased STIR signal is also frequently present within the intramedullary cavity of the navicular bone. Oftentimes, this is a relatively subtle to mild finding, perhaps within the central portion of the intramedullary cavity and/or along the endosteal margin. These pathologies may not correspond with much change on the thin 3D GE T1 sequence, with the exception of a prominent number of linear hypointensities, indicating an increased vascularity. However, when the STIR signal is more pronounced and corresponds with decreased T1 signal fatty replacement on the heavily T1-weighted sequences, this is more indicative of a diagnosis of edema/inflammation/hemorrhage and, in marked cases, osteonecrosis. (Figure 66)

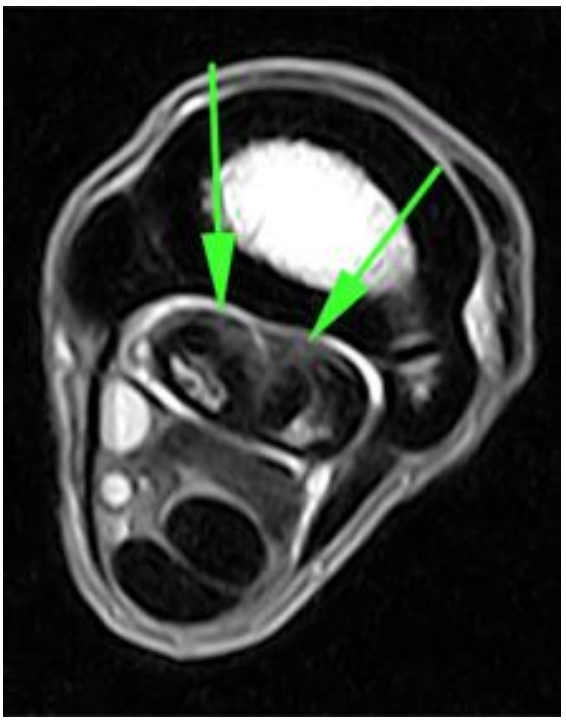


Figure 81a. PD axial shows PD hyperintense and predominant T2 hypointense (Figure 21b) thickened dorsal fibers of both proximal suspensory lobes. This is typical of a healed injury, but with immature collagen that maintains PD signal relative to surrounding normal fibers.

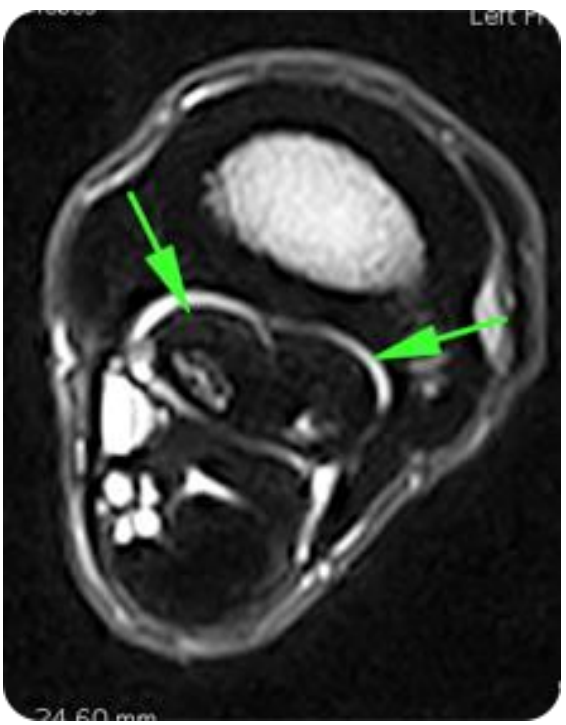


Figure 81b. T2 axial.

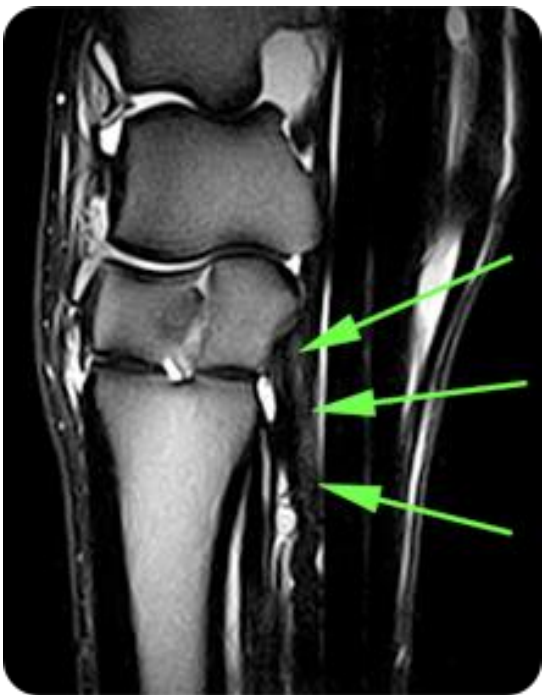


Figure 82a. 12yo Holsteiner horse with a LF lameness that improves with a wheat block and an intercarpal block. U/S evaluation reveals mild, but underwhelming changes of the proximal suspensory ligament. MRI shows an active desmitis/desmopathy of the proximal portion and origin of the inferior check ligament.

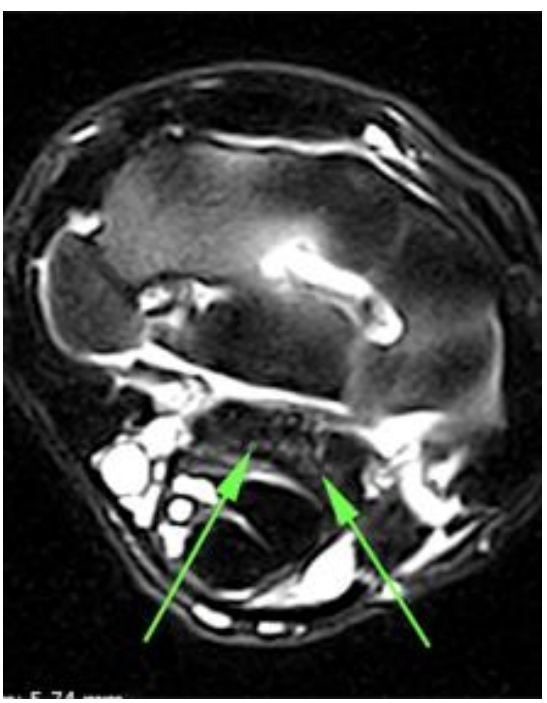


Figure 82b. T2 axial level of metacarposesamoidean joint, same horse.

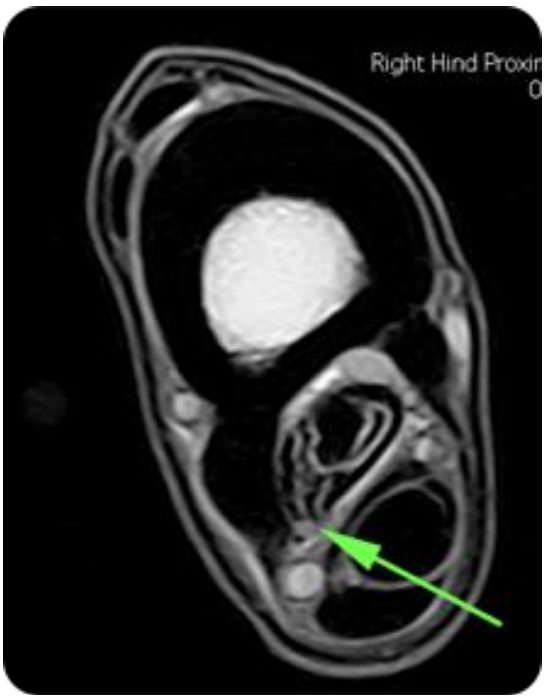


Figure 83. Plantaromedial protrusion of the suspensory ligament body toward the lateral plantar nerve, suggesting an adhesion.

The pastern joint is almost always included in a routine foot examination. As is seen even radiographically, this joint is also prone to degenerative lesions. On MRI, some of the cartilage abnormalities can be seen to be associated with subchondral plate demineralization/erosions, and in some cases, larger subchondral defects or cystlike lesions. The collateral ligaments of the pastern joint need to be assessed on MRI as well. Abnormalities of the pastern ligaments, namely the medial and lateral oblique distal sesamoidean ligaments, and to a lesser extent the straight distal sesamoidean ligament are frequently found. (Figure 57, 69 a, b).

Fetlock MRI, like the foot, often reveals multiple abnormalities. A lot of horses that block to a low four-point or intra-articular block can and often do have distal suspensory branch abnormalities, oblique distal sesamoidean ligament abnormalities, as well as articular pathologies. Articular cartilage degeneration can be seen in any part of the joint, but the dorsal and palmar/plantar McIII/MtIII condyles and the metacarpo/metatarsosesamoidean joints are frequently seen. The articular abnormalities are considered more significant when the underlying subchondral plate is ill-defined or osteolytic, consistent with erosions. The distal suspensory branches are readily visible and size, shape, architecture, and collagen quality is typically very well assessed. Acute, chronic, and chronic-active tears will be evident and are often seen, but generalized insertional desmopathies of the distal branches and of the origins of the oblique distal sesamoidean ligaments are more frequently seen in the routine fetlock study. Abnormalities of the dorsal joint capsule, including the site of insertion onto the medial and lateral supracondylar fossae near the collateral ligaments of the fetlock joint are occasionally involved with fetlock injuries. The collateral ligaments themselves should be carefully examined for injuries. The most frequently seen combination of pathologies in a

standard fetlock lameness are varying degrees of distal suspensory branch desmopathies, oblique distal sesamoidean ligament desmopathies, and fetlock joint arthropathies. (Figure 70, 71, 72, 73, 74 a-d, 75, 76, 69 a, b, 57).

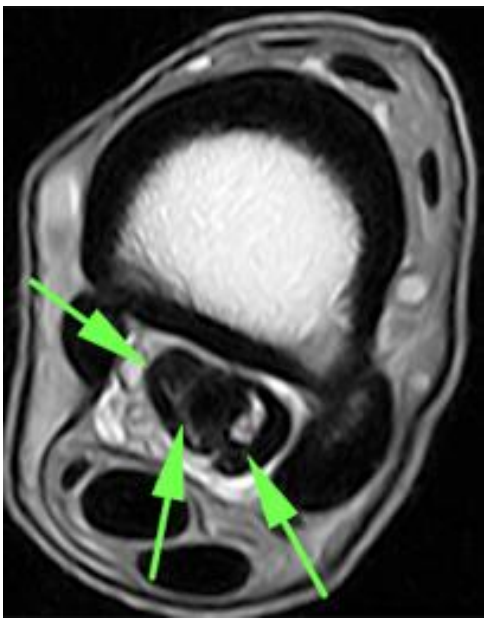


Figure 84a. 10 yo Westphalian horse with a prior history of proximal left hind retinaculum release and neurectomy of the lateral plantar nerve. Had improved then became lame again. PD axial -- thickening of the dorsomedial and central fibers of the of the proximal suspensory ligament, but with complete PD (and T2) hypointensity indicating a quiescent, prior and well healed injury.

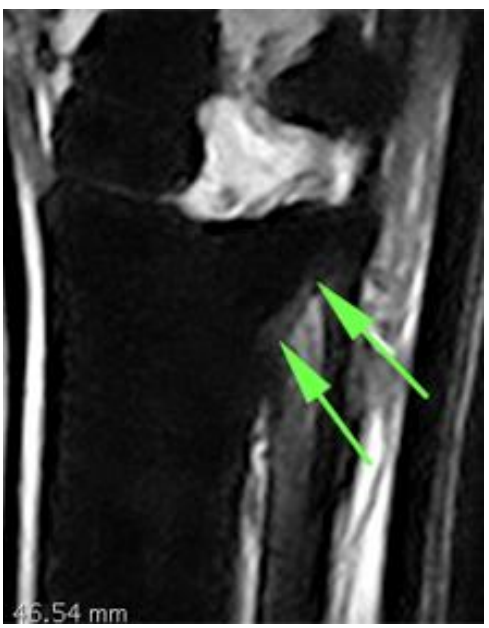


Figure 84b. Active cortical bone reaction/stress fracture at the bony origin of the proximal suspensory ligament.

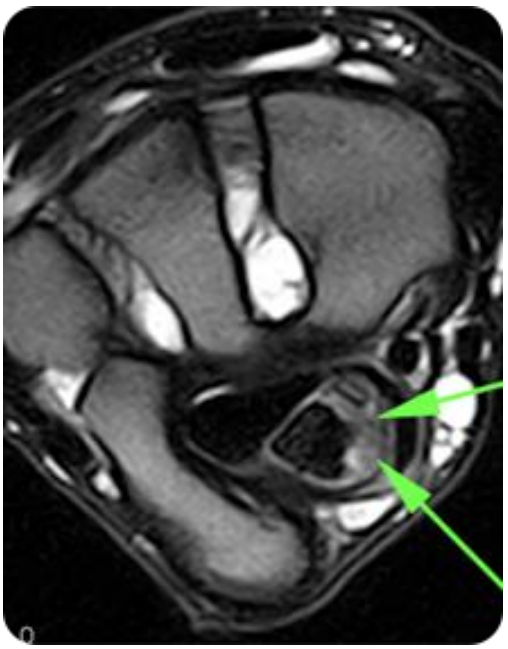


Figure 85a. T2 axial of the carpus. 10yo driving horse with a known chronic SDFT lesion. MRI was requested to determine the proximal extent of the lesion and shows abnormal proliferation of low signal tissue adjacent to some medial SDFT margin lesions typical of a tenosynovitis.

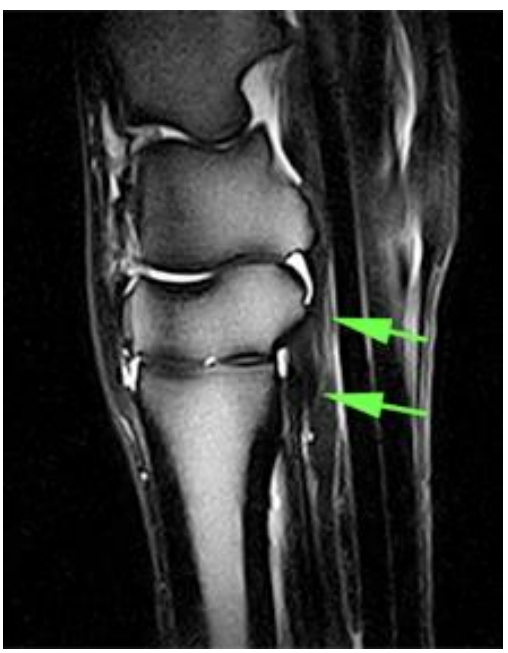


Figure 85b. T2 sagittal of the carpus. Abnormal T2 signal involving the palmar fibers of the origin of the inferior check ligament.

Figure 85. (Continued).

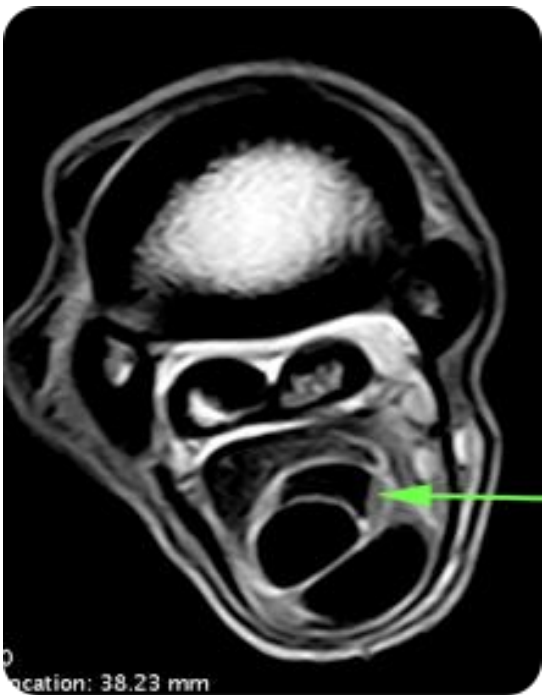


Figure 85c. PD axial of the proximal-mid metacarpus shows unusual presence of an anomalous tendon (arrow) located between the DDFT and the inferior check ligament, believed to be incidental.

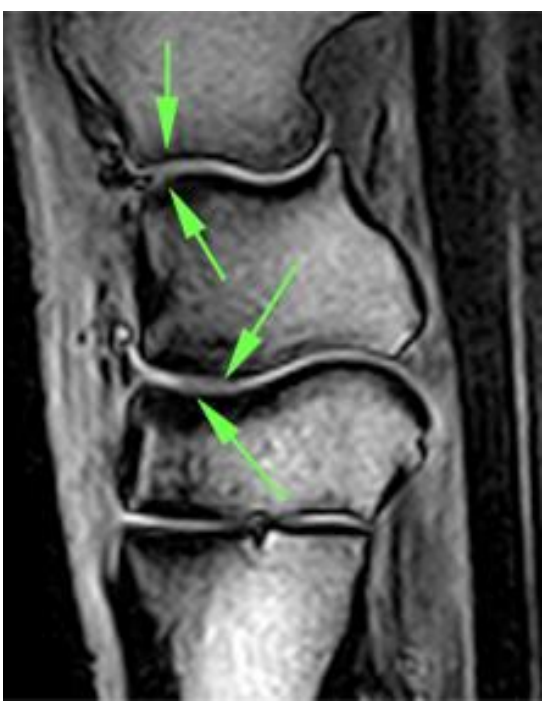


Figure 86. 3D GE T1 sagittal carpus--subtle to mild signal decrease within the articular cartilage, suggesting cartilage degeneration. No associated subcondral bone lesions. This is not considered clinically significant.

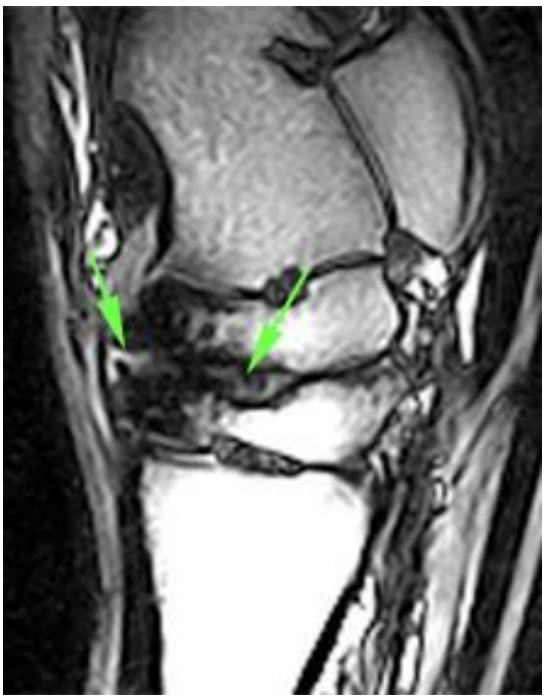


Figure 87. 3D GE T1 sagittal tarsus--moderate to marked degeneration within the distal intertarsal joint, including erosion of the articular and subchondral structures as well as intramedullary pathology of the cuboidal tarsal bones (T3 and TC).

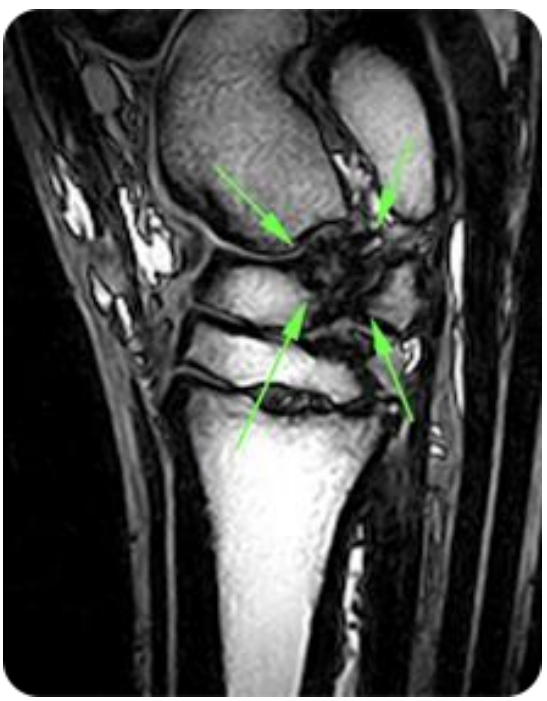


Figure 88. Isotropic 3D GE T1 sagittal showing unusual osteoarthritic pathology confined to the centroquartal joint of the tarsus.

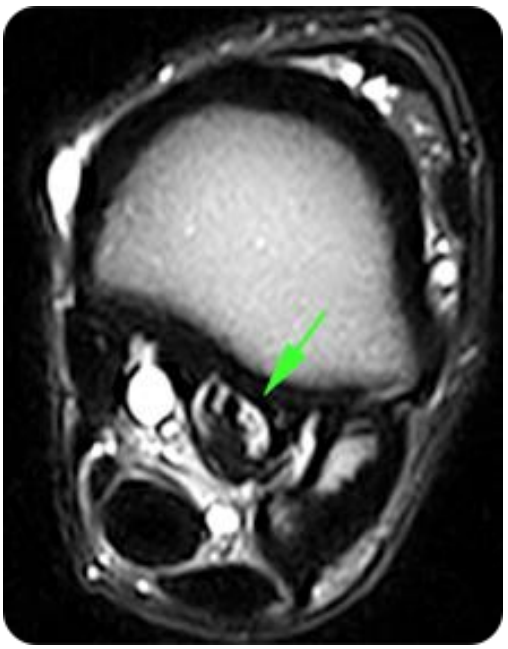


Figure 89a. T2 axial image of the proximal metatarsus in a horse that was noted to have a core lesion in the proximal suspensory ligament on ultrasound. The horse blocked consistently and repeatedly to the proximal suspensory region. The MRI appearance of the suspensory ligament origin was completely underwhelming, with the exception of an increased amount of fatty tissue as appreciated on the fat suppressed STIR sequences. The horse was found to have lesions in the distal tarsal joints and an active desmopathy of the long plantar ligament.

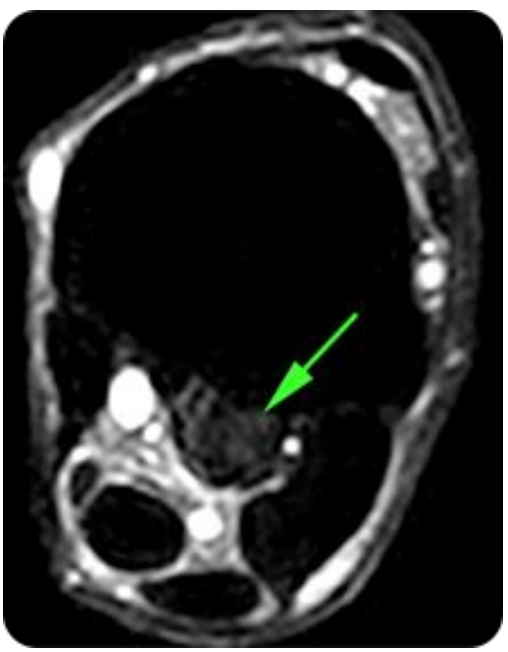


Figure 89b. STIR axial image revealing an unusual amount of fat suppression within the central connective tissues of the proximal suspensory ligament. No core lesion is present. Clinically, the horse immediately responded to distal intertarsal and tarsometatarsal joint steroid and calcitonin injections.

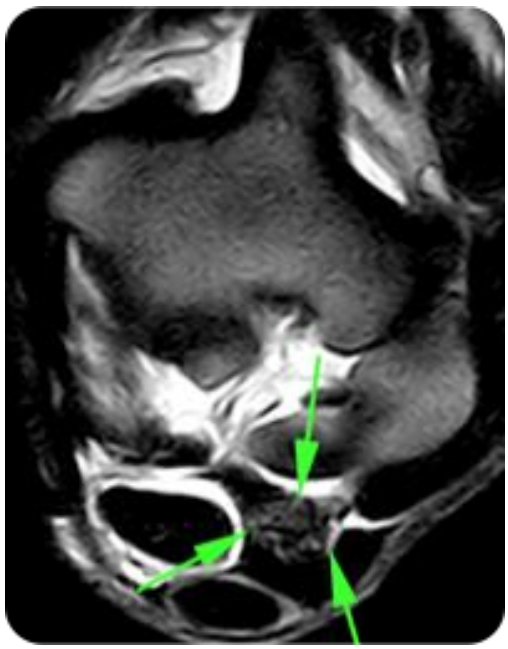


Figure 90. 8yo QH three day eventer with an intermittent lameness on the RH for the past several weeks. 2/5 lame day of MRI exam and very tender on palpation of the proximal suspensory ligament region. T2 axial tarsus, level of distal intertarsal joint. Moderate/marked heterogeneous increase in T2 signal within the long plantar ligament consistent with an active desmitis/desmopathy. The proximal suspensory ligament was completely unremarkable.

MRI of the front and hind proximal suspensory ligament is an area that is commonly requested. Some of these horses may have prior ultrasonic examinations with a prior diagnosis and are requesting a more thorough assessment of the lesions. Interestingly, as stated above, it is not uncommon to see on the MR exam changes that differ from the prior diagnosis. Lesions that are noted on ultrasound (thickened ligaments, mild/moderate chronic pathologies, even presumed core lesions) are not present; and conversely, an unremarkable ligament can be quite abnormal. These discrepancies have caused some practitioners to develop a distrust in their ultrasonic findings; this is arguably more prevalent in the hind limb than the front limb.

The horses that block to the region of the proximal suspensory ligament (as well as the carpus/tarsus) but have no visible morphologic pathologies of the suspensory ligament can have metacarpus/metatarsus bone injuries, distal intercarpal/intertarsal joint lesions, inferior check ligament desmopathies, palmar carpal ligament desmopathies, and long plantar ligament desmopathies. It is also quite common to see numerous lesions identified in combination, and for this reason it is suggested to include the mid carpus and tarsus in a study of the proximal suspensory ligament.

Certainly plenty of cases with ultrasonographic pathologies are indeed confirmed and the additional unknown lesions, where present, can be appreciated as well. Assessing the quality of the collagen matrix filling in the lesions of chronic and chronic-active proximal suspensory lesions is considered a very important component to the exam. Some chronic pathologies, have healed well with good to excellent quality collagen, appear completely quiescent, not associated with any notable adhesions, and are unlikely to be a source of lameness. However,

additional chronic lesions can be wrought with complications. This may include very poor quality healing with a chronic-active appearance, more acute appearing exacerbated tears, adhesions to the proximal splint bones, adhesions to the cannon bone, adhesions to the lateral plantar nerve (hind limb), grossly enlarged ligament with suspected compartmental syndrome, and bony insertional lesions. (Figures 77 a, b, 78 a, b, 79, 80 a, b, c, 81 a, b, 82 a, b, 83, 84 a, b, 85 a, b, c).

Additional tendons and ligaments of the carpus and tarsus are also readily assessed on MRI. Subcarpal pathology, namely palmar carpal ligament injuries, SDFT and DDFT lesions within the carpal canal, and the origin of the inferior check ligament are not uncommonly seen in the carpus. Other structures such as the medial and lateral collateral ligaments of the carpus/tarsus and the intercarpal/intertarsal ligaments and/or their insertions onto the cuboidal bones, can also be found to be abnormal in some horses. In the carpus for instance, active insertional desmitis and avulsion fractures of the lateral palmar intercarpal ligament on the ulnar carpal bone (palmar medial corner) are visible. In the tarsus, desmitis/desmopathy of the long plantar ligament is frequently present. Careful evaluation of all joints is likewise very important to determine the presence or absence of complicating bony and articular pathologies. (Figures 86, 78, 79, 82 a, b, c, 85, 87, 88, 89 a, b, 90).

One additional and a major consideration with the use of MRI is the ability to more fully evaluate additional injuries beyond those that are already known and/or diagnosed on other modalities such as ultrasound or radiography. For instance, if a horse is lame and blocks to the coffin joint with radiographic evidence of low ring bone, it is important to understand what additional injuries are also present beyond the pathologies in the coffin joint. The experience gained from MRI has revealed that the pathologies in the navicular apparatus and the collateral ligaments of the coffin joint, for example, are frequently found together with coffin joint injuries.

It is very important for the interpreting veterinarian to describe all pathologies seen, so that proper treatment can be administered. Clinicians with experience treating lame horses following a focused MRI examination report optimal outcomes when most of the noted abnormalities, from mild to marked/severe are treated. Even when a lesion is not believed to be a major contributor to the current lameness, particularly in light of additional, much more significant lesions, some clinicians opt to treat these subclinical lesions to prevent them from progressing to becoming more significant sources of lameness in the future. Examples include general degenerative pathologies of the coffin, pastern, and fetlock joints, and mild degenerative desmopathies of the collateral ligaments of the coffin joint. Furthermore, in the presence of numerous lesions, it is very difficult to accurately predict which ones are clinical and which one are subclinical.

In other cases with known trauma, such as SDFT wounds in the distal metacarpus, for example, a horse that is persistently lame and blocks to a low 4 point block, may well have additional injuries to the fetlock joint that may not be apparent following ultrasonographic and radiographic evaluation. Several months of treatment and rehabilitation focused specifically on the known SDFT injuries may not completely address all injuries. In cases where these horses do not improve following therapy which addressed the known injuries, a subsequent MRI evaluation often reveals numerous other significant pathologies. This experience brings new meaning to the term “treatment failure”, because it is often not a treatment failure per se, but an initial diagnostic failure which leads to longstanding lack of soundness.

Growing availability and use of regenerative therapies, in the author's opinion, are leading to improved matrix quality within lesions on follow-up examinations. As previously mentioned, the signal of optimal collagen matrix resembles normal tissue, and thus is typically hypointense on all pulse sequences. Ideally, there will also be minimal to no surrounding scar tissue or adhesions to surrounding soft tissue structures. More commonly however, if the lesion has healed, T2 signal will be completely hypointense and comparable to normal surrounding fibers, but PD and T1 signal often maintain a hyperintense matrix relative to normal surrounding tissues, suggesting a more immature form of collagen. Many lesions that break out the margin of a tendon or ligament are also surrounded by varying degrees of scar tissue with adhesions to surrounding structures. (Figure 63 a, b, c, 64 a, b, c, 77 a, b, 78, 82 a, b, 74, 85 a, b, c, 84, 51a-f)

The use of MRI for musculoskeletal applications in the horse has shown to be, like in human imaging, invaluable for accurate assessment of all notable injuries in a given joint/region. More commonly than not, experience reveals that most MRI examinations reveal multiple significant injuries, many of which cannot be seen radiographically or ultrasonographically. Improved diagnostics can lead to focused treatments, and thus, the potential for return to function can be optimized when all significant injuries are addressed.

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In: The Equine Tendon in Health and Disease
Editor: Henry Jann
ISBN: 978-1-62808-048-3

Chapter 3

TREATMENT OF ACUTE SOFT TISSUE INJURIES

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ABSTRACT

While the treatment of acute soft tissue injury is common in equine sports medicine, many different opinions exist as to the best approach to optimize healing. The divergence of treatment methods is the result of variation of injury, individual experience and conflicting scientific literature.

Previous attempts usually have focused on the merits of a specific modality. This chapter presents an outline of therapeutic approach that can be modified as future research illuminates merits and disproves assumptions of specific elements of the treatment program.

Current germane literature was reviewed and presented to demonstrate the current understanding and rationale for use in the treatment of acute tendon injury. The chapter provides an open approach for the treatment of the acute tendon while allowing individual preference for specific injuries which can later be modified as newer information becomes available.

INTRODUCTION

The treatment of acute tendon injuries can vary dramatically from clinician to clinician. Treatment is based on the experience of the veterinarian, the expectations of the client and the tolerance of the patient. Because of the dearth of specific validated therapeutic modality studies in peer reviewed journals, it is difficult for the attending veterinarian to determine the best treatment modality. The purpose of this chapter is not to propose the ideal treatment but rather to outline the elements of a treatment plan. These elements can then be modified by specific therapies as more information becomes elucidated by the advancement of veterinary science.

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Approach to acute injury should include the consideration of five elements:

1) Specific injury identification
2) Inflammation management
3) Regenerative medicine
4) Biomechanical optimization
5) Rehabilitation

Specific Injury Identification

In order for treatment to be rational, the nature of the injury must first be characterized. Specifically, two items must be addressed: first, what is the injured structure(s) and secondly, what type of injury has occurred. It is incumbent that the injured structure be accurately identified. As an example, swelling in the palmar aspect of the mid pastern could possibly be a tendonitis of the deep flexor tendon or a desmitis of the straight sesmoidian ligament. The biomechanical loading of these two structures is diametrically opposite in that the DDFT tension is reduced by heel elevation of the hoof capsule whereas tension of the SSL is increased. It is essential to identify the injured structure so that appropriate therapy can be applied.

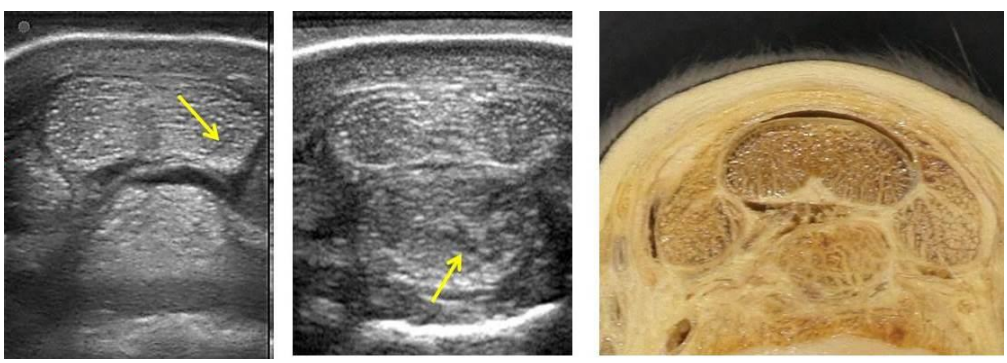

Figure 1. Example to show how a similar clinical presentation (swelling of palmar pastern) can be caused by two distinctly dissimilar structures.

left image>palmar ultrasound with fissure in dorsal aspect of lateral lobe of DDFT; center image>enlargement with heteroechogenic fiber pattern of SSL; right image>reference

Once the specific structure is identified, it is then important to characterize the type of injury. Often acute injuries are exacerbations of preexisting conditions. Treatment approach may be varied in accordance with the characterization of injury. Typically, soft tissue structures are injured by mechanical failure, inflammation and/or degeneration. As an example, a proximal enlargement of a superficial flexor tendon in a two year old Thoroughbred race horse in training and a 20 year old “school master” dressage horse may be caused by two quite different processes: the sudden overloading of a tendon in a two year old Thoroughbred and a degenerative tendon rupture in a warm blood dressage “school master”. These horses would be treated quite differently based on upon their work expectations.

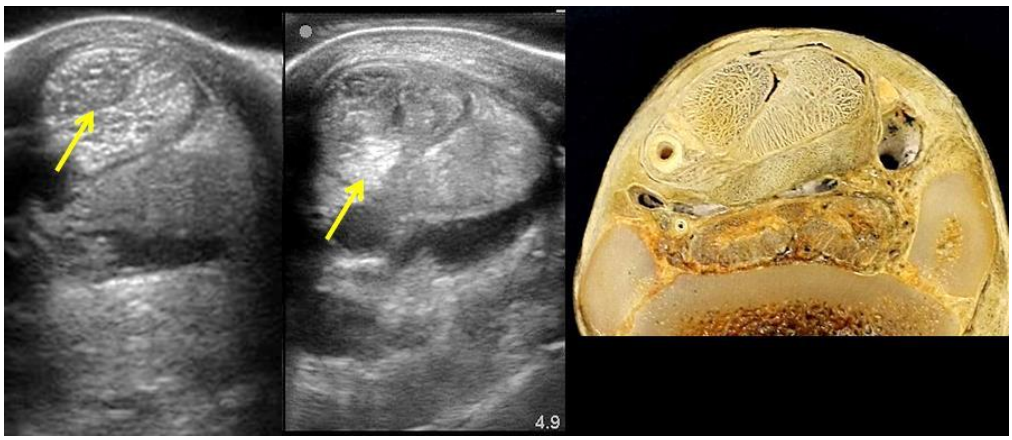

Figure 2. Example to show how enlargement of similar anatomic structures (superficial digital flexor tendon: 2 cm distal to carpal metacarpal joint) can be caused by two distinctly dissimilar processes. Left image>acute injury with palmar portion of tendon appearing mildly hypoechoic with a lack of ultra-architecture; center image>chronic active tendinosis with area of sclerosis (marked with arrow) with adjacent hypoechoic foci and overall loss of ultra-architecture; right image>reference.

Inflammation Management

Topical Therapy

The application of cold therapy has been the foundation of acute soft tissue injury management. Recent veterinary literature clearly documents the beneficial effects of tissue cooling in a variety of studies. These effects include down regulation of chemokines, proinflammatory cytokines, COX-2, MMP-2 and endothelial adhesion molecules. In addition, other cytologic effects noted were reduction in protease enzyme activity, reduction in metabolic energy requirements and reduction of delivery of hematogenous "trigger factors". Systemically, cryotherapy was documented to cause analgesia, hypometabolism and vasoconstriction. [1, 2, 3]

The difficulty arises in the application of this information in the injured horse. Our concerns are mirrored in human medicine where a review of the use of ice in the treatment of soft tissue injuries determined that few studies assessed the effectiveness of ice on soft tissue injuries and found no evidence of optimal mode or duration of treatment. [6] Frequently, the effect of cold was confounded by the concurrent application of compression, variation in degree of cooling and time after onset of treatment post-injury. At this time, there is no consensus on the mode, duration or frequency of cryotherapy in human medicine.

What can be gleaned from the literature regarding the use of cryotherapy for soft tissue injures is:

1) Topical application of cryotherapy significantly reduces core temperature of the SDFT. [4]
2) Topical application of cryotherapy is not detrimental to viability of tendon cells.[4]
3) Soft tissue temperatures did not elevate to normal for 2 hours after 60 minutes of treatment (30 minutes after 30 minutes of treatment). [4]
4) Compression facilitates cold therapy by reducing influx of blood. [4]

5) Pain reduction was noted within 10 minutes of application. This effect is optimized by intermittent application versus sustained application. [5]
6) A 10° C reduction of temperature reduces metabolic activity up to 50%. [5]
7) Ice boots and ice slurries were significantly more effective than gel boots. [1]
8) During cryotherapy post capillary venous filling pressures are reduced which facilitates clearance of local metabolic end products and reduces swelling. [5]

Using this information, it is then rational that cryotherapy probably has beneficial effect that should be implemented in the presence of inflammation for up to 5 days after injury. The cold should be in contact with the limb for at least ten minutes, preferably 60 minutes, at least four times and a day with the use of ice boots. We are gaining awareness that gel packs are probably not effective in attaining the needed reduction in tissue temperature. We are attempting to achieve at least a 10° C reduction in temperature which suggests that cold water hosing has minimal effect although actual studies have not been proven this statement.

In spite of this information, we as a profession face similar challenges as our human colleagues regarding the lack of evidence based guidelines in the treatment of acute soft tissue injures with cryotherapy. We simply do not know the optimal mode, time or frequency for cold administration. As time progresses, we need to recognize that current therapy is based on time worn experience that may indeed stand up to investigation but will almost certainly change as new information is uncovered.

In addition to inflammation reduction by cryotherapy, low level laser therapy (LLLT) has been advocated for the treatment of acute tendinopathy because of reported effects of decreased inflammation, increased angiogenesis and increased fibroblast activity. [9] At this point, multiple studies have supported the use of LLLT for various tendinopathies while an equal number have demonstrated weak or negative effects. Therefore, current literature is inconclusive regarding the usefulness of LLLT. Controversy arises regarding the dose of photon density to the affected tissue. Photon density is dependent on a number of variables; namely: wavelength, time, power density. These variables have not been correlated to adequate therapeutic doses for given depth of affected equine tissue. Further investigation will certainly provide more information in the future.

Other topical therapy of acute tendinopathies may include the application of topical medications to affected areas. Usually this includes a non-steroidal anti-inflammatory or a corticosteroid mixed with a vehicle that augments absorption into the affected area. Regarding NSAID ointments, it has been shown in humans that tissue levels at the area of application are approximately the same level as systemically administered medication with the advantage of producing plasma concentrations that are not associated with adverse effects. [7] However, in various animal models COX inhibitors have been shown to impair mechanical strength return following injury to bone, ligament and tendon even when use is limited to early stages of injury [8].

Literature search yields no information regarding the use of topical steroid mixtures for the treatment of soft tissue injuries. There are many references regarding the injection of steroids into the affected tissues with predominately a negative outcome regarding impact on healing but a positive response regarding improvement of comfort. It would be reasonable to assume that the application of NSAIDS and corticosteroid mixtures would decrease the inflammatory response of the tissue with the positive effect of improved comfort and decreased inflammatory mediated degradation. These positive effects must be weighed

against the potential negative effects of decreased mechanical strength. Certainly their use should be limited to the immediate inflammatory phase of injury (less than 5 days after injury).

Frequently, dimethyl sulfoxide (DMSO) is used as a sole ingredient or mixed with a corticosteroid to be applied to the area over the affect soft tissue. Initial studies were not blinded or well controlled but did suggest beneficial effects of improved comfort and anti-inflammatory effects. More recent double-blinded studies demonstrated inconsistent results although the topical use did appear to be safe. [13]

Regional Injection

Regional injection into the affected tissue has long been a treatment in acute tendinopathies. Most often intra or para-lesional injections of corticosteroids are performed to reduce inflammation associated with acute inflammation. The reduction of inflammation may limit further degradation which could reduce the extent and severity of injury facilitating a speedier return to work. However this relationship between inflammation abatement and earlier return to work is not clear. For example, in humans, treatment of medial epicondylitis with corticosteroids did demonstrate a short term improvement despite absence of histological evidence of inflammation. [10]

The most notable side effect of corticosteroid injection includes specific tendon/ligament weakening which may lead to rupture. Degenerative tendon changes have been noted after direct and peritendinous injections. It remains difficult to say if corticosteroids actually induced tendon degeneration or if the corticosteroids reduced discomfort and enabled overloading of previously compromised tissue inducing more acute trauma. Additionally, in metabolic horses, it may be prudent to consider that hyperglycemia has been shown to persist for up to 5 days in humans after a single injection. Specifically insulin resistant horses may be at more risk of developing laminitis.

Often corticosteroid, local anesthetic injections are mixed with Sarapin. Sarapin has been used for over 50 years. Its use has been supported by investigative work that demonstrated a reduction in C fiber (slow, chronic pain) transmission. The mechanism of action is still not identified although it is thought to be associated with the potentiation of the ammonium ion. Early studies suggested a potentiation of duration of effect. More recent studies [11, 12] have clearly demonstrated no improvement in pain tolerance or duration. It is conceivable that Sarapin may still be of some benefit in more chronic pain although evidence is scarce.

Anecdotally, DMSO has been added to mixtures of medications for local injection. Rationale for addition is that DMSO would augment the penetration of ingredients in to affected tissues while also providing a direct anti-inflammatory effect. Unfortunately, little information regarding safety, efficacy and dosage is available. Interestingly, one obscure study demonstrated benefit of intrasynovial DMSO when added to corticosteroid therapy. [14] Unfortunately, the lack of commercial interest and lack of availability of aseptic packaging will likely reduce further clinical investigation into its use.

Recently, the use of autologous biologic products has become popularized. The relative safety and unregulated restrictions has led to use that has outpaced scientific study. One such product, IRAP (autologous conditioned serum) has been anecdotally used to downregulate inflammatory process of acute tendonitis. Regimes regarding amounts and frequency have not

been published. Rationale for use is based on the anti-inflammatory effects of IRAP/ACS without concomitant risk of weakened repair associated with steroid or nonsteroidal anti-inflammatory therapy. Limiting factors at this time are related to lack of literature validation and procedural cost. While the action of IRAP/ACS is predominantly ani-inflammatory, other biologic products can act in more growth promoting manner.

Platelet rich plasma (PRP) as a biologic product is used to enhance tissue healing as a promoter of tissue growth. Various systems are commercially available. Optimal protocols regarding number of platelets and frequency of injection have not been established. Although the release of growth factors (PDGF and TGF-b) from the platelets seems to be the dominant mechanism of action, some research has described primary analgesic effects. [15] The rationale of use during acute tendon inflammation would be its inclusion into a new active injury site to augment recruitment of stem cells, provide tissue growth factors and possibly improve quality of healing. There are currently no guidelines with regard to the optimal time to administer the platelet therapy. As with IRAP/ACS usage, controlled blinded studies for PRP are lacking although its use is promising.

Interestingly, autologous mesenchymal stem cells have both the anti-inflammatory and trophoblastic effects. The use of MSC therapy has been predominantly popularized because of their trophoblastic effects no tissue repair and regrowth. However the anti-inflammatory effects through down regulation are well documented and may have a place in the acute management of tendonitis. The down regulation occurs because of effects on altering of antibody production by B lymphocytes, shifts in T lymphocytes and increased immune tolerance. [16] These effects include inhabitation of scar formation, inhibition of apoptosis, increased angiogenesis, and stimulation of intrinsic progenitor cells to regenerate function. It is known that activation of MSC is required for their benefits to be expressed. Unfortunately, the exact timing, dose and frequency of administration have not been adequately characterized to maximize the benefits of immune system modulation by MSC therapy.

Systemic Therapy

The treatment of acute tendonitis typically employs the use of systemic non-steroidal anti-inflammatory drugs. The rational is to provide pain relief and inflammation control. As described above, the presence of NSAIDS during the acute process does decrease the amount of inflammation but has been shown to significantly decrease the quality of healing/repair of bone, tendon and ligaments. [17, 18, 19, 20]

Literature is confusing as it includes various NSAID drugs, vitro and vivo models and various tendinopathy models. As an overview, it appears at this time, that we should strongly reconsider the use of NSAIDS if the injury involves a tendinous-bone junction and that the repair of tendon may be significantly decreased when NSAIDS are used during the acute phase (first 5 days) of healing. Certainly, future studies are indicated to better characterize NSAID impact on tendon healing.

In addition, systemic corticosteroids can be used to reduce inflammation. Advocates of such use reason that the benefits of reduced inflammation are more significant than the potential detrimental effects of delayed soft tissue healing. Literature typically reports significant deleterious effects on biomechanical properties on tendons while a few other studies report a lack of adverse effects. This suggests that the effects of corticosteroids on

tendons is complex and is influenced by drug type and dosage, route of administration and type of tendinopathy. Two more recent studies demonstrate that dexamethasone inhibits proliferation and collagen accumulation and migration of tenocytes. [21,22] These changes represent a significant compromise to the cellular repair process especially during the regenerative phase of healing.

Although clinical signs may show improvement with the administration of corticosteroids, there is minimal support for their use.

The use of intra-venous dimethyl sulfoxide (DMSO) has also been advocated for its ability to penetrate tissue and promote anti-inflammatory effects. Again, lack of research support has left its use to the realm of subjective use. Literature is antiquated and anecdotal. Of note is a paper written in 1982 by Albrechtsen and Harvey that demonstrated significant decrease in tendon strength for 7 days after topical application in mouse Achilles tendons. [23] The lack of quantifiable benefit with the possible detrimental effects leaves little support for the use of intravenous DMSO.

Recent developments in regenerative therapy with mesenchymal stem cells (MSC), has opened a door to an undeveloped therapeutic modality in the treatment of acute tendonitis. Systemic administration of MSC has been encouraging regarding the treatment of inflammatory conditions. [24, 25, 26, 27, 28] These conditions include cytotoxic and autoimmune based inflammatory diseases. Several characteristics were noted regarding treatment with stem cells:

1) MSC's "homed" to diseased tissues.
2) Suppressive effects require an activation step that occurs in the presence of an inflammatory environment which limits generalized immunosuppression.
3) MSC's were systemically administered intravenously or intra-peritoneally.
4) Clear objective evidence of attenuation of inflammatory response was identified.

However, several obstacles need to be overcome:

1) Cost of therapy
2) Lack of research based evidence in the horse
3) Inadequacy of an appropriate acute tendonitis model
4) Treatment protocols are not validated regarding dose and time intervals
5) Administration during acute phase of tendonitis requires immediately available cell cultures or adequate fresh harvesting of cells.

Evidence is mounting that the immuno-regulatory effect may be more profound and far reaching than the trophoblastic effect of stem cell therapy. It is this author's opinion that the future therapies of acute tendonitis will be based in systemic regenerative medicine.

Biomechanical Management

Although compressive forces are present in tendon structures at deviations of axes i.e. at joints, scuta, retinaculi; most tendon injuries are associated with excessive strain during elongation. The biomechanical management of acute tendonitis has two goals: first, to reduce

the amount of elongation and secondly to allow the continued cyclical loading of the injured structure during rehabilitation exercise. Both of these goals can be accomplished by alterations of foot capsule length (with or without wedge pads), application of asymmetric shoes and/or surface firmness.

Discussion regarding the direction and amount of alteration has typically led to controversy among farriers and veterinarians. However research papers are showing a cohesiveness regarding these alterations and their effects on loading of soft tissue structures. Clearly, the elevation of palmar angle (with wedge pads on firm surface or egg bar shoes on deformable footing) has been shown to decrease the loading of the DDFT while increased loading of the SDFT and SL structures. [29, 31, 32, 33, 34]

Conversely, the reduction of palmar angle (with elevation of toe by wedge or application of wide web at toe in deformable footing) has been shown to decrease the loading of the SDFT and SL while increasing the loading of the DDFT. [29, 31, 35]

On firm surfaces, alteration of palmar angle can simply be performed by the trimming of the hoof capsule given that has enough material and quality available or by the application of wedge pads. Both of these methods cause an immediate effect but this may only be temporary as the self-regulation of muscle tension will cause elongation/contraction of the associated muscle body to self-determined levels of tension.

On deformable surfaces alteration of palmar angle can be accomplished by the use of asymmetric shoes. This has the advantage of transient reduction of loading while moving on deformable footing during walking yet not inducing muscle contraction/elongation while standing on firm surfaces.

In figure 3 above the palmar angle is indicated by the red vectors. In figure 4, the application of an egg bar shoe shows a ~2° palmar angle elevation. In figure 5, the application of a reverse shoe shows a ~5° palmar angle elevation. While in figure 6, the application of a wide toe shoe shows a reduction a ~2° palmar angle reduction. It should be noted that egg bar and asymmetric shoes have limited effect on loading of soft tissue structures while on firm, non-deformable footing. [34, 35]

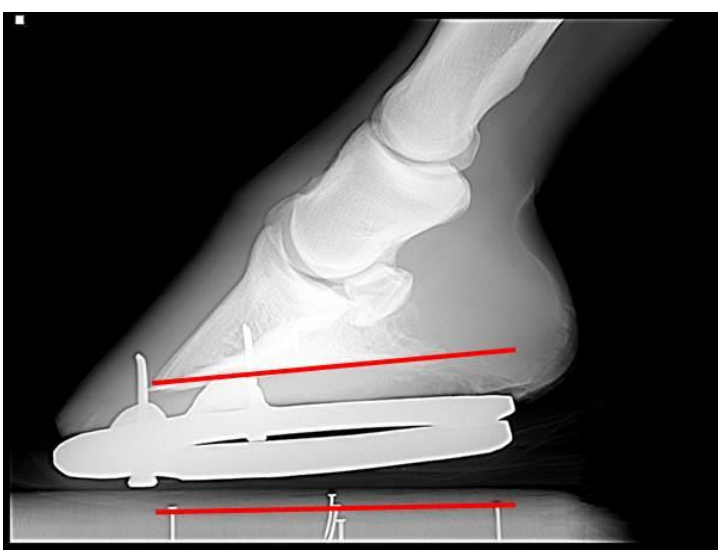

Figure 3. Palmar angle with standard shoe on deformable footing= 5.15°.

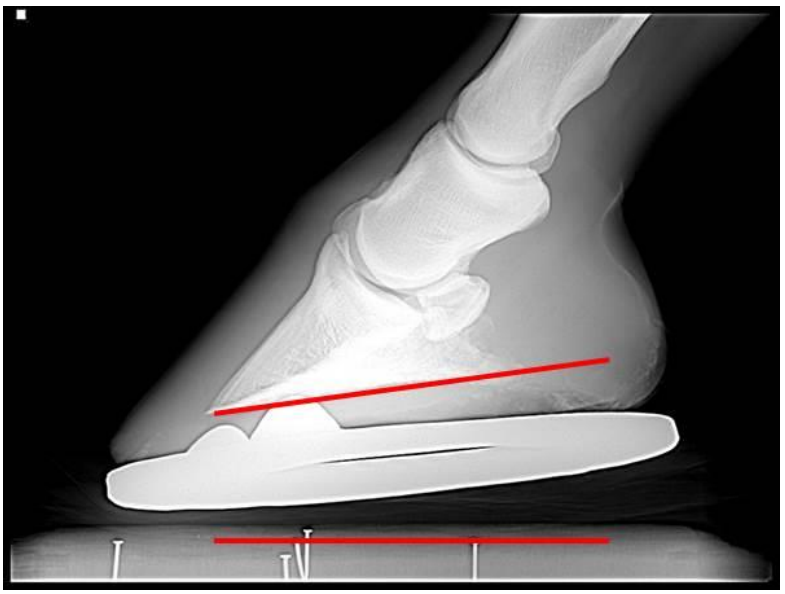

Figure 4. Palmar angle with egg bar shoe on deformable footing= 7.61° (increased 2.26°).

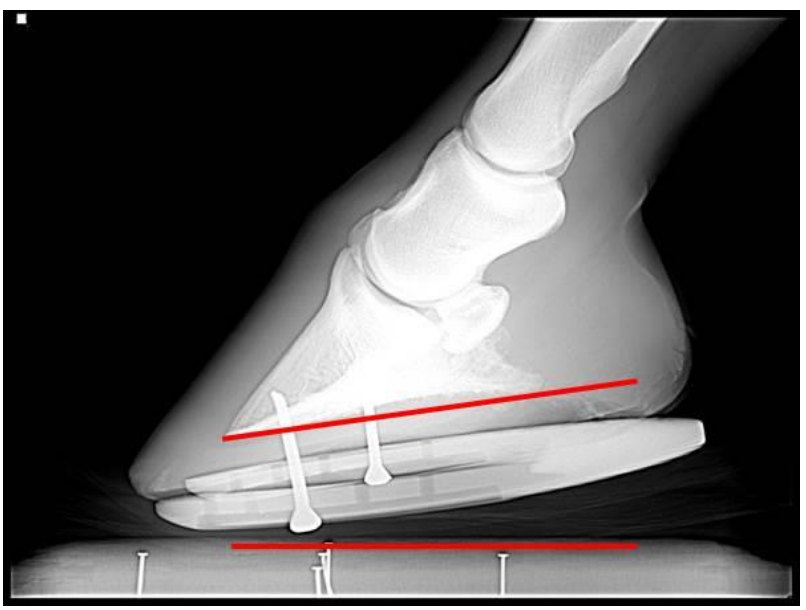

Figure 5. Palmar angle with reverse shoe on deformable footing= 10.02° (increased 4.87°).

In review: an increase in the palmar angle (angle between solar surface of P3 and standing surface) causes a reduction in the loading of the deep digital flexor tendon and accessory ligament of the DDFT while increasing the loading of the superficial flexor tendon and the suspensory ligament. Thus:

1) Injuries to the DDFT should have the heels elevated
2) Injuries to the ALDDFT should have the heels elevated
3) Injuries to the SDFT should have the heels lowered (or toe elevated)

4) Injuries to the SL should have the heels lowered (or toe elevated)
5) Injuries to the ALSDFT should have the heels lowered (or toe elevated)

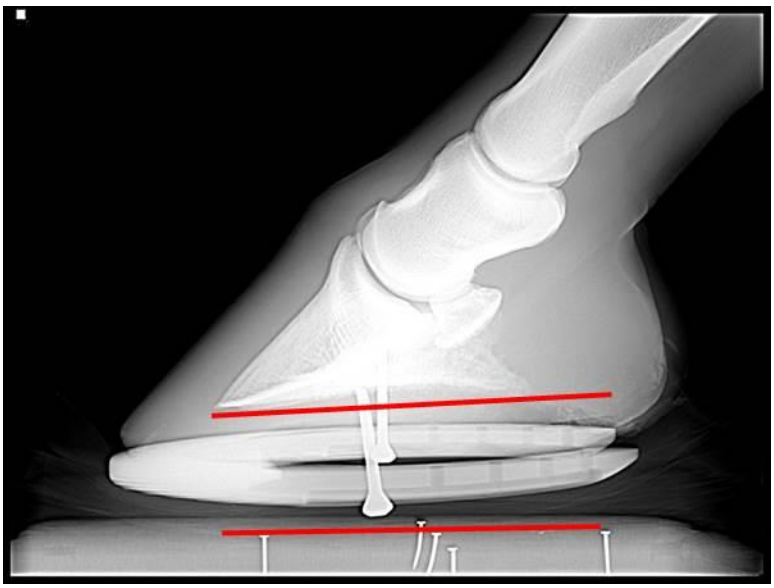

Figure 6. Palmar angle with wide toe shoe on deformable footing= 2.95° (decreased 2.20°).

As in all therapies, some degree of caution should be exercised as experimental data by LS Meershoek et al. showed that induced SDF tendonitis caused increased loading of the DDFT on the affected limb. [30] However, the contralateral limb showed increased loading of the SDFT as a compensation for lameness. This information suggests that when applying palmar angle alterations, that only the affected limb should be altered. During the acute phase, it would be prudent to only allow unilateral weight bearing on the non-affected limb until healing has progressed to a point where unilateral weight bearing is able to be supported by the injured structures of the affected limb. In addition, that alteration of palmar angle should be applied in a moderated manner. This author would suggest less than 6° alteration from normal. Collateral soft tissue structure injuries (collateral ligaments of joints, suspensory branch injuries, oblique sesmoidian ligaments, etc) may also benefit from similar manipulations of the hoof capsule although reviewed literature has not been published. For example, if reduction in tension (reduced elongation) of the lateral collateral ligament of the distal interphalangeal joint is desired then several options are available:

1) Reduce medial wall of hoof capsule
2) Apply a wedge to the lateral wall
3) Apply an asymmetric shoe (wide web shoe to the lateral wall with a narrow branch on the medial wall). This requires deformable footing to become effective. See figure 7.

Note: the amount of medial lateral manipulation is usually less than 2° with the use of asymmetric shoes when ratio of wide to narrow branch is around 2:1 (35:17 mm) in width.

The effect of alterations to the hoof capsule can profoundly affect the loading of specific structures. This underscores the importance of first obtaining a specific diagnosis as improper adjustments can exacerbate further injury. Alterations of the hoofcapsule should be initiated as soon as possible after specific injury identification and then progressively tapered back to a normal shoe during rehabilitation. For example, an injury to a DDFT at the level of the second phalanx would first be shod with a reverse shoe during the acute phase, then an onion shoe, followed by a "sport" shoe. See Figure 8.

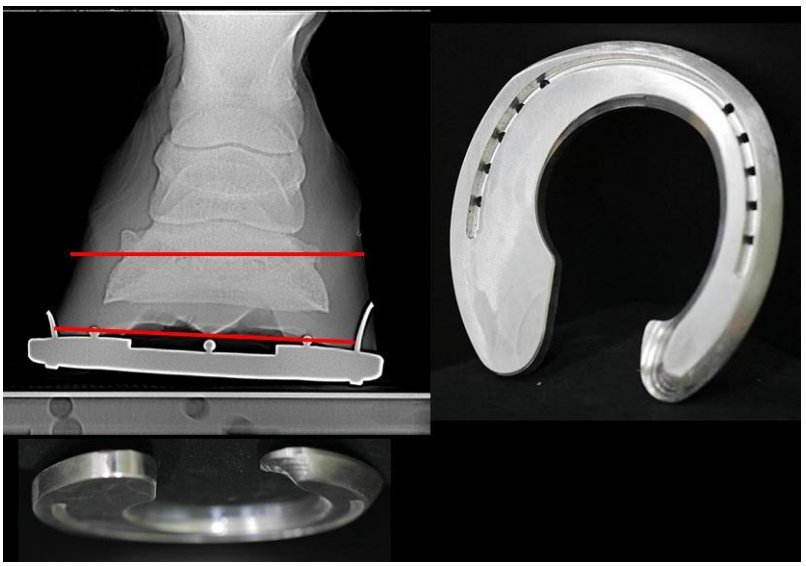

Figure 7. Alteration of medial lateral balance with application of asymmetric shoe on deformable surface. Wide branch supporting elevated wall of hoof capsule.

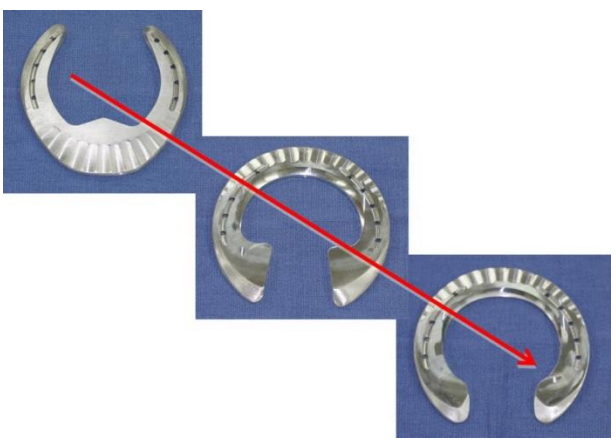

Figure 8. Progression of asymmetric shoes used for injury to DDFT from acute treatment (reverse shoe) to onion then a "sport" shoe at end of rehabilitation.

Fortunately, there are many different methods and combinations that can yield supportive results for acute injury. The goal is not necessarily to force the use of a particular shoe but rather to conceptualize the goal of reducing elongation of the injuried structure while allowing a movement based rehabilitation program.

Rehabilitation of Acute Injury

Healing of acute soft tissue injuries is optimized with the cyclic loading of the structure. As in human therapy where in the past, sprained ankles would be imbolized for several weeks, current standard of care maintains the range of motion and cyclical loading as can be tolerated by the patient. In horses, we attempt to provide the same care although we are sometimes presented with specifc behavioral challenges. Most horses will adapt to exercise restriction but occasionally some horses become unmanageable for the owner and a detriment for the injury repair. Sedation may be required to allow the horse productive rehabilitation. Frequently acepromazine or reserpine is used to help make the horse more agreeable. Alternatively, management changes like feeding the horse first, keeping a companion nearby, or keeping outside in a round pen can be helpful.

Certainly, unmonitored exercise is contraindicated. Increased soft tissue loading occurs as speed increases or jumps get higher. For this reason, we choose to keep horses in small pens and not simply get unsupervised turn out in a large pasture. Alternatively, box rest reduces quality of soft tissue healing and often leads to more neurotic behavior. In the acute stage exercise should be limited to hand walking. No real consensus exists regarding the optimal amount of exercise for given injuries. As a clinician, I strike a balance between perceived activity during the day in the small pen and frequent hand walking. If the horse is sedentary, frequent walks of 15 minute durations (6x per day) are recommended. If the horse is social and active, then two walks per day may be enough. It should be noted that this information is anecdotal but practical.

In addition, the area of exercise should be evaluated. The firmness of footing should be selected so as to reduce elongation of the injuried structure. Firm footing reduces loading of the SDFT and SL while soft footing reduces loading of the DDFT. The decision to use asymmetric shoes should be based on the recognition that they function only when work is perfomed on deformable footing. Egg bar shoes, although proported to enhance the stabilitiy of support have shown minimal effect on soft tissue structure loading while on firm surfaces but have shown good effect when on deformable surfaces. [34] The radius of required turns during walking should be considered. Turning elongates and strains collateral structures therefore collateral structure injury necessitates caution during turning and turning should only be performed in large radius turns. Finally, walking areas should be quiet and away from excitement.

CONCLUSION

Acute injury treatment begins with an accurate diagnosis. The following four aspects should be considered as part of early treatment: inflammation control, regenerative medicine, hoof balance optimization and rehabilitation exercise. At this time too many variables exist to give specific protocols for therapy. As time progresses more information will become available to give more specific information for specific injuries. For now we are forced to rely on our anecdotal experience as to how we can best manage the acute tendonitis case. The information presented here was intended to bring awareness and rationality to our decision making process.

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In: The Equine Tendon in Health and Disease
Editor: Henry Jann
ISBN: 978-1-62808-048-3

Chapter 4

STEM CELL THERAPY

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ABSTRACT

Horses have to be able to function at one hundred percent of their capabilities in order to perform in the highly competitive field of performance horses. When injuries occur, horses must be assisted back into health quickly and restored to normality. Regenerative medicine, new surgical techniques, new diagnostic imaging, therapeutic laser, acupuncture, shock wave therapy and a better understanding of rehabilitation has lessened convalescent time and improved the quality of healing. No longer are stall rest and anti-inflammatory agents the only treatment options. By no means, do the new therapies replace surgical intervention or all of the traditional therapies; many times the new therapies are used simultaneously or as a supplemental therapy.

Stem Cell therapy is probably the current "premium" regenerative medicine treatment. Shockwave therapy has been used for several years but has not really been fully understood. This chapter is an attempt to explain the major aspects of stem cell therapy.

Stem cells over the past several years have now become the center of attention for regenerative medicine in both the veterinary and human field. Stem cells can be obtained from many sources. Their origin can be embryonic, neonatal, or adult. Adult derived stem cells can be grown from almost anywhere in the body. The two most commonly used adult stem cells are adipose and bone marrow derived. Other sources such as skin, cardiac muscle, neuronal tissue, tendon, etc. have been used and researched. Cultured stem cells are used autologously primarily but have also been used allogeneicly. Xenogenic stem cells have been used successfully in research.

Embryonic stem cells are derived from the inner cell mass of preimplanted blastocysts. Embryonic stem cells are not used extensively at this time because of the difficulty in

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producing cells other than in research. Also there is a high incidence of teratomas associated with their use. Although they are truly a pluripotent cell; their use is impractical at this time.

Neonatal stem cells are derived from the blood of the umbilical cord after birth or from umbilical cord tissue mostly from the Wharton's jelly. Also the amnionic membrane can be used for obtaining stem cells. At this time, umbilical cord blood can be saved and cultured. The cells are stored for future use. In veterinary medicine, its use is still limited.

Adult stem cells are the most commonly used form. Cells can be harvested from many different tissues even from circulating blood, but the two most common areas are bone marrow and fat. At this time we will not discuss any differences in the two but bone marrow contains a high hematopoietic cell density and low stromal cell density. Bone marrow samples must be cultured in order to harvest mesenchymal stem cells (MSCs). Fat contains a high number of regenerative cells and can be used immediately after processing. The cells obtained from processing can also be cultured to further expand the MSC population. Adult MSCs are possibly the descendant of the pericyte [1, 2]. MSCs from fat are probably housed in a perivascular niche. The number of colony forming units of MSCs are linearly associated with the blood vessel density in the fat. [3] MSCs can be found in bone marrow in a small niche along with a number of nucleated cells types.

Induced pluripotent stem cells are obtained using any cell after detransforming their stemness. This may require the use of viral vectors or other methods. These types of stem cells are still in the research phase. At this time in the equine field they are not utilized.

WHAT ARE STEM CELLS?

In order for a cell to be a classified as a stem cell it has to have the ability to differentiate into one or more cell types, be plastic adherent, and propagate in vivo. Potency of the stem cell is the ability to differentiate and is classified as unipotent, multipotent, pluripotent, and totipotent. Unipotent cells differentiate in to one tissue cell type. Some do not classify this type as a true stem cell. Unipotent cells reside in tissue and are responsible for maintenance of that tissue. These cells are host cells or residing stem cells that have been influenced to differentiate to the tissue around it. For example, a skin wound heals and it is well known that epithelial cells will move to the wound margins along with fibroblasts, myofibroblasts, etc. and the wound will heal with skin. The multipotent cell can differentiate or give rise to a number of different cells or tissues and are defined as having the capability of developing into cells of 2 different germ layers. Most notable, the mesenchymal stem cell, which has the capacity to differentiate into many number of connective tissue and cell types. Multipotent cells have been isolated from many types of mesenchymal tissues in adult mammals, including hepatic [4], neural [5] hematopoietic [6], synovial membrane [7], fat [8], and skeletal muscle [9] tissues and the umbilical cord [10, 11].

Pluripotent cells can develop across all three germ layers and theoretically develop into a whole organism. The blastula or blastocyst would fit this classification. This definition has been mostly used for embryonic stem cells but with more recent research, the mesenchymal stem cell can also fit this classification [12]. The totipotent cell is capable of developing into any cell in the body including embryonic tissue and fetal tissue. This classification is more denoted to the zygote and early embryonic stage cells. Some research has suggested that the

cells on the periphery of the blastocyst, trophoblast cells, may also be able to fit this classification [13].

The phenotypic characterization of mesenchymal stem cells(MSC) have been defined in the human literature by the Mesenchymal and Tissue Stem Cell Committee of the International Society for Cellular Therapy with four minimum criteria: One, MSC when maintained in culture conditions have to adhere to plastic (plastic-adherence); two, MSC must have adipogenic, osteogenic, and chondrogenic differentiation ability; three, must express CD73, CD90, and CD105; four, must have negative cellular markers and genes for CD11b, CD14, CD19, CD34, CD45, CD79α, CD 80, CD 86, HLA-DR (human leukocyte antigen), and c-kit which are hematopoietic lineage markers(31). Some of the surface markers of designation may have changed, as CD29, CD 44, CD 49a-f, CD 51, CD 106, CD 166 and Stro 1 have also been indicated as markers for MSCs. The exact markers of equine MSCs have not yet been classified.

MSCs contain the following hematopoietic markers: (CD45, CD35, CD14, or CD11), leukocyte function antigen -1 (CD18), dendritic cell marker (CD80, CD86, and CD83), platelet/endothelial cell adhesion molecule, or neuronal cell adhesion molecule-1. They should express the vascular cell adhesion molecule (CD106), activated leukocyte cell adhesion molecule, intercellular adhesion molecule, and previously described cell surface markers. [14] Cell surfaces markers for MSCs vary depended on organ and species origin.

HOW STEM CELLS WORK

Homing/Engraftment

MSCs in vivo can be found within tissue blood. Hematopoietic stem cells from bone marrow are examples of circulating MSCs. During repair, MSCs are recruited to the site of injury in a process called homing. After arrival to the area, MSCs invade and incorporate into normal and abnormal tissue in a process called engraftment. The environment of the tissue in which the stem cells engraft controls the development and differentiation of the stem cell by contact with host cells, triggered by growth factors ,interleukins, oxygen content, pH, inflammatory factors and cells, etc. This was demonstrated when a surgically created lesion in the superficial digital flexor tendon of a horse was injected with labeled MSCs. The study found labeled MSCs in the lesion and surrounding healthy tissue. The labeled MSCs that had engrafted into the tendon crimp demonstrated tenocyte morphology [15].

Chemokines are involved in many biological processes such as white blood cell movement, angiogenesis, organ development and hematopoiesis. Chemokines control the transendothelial migration of leukocytes. Once a leukocyte is across the blood vessel and into the tissue, the variant gradation of chemokine concentration becomes a cell's polarity, thus controlling its destination. It is probable that MSC's retention, mobilization, homing and engraftment are controlled in the same manner as the leukocyte by using chemokine receptors and its ligand. There have been many reports of the amount of MSCs containing different families of chemokines [14]. Chemokine receptors of MSCs can change when exposed to TNFα [16]. This change in chemokine receptors is a probable way in which MSCs change in order to migrate to different areas of the body. A change in receptor is needed to migrate into

areas of ischemia, increased inflammation or different areas of the body such as intestines or neural tissue. It is believed that the type of chemokine receptors may play a role in the quality of the MSCs engraftment and homing capability. Adipose derived MSC (AD-MSC) have more mRNA expression of CCR1, CXCR4, CCR7, CXCR6, CXCR3 than bone marrow derived MSC (BM-MSC). Since chemokine receptors play a role in cell migration it was concluded in this study that human AD-MSC are better for transplantation then human BM-MSCs [17].

MSCs also have further similarities to leukocytes. Molecules that are involved in the rolling, tethering, adhesion and membrane migration of leukocytes from the blood stream into tissues are expressed on MSCs. Integrins, selectins, chemokine receptors, L-selectin (leukocyte rolling molecule), P-selectin (adhesion molecule) and VCAM-1 (vascular cell adhesion molecule) are expressed by MSCs[14].

Human MSCs cultured in a hypoxic environment show increased targeting towards wound healing compared to those MSCs grown in a normoxic environment. The MSCs grown in the hypoxic environment also had increased migration toward hepatocyte growth factor (HGF), stromal cell derived factor 1α (SDF 1α), interluekins, and tumor necrosis factor α(TNFα) compared to MSCs grown in normoxic environment. The Hypoxic MSCs were also more responsive to epidermal growth factor(EGF), fibroblastic growth factor (FGF),vascular endothelial growth factor (VEGF), platelet derived growth factor (PDGF) along with an increased expression of CX3CR1 and CXCR4 chemokine receptor RhoA, hypoxic inducible factor (HIF-1α and HIF1β) [18]. RhoA is a signaling cascade that is possibly responsible for some of the migration of MSCs. HIF is a factor responsible for oxygen homeostasis and may control the increase of RhoA. These changes in the MSCs response are another example of how MSCs change in-order to migrate to the areas of need. Other possible mechanisms for the homing capability may be controlled by stem cell factor (SCF) which binds to c-Kit. Stem cell factor can be found in fibroblasts and endothelial cells. The proposed controls are involved with many different pathways, notch signaling, and growth factors which are too involved to discuss in this chapter.

MSCs not only engraft into an area but secrete growth factors. The adipose- derived stem cells secrete vascular endothelial growth factor (VEGF, hepatocyte growth factor (HGF), and insulin-like growth factor 1 (IGF-1) [19]. These factors along with HGF, SDF-1, thrombopoietin, placental growth factor, FGF-2, TGF-β and angiopoietin-1 are proangiogenic regarding vessel incorporation, neovascularization and vessel like formation [20, 21, 22, 23, and 24]. MSC's exposure to hypoxia, fibroblast growth, epidermal growth factor, tumor necrosis factor-α can increase the secretion of VEGF, HGF, and IGF-1 [23, 25, and 26]. Paracrine and autocrine function of bioactive factors produced by MSCs have profound effects on surrounding cellular function. The release of these growth factors and cytokines effect cells in their vicinity by protecting against ischemic necrosis, maintaining hematopoiesis, and support stem cell cypts, etc.

Trophic

Endosomes are secreted vesicles that have membranes enriched with cholesterol sphingomylin and ceramid, phosphatidylserine, and lipids. They contain protein and RNA. Endosomes function in intercellular communication and as a carrier vehicle. They are

secreted by many different cells including MSCs. Endosomes are partially responsible for the trophic effect of the MSCs. Endosomes from human embryonic stem cell derived MSC grown in conditioned medium were responsible for the reduced infarct size in a mouse model of myocardial ischemial reperfusion injury [27]. Even though only few transplanted MSCs engrafted and survived, and 6% or less of the transplanted MSCs remained in the heart after two weeks and poorly differentiated into cardiomyocytes, cardiac function improved [20]. This protection is proposed to be through the paracrine function of the MSC. MSCs have trophic effects on cells in their vicinity resulting from secreted molecules that can either directly effect by intercellular signaling or by indirect effect causing other cells in the vicinity to secrete the active agent. This indirect effect is trophic and causes up regulation of surrounding host cells, inhibiting scar formation, inhibiting apoptosis, increasing angiogenesis, stimulating intrinsic progenitor cells to regenerate host tissue and pathways [28].

Differentiation

Mesenchymal stem cells can differentiate into ectogenic, endogenic, and mesogenic cell lines that include skin, liver, neural and many other tissues (Figure 1). The exact mechanism is not known yet but it may involve the Wnt pathway. Wnt, a class of secreted morphogenic ligands, are important in establishing a pattern of development in multicellular organisms.

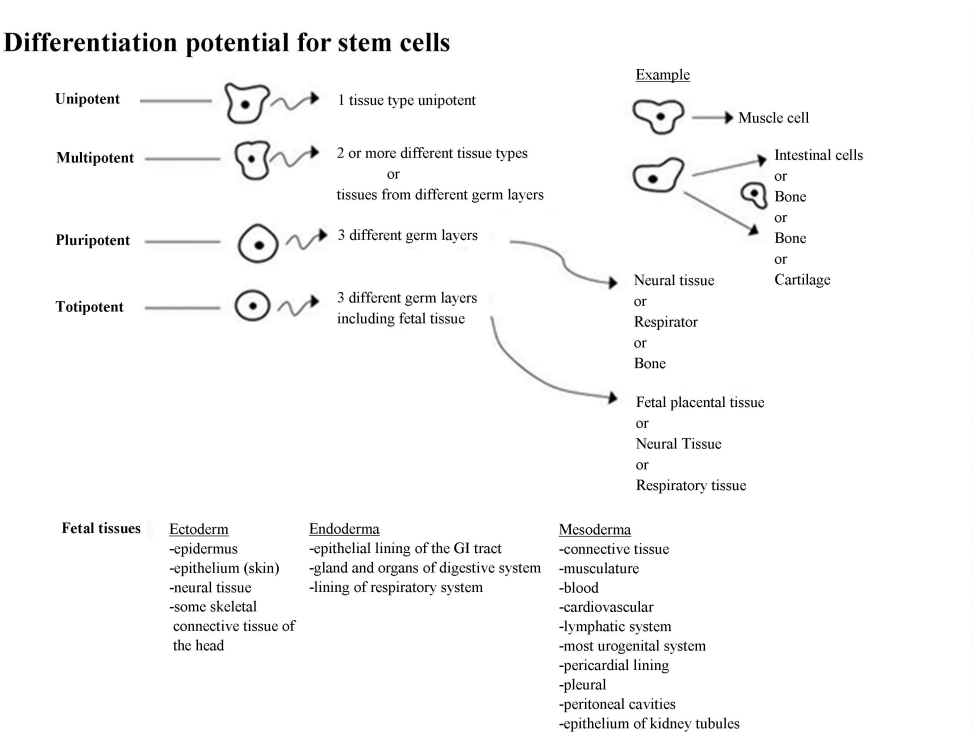

Figure 1. Mesenchymal stem cells can differentiate into ectogenic, endogenic, and mesogenic cell lines that includes skin, liver, neural and many other tissues. Image courtesy of Nova Science Publishers Inc.

The Wnt pathway involves binding of Wnt proteins to surface receptors of cells. These are mainly frizzled family receptors, causing activation of disheveled family proteins and changing the amount of β-catenin that interacts with the cell's nucleus. The Wnt pathway has been shown to induce murine embryonic stem cells into mesoderm and endoderm potent cells, with a high potential for bone and cartilage differentiation [29]. In a human embryonic stem cell study Wnt signaling induced hematoendothelial cell development. Wnt3 influenced more mesogenic committed cells with hematopoietic destiny. Wnt 1 stimulation was associated with nervous system cell regeneration [30, 31].

Immunosuppressive, Anti-inflammatory and Analgesic

MSCs have immunosuppressive properties in vitro and in vivo thus they are said to have a tolerogenic effect on graft acceptance. This effect has been under extensive study in the human field. In a murine model a heart was transplanted from the donor to the recipient abdominal region and attached to the aorta. The mice that received donor or recipient MSCs prior to transplant did not reject the heart. The mice that received MSCs prior to the transplant had a high acceptance rate but those that received MSCs post transplantation had a low acceptance rate. Those that received no MSCs did not accept the graft. Immunosuppressive properties were associated with the expansion of regulatory T cell [32]. Immunosuppressive properties were strongest in this study when MSCs were given via the portal vein resulting in engraftment of the MSCs to the liver and possibly interacting with the sinusoidal endothelial cells or the sinusoidal macrophages either by indirect or cell to cell contact.

IL-6 and PGE2 have been shown to play a key role in immunosuppression. IL-6 secretion regulates Cox-2 and iNOs activities. IL-6 is a cytokine that is both pro and anti-inflammatory depending on its environment. In an arthritis model, MSCs controlled inflammation through IL-6 dependent PGE2 secretion. PGE2 was found to impair the maturation of dendritic cells shifting Tcell to a more TH2 immunoresponse. PGE2 also increases macrophage secretion of IL-10 [33].

MSCs also have been reported to regulate immunosuppression by soluble factors such as hepatocyte growth factor and transforming growth factor-β [32]. MSCs have been reported to inhibit B cell proliferation and differentiation [34]. MSCs can secrete soluble factors in the presence and activation of IL-1β and interferon γ (35). MSCs can initiate apoptosis of activated T lymphocytes via indoleomine 2, 3 – dioxygenase (IDO). MSC expresses IDO and in the presence of Interferon γ, tryptophan is changed to kynurenine. Tryptophan is needed by lymphocytes to proliferate. MSCs also possess inducible nitric oxide synthase which inhibits T cell proliferation via stat-5 phosphorylation. MSCs have shown to express both Cox-1 and Cox-2 that influences synthesis of PGE2 and proliferation of T cells [36, 37]. MSCs depress phenotype maturation of the dendritic cell thus decreasing the capacity for it to stimulate lymphocyte proliferation and decreases interferon γ, tumor necrosis factor α, and interleukin-2 [38].

In a orophargyngeal aspiration study, MSCs were given four hours prior to lipopolysaccharide administration. In those rodents given the MSCs, there was a decrease in leukocyte migration into the alveoli, suppressed expression of proinflammatory cytokines and an increase in IL-10 [39]. Il-10 is an anti-inflammatory cytokine from the MSC. In a rodent model of hypersensitivity of sciatic pain, MSCs therapy down regulated sciatic nerve levels of

cytokine IL-1β, reduced pain response and increased IL-10 in the sciatic nerve. IL-10 plays a role in in anti- inflammatory and analgesic response of the MSCs [40].

MSCs could help to regulate many immune related problems in the horse by their down regulation of many complexes, such as, human leukocyte antigens-DR. This complex has been found to be associated with many diseases in humans such as rheumatoid arthritis, Crohn's disease, heart disease, hepatitis, sarcoidosis, narcolepsy and uveitis. Intravenous infusion of MSCs has decreased production of interferon γ (IFN-γ) and tumor necrosis factor [41]. IFN-γ is released by antigen-stimulated T helper lymphocytes, which are involved in immune responses. IFN-γ binds to a cell surface receptor protein inducing gene expression, thus producing an antiviral state to the cell making it less susceptible to infection of the virus. It does this by inducing phosphorylation of Stat1αs helping this protein stimulate transcription, hence in a viral infection it may abrogate the body's ability to respond to the infection. It could also be used to regulate the destructive events in a mass immune response. Tumor necrosis factor is a cytokine that is involved in systemic inflammation and can stimulate the acute phase reaction. TNF-α is produced mainly by macrophages but is also produced by many other cells. Large amounts are released in response to interleukin-1, lipopolysaccharide, and other bacterial products. It can cause problems in the hypothalamus, liver, and increase insulin resistance. Also high concentrations can cause shock-like symptoms, cachexia, apoptosis, up regulate collagenase, etc. So in the horse MSCs may be useful for endotoxemic events, cachexia, enteritis, arthritis, and insulin resistance syndrome. MSCs have an immunosuppressive property whether given locally or intravenously. MSCs are triggered by inflammatory factors, and then release soluble factors that will down regulate the immune response. MSCs are dose dependent. MSCs also have strong angiogenic factors that are triggered by inflammation and hypoxic areas which could be useful in areas of ischemic necrosis and post colic surgery.

HOW TO HARVEST SAMPLES

I will not discuss how to harvest embryonic, fetal or peripheral blood for stem cell production because bone marrow-mesenchymal stem cells (BM-MSC) and adipose derived-mesenchymal stem cells (Ad-MSC)are the most commonly used stem cell lines in horses, dogs, and cats at this time. One of the differences between BM-MSC and Ad-MSC is the turn- around time for treatment. The regenerative cells from fat can be received in 48 hours which is in the stromal vascular fraction. The time for BM- MSCs is 3-6 weeks. The stromal vascular fraction can be used primarily or be cultured to produce only MSCs. The stromal vascular fraction and AD-MSCs have differences but will be referred to as AD-MSC.

Fat Harvest

There are two ways to obtain fat, liposuction or surgical extraction/excision. Liposuction is the most cosmetic but requires specialized equipment. Surgical excision is inexpensive and no specialized equipment is needed. Surgical excision can also be cosmetic after the incision heals. There are several areas on the horse that fat can be removed. The abdomen, inguinal,

and stifle region fat can be used. Harvesting from these areas is very cosmetic but requires general anesthesia. The most readily accessible area for a standing procedure is in the rump. Ultrasonic examination is the best way to find the largest area of fat deposition.

The horse should be restrained in stocks. The environment should be free from dust and dirt. Use the 7.5 mHz ultrasound probe to find the largest area of fat deposition. This is usually four to five inches to either side of the dorsal midline between biceps femoris muscle and the semitendinosis muscle. The area is clipped and shaved with a razor. The horse is sedated. The area is surgically prepared using 4% chlorahexidine gluconate scrub and alcohol. Local anesthesia is performed using 2% mepivicaine subcutaneously. Local anesthesia should include a three inch margin circumscribing the intended incisional area and the incision line. In some cases intradermal infusion of local anesthesia is necessary. An additional sterile preparation using chlorahexidine gluconate and alcohol is performed. The surgeon should use sterile gloves and a gown and sterile draping technique.

A three centimeter incision is made through the skin over the area of the largest fat deposition. A curved Metzenbaum scissors is used to undermine the skin from the fat. A small incision is made through the fat and then the Metzenbaum scissors is used to undermine the fat from the fascia and muscle. The fat is then cut or dissected free using the Metzenbaum scissors or digitally. A Guyon kidney pedicle clamp forceps is used to extract the fat. Fat is then placed in the vials that are provided by a stem cell company. Once the desired amount of fat is removed the incision is closed. A simple continuous pattern using 2-0 polydioxanone suture is used to close the subcutaneous tissue. Then a continuous intradermal suture or simple interrupted sutures can be used to close the skin. 2-0 polydioxanone sutures are used in either closure. The drapes are removed and the incisional areas are washed with sterile saline. 1% silver sulfadiazine cream is applied to the incisions and 1% diclofenac sodium topical anti-inflammatory cream (5 inch ribbon Q 24hr topically) is applied around the incision.

The horse is given sulfamethoxazole/trimethoprim antibiotics (24mg/kg q 12 hr, PO) for seven days and firocoxib (0.1mg/kg q 24hr, PO) is given for seven days for pain management. The incision is cleaned daily and silver sulfadiazine cream is applied. 1% diclofenac is applied around the incisions daily for 5 days. Sutures are removed at twelve to fourteen days. Medical hydrolysate of type I collagen gel is applied daily for 5 days after suture removal.

The fat pad is microdissected, washed with PBS, and incubated in an enzyme for 30-60 minutes at 37°C with slight agitation. Then the collagenase is neutralized with DMEM-lg containing 10% fetal bovine serum. This fatty solution is then centrifuged at 1200g for 10 minutes and then resuspended with DMEM/lg and filtered through a 100μm nylon mesh. The solution is then centrifuged, decanted and the supernatant is recentrifuged and the erythrocytes are removed from the pellet with erythrocyte lysis buffer and washed in PBS. The end product is the stromal vascular fraction (SVF). This process takes about one to two hours. The SVF can then be plated, cultivated, and cultured or used as an injection for horses, dogs, or cats. The turn-around time is 48 hours with shipping if it is sent to a laboratory. There are two companies that process AD-MSCs in the USA. These are Vet Stem (Vet-Stem, Poway, California) and StemLogix in Florida.

The fittest horses have very little fat but usually have high numbers of stem and regenerative cells. There is usually only enough for initial treatment. To combat this problem Vet-Stem Regenerative Veterinary Medicine® now has a straight to culture program that only requires a grape size piece of fat. When using this program, a one cm incision is made. A Ferris Smith rongeurs is guided with ultrasound to extract the fat. The skin is closed and the

incision management is the same. The disadvantage of this program is that two weeks are required for the adipose derived stem and regenerative cells to be returned. Four doses are guaranteed.

We have removed adipose from the rump in hundreds of horses and only a few horses did not have a cosmetic outcome. Those horses developed a dimple where the fat was removed. The muscle tissue may have been disrupted during fat extraction and the skin adhered to the muscle tissue. This defect is easily repaired by injecting saline under the skin if recognized early enough or by making a small incision and dissecting the adhesion. Most incisions were not apparent or barely detectable by the time the horse was showing. The advantage of fat derived stem cells is that the horse can be treated quickly with in the first few hours to weeks of healing rather than waiting for several weeks for bone marrow cultured stem cells.

Bone Marrow Harvest

Mesenchymal stem cells (MSC) that are isolated from bone marrow are referred to as BM-MSCs. BM-MSCs or hematopoietic stem cells (HSC) when in the marrow are found along with hematopoietic progenitor cells adhered to stromal cells (fibroblasts and osteoblasts.) This is called a niche. The hematopoietic stem cells remain in the niche by adhering to extracellular matrix proteins and to stromal cells. Stem Cell Factor (SCF) is thought to control the placement of these cells. Some HSCs leave the bone marrow and circulate through the body but then if not used, return to the niche. Bone marrow has to be cultured because bone marrow contains low numbers of MSC and culturing removes or lowers the numbers of hematopoietic cells. There have been reports of bone marrow samples with no MSCs, requiring another sample to be harvested.

The horse is sedated and the area is surgically prepared using 4% chlorahexidine gluconate scrub and alcohol. Bone marrow is easiest to harvest from the manubrium sterni or the tubera coxa. We will normally use the manubrium in the standing horse. Local anesthesia is performed using 2% mepivicaine subcutaneously and on the manubrium sterni. The sternal region just between or just caudal to the front limb is prepared for surgery. The manubrium sterni is easily palpated in and the manubrium is usually widest in this area. A #15 surgical blade is used to make a stab incision. Then a bone marrow biopsy needle is insered into the bone approximately 1 to 2 cm. The biopsy needle has to be rotated in half circles to get through the bone. Care must be taken not to go to deep and through the manubrium because it will cause a pneumothorax or puncture the heart. Bone marrow will not immediately drain from the needle so a 12 cc syringe is used to aspirate. Bone marrow is transferred into a 30ml syringe containing 5ml heparin (1000 IU/ml) diluted with phosphate buffered saline (PBS). The needle is then removed and direct pressure is applied with a sterile gauze sponge for five minutes. Then use 35w stainless steel skin staples to close the stab incision. Apply topical antiseptic ointment of choice. The horse is given sulfamethoxazole/trimethoprim antibiotics (24mg/kg q 12 hr, PO) for seven days and firocoxib (0.1mg/kg q 24hr, PO) for seven days for pain management. The incision is cleaned daily and silver sulfadiazine cream is applied. 1% diclofenac is applied around the incisions daily for 5 days. Staples are removed at twelve to fourteen days.

Once the cells reach the laboratory they are processed to reduce cellular contaminates and to isolate only MSCs. Thirty milliliters of bone marrow is diluted with 15ml of PBS and

centrifuged at 300g for 15 minutes. The supernatant is removed and the pellet resuspended in PBS and centrifuged again. The cells in the pellet (at the bottom of a conical tube) are resuspended in 12 ml of Dulbecco's modified Eagle medium-low glucose(DMEM/lg) with 10% fetal bovine serum, 300µg of L-glutamine/ml, 1mmol sodium pyruvate/ml, and antibiotics usually sodium penicillin and streptomycin sulfate (100u and 100µg/ml respectively. The suspension is placed in a flask and left in incubation at 37°C at 90% humidity and 5% carbon dioxide atmosphere for 5 days. This will give the cells time to attach to the polystyrene surface of the flask. The media is replaced with fresh medium until cells reach subconfluency. The monolayers of MSCs are then treated with trypsin and washed again with (DMEM) and replated with medium. This medium is sometimes supplemented with triggering factors to change the commitment of the cells. At this time there are multiple laboratories culturing bone marrow. In recent years, in hospital laboratory kits have become available. I have not yet used any of them. I cannot comment on stem cell quality but they may facilitate early injection of a lesion in order to stop the inflammatory reaction, thus reducing healing time. BM-MSC and AD-MSC are the most commonly used stem cell lines in horses, dogs, and cats at this time. The big difference is turn-around time for treatment. The stromal vascular fraction (SVF) can be achieved in a few hours to 48 hours from fat and the BM- MSCs takes about 3-6 weeks. The SVF contains about 43% stem cells and other regenerative products. BM contains about .002% stem cells per cells collected. A human study "Comparative analysis of Mesenchymal Stem Cells from Bone Marrow, Umbilical cord, or Adipose Tissue" found the colony forming unit frequency was highest in the AD-MSCs and lowest in the umbilical cord blood (UCB) but the UCB could be cultured the longest and had the highest capacity for proliferation. The study also cultured for the ability of the cells to have mesodermal differentiation. The UCB-MSCs had the highest percentage for osteogenic differentiation capacity and BM-MSCs had the lowest. Both AD-MSCs and BM-MSCs had high percentage of adipogenic differentiation capacity but UCB-MSCs had none. All had good chondrogenic differentiation capacity. Adipose tissue in this study contained the highest frequency of MSCs. In another study the adipose tissue around the synovium showed a higher chondrogenic differentiation capacity then subcutaneous adipose tissue. Synovial adipose tissue derived MSCs also had a higher proliferation rate and higher cartilage matrix production [42]. Both AD-MSCs and BM-MSCs have been effective but I prefer to use the AD-MSCs because of the fast turn-around time. The true differences between adipose derived MSC and bone marrow derived MSC are still confusing and somewhat controversial. Studies have reported conflicting results so both types of stem cells will be used interchangeably in the remainder of this chapter. Both ASC and BM-MSCs have been effective but I prefer to use the AD-MSCs because of the fast turn-around time. It should be noted that unprocessed bone marrow aspirates contain hematopoietic cells and some growth factors rather than stem cells.

Clinical Application

Stem cell therapy in my opinion should be started acutely in order to reduce the inflammatory stage of wound healing (the first 3-10 days after injury). At 10 to 50 days the healing stage is present. Reducing the inflammatory stage of wound healing in theory would send the injury into the healing stage. Reducing the inflammatory reaction can reduce further

injury to the surrounding tissue and lessen the chance of scarring. Even in chronic wounds we have found that the size and the amount of scarring decreased with MSC treatment.

Tendons gain strength after grafting between the 3-6 weeks. Grafts go through an acellular phase around 7-14 days prior to being recellularized. The acellular phase may be due to decreased vascularization of the graft causing cell death or from the inflammatory response. The optimum time to inject MSC is prior to the acellular phase to try to cause neovascularization. Frequently, we don't see the acute part of the injury because the owners have first started stall rest, analgesics, and other therapy prior to getting an accurate diagnosis. By the time we diagnosis and find the lesion and get the harvesting procedure completed, we are usually well into the healing stage. When used for joint therapy post arthroscopy, we wait ten days before we harvest fat for stem cells. We used to inject at around 10 days postop. This waiting time was to make sure we were not going to get a joint infection, horses are off antibiotics, and they are not as painful. If a joint infection is present, lavage is performed immediately and stem cell therapy will wait until arthrocentesis and cytology indicate it has resolved. This avoids the possibility of lavaging the cells out along with the infection and since there is a tolerogenic property to stem cells, they may inhibit the immunocapability of the joint in the face of infection. Now we inject AD- MSCs as soon as possible after surgery. If we have to delay this process, many times we inject the joints with platelet rich plasma, hyaluronic acid or IRAP during surgery in order to reduce the inflammatory process. It has been found that in vitro, joint fluid from an osteoarthritic joint will reduce the viability of the MSCs [43]. In theory, after arthroscopy when the joint has just been flushed, it is the optimum time for MSCs viability. With the future of in house adipose processing and in house allogenic cells, it will be interesting to see if intraoperative injections of arthritic joints will improve therapeutic results.

Even if soft tissue injuries and joint injuries have been chronic, stem cells can still be used. We have had several cases of chronic deep digital flexor tendon injuries usually in the navicular bursa and tendon sheath regions that we have injected with good to excellent clinical results. Chronic proximal suspensory injuries that have had less than ideal results from other therapies have had good to excellent results with MSC treatment. We have also successfully treated chronic desmopathies of the distal sesamoidean ligaments, collateral ligaments of the distal interphangeal joint and metacarpal/metatarsophangeal joint, accessory ligaments of the deep digital flexor tendon, and meniscal tears.

Joint lesions such as denuded cartilage and degenerative joint disease appear to respond well to stem cell therapy. The use of either BM-MSCs or AD-MSCs does not appear to make a difference in the outcome. Incidences of reactions to the BM-MSCs injections are far greater than those of AD-MSCs. Reactions could be from the incomplete final wash of the bovine serum in the culturing process. MSCs have the potential for cartilage and bone repair. Many clinicians advocate the use of platelet rich plasma along with MSC therapy. Injecting MSCs with hyaluronic acid may help provide a viscosity for the joint and may give more viability to the MSCs. It was found that in vitro, there was an increase in cellular proliferation when AD-MSCs were exposed to low doses of hyaluronic acid or Polyglycan [44]. The addition of hyaluronic acid or Polyglycan may help in vivo. I often use intraarticular hyaluronic acid with stem cell injections post operatively. Intra-articular antibiotics are avoided because they will reduce cell viability substantially.

INJECTION TECHNIQUE

The lesions I inject are confirmed by ultrasound or by MRI, except in cases with spinal cord trauma where the lesions are rarely seen. The normal dose is about 4 million to 10 million cells, but this can vary depending on the company producing the cells and the extensiveness of the lesion. MSCs do respond in a dose dependent manner and to my knowledge there is no set number of cells per area of treatment that has been formulated. MSCs have been estimated to live in tissue from 5 months to 3 years. I could not find a percentage of MSCs that remain in the injected area but a high percent of them leave. Most of the non-engrafted MSCs end up in the liver, spleen, and lung. In this case you just inject cells, assess response, and if needed inject again 6 weeks later. MSCs usually migrate to other tissue planes and engraft out to the surrounding tendon crimp after exiting the lesion. This may be due to migration or by the injection itself. During ultrasound guided injections the fluid that is injected can be seen infiltrating surrounding tissue. In many cases though, you can see small amount of air that was left in the needle being pushed through the tissue. When MSCs are injected into the joint some of the MSCs infiltrate along the joint surface and but they also engraft to the synovium and probably the cartilage. MSCs differentiate and can secrete transgenic products and other therapeutic proteins into the joint space. It may be possible to have too many MSCs [45]. A mouse study showed genetically modified cells expressing BMP-2 were capable of osteophyte and ectopic cartilage formation. In this study there was an over expression of BMP-2 [46]. So it may be possible to inject too many stem cells but I have not experienced this problem. I have however, had to reinject in 6 weeks to get the response I wanted. The times I had to reinject were on large soft tissue lesions and it was very possible that I did not use enough MSCs initially.

Sterile preparation of the injection site is important. We use a 7 minute chlorhexidine scrub followed with alcohol. It is recommended not to inject antibiotics along with the stem cells because it may alter or damage them. In some cases where we feel that antibiotics will be needed along with the stem cells, we will either use regional perfusion before injection or inject the joint with antibiotics five minutes before stem cells are injected. After the preparation, many times we will use a local anesthesia at the site of injection. When injecting the lower limb we will use regional anesthesia to prevent movement and increase accuracy. The animal is always sedated when performing this procedure. Commercially processed MSCs are transported in a medium. For soft tissue lesions we use only the content of the transported syringe in order to reduce the volume. If we need to add volume or are injecting a joint, hyaluronic acid is added to the cell mixture.

We use imaging to guide our needle placement. We use both radiographic and ultrasound guided injections. I feel that the needle can be placed more precisely with ultrasonographic guidance than with radiography. Fluoroscopy is a better imaging modality than is radiography. In a diffuse soft tissue lesion we place multiple needles approximately 2-3 cm apart. All the needles are placed prior to injection. If fluid flows from the site of the lesion then it is aspirated prior to injection of stem cells. If injecting into one site and the stem cell suspension appears in the hub of the needle in another site the stem cell suspension is pushed back into the needle and the needle is removed. Then the next site is injected. In soft tissue injection, ultrasound is used during the time of the injection to watch the diffusion of stem cell suspension. The lesion is not distended with stem cell suspension. If there is too much

volume then a peripheral site to the lesion is injected. After injection of the stem cell suspension the area is bandaged or covered with iodine impregnated impermeable adhesive drape for several hours. This allows the needle insertion sites to close to prevent iatrogenic infection. Imaging is performed at 3 to 4 week intervals post injection until the lesion is appears healed after ultrasonographic examination. If at 8 weeks especially in soft tissue injuries, the lesion has not made substantial progress then more stem cells are ordered. Most commercial processors will have cells banked and frozen. These cells can be cultured to yield another dose of stem cells thereby avoiding another harvesting procedure.

Contraindications

Stem cells should not be used where neoplasia is present. Caution should be taken in cases involving sepsis or viral infections. In the case of neoplasia it is possible for the stem cells to engraft to and differentiate into the neoplastic cells. Also MSCs secrete many factors that may enhance the growth or mutate the neoplastic cells. There are reports of MSCs being used in a timed manner to cause regression of the tumor size and replacement with normal tissue. This research is beyond the scope of this chapter. At this time, it is not recommended to use MSCs when neoplasia is present.

In the presence of sepsis, MSCs have immunosuppressive properties and may inhibit the clearing of bacteria. On the other hand, MSCs could provide protection from the lipopolysaccharides and the inflammatory effects. One of the common therapies for septic areas is to lavage so placing cells into this area maybe ineffective. MSCs are also fragile and the septic environment may cause decrease cell viability. Do to the endosomal secretions of the MSCs, a latent virus could attach to the endosome and be carried into a cell. The MSCs could cause a decreased immune response do too their immunosuppressive effects. New research is now utilizing viral particles to cause mature adult cells to de-differentiate into stem cells (Shinya Yamanaka and John Cooke). Reengineered cytotoxic T lymphocytes are being studied to treat lentivirus i.e., HIV. These new studies are beyond discussion in this chapter.

It is contraindicated to use shockwave on the area injected for up to 60 days. There is a possibility of injuring or reducing the effectiveness of MSCs. Shockwave is performed initially followed by MSC injection. Shockwave treatment can be repeated at a later time.

It is contraindicated to use corticosteroids with MSCs. An in vitro study concluded that dexamethasone supplementation may cause BM-MSCs to move towards a chondrogenic phenotype that results in bone formation [47]. This situation to my knowledge has not been studied in vivo. Vet Stem has made a recommendation of using one dose of short acting steroids if needed for immediate relief of inflammation. Do not use high doses of antibiotics in the area of injection. I have used stem cells with Depo Medrol when injecting bone cysts without any apparent adverse effects.

Indications

Indications for the use of MSCs are not clearly defined at this juncture. We will use stem cell therapy if the owner can afford it and if it is not contraindicated. We will also treat

primarily with stem cell therapy when there is no other effective modality for treatment. In many cases, we need to obtain special permission from the company for use in cases where stem cell therapy is not routinely used.

Some of the current uses of stem cell therapy include osteoarthritis, fracture repair, soft tissue lesions (both chronic and acute), spinal cord trauma, navicular bone cartilage degeneration, longitudinal tears in the deep digital flexor tendon(DDFT), cartilage degeneration in any joint, meniscal tears, desmitis in any ligament (chronic or acute), tendinitis (chronic or acute) and wounds. We are currently doing a preliminary study on treating laryngeal hemiplegia. The results so far are promising.

It is difficult to get tendon lesions in synovial structures to heal and I have found that stem cell therapy has been useful for these types of lesions. These horses often become comfortable within a few days to a few weeks. The comfort comes before any possibility of the lesion healing. The decreased pain may come from the anti-inflammatory affect in the region or reduced levels of proinflammatory cytokine IL-1 with in the nerves themselves.

Meniscal tears which are seen most often in "cutting horses". These lesions can be career ending for horses. Arthroscopy can be performed along with debridement for small tears or suturing for larger ones. Some horses do get better with surgery but many do not. I think that if the lesion lends itself to arthroscopy then it should be performed and stem cell therapy should be used in conjunction with the surgery. While performing the arthroscopy, the fat around the stifle is an excellent source for stem cell extraction. On all of the meniscal tears that we have treated with MSCs, all have improved both in soundness and on ultrasonographic appearance.

Cartilage degeneration in the pastern joint, areas in the fetlock, or other difficult to reach places may lend itself to arthroscopy but if arthroscopy is not an option then stem cell therapy should be considered for maintenance of the joint. I have seen cartilage resurface in a joint that I performed arthroscopy and then injected with stem cells. A year later, I performed another arthroscopy on the same joint. The area where the cartilage was removed had resurfaced with cartilage over the osteochondral bone defect. This case will be discussed later. Horses with MRI imaged cartilage degeneration returned to soundness and cartilage degeneration was gone when the joint was imaged again.

Desmitis and tendinitis whether it is a core lesion or a diffuse thickening of the ligament (chronic or acute) are candidates for shockwave and stem cell therapy. There are other modalities such as fasciotomies, neurectomies, bone marrow injections, IRAP injections, PRP injections, corticosteroid injections, glycoaminoglycan injections, pin firing, and etc. and all have had some degree of success and failures. Many times with chronic desmitis cases that have been going on for over four to six months, I will start with one to two shockwave treatments and then follow with one to two of stem cell treatment eight weeks apart. If there is not satisfactory healing at three months after the last dose of stem cells, I will use shockwave therapy again and inject PRP.

Osteoarthritis of the distal hock joints and pastern joints are difficult to treat medically once the process starts. We treat these joints with shockwave therapy followed by IRAP injections to reduce the inflammatory response prior to stem cell therapy. This has led to favorable results. If it is not possible to l inject via arthrocentesis it may be feasible to use intravenous stem cell injections or regional perfusion. Usually these joints will need at least two doses of stem cells eight weeks apart. I will many times follow up over the year with

therapeutic laser therapy or shockwave therapy. It has been recommended by some practitioners that stem cell therapy should be repeated in two weeks for osteoarthritic joints.

Spinal cord trauma that is non-responsive to other treatments may respond to stem cell therapy. The horse will need to be anesthetized and properly prepared for an intrathecal injection at the atlanto-occipital site. Spinal fluid is removed (approximately 10ml) the stem cell solution is injected. A hemo-filter will be needed for this injection. Preanesthetic doses of flunixin meglumine and dexamethasone are given. Post-operatively, the horse may be more ataxic for the first 24 hours but quickly returns to the prior neurologic status. The horse should steadily improve over the next two to six months. We have had excellent results treating neurologic cases in this manner.

Laminitis has also been treated with stem cell therapy. We normally treat laminitis using two regional perfusions with IRAP. Then if economically feasible we will follow with stem cell therapy. MSCs should reduce the inflammatory response, regulate neovasculization, control pain and rebuild laminar tissue. More research needs to be done in this area. I regionally perfuse intra-arterially and intravenously. Success is questionable because of very low case numbers and owner's noncompliance. I believe that allogeneic MSC treatment will improve the prognosis for laminitis. Many of the laminitic horses that we treat have metabolic problems. When we remove adipose tissue, it normally has very low numbers of regenerative cells. The allogeneic cells taken from healthy horses may have more viable cells or may improve the trophic effects. More research is needed in this area. We have found that horses with laminitis and other metabolic problems and in some older horses have had a low regenerative cell numbers. The low number is not a problem because the cells can be cultured to a higher number if needed. Stem cells derived from diabetic mice caused a decrease in size and number of neovasculature components and increased healing time when injected into open wounds. Mice that were old had increased healing time but almost normal neovascularization. When they were injected with donor stem cells from young normal mice, the wounds healed at an increased rate with improved vascularization. Stem cells may not all function the same and stem cells obtained from diseased or older animals may not give the appropriate affects. At this time, more research needs to be done in horses but I use stem cells from both aged and metabolic horses.

Chronic hives in horses can be treated with some success with stem cell therapy. I have treated two horses that had severe allergy problems that resulted in hives. Corticosteroids would only lessen the problem. Environmental and feeding changes did not make a difference. I treated both horses with MSCs intravenously twice using a ten day interval. Both horses improved but we also continued with feeding changes.

Wound treatment with stem cell therapy is difficult to evaluate in terms of increased rate of healing. Most of the wounds that I have treated where open wounds involving tendons, ligaments and/ or joints. Surgical wound treatment was used along with collagen sheets where tissue could not be closed. Fat was harvested during surgery or 24 to 48 hours after. The affected areas where injected after the stem cells were returned. All the wounds healed.

When using stem cell therapy for wounds, a clean or sterile wound environment is necessary. After injection, the wound should not be flushed and ointment should not be applied to open wounds. Stem cells need a matrix to work and collagen sheeting is a good matrix. I have not yet tried the collagen powder mixed with stem cells but it may be a good combination. If the wound environment cannot be cleaned initially then wait to inject after

there is a granulation bed formed. Inject the area surrounding the injury, the granulation bed, and other structures that have been affected.

I have had two horses that had increased lameness after joint injections of the lower limbs with MSCs. We found no evidence of infection or problems with the stem cells. Both horse's joints were flushed and later reinjected with stem cells. Both horses improved in soundness before their second injection. Both of these horses have had problems with other medications also. Both have not regained soundness. I have not found a reason for the adverse reactions.

CASE EXAMPLES

Case 1

An 8 year old Quarter Horse gelding western pleasure show horse was found recumbent and unable to stand. The horse was treated initially with DMSO, corticosteroids, and NSAIDS. Later that day he was able to rise and stand but unable to walk out his stall. Treatment continued and test results for equine protozoal myeloencephalitis, equine herpes, west Nile where all negative. The lesion appeared to be localized to the cervical region. Radiographs and ultrasonographic examination revealed osteoarthritic changes on articular facets of C4, C5 and C6. There appeared to be a small fragment associated with C3. The vertebral canal diameter was normal. The owner declined a myelographic examination and MRI. The horse continued to improve for the next 2-3 months but then his improvement became stagnant. Three months post trauma, the horse could not trot without falling or tripping. He was too unstable to ride. We elected to try stem cell therapy. We used 3.1 million regenerative cells in the stromal vascular fraction.

The horse was anesthetized, positioned in lateral recumbency, and aseptically prepared for an AO spinal tap. The spinal needle was placed through the dura mater and cerebrospinal fluid was obtained. Ten mls of stem cell suspension was injected through a hemo-nate filter. The horse was recovered in a sling and recovery was excellent. The horse was mildly more ataxic for the first 24 hours after the injection. He was treated with one dose of corticosteroids and NSAIDs.

Over the next 6 weeks he improved enough to be ridden. Later that year he was showing at the same level as he was prior to the ataxic event. The reason for his recovery is unknown. It may be due to the stem cells ability reduce the inflammatory cytokines within the subarachnoid space. But with all the corticosteroids and NSAIDS this horse had been given along with the fact that the cerebrospinal fluid was normal tells me that the anti-inflammatory response was a small part of the improvement. In rodent models of sciatic nerve constriction the nerve has increased proinflammatory cytokine content but with MSC treatment the intraneural proinflammatory cytokine level is reduced. Perhaps the anti-inflammatory mechanisms of the stem cells affected the spinal tracts. It is also likely that there was a defect in the spinal cord or a focal lesion in the dura mater and the stem cells repaired the area.

I believe the stromal vascular fraction is safe to use on central nervous system problems in the horse. More research is definitely needed to find out what happens to the stem cell once they are in cerebrospinal fluid.

Case 2

A 4 year old Quarter Horse gelding used for western pleasure had a left hind high suspensory injury that had been treated by another veterinarian about 10 weeks previously. The horse was confined to a stall for this entire period and developed a right front lameness. The right front lameness was localized to the foot and the left hind limb suspensory ligament was very thick and mildly sensitive to palpation. MRI of the left hind suspensory ligament revealed an extensive chronic desmitis that extended 10cm distal to the MT3 origin. The cross-sectional area was 4.1cm² and there were possible adhesions to Mt4. The right front foot had focal fibrocartilage degeneration of the navicular bone and solar frog and heel hemorrhagic exudates. The navicular bursa was treated with IRAP (interleukin-1 receptor antagonist proteins) and therapeutic shoeing. The left hind limb was treated with 5.3 million AD-MSCs. The horse was sedated and a local infusion of carbocaine was injected in 4 different areas along the proximal suspensory ligament. Four needles were inserted along the proximal suspensory ligament area using ultrasonographic guidance and an equal dose of stem cells was injected at each site. The tarsometatarsal joint was also injected with 40mg of depomedrol, 11 mg hyaluronic acid and 50mg amikacin. A controlled exercise protocol was utilized for 1 month. Over the month period of time there was a 50% reduction in diameter of the suspensory ligament. Ultrasound measurements were 2.0c m² in the left hind limb proximal suspensory ligament and the right hind limb measurement was 1.7cm². Rehabilitation consisted of hand walking for 15-20 minutes three times a day for one month. He competed at the World Quarter Horse Show that year. This case is typical of the healing pattern of chronic diffuse soft tissue lesions. The cross sectional area usually decreases over the first 4 to 6 weeks. If this ligament's cross sectional area would not have continued to decrease another dose of stem cells would have been injected. If the horse would have stayed consistently lame then another MRI would be indicated looking for possible adhesions which are common after proximal suspensory injuries. I feel the cross sectional area of the lesion is one important indicator of healing along with parallel fiber alignment with an isoechoic appearance. The opposite limb is used for comparison. When performing an ultrasound examination of the proximal suspensory ligament it is necessary to determine which body of the suspensory is injured. In the front suspensory ligament there is a medial and lateral body. The suspensory ligament of the pelvic limb contains a short branch of the plantar ligament that makes up the superficial part and at the Mt3 origin area there is a lateral, middle and medial body divided by bundles of connective tissue, muscle fiber, and fat. The bodies can be seen with ultrasound by doing a comparison of off set (angling the probe slightly to enhance contrast) and normal ultrasound. In the Quarter Horse there are more problems with the middle body and this is the area to concentrate your injections. The prognosis for proximal suspensory injuries is excellent, 85.7% return to original performance [48]. (Rich R. et al: Outcome of 84 horses with suspensory injuries treated with adipose-derived regenerative cells. 3rd North American Veterinary Regenerative Medicine Association Conference; Nov. 8 2012.)

Case 3

A 14 year old Hanoverian gelding that was retired from show jumping and was in dressage training presented with a 3/5 lameness. Examination revealed sensitivity to palpation in the area of insertion of the superficial digital flexor tendon. The horse was treated with NSAIDs and cold compression therapy. The lameness worsened to 4/5. Radiographic examination revealed an osteophyte on the dorsal proximal aspect of P2 and a slightly increased sclerotic appearance to proximal P1. Ultrasound revealed a possible longitudinal tear in the DDFT and thickening of the lateral distal oblique sesamoidean ligament, effusion of the pastern and metacarpophalangeal joint, and osteophytosis in various areas along the margin of proximal P1. Joint fluid from the coffin, fetlock, and pastern joints was normal. The coffin joint, digital tendon sheath, pastern joint, and fetlock joint were treated with 22mg hyaluronic acid, 4mg triamcinolone, and 100mg of amikacin. NSAIDs were given. Regional perfusion of antibiotics and lidocaine was performed which improved the lameness to a 3/5 for about 2 days after the treatment. An MRI revealed P1 bone injury associated with an increase in vascular response in the medullary cavity, pastern joint effusion, desmitis of the lateral and medial distal oblique sesmoidean ligaments, osteoarthritic changes of the metacarpophalangeal and metacarposesmoidean joints consisting of articular cartilage degeneration and subchondral sclerosis in the sagittal ridge of Mc3. The horse was treated with Tildren via regional perfusion. The fetlock joint, digital tendon sheath, pastern joint and the lateral oblique distal sesamoidean ligament were injected with AD-MSCs. After a period of hand walking followed by free turn out for 45 days, light riding was initiated. Five months after injury this horse was back to training as a show jumper and one year he was back to grand prix jumping. To my knowledge the right front joints have not been injected as of one year.

This case demonstrates the value of MRI in a complicated lameness. We frequently see severe pain associated with bone edema. We rarely find just one problem in a lameness case. Most of the lameness cases we see have multiple problems. This horse has been show jumping for two years since treatment.

Case 4

A 7 year old Quarter Horse/Paint gelding used for trail riding and showing came to the clinic with severe effusion in the right femoropatellar joint. The horse had been treated for about a year with chiropractics and other joint therapies. Diagnostic arthroscopy revealed severe cartilage erosion on the articular surface of the patella. About 2/3 of the patellar cartilage was gone along with cartilage fibrillation throughout the joint. Severe synovial flare was also present. The cartilage was curetted and the joint was treated with IRAP. The horse was intermittently lame for over a year. Radiographs revealed fragmentation of the patellar margins. Arthroscopy was performed again revealing multiple fragments along the apex of the patella, the eroded cartilage on the patella was covered with fibrocartilage that had some fibrillation. The femoral cartilage was cracked. Some areas of cartilage were thin and some areas were lifting off the subchondral bone. The synovium was normal in appearance. The cartilage flaps were removed and the fragments removed. Due to the extent of the cartilage problems stem cells were used as a post op joint treatment. Ten days after surgery 4 million

AD-MSCs were injected along with 22 mg of hyaluronic acid. The horse continued to improve and the effusion decreased. At 2 months the owner started light riding. After three months the horse was brought back for a recheck and was scheduled for another stem cell treatment. The horse was injected with another dose of stem cells and with IRAP. There was a significant decrease in effusion and the total protein of the synovial fluid was <1g/dl. The lameness improved to a 1/5. The horse was sound during training and returned to ridden exercise.

Case 5

A 17 year old Welsh Thoroughbred cross pony used as a show pony presented with a 4/5 lameness. The right front lower limb was hot and swollen. No fracture was evident radiographically. Ultrasound examination of the flexor tendons revealed a 90% tear of the superficial digital flexor tendon. The limb was treated with ice and bandaging. Stall rest was enforced for one year.

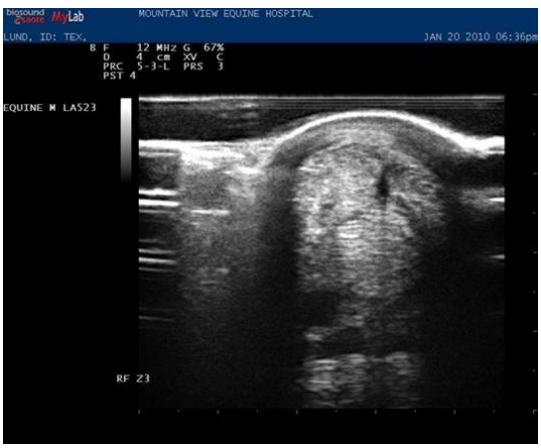

Figure 2.a.

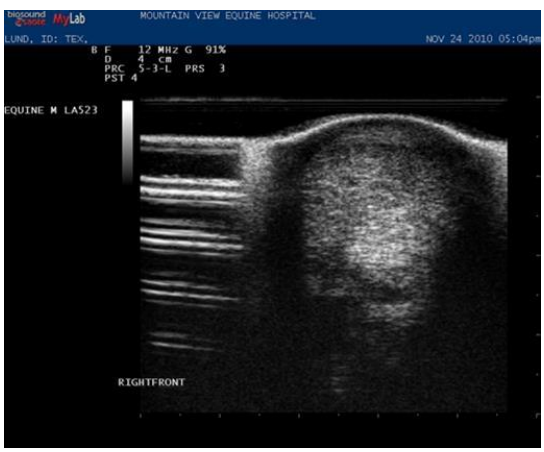

Figure 2.b. Sonograms obtained from case five.

Ultrasonographic examination revealed that the SDFT zone 1b to 3a had poor fiber alignment and anechoic regions. The SDFT was mildly enlarged at zone 1a and continued to enlarge to zone 3a where the cross sectional area was at its largest (Figure 2.a. and 2.b.). At zone 3b the tendon was almost within normal limits. Fat was harvested and the AD-MSCs were injected into eight sites along the SDFT. A total of ten million cells were used. The pony was sent home with instructions for hand walking 20 minutes twice a day and stall rest. The four week ultrasound recheck revealed that the cross sectional area of zone 3 was decreased mildly, anechoic areas were being to fill in. The proximal zone's fiber alignment had improved. At eight weeks post injection, the zone 3 cross sectional area decreased and the anechoic regions were no longer evident. The SDFT was reinjected with AD-MSCs. The pony was shod with a therapeutic shoe that was wider at the toe than at the heel. Ridden exercise was begun gradually. At the six month check-up the SDFT was thickened with some improvement in fiber alignment. The pony was sound and able to ridden at the walk, trot, and canter. The pony was slowly returned to training over the next several months. At 10 months from the first injection, the pony competed in a show. It has been competing now for over two years with no lameness.

The injury of the SDFT was chronic that improved with AD-MSC treatment. I believe that due to the chronicity of the condition more than one treatment of MSCs was necessary. If the owners were able to afford shockwave treatment and rehabilitation therapy, the tendon may have had a better appearance. Often we will incorporate shockwave therapy at the beginning of treatment of chronic injuries. Chronic injuries can be stimulated to heal.

Case Example 6

A 13 year old Irish draft sport horse had swelling around the right front pastern region for over 2 weeks after fox hunting. Lameness examination revealed a 3/5 right front lameness. The lower limb was radiographed and no fractures were found. Hoof tester examination was unremarkable. Heel pressure test was a 3/5 and digital flexion was a 3/5. All other flexion tests were within normal limits. Palmar digital nerve anesthesia improved soundness by 90%. Ultrasound examination revealed enlargement of the medial and lateral collateral ligament of the distal interphalangeal joint (DIPJ), moderate to severe subcutaneous edema around the eastern region, effusion in the distal and proximal (PIPJ) interphalangeal joints and navicular bursa, and enlargement at the attachment of the lateral branch of the SDFT and straight distal sesamoidean ligament.. The fluid removed from the joints had 5 g/dl total protein and moderate cellularity. Joint sepsis was suspected. A week later the horse returned with a mild improvement. Arthrocentesis of the PIPJ and DIPJ and the navicular bursa was performed. The total protein of the joints had reduced to a 3 g/dl and the navicular bursa was 5.2g/dl with high neutrophil count. Culture of the fluid produced no growth. The joints and navicular bursa were flushed and injected with amikacin. Distal limb regional perfusion with amikacin was performed. Over the next few days the horse was more comfortable but we suggested an MRI. MRI revealed severe pathology of P3 emanating from the distal impar ligament, hyperemia of the navicular bone, osteolysis of the origin of the impar ligament, deep digital flexor tendon tear at the level of the navicular bursa, moderate desmitis of the origin of the medial and lateral collateral ligaments of the coffin joint, desmitis of the lateral abaxial ligaments of the pastern joint, and desmopathy of the straight distal sesamoidean ligament

(Figures 3-7). Regional perfusion with Tildren was performed on the distal right forelimb. Fat was harvested for MSCs and the DIPJ and PIPJ, navicular bursa and the other soft tissue lesions were injected. Stall rest with handwalking and Equivibe therapy was initiated. The horse improved rapidly over the next two weeks but then at four weeks he became severally lame again.

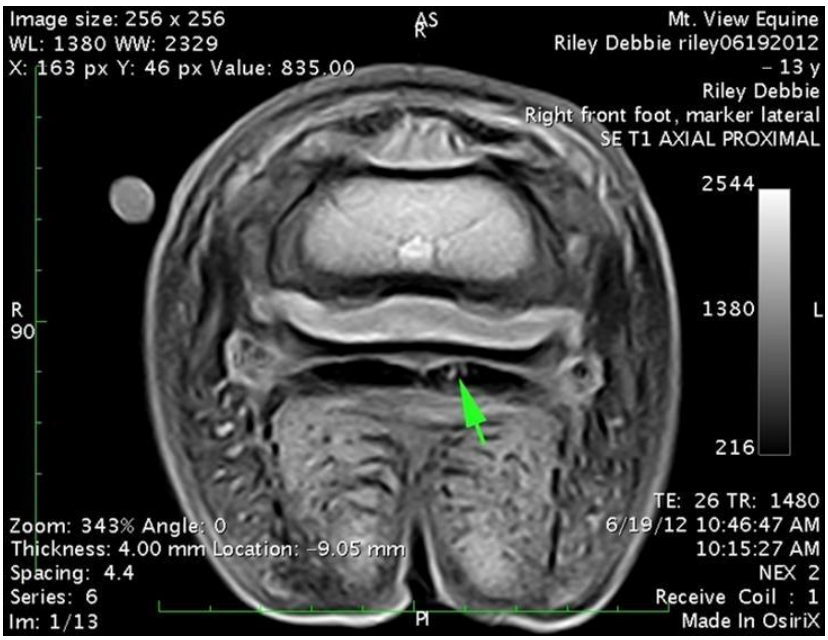

Figure 3. Right front deep digital flexor tendon tear.

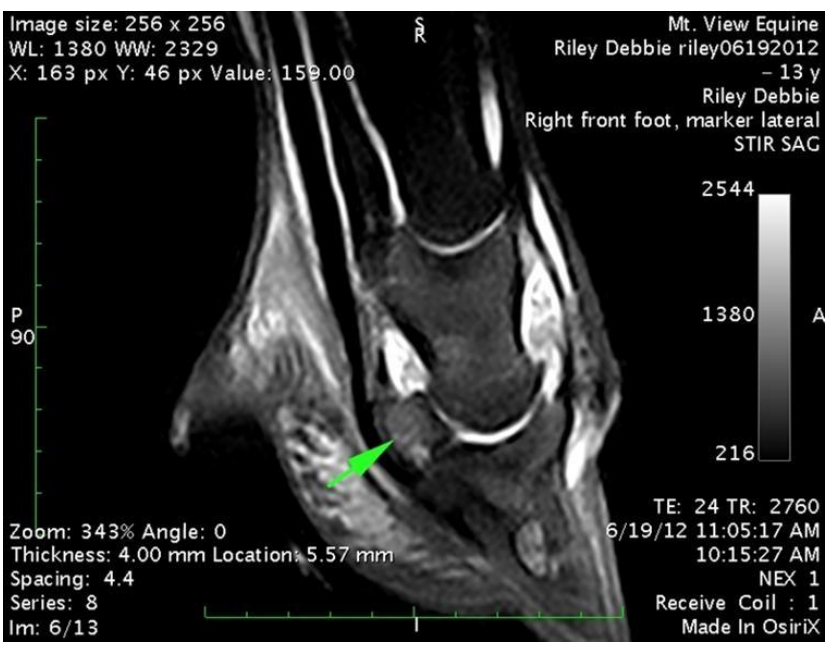

Figure 4. Hyperemia of the distal sesamoid bone (Navicular bone).

At 5 weeks another MRI was performed on the same area. The MRI examination revealed osteonecrotic foci in the lateral aspect of the intramedullary cavity of P3 at the insertion site of the lateral collateral ligament, progressive enlargement of the deep digital flexion tear, hyperemia of the P2, P3, and the navicular bone, and moderate desmitis of the collateral ligaments of the DIPJ. The areas were injected again with cultured adipose derived MSC.

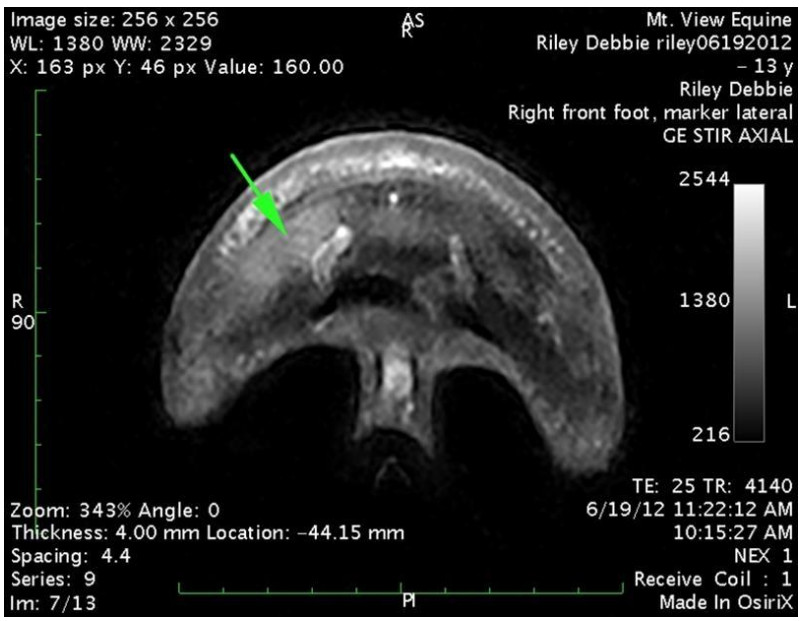

Figure 5. Hyperemia/bruising of the coffin bone.

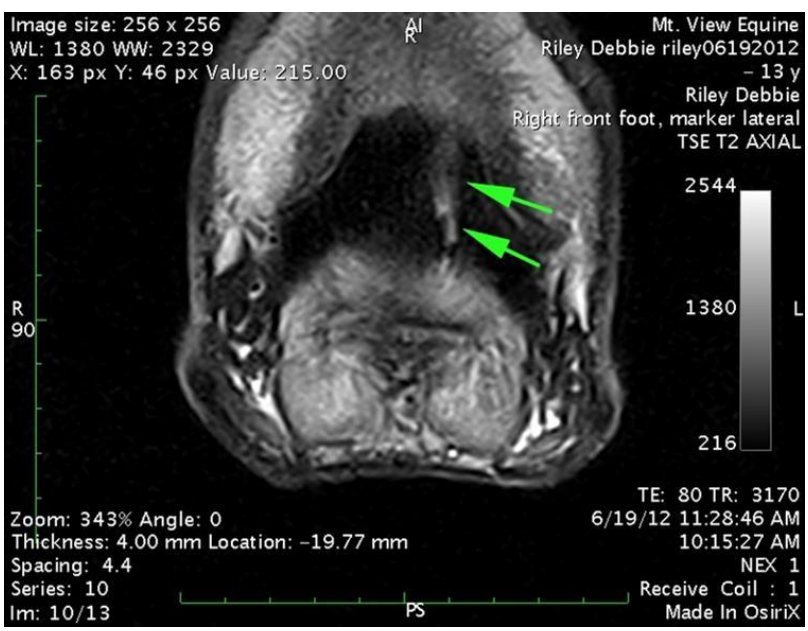

Figure 6. Tear in the deep digital flexor tendon distal to the navicular bone.

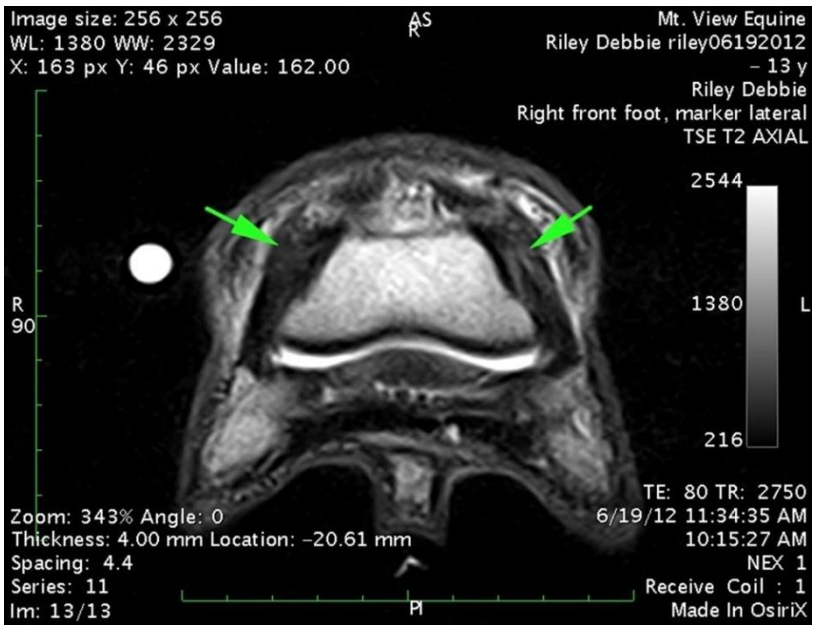

Figure 7. Desmitis of the medial and lateral collateral ligaments of the distal interphalangeal joint.

A six degree wedge shoe was placed on the right front foot. The horse was hand grazed and stall rested for one month followed by stall rest and handwalking for 20 minutes twice a day. At 8 weeks from the last MSC injections, shockwave therapy was utilized on the lateral collateral ligament insertion. The horse was sound at a trot, six months after the initial injury. The six degree wedge shoe was slowly modified to a two degree onion heel shoe. Ridden exercise was initiated on the fifth month post injury. Nine months after the last stem cell injection the horse remains sound.

This horse had severe injuries to the DDFT, collateral ligaments, navicular bone and coffin bone. The initial assessment of the joint fluid was consistent with sepsis. The MRI was necessary for appropriate diagnosis and treatment. The second MRI showed that there was further pathology to the structures of the foot. This was probably a reinjury during rehabilitation. The wedge shoe helped to reduce some of the DDFT pressure over the navicular bone. Usually we will start with a seven degree wedge if we see this severity of DDFT injury. We elected to let the horse go barefoot after the first MRI because of the collateral ligament desmitis. In hind sight, we should have put the shoe on. The horse was not comfortable in the seven degree wedge so we tried the six degree wedge which he was able to tolerate. The wedge is brought down one degree over every six weeks and then an onion heel shoe is applied.

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In: The Equine Tendon in Health and Disease
Editor: Henry Jann
ISBN: 978-1-62808-048-3

Chapter 5

SURGICAL TECHNIQUES FOR EQUINE TENDONS AND LIGAMENTS

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INTRODUCTION

Given the general anatomy and athletic ability of horses, conditions involving the soft tissue structures of the limb are commonly seen in equine practice. These soft tissue injuries can be potentially devastating to the athletic career of the horse, in addition to being financially burdensome to the client. Currently there are multiple treatment regimens available to attempt to help resolve these conditions and help achieve the ultimate goal of getting the horse back to work. They include but are not limited to cold therapies, compression therapy, extracorporeal shockwave therapy, therapeutic ultrasound, low level laser therapy, topical counter-irritation, systemic anti-inflammatory drugs, intralesional injections, controlled exercise programs and surgical interventions. This chapter will focus on some common surgical treatments for equine tendons and ligaments.

SURGICAL TECHNIQUES

Tendon splitting is a technique that was historically used to increase the vascularization to chronic tendon injuries. However it has become essentially obsolete for these types of injuries as it has been shown to have very little benefit. In recent years tendon splitting has gained attention for the treatment of acute tendon injuries. One application is its use is in the treatment core lesions within the matrix of a tendon, as they are believed to compromise blood flow to the damaged area due to a compartment-like syndrome. Thus, removal of the fluid within the lesion helps establish better blood flow to the injured area and potentially aids

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in faster and more normal healing of the lesion. Additionally, this technique can be employed to treat horses with an upward fixated patella. In this situation, the proximal third of the medial patellar ligament is split in an attempt to induce desmitis and ultimately strengthen the ligament and help prevent future episodes of fixation over the medial trochlear ridge of the femur. The procedure can be performed with the horse under general anesthesia or under sedation and local anesthesia with the horse standing. Additionally, the assistance of an ultrasound can provide more accurate needle or scalpel blade placement into the tendon. If a needle is used, it is inserted into the appropriate area and fanned both proximally and distally in effect "splitting" the tendon. Multiple needles may need to be inserted into a lesion to achieve appropriate therapeutic benefit. Alternatively, a No. 11 scalpel blade can be inserted into the lesion and fanned in a similar manner as previously described. [1, 2] A study evaluating the effect of tendon splitting for the treatment of upward patellar fixation and found that in seven equids splitting of the proximal third of the medial patellar ligament, and thus induction of desmitis, resulted in complete resolution of clinical signs. [3]

Desmotomy of the accessory ligament of the superficial digital flexor tendon (superior check desmotomy) has been described for the treatment of flexural limb deformities of the metacarpophalangeal joint and superficial digital flexor tendonitis. Currently there are conflicting studies in the literature arguing whether horses with tendonitis actually benefit from this procedure. Historically the belief was that a longer musculotendonous unit would exert less strain on the superficial digital flexor tendon. However, recent research has shown that this may not be the case as the accessory ligament plays a vital role in the biomechanics and load distribution of the limb and in actuality more strain is placed on the superficial digital flexor tendon and the suspensory ligament following desmotomy of the superior check ligament which is ultimately of no benefit to the horse. [4] Yet, this procedure is performed routinely to correct flexural limb deformities of the metacarpophalangeal joint. There are multiple approaches to this procedure, three will be described here. The first is incisional over the medial aspect of the limb, in which a 10 cm incision is made over the medial aspect of the forelimb beginning at the level of mid-chestnut and extending to the medial malleolus of the radius. This incision is positioned half way between the cephalic vein and caudal aspect of the radius. The flexor carpi radialis sheath can be found following caudal retraction of the cephalic vein. The sheath is incised and the flexor carpi radialis tendon is identified and retracted caudally to provide visualization of the superior check ligament, which is then transected. The flexor carpi radialis sheath, subcutaneous fascia and skin are all closed separately. [5] Alternatively, an incision similar to the one outlined above can be made and the depression that is visible in the antebrachial fascia with an associated branch of the cephalic vein can be used to guide deeper dissection. Following retraction of the flexor carpi radialis sheath, without entering the sheath, the fan shaped superior check ligament can be identified and transected. [6] These can be a technically difficult procedures with potential intra-operative and post-operative complications, thus a lateral tenoscopic approach has been described. For this approach the lateral aspect of the affected limb is placed up. The carpal sheath is distended and an arthroscopic portal is made 2 cm proximal to the distal radial physis. An instrument portal is then made 0.5 cm proximal to the distal radial physis. The limb is flex to a 90 degree angle and the ligament is identified and transected. [7] The tenoscopic approach is thought to be more aesthetically pleasing than the medial incisional approach.

Desmotomy of the accessory ligament of the deep digital flexor tendon (inferior check desmotomy) is the treatment for stage 1 distal interphalangeal joint flexural limb deformities. There are two stages of flexural limb deformities. Stage 1 is defined as a dorsal hoof wall angle of less than 90 degrees. If the angle is more than 90 degrees then it is considered a stage 2 flexural limb deformity and would have a poorer prognosis requiring a deep digital flexor tenotomy. For the desmotomy of the accessory ligament of the deep digital flexor tendon the horse can be placed in dorsal or lateral position and medial or lateral approach can be used with the later somewhat safer as one avoids the neurovascular bundle that sits along the medial aspect of the limb. A 3 cm skin incision is made over the deep digital flexor tendon and centered on the junction on the proximal and middle third of the metacarpus. Blunt dissection is then used and the accessory ligament is identified and transected. [5] A standing ultrasound guided tenotomy of the accessory ligament of the deep digital flexor tendon and a tenoscopic approach for transaction through the carpal flexor sheath have also been described for this procedure. [8] In a study comparing standardbred foals with a flexural limb deformity that received an inferior check desmotomy to those that did not, 6 of the 11 surgically treated responded favorably and raced or were sound and in training, whereas none of the 12 non-surgically treated went on to race or train. [9]

Tenotomy of the deep digital flexor tendon is the treatment of choice for stage 2 flexural limb deformities as previously described. Additionally, this procedure is used to treat horses with chronic refractory laminitis. There are two anatomic locations that this can be performed. One is at the mid-pastern level just distal to the bifurcation of the superficial digital flexor. In this instance, a skin incision is made over the pastern right on midline. The deep digital flexor tendon is identified, isolated and transected. The second location is at the mid-metacarpal region and can be performed with the horse standing or anesthetized. The tendon can be approached either medially or laterally. [5] A potential advantage of this technique is the tendon sheath is not entered as it is with the mid-pastern approach. Most clinicians view the deep digital flexor tenotomy as a savage procedure with a guarded prognosis for return to athletic function. A study evaluating the long term prognosis for laminitic horses treated with deep digital flexor tenotomy found that 27 of 35 horses survived at least 6 months following treatment and 19 of 35 survived at least 2 years, implicating that this procedure should be considered for those horses with chronic refractory laminitis. [10]

Annular ligament desmotomy is used to treat horses with compartment syndrome of the digital flexor tendons over the fetlock region. This pathophysiologic process is defined by chronic tenosynovitis within the digital tendon sheath that results in thickening of the palmar/plantar annular ligament and ultimately causes constriction of the flexor tendons and thus chronic lameness. An open technique has been described in which a 1 cm skin incision is made proximal to the annular ligament and a curved bistoury knife is used to incise the ligament. [2] More recently a tenoscopic approach has been used in which an arthroscope is inserted distal to the annular ligament and an instrument portal is made proximal to the annular ligament allowing for a more comprehensive evaluation of all the structures within the digital sheath and visualization of the ligament transection. [2] A study evaluating the effect of annular ligament desmotomy on horses affected with fetlock compartment syndrome looked at 24 horses with 16 responding favorably to annular ligament desmotomy and returning to work without further lameness. [11]

Fasciotomy and neurectomy of the deep branch of the lateral plantar nerve is used to treat chronic hindlimb poroximal suspensory desmitis.

This procedure is performed with the horse anesthetized and in dorsal recumbency. A 6 cm incision over the lateral aspect of the superficial digital flexor tendon centered at the level of the tarsometatarsal joint. The deep branch of the lateral plantar nerve can be found in the connective tissue between the superficial digital flexor and the long plantar ligament. Following isolation and removal of a 2 cm piece of this nerve, a fasciotomy is performed in which the fascia over the proximal suspensory ligament is incised with a No. 11 scalpel blade parallel to the lateral splint bone extending from the level of the tarsometatarsal joint to approximately mid body of the suspensory ligament. [2] In a study of 90 horses with lameness associated with proximal suspensory desmopathy, 70 responded favorably to fasciotomy and neurectomy of the deep branch of the lateral plantar nerve with a successful outcome defined as a horse being in full work for at least one year post-operatively. Additionally, the authors of this paper noted that horses with a straight hock or fetlock hyperextension are poor candidates for this procedure given that all horses in the study with this conformation continued to be lame following the procedure [12].

Bursoscopic assessment of the navicular bursa was initially described for penetrating wounds into the navicular bursa. However, more recently it has been used as a diagnostic modality to evaluate intrabursal lesions of the deep digital flexor tendon. The technique is a blind approach in which a stab incision is made proximally over the lateral collateral cartilage and the arthoscope is inserted dorsally to the deep digital flexor tendon and advanced into the navicular bursa. [2, 13] In addition to bursoscopy, tenoscopic exploration of tendons and ligaments within other synovial structures has been used for the evaluation of lesions within these structures.

Tendon Lacerations are a common injury seen by equine veterinarians. The prognosis for recovery and return to function is determined by the anatomic location and severity of the lesion. The primary goals of repair are to achieve adequate end to end apposition and restore as much normal functioning tendon as possible. Flexor tendons injuries are much more of a concern in comparison to extensor tendon injuries, because they are more important for movement and maintenance of posture of the horse. The mainstay of flexor tendon repair is debridement, tendon suturing and wound closure.

Following evaluation of the injury and debridement the proximal and distal tendon ends must be identified and sutured back together with absorbable suture material. The two most common suture patterns for tendon repair are the three loop pulley and the interlocking loop. [2] Research evaluating the these methods of tendon repair found that the three-loop pulley pattern provided more support, less tendon distraction, and less tendon matrix constriction and distortion than the locking-loop pattern. [14] The superficial wound is then closed and a cast applied for the limb. Depending on the location and severity of the lesion, casting needs to be continued for 4-6 weeks during the recovery process.

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In: The Equine Tendon in Health and Disease
Editor: Henry Jann
ISBN: 978-1-62808-048-3

Chapter 6

THERAPEUTIC REPAIR OF DAMAGED TENDONS AND SUSPENSORIES: CONSERVATIVE AND SURGICAL THERAPEUTIC OPTIONS

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ABSTRACT

The importance to the horseracing world of Superficial Digital Flexor (SDF) Tendonitis was recently made very clear when I'll Have Another was forced to scratch from the 2012 Belmont Stakes. This important tactical decision not only cost the horse and his connections a chance of winning the prestigious Grade 1 Stakes, but forfeited I'll Have Another's probable potential for capturing the elusive Triple Crown (Figure 1). That feat hasn't been repeated since Affirmed won it in 1978; some 34 years ago. However, trainer Doug O'Neill and owner J. Paul Redman are to be complimented on their difficult decision not to jeopardize their horse and rider, or the other contestants in the race. Especially with so much money and prestige at stake, the decision to scratch such a horse from a race, or to retire a horse from any other form of competition due to a damaged tendon or suspensory isn't easily made; however, of even greater challenge is the course chosen for trying to rehabilitate that horse and bring it back to its previous level of performance.

INTRODUCTION

Tendons are elastic high tensile strength cable like soft-tissue structures that connect major bones via their direct attachment of the bones to the muscles thereby connecting the major bones.

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Figure 1. I'll Have Another forced to scratch from the 2012 Belmont Stakes.

Tendons are well constructed to allow passive transfer of significant degrees of muscular force between bones and to provide movements of the intermediary joints. *Ligaments* directly attach two bones to each other to resist distractive forces. They have a much lower degree of elastic flexibility and a higher degree of inherent strength; meaning they are inherently stronger but equally less resilient than tendons.

There are 2 tendons (superficial digital flexor tendon and deep digital flexor tendon) and 1 ligament (suspensory ligament; also known as the suspensory apparatus) on the back side of all 4 of the horse's legs. Each of the distal branches (medial and lateral) of the suspensory apparatus insert on the abaxial (outside) surface of its corresponding proximal sesamoid bone; found distally at the back of the fetlock (ankle) joint. Additionally, the distal sesamoidean ligaments are the functional continuation of the suspensory in the digit and comprise the straight sesamoidean ligament, oblique sesamoidean ligaments, cruciate sesamoidean ligaments, and short sesamoidean ligaments; which are attached below the fetlock joint. There is additionally a palmar ligament of the fetlock which strongly attaches each medial and lateral sesamoid together.

When the 4 fetlock (ankle) joints over-extend (excessively flex toward the ground during racing, jumping or other athletic performance), these composite tendons and ligaments absorb a very large amount of weight bearing load (stress) in the process of supporting the joints.

The cumulative elastic energy storage within and the simultaneous shock absorption properties of the flexor tendons allow the horse to efficiently accelerate to and maintain high speeds, while conserving energy. The force placed upon a tendon in relation to its degree of stretch allows calculation of its modulus of elasticity; more scientifically known as its stress-strain curve. Much like a bungee cord, the tendon can only be stretched so far and so many times before it begins to fatigue, loose its elasticity, begin to deteriorate, distort, fail or even worse; rupture. Flexor tendons are calculated to stretch approximately 10 to 12% of their original length before they catastrophically rupture due to an excessively high tensile load.

When a tendon or the individual fibers within the tendon reach their yield point causing irreversible damage, or their breaking point, their normal structure will be permanently altered and/or they will rupture. The same is true for suspensories. The amount of force needed to alter or rupture normal tendon or suspensory anatomy vary tremendously amongst individual horses, are associated with their athletic training and conditioning base, and are a function of the degree and/or level to which the horses are expected to compete. While much of the emphasis in this chapter will concern tendon injury, suspensory damage is equally detrimental and is believed to be similarly challenging to overcome, and slower to heal and rehabilitate.

Tendons are built much like steel support cables except they are elastic, like bungee cords. There are many cross-linked sub-cable bundles of collagen fibers which are joined in parallel and interconnected within the major tendon itself. These are all contained within a durable fibrous covering or tendon sheath. The tendon fibers receive nutrition and oxygen from a uniquely complex blood supply. Damage is caused to tendons by excessive physical exertion, local heat production and loss of blood supply. The most common race horse tendon injury is to the superficial digital flexor tendon on the posterior surface of the mid-cannon bone region; often in the left forelimb lead leg more so than the right foreleg, although damage can occur in either or both front legs; as well as to the rear legs (Figure 2). Suspensories can be damaged in any one of their 3 separate anatomic regions: the proximal part (origin); the body; and the distal branches.

Clinical *tendonitis* is an inflammatory straining often preceded by a less obvious subclinical degeneration of the tendon or its sub-fibers. Affected tendons can be warm, swollen, thickened and/or locally painful to the touch and all are affected to varying degrees.

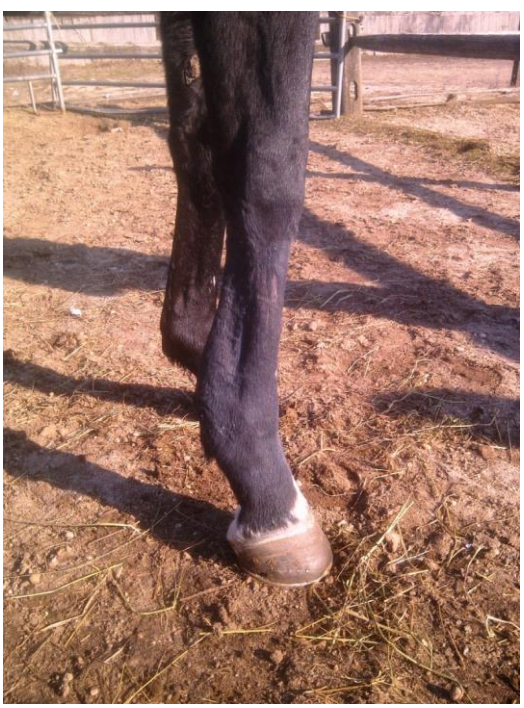

Figure 2. Typical clinical appearance of an injury in the distal region of the flexor tendons of the right forelimb.

An excessively bulging grossly visible posterior profile usually indicates more severe damage and is referred to as a 'bowed tendon'.

There may be unsoundness of the affected limb, especially as time goes on and as the pathology worsens. There can be inflammatory fluid filling of the sheath of tissue surrounding the Superficial Digital Flexor Tendon (tenosynovitis) without damage to the tendon itself. The suspensory ligament has no such surrounding sheath. Pathologic changes naturally occur in tendons in response to aging and they are accelerated with exercise. Cumulative fatigue micro-damage weakens tendon fibers and initiates clinical tendonitis. Sudden over-extension of the fetlock (ankle) joint may stretch the tendon and cause a significant mechanical disruption; along with severe unsoundness. Similar inflammatory changes and concomitant early degeneration of the suspensory ligament is referred to as *desmitis*.

Physical examination and diagnostic ultrasound are the chosen methods for determining the clinical significance of damage. There may only be inflammatory changes to the tendon or within the tendon sheath, however, there may also be structural tendon fiber disruption of varying degrees and/or varying sized holes within the tendon that are filled with serum and are called 'core lesions'. Ongoing enzymatic degradation and pressure necrosis affects the clinical significance of the damaged tendon area. Reassessment of the size, shape and location of these areas of damage determine the prognosis for successful therapy and a return to competitive athletic use. Repeat diagnostic ultrasound evaluations are necessary because initial damage may not be visible for several days, areas of damage (lesion size) can increase in time, and confirmation of healing is necessary in assessing an optimal rehabilitation program and to determine the best time to return to training or competition.

Therapy requires a multifaceted approach that reduces hemorrhage and the associated inflammatory response in the acute phase and encourages optimal fiber repair and alignment during the rehabilitation phase.

Normal tendons are composed of elastic type 1 collagen fibers, however, less elastic but similar type 1 collagen repair fibers along with less elastic type 3 collagen fibers are usually the body's natural repair tissues. The ultimate goal is to maximize repair with tissues of adequate strength and elasticity to return the horse to a competitive level of performance with a minimal risk of tendon re-injury.

Like cooking, successful therapeutic care of sprained, strained, torn and/or damaged tendons and suspensory apparati is as much a matter of using one's experience and creative talent as it is simply a generalized formula for treatment success. There is no one perfect way or formula to universally treat and rehabilitate all injured tendons or suspensory apparati.

In the past, some tendon or suspensory injuries (especially the minor ones) have healed well with little or no care i.e. akin to benign neglect; because nature inherently attempts to self-repair. Successful return to use by rest alone, however, does not substantiate the total validity of the 'do nothing –wait and see' approach.

Currently, rest alone is considered too conservative and insufficient, knowing the various adjunct therapies now available to improve or to surpass the 'do nothing' approach. Furthermore, in my opinion, local sweating, painting and/or blistering a previously cooled out injured tendon or suspensory still has its beneficial considerations and is still worthy of consideration. These same horses being treated cannot be expected to return to competition at close to previous pre-injury levels in less than 9 to 12 months' rehabilitation time.

With these openly understood limits, it can be worthwhile to rehabilitate a good horse with an injured tendon or suspensory apparatus.

Upon first identifying a damaged tendon or suspensory, it is *critical* to *respond immediately*. The more promptly attention is given to the situation; the more likely there will be a successful outcome. The statistics for a worthwhile outcome are nearly impossible to predictably pre-determine because the word 'success' has numerous connotations. In my experience a damaged tendon or suspensory nearly always sets up course for future ongoing soundness concerns, if the present level of use is continued. Many horses with an injured tendon or suspensory can be successfully rehabilitated to return to future performance soundness; however, they may from that point on lose a step or two, and be better suited to a slightly lower level of performance. There are exceptionally few horses that can heal and return to their maximum high level of peak performance; although it is definitely possible. Therefore the clinician must always be straightforward in their initial damage assessment with owners and trainers as to the degree of necessary medical intervention and rehabilitation, the expected costs incurred both in hard expenses and in lost training time, and a realistic prediction for future performance soundness.

The most important initial therapeutic response to an acutely injured or damaged tendon or suspensory is to attend to the bodies' immediate first response phase of injury: *inflammation*. In my experience, almost any method of promptly controlling inflammation is inherently appropriate. I believe in immediate cold hosing, applying ice, poulticing, and then supporting the injured leg with a very well-padded soft bandage. Use of a mini Robert-Jones bandage is highly recommended. It is also important to apply a well-padded support wrap to the uninjured contralateral leg. While arguably controversial, I immediately administer 18 mg (3cc) Vetalog®, a powerful steroidal anti-inflammatory medication, for the average 1,000 to 1,200 pound horse. Care must be taken not to overdose steroidal medication and to avoid the potential complications of digital laminitis and/or founder in either the affected or the contralateral support leg. To date, I have experienced no such contradictory results. The key goal in this immediate care state is to promptly bring the fire of inflammation and swelling under control and to provide adjunctive support to the affected leg(s).

Barring any extenuating circumstances, my first choice for foot care is to initially leave the original shoes in place on the affected horse for at least the first few days, so as not to create further imbalance or to impose greater trauma to either foot. Again my greatest concern is to first address the initial locally traumatic inflammation and swelling; and to stabilize the injured leg as best as is possible. Changing an affected horse's shoes is briefly delay only until a more appropriate time, unless those shoes are already loose and/or they detrimentally have excessively long toe grabs or heel stickers.

Unless it is extremely important to relieve severe pain or unless the damage is already determined to be irreversible, I purposefully do not give any immediate nonsteroidal anti-inflammatory medications, such as phenylbutazone (Bute®) or flunixin meglumine (Banamine®) to an acutely injured horse.

Unless their use is absolutely necessary, I initially avoid these non-steroidal anti-inflammatory drugs (NSAIDs) for at least the first day or two; and/or until the initial hemorrhage is considered under control; because of their equal ability to prolong the problems with additional bleeding. However, sometimes it is unavoidably prudent to immediately administer NSAIDs to counter the potential for laminitis in the unaffected contralateral support leg. This is one of the clinician's important choices.

If deemed appropriate, usually after the first 24 hours, I prefer to administer trichlormethiazide and dexamethasone (Naquasone®) diuretic/anti-inflammatory medication; bearing in mind that Naquasone® also contains a small percent of steroidal anti-inflammatory medication (dexamethasone). Care is always given not to cumulatively use too much steroidal medication in too short a period of time for the aforementioned reasons.

Only after the immediate inflammatory crisis is under control, will I next proceed to a more accurate assessment of the structural damage via diagnostic ultrasound imaging. Because the severity of inflammation and pain does not always correlate with the extent of the injury to the intratendinous structures, treatment and prognosis for tendinitis are based most rationally on ultrasonographic evaluation of the tendon. Therefore, you can't properly determine the true degree of damage, your chances for therapeutic success, or your therapeutic functional game plan without an accurate and definitive initial ultrasound diagnosis. Clearly, the less the structural damage incurred, the more optimistic the outcome, both cosmetically and functionally.

Within the first 12 to 24 hours I also like to initiate anti-inflammatory cold laser therapy (CLT). In the acute phases of care (during the 1st week), CLT is given twice daily for 5 to 10 minute treatments each. Cold lasers are known to have both anti-inflammatory and therapeutic effects. The beam of coherent light penetrates tissues and directly stimulates cellular metabolism and the release of pharmacologically active substances from cells that, in turn, indirectly enhance the inflammatory and repair phases of the healing process. The therapeutic effects are believed to include enhancement of protein synthesis, augmentation of blood flow, promotion of lymphatic regeneration and pain relief. Other proposed beneficial effects include the enhancement of cellular phagocytic activity, fibroblast proliferation and collagen production.

I will first treat the affected leg with a therapeutic cold laser for about 5 to 10 minutes; then I will cold water hose it for another 5 to 10 minutes (or sometimes visa versa); then poultice the leg overnight.

At about the same time, adjunct nutritional-medical supportive supplementation is given. This includes supplementation with the support of the physiologic building blocks for collagen repair and healing. Injectable polysulfated glycosaminoglycans (Adequan®) are administered intramuscularly once weekly for the initial seven weeks. This is coupled with feeding a chondroprotective neutracidal including both glucosamine and chondroitin sulfate. Cosequin® can be administered therapeutically at three scoops in the morning and three scoops in the evening (a higher than normal maintenance dose) for an average 1,000 to 1,200 pound horse. Other similar chondroprotective products should be dosed similarly. Hyaluronate sodium (Legend®) is begun intravenously to be given every 2 weeks, for the first few months.

Every effort should be made to feed a good quality ration. A proper ration should have sufficient proteins and roughage, and not include an exorbitant amount of simple sugars and carbohydrates; as can be found in the molasses-based sweet feeds. The goal is to provide healthy hearty nutritional building blocks, but to avoid potentiating or causing laminitis. Therefore fresh green grass and\or rich alfalfa products should initially be avoided for at least the first few weeks, if not months.

Within the first 3 to 5 days, preferably after the initial inflammatory phase is under control, evaluation and attention should be paid to any shoeing adjustments, only as is deemed necessary and appropriate for each individual horse.

I like applying a wide-webbed egg bar shoe set back posteriorly (long) in the heels, to at least the affected leg (if not to both legs thereby including the opposing support leg). This will give the horse's affected leg a larger and more stable landing surface which also relieves some of the tension on the leg's flexor surface. Side clips are a necessary component to this type of shoe to minimize it's coming loose from the foot. I also initially prefer to briefly and temporarily elevate the heel of the affected leg 1 to 2°, depending on the degree of damage, the level of residual inflammation, and the disposition of the horse. Too steep an angle of elevation is no good and leads to an abnormal balance between the extensor and the flexor surfaces of the leg. It also creates a greater chance of a non-correctable flexural contracture, thereby permanently shortening the flexor surface of the injured leg. However, raising the affected foot's angle even subtly significantly reduces some of the tensile forces on the posterior flexural side of the leg. This is initially important and helps reduce the local inflammation at the area of damage, and thereby also accommodates commencement of the healing processes.

In the past there has been surgical application of a superior check ligament (accessory ligament of the superficial digital flexor tendon) desmotomy to relieve flexor surface tension in horses with bowed or strained superficial digital flexor tendons. This described use and modified surgical approach was (get rid of the word first here) reported by Bramlage, subsequent to its classical approach as first published in 1986 by Jann, Beroza and Fackleman. The surgery was initially used to treat young horses that had developed significantly dramatic flexure deformities of the metacarpophalangeal joints from too rapid bone growth and unequal insufficient flexor tendon stretching. Generally speaking, most surgeons including the present author no longer use this procedure to treat tendinitis or core lesions of the superficial digital flexor tendon. However, its use is still engaged for treatment of flexure deformities of the metacarpophalangeal joints.

Generally at this time (after the initial 24 to 48 hours), I also begin a very modest exercise program of twice-daily hand walking for 5 to 10 minutes each. I often precede each therapeutic walking period with 5 minutes of local hand massage. Such massage is only as forceful as deemed appropriate for the degree of injury, swelling, and patient acceptability. Local massage is a physical form of therapy to move the stagnant blood through the leg, and it similarly also aids the lymphatic drainage of any toxic products that are otherwise being pooled within the affected leg. Following hand-walking, the legs are immediately cold water hosed (hydrotherapy) for another 5 to 10 minutes; followed by a similar treatment period with a cold therapeutic laser.

Again, the cold laser is herein important to reduce inflammation. The therapeutic goal is to begin a return to normal function as quickly as possible without creating a larger inflammatory event. Ideally I want to return the soft-tissue anatomic structures to their original anatomic proportions while maintaining their normal functional capabilities.

It is critical for the once formally resilient elastic soft-tissues to stay as compliant and elastic as possible. Yet the goal is for those tissues to still be as strong, if not stronger, than their original pre-injury levels.

Therapeutic ultrasound may also be added after all stages of inflammation are concluded; generally after the first or sometimes second week of care. Ultrasound used too early can cause blood flow stasis, endothelial damage, decrease collagen synthesis and locally elevate the tissue temperature too high. It can similarly contribute to inflammation, worsen edema and cause hematoma formation.

Therefore, it is very important that all internal tissue hemorrhage is first under control, before judiciously introducing therapeutic ultrasound. Therapeutic ultrasound is used 3 days per week, to allow healing time in between treatments.

When used appropriately, it is believed that therapeutic ultrasound causes a beneficial low level of circulatory stimulation, it promotes lymphatic drainage, and it increases collagen extensibility, protein metabolism, membrane permeability, enzymatic activity, and it raises the pain threshold. It may increase the tensile strength of tendons and increase the range of motion of joints by increasing ligament extensibility. Therapeutic ultrasound may reduce restrictive collagen cross-linking because the cross-links are soluble and easily denatured by heat. Ultrasound is added to the therapeutic regime just before any hand-walking is done so that following use of the stimulatory therapeutic ultrasound, treatment subsequently includes hand walking followed by both therapeutic cold laser and then cold water hosing. These later two therapeutic modalities subsequently have anti-inflammatory qualities, prior to the affected leg being bandaged.

I leave the initial elevated corrective shoe in place for only the first shoeing cycle of 4 to 6 weeks. Again, this is to minimize the chances for a permanent non-resolvable flexural contracture that might otherwise be created by leaving the shoe in place for too long a time period. After the first corrective shoeing cycle, I will often apply a second corrective shoe with approximately half the degree of elevation (i.e. from 2 degrees to 1 degree) as the original corrective shoe, to minimize any excessive forces being applied too quickly during the healing phase. By the next shoeing cycle, I like to have eliminated any elevated angle at all and to return the affected foot to a normal angle, still using the corrective wide-webbed egg-bar shoe.

I only give the amount of exercise that I believe each horse can handle. My theory is that all the involved soft tissues heal along the same accepted medical principles of structural dynamics as already recognized by Wolf's Law. All tissues heal in a manner conducive to the stresses placed upon them; so those tissues can later capably handle that type of stress. If the affected tissues are not minimally challenged on a consistent and appropriate basis during their maximal time of healing, they are believed to heal with weaker, more poorly organized and even more excessive scar tissue than if they are properly physiologically and physically challenged. While this belief and theory is heretofore only this author's personal as yet unproven clinical theory applied to the healing of damaged horse tendons and suspensories, the same principles have already been successfully expounded and applied for several decades to human rehabilitative sports medicine; especially beneficial in post-surgical orthopedic patients. The initial first 4 to 6 weeks of tendon or suspensory injury care is literally the most important portion to affect the long-term outcome of therapeutic success. This may be the reason why injured tendons and suspensories that are presented 1 to 2 weeks after they have occurred and especially after they have already been given insufficient initial care, disappointingly often don't heal to their maximal potential.

It may be prudent to diagnostically re-ultrasound the affected leg 4 weeks after injury, if for no other reason, only as a reality update of the situation. However, many significant therapeutic changes are often not so well appreciated this early in the therapeutic period. Rarely are my methods or perspectives for patient care affected (encouraged or discouraged) by the diagnostic ultrasound findings so early in the healing process. During the first 30 days of therapy, my clinical perspective on healing relies more on my physical assessment of the horse being treated.

However, I highly recommend a diagnostic ultrasound for damaged tendons by approximately 60 days of care and for suspensories by approximately 90 days of care. I fully expect at least a 50% resolution of the initial physical signs of damage of each by these post-injury time frames. In some cases, the initial damage is nearly completely unobservable (80% resolution) by these same time frames. My observations are that damaged suspensories heal at a significantly slower rate than do damaged tendons. My speculative reasons are that this phenomenon is due to the inferior blood supply that a suspensory apparatus has over a tendon. A tendon has both its own inherent blood supply as well as a peritendinous blood supply. The suspensory does not have an analogous peritendinous blood supply; only its own less pervasive blood supply.

There are other adjunct therapeutic options which can and should be considered in providing the entire treatment package to successful wound healing, as concerns the repair of damaged tendons and suspensories.

Some facilities have access to hyperbaric oxygen chambers which offer oxygen therapy. In theory, increased atmospheric oxygen is made available to the horse with the proposed belief that by inhaling higher levels of oxygen, higher levels of oxygen are made available to the local damaged tissues. There may also be increased oxygen available locally to open wounds. This principle in theory could potentiate the healing process. How accurate these proposed theories are in the horse and, if so, how economically affordable such options are is beyond the scope of my discussion. Oxygen therapy is believed to be rather costly. However, this option should be considered as potentially beneficial. It is my belief that oxygen therapy can begin nearly immediately after injury as there is little down side for its prompt implementation.

Additional therapies that have been explored, have been advocated, and are currently being used include injection of various local agents which are touted to stimulate the healing with repair by normally functional tissues. Nearly every group of researchers is presently trying to find the injectable ‘magic bullet’ to stimulate optimal repair of damaged tendons and suspensories. Of historic interest but no longer being used is Bapten® (B-aminoproprionitrile fumarate). This product was used extensively in the 1990s under the belief that it helped organize more normal repair tendon types and structures. It was believed to interrupt excessive cross-linking of fibers during the early phase of healing; which were known to be less elastic and re-tear more easily. Bapten® was credited with returning Cavonnier, a 1996 Kentucky Derby (G1) runner-up, to competitive racing. After many years of use as a heralded ‘Holy Grail’ for treating tendon injuries and after many patients were treated, it was finally pulled from the market. Many users found it to be ineffective or to not yield its purported long-term benefit, to have negative side effects, and to be a troublesome solution for treating bowed tendons. The most noteworthy point is, however, that associated with the select use of Bapten® in qualified patients; there was a concomitant extensive regime for physical therapeutic care. It is my personal professional opinion that the significant pre-treatment screening and case selection, combined with the simultaneous extensive physical therapeutic parameters that were mandated as part of the injectable therapeutic regime, were equally the major contributing factor to the initially positive therapeutic results reported in several patients. We may now be in a similarly analogous era wherein newer products are regularly being injected into damaged tendons and suspensories under the hope and belief that these agents are the essential newly recognized factors that lead to proper soft-tissue repair; the newly injectable ‘magic bullets’.

These agents currently being touted include: growth factors, platelet-rich plasma (PRP), both adipose tissue and bone marrow stem cells, Hyaluronan, and others. Including any one or combination of these primordial growth agents/factors into the composite therapeutic regime may be cumulatively beneficial. However, this subject will not herein be discussed as this author still remains a positively inquisitive skeptic about any potentially increased benefits afforded by use of these forms of adjunct injectable therapy.

Similarly, there are additional potential modalities of adjunct therapeutic care which may have significant potential to stimulate the healing of damaged tendons and suspensory apparati; i.e. shock-wave therapy. This physical means of treatment is believed to be beneficial in increasing the local blood flow to treated areas, thereby also increasing local circulation and perhaps lymphatic drainage, and concomitantly decreasing the perception of pain. However, akin to therapeutic ultrasound, this modality should not be used during the early inflammatory phase of soft-tissue damage and repair. There was historically an old veterinary treatment adage, to 'never add heat (liniments, paints or blisters) to a fire (i.e. a damaged inflamed tendon that hasn't cooled out yet)'. I believe this simple therapeutic principle to be true concerning the use of both shockwave and ultrasound therapy. I would not recommend use of shockwave therapy to a recently damaged area of acute concern.

Use of shockwave therapy has its place only after the immediate inflammatory phase has been brought under control (usually within the first 2-4 weeks). It may also be beneficial in those regions where healing is too slow or insufficient. However, there aren't presently any adequate studies to grade the degree of benefit afforded by the adjunct use of shockwave therapy. In my treatment regime, for the most part I do not regularly employ shockwave therapy because of my extensive successful use of cold laser therapy, therapeutic ultrasound and a unique proprietary method of core lesion reduction via radio-surgical techniques. Only after exhausting these options, do I consider adding therapeutic shock-wave therapy as an adjunct form of treatment.

If I do not appreciate at least a significant degree of healing i.e. 50% reduction of a core lesion by 60 days in a damaged tendon and by 90 days in a damaged suspensory, I highly recommend considering use of a technique exclusive to my practice only: core lesion reduction via radio-surgery. If I do not believe healing is progressing at a significant rate, I do not delay the use of this more aggressive intervention in favor of continued physical therapeutic care only. This would now also be the same appropriate time frame to initiate use of shockwave therapy, if not sooner, as deemed appropriate.

However, with client consent, my strongly recommended personal preference is to shrink the nonresolvable residual core lesion by using my unique radio-surgical techniques. These techniques are similar to my application of radio-surgery for cosmetically shrinking and tightening floppy displacing soft-palates. I try to correct therapeutic healing inactivity by converting the lesion into a more active area of healing. Essentially, I fulgurate (vaporize) the residual core lesion fluids (serum) to shrink the involved area of concern.

The technique also increases residual drainage of the formerly entrapped fluid and stimulates new blood vessel ingrowth. The procedure is done in our recovery stall, under a short-acting intravenous anesthetic protocol. Specialized radio-surgical equipment is used to ablate the residual core lesion which has been formerly identified via diagnostic ultrasound.

Physical therapy is delayed only on that single day of surgical intervention. A well-padded support wrap is applied to the leg immediately after surgery and it is left in place until the next day. This wrap may or may not be changed daily for only a few days, as needed.

All the previously mentioned therapeutic methods and treatments are continued, as described, for another 30 days and then a follow-up diagnostic ultrasound examination is done. Significant resolution (another 50%) of the treated lesion is now expected or else there is a real concern for therapeutic failure. Any lesions that persist 60 days after surgical core lesion ablation therapy render an ominous prognosis for a future return to any form of substantial athletic performance. In my experience, these are the injuries that will not be therapeutically successful for treatment and a reasonable return to use. However, I would herein employ concurrent use of shockwave therapy as a non-surgical means for trying to stimulate healing.

Prior to the introduction of any forms of injection, the historically preferred surgical method of aggressively treating 'bowed' tendons and thick chronically damaged suspensories was Forssel's method of tendon stabbing (1940s) and Asheim's percutaneous tendon splitting procedure (1964). In theory, these calculated surgical perforations and divisions of the tendon fibers was done to theoretically allow drainage of any serous fluids trapped inside a core lesion and/or to allow neogenic blood vessel ingrowth into a chronically unhealed area of soft-tissue damage. Newly modified methods of similar therapeutic design and intent include fine needle perforation of these same areas of tendon and suspensory apparatus concern. These techniques were generally beneficial for all soft-tissue injuries, with greater success being touted in treating cases of chronic desmitis rather than tendinitis.

The first stage of wound healing following damage is inflammation. This occurs so the body can send in new blood vessels to remove the degraded bad humors and allow the primordial clean-up cells to enter the injured area. By contrast, the current introduction of stem cells is done via a needle puncture which allows some drainage of the bad humors and direct injection of supposed primordial cells useful to expedite proper tissue repair. Similar speculations have been applied to the other locally injectable medications. None of these methods of treatment have yet been substantiated with hard scientific evidence. However, employment of these therapeutic techniques has the hopeful intent to help remedy any imperfections or inadequacies in the healing process.

For various and sundry reasons, historically relevant methods of treatment have included the use of liniments, paints, blisters and even pin firing. All these methods are aimed at increasing the local blood supply in an effort to speed healing. They were therefore only to be employed after the initial inflammation (inflammatory phase) was under control, and the previous warm and painful leg was totally cooled out. An additional theory for using methods of blistering and pin firing is to lay down increased amounts of scar tissue to strengthen the formerly damaged normal tissues. However, more modern assessment has negated their use because the scar tissue laid down is not normal and it lacks elasticity. It is therefore less resilient and it more easily ruptures again.

Furthermore, especially in regards to local pin firing, there is also a concern for increasing the degree of peritendinous adhesions and further restricting the normal elastic structure that is necessary for normal tendon function.

This similar concern for increasing peritendinous adhesions and restriction of normal tendon elasticity can be raised in applying the various techniques of tendon splitting. In theory, all therapeutic forms of tendon and suspensory treatment have their validating strengths as well as their offsetting concerns for insufficiency; and even their own inherent negative consequences.

Meanwhile, in regard to additional rehabilitation aspects of physical therapy, hand-walking is given twice daily for 5 to 10 minutes each day throughout the first 30 to 60 days of post-injury care. This is done throughout the entire period of rehabilitation in conjunction with all the aforementioned methods of first controlling inflammation, then controlling the laying down of functional repair tissues; thereby filling in any core lesions and remodeling any damaged or repaired tissues. After a successful diagnostic ultrasonic examination reveals a significant correction of the initial damage or advancement of healing; and after all outward physical signs of clinical concern are alleviated, the degree of physical therapeutic activity is increased. Such increased activity may consist of 1) increasing the amount of daily hand-walking; 2) slowly introducing initial lunging; 3) adding weights to the horses back and doing one or both of the first two options; 4) walking in sand and/or in water; 5) eventually having a rider only tack-walk in a gradually increasing level of rehabilitation; and 6) other methods.

There are additional options available to other doctors and other rehabilitation facilities such as treadmills, and under-water treadmills. The types of rehabilitation activity are limited only by ones imagination; so long as the goal is to slowly increase the amounts of stress placed upon the healing tissues, while simultaneously not increasing the stress beyond a horse's ability to handle such. Uncontrollable new inflammation should be avoided at all costs. During the initial period of physical therapeutic care of 60 days for tendons and 90 days for suspensory apparati, it is expected that the level of exercise should be increased. Return to use should occur as soon as is structurally possible, bearing in mind that some damaged suspensories heal more slowly than injured tendons heal. If a proper return to use, even minimal, is not initiated fairly quickly, the repair tissues laid down may not be sufficiently strong or properly resilient to return a horse to its former normal stressful level of activity at peak performance. That is why by this time period, it is important that all inflammation in the affected leg is gone and both feet should be shod at a normal angle; although a wide-webbed egg bar shoe would probably still be engaged in the healing interim to preserve the integrity and stability of the foot's landing surface.

In uncomplicated routine cases of tendon repair, by 90 days the level of activity is expected to be ready to be gradually increased as discussed in the aforementioned section. The same is applicable for suspensory repair by 120 days. By 90 days, the frequency of administering Adequan® may now be decreased to every other week, and Cosequin® may be slightly reduced to a supplementation at 2 scoops twice daily. Cosequin® supplementation stays at this new maintenance level throughout the first 4 to 6 months. After about 4 months, however, Adequan® may be further reduced to once monthly injections. Continued attention to physical therapy and rehabilitation is maintained, often at a slightly reduced level, for the next several months.

Rehabilitation therapy that is slowly orchestrated is important to securing maximum long-term athletic performance. Again, a lightly regimented program in which physically apparent tendon and suspensory healing might be expected would be by 6 months. Furthermore, the end of this time period of physical healing coincides with the expected beginning time period of tendon and suspensory tissue remodeling.

Therefore extreme patience should still be employed before commencing an aggressive training program. Return to the previous pre-injury level of use should only be considered after 9 months to 1 year of therapeutic rehabilitation.

Within this closely monitored conservative time frame, the earlier the horse's return to use the better the expected performance outcome.

Figure 3. Di's Time wins Correction Handicap.

Properly rehabilitated, even horses with initially appearing catastrophic injuries are still capable of a complete return to their previous level of athletic performance. It is ultimately important to always remember that advancing rehabilitation too quickly may worsen the problem; however, advancing rehabilitation too slowly decreases athletic potential.

As reported in my 9/10/00 presentation at the 20th Meeting of the Association for Equine Sports Medicine in New Brunswick, New Jersey; I have resolved several core lesions 80% within the first 60 days (tendons) to 90 days (suspensories). These lesions were essentially gone (90% reduction) by 90 days (tendons) and 120 days (suspensories).

An early return to light training was often attainable at approximately 120 days (tendons= 4 months) and 150 days (suspensories= 5 months).

Racehorses usually resumed full training after 6 to 8 months, respectively; and raced competitively at 8 to 12 months, respectively. There was significant individual variation.

The majority of patients that returned to full performance were not sidelined by recurrence of the original tendon/suspensory tear.

One 3 year old thoroughbred racing filly with type-3 core lesions of both distal branches of her superficial digital flexor tendons of her left hind leg returned to racing at 4 and 5 years old, after 10 months of therapy. She raced 20 more times and earned another $200,000; including successfully competing in allowance and grade 2 and 3 stakes races (Figure 2 and 3).

Start-by-Start Past Performance Reports (#5043)

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5043 For DI'S TIME 1995
On 08-Sep-2000.
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DI'S TIME            CH. F. 5, by GILDED TIME-YALLAH MISS, by YALLAH NATI
==========           Br.-BLOODSTOCK LEASING IN(KY)   2000   4  1  0  2    $92,383
Own-SCHURMAN JAMES Tr-SCIACCA GARY                   1999  11  3  4  0   $110,430
               LIFETIME  19  6  4  3   $241,793     TURF   1  0  0  0         $0
                      PP ST First    Second   Stretch  Finish
25Mr00AQU8    ft 7f                                                fDistaffBC H-G2
 :22.4  :45.1 1:22.0  8  1  7- 5.25  6- 5.50  6- 3.75  4- 3.75  Lb  20.60  86-19
19Fe00LRL9    ft 7f                                                  fBarbFrtchH-G2
 :22.2  :46.0 1:24.3  9  4  5- 6.50  6- 3.25  3- 4.00  3- 2.25  Lb  10.00  82-17
05Fe00AQU9    ft i6f                                                  fCorrectionH7
 :22.2  :44.3 1:09.3  7  8  8- 5.75  5- 5.50  4- 1.25  1- 1.75  Lb   7.00  94-11
01Ja00AQU6    ft i6f                                               fInterborgh H-G3
 :23.0  :45.4 1:10.3  2  7  6- 5.00  4- 4.00  4- 2.00  3-  .75  Lb   4.40  88-12
05De99AQU7    ft i6f                                                 fAlw47000n3x
 :22.1  :45.1 1:10.0  4  5  6- 3.50  3- 2.00  1-   hd  1- 3.00  Lb   8.40  92-11
12No99MED1    ft 6f                                                  fAlw30000n3x
 :22.1  :44.3 1:09.3  5  4  4- 2.00  4- 5.00  4- 3.50  2- 1.75  Lb   2.90  89-10
21Oc99MED7    gd 6f                                                  fAlw30000n3x
 :22.1  :45.1 1:10.3  5  2  4- 2.50  4- 3.50  5- 5.50  4- 7.25  L    4.30  79-20
11Ag99SAR3    ft 6f                                                    fClm65000
 :22.0  :45.2 1:11.0  2  5  3- 3.50  5- 4.50  5- 6.50  5- 9.25  L    3.60  77-13
23Ju99BEL5    ft 6.5f                                                  fClm50000
 :23.0  :46.1 1:17.1  1  6  4- 1.00  4- 1.25  2- 1.00  1-   nk  L    7.90  86-14
31My99BEL8    ft 6.5f                                                fAlw46000n3x
 :22.2  :45.2 1:17.1  5  8  6- 4.50  8- 9.25  8- 8.75  8-14.25       8.90  72-15
06My99AQU6    gdT1 1/16                                              fAlw47000n3x
 :49.3 1:14.0 1:45.0  5  2  2- 1.50  5- 6.50  7-14.00  7-22.00       6.70  64-14
11Ap99AQU7    ft 7f                                                  fAlw44000n2x
 :23.2  :46.4 1:23.4  3  3  3- 1.50  3- 1.50  2-   hd  1-   hd       2.45  83-17
04Mr99AQU6    ft i6f                                                 fAlw42000n3l
 :22.4  :46.1 1:11.0  1  6  3- 1.00  4- 1.25  3- 1.50  2-   nk      *1.00  87-16
21Fe99AQU7    ft i6f                                                 fAlw42000n2x
 :23.1  :46.2 1:10.4  8  3  5- 2.75  5- 3.00  4- 4.00  2- 2.75      *2.65  85-16
27Ja99AQU3    gd i6f                                                 fAlw42000n3l
 :23.0  :46.1 1:10.4  1  5  2-   hd  2- 1.50  2- 3.00  2- 5.25       3.60  83-19
16De98AQU8    ft i6f                                                 fAlw40000n1x
 :23.0  :46.4 1:12.0 11  3  6- 5.25  6- 4.00  3- 3.50  1-  .50      20.70  82-18
14No98MED6    ft 6f                                                      fMdSpWt
 :22.0  :45.3 1:11.0  4  4  3- 2.50  1-   hd  1- 4.00  1- 3.75      *0.40  84-15
25Oc98BEL2    ft 7f                                                      fMdSpWt
 :22.4  :46.3 1:24.4 10  4  3-  .50  2-  .50  3- 7.00  6-17.50       7.60  60-20
27Sp98BEL2    ft 6.5f                                                    fMdSpWt
 :23.0  :46.2 1:17.1  3  6  5- 1.50  2-  .50  2- 4.50  3- 8.50      14.00  77-16

Works:  Mar21 BEL tr.t 3f ft :37.2 B     Mar09 BEL tr.t 5f ft 1:03.0 B

END OF REPORT
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Figure 4. Di's Time past performance report.

A 3 year old thoroughbred racing stallion with a type-2 core lesion of the 2AB area of his right front superficial digital flexor tendon returned to racing after 1 year; having 10 starts and earning $28,000.

A 4 year old thoroughbred stallion that was being treated for a type-3 core lesion of his right forelimb superficial digital flexor tendon died of unrelated causes after an initial 75 days of rehabilitation therapy. His right forelimb was subsequently sent to the University of Pennsylvania's New Bolton Center Pathology Laboratory for their independent evaluation of the area of former injury and current treatment.

Their findings were, "Hyalinized or mineralized large caliber collagen fibers were not present in the affected fascicles. Repair has occurred in a very organized fashion, with no debris remaining from degenerate collagen fibers."

The less severe the initial damage, the more immediate the presentation, the more appropriate the subsequent therapy, the lower the expectations for performance soundness, and the greater the continued trainer compliance subsequent to patient discharge; the greater the successful results are expected to be. However, the costs incurred (commitment, time and finances) to be successful are very significant. It is believed that a rapid return to controlled exercise, aggressive physical therapies and nutritional support to maximize fibroblastic production of collagen, yielded the best results. Committed owners and trainers need to know that at least 120 to 150 days of intense expensive rehabilitation are needed before a horse can even be considered for a return to a well-monitored level of athletic performance.

Currently there is no consistently successful, universally agreed upon treatment for tendonitis or desmitis in any performance horse. Reported use of newer adjunct treatments appears to be 'promising' but as yet 'inconsistent' and unproven.

Most treatment regimes are combined with one or another form of medical therapy, a controlled exercise program, and constant ultrasonographic re-evaluation. Regardless of the methods of therapy, the time before a return to active training due to a clinically significant tendonitis or desmitis can be many months (6 to 12 months) and is very dependent on the severity of the initial injury. Additionally, the chance for a repeat incidence of tendinitis or desmitis following recovery and a return to use is significant and increases in direct relationship to the size and severity of the original area of injury (lesion), the reasons for the initial problem, and the degree of stress of the intended return to athletic performance.

In: The Equine Tendon in Health and Disease
Editor: Henry Jann
ISBN: 978-1-62808-048-3

Chapter 7

PROFILING, PHYSICAL CONDITIONING, AND PREVENTION OF TENDON INJURIES

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ABSTRACT

Preventing tendon injuries is a goal that is critical to any conditioning strategy. This chapter discusses the common etiologic factors associated with tendon injuries. Unfortunately some of these factors are difficult to avoid in the context of a rigorous competitive career. The more we are aware of these predisposing factors; hopefully the better we will be able to avoid them. The longevity of an athlete's career is directly related to the ability of avoiding injury. This chapter discusses the factors that cause tendon injury and how to avoid them.

PROFILING

Profiling overstrain tendon injuries refers to a process of accumulating all the surrounding circumstances present when an injury occurs. This is in fact difficult because the actual initial injury may have gone unnoticed and the recognized injury could have occurred in a variety of situations that actually have little bearing on why the initial injury occurred. Profiling tendon injuries may be only a part of the pertinent epidemiologic issues. The occurrence of a tendon overstrain injury is the result of an accumulation of contributing factors. Epidemiologic studies have clearly shown that the incidence of tendon injury increases with age. [1] There is also a positive correlation with male gender [1] and hardness

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of track. [2] The important aspect with regards to profiling is not merely to focus on the events immediately surrounding the injury.

More importantly we must attempt to characterize the details of the training strategy with all its intricate components. Research has shown that tendon injuries are not caused by a single traumatic event but rather the accumulation of trauma leading to alterations in cellular metabolism caused by altered strain patterns imposed on tendon cells. The result is collagen degeneration, weakness, and microscopic failure. Accumulation of micro damage eventually results in gross failure of large portions of the tendon. Therefore what is recognized as an isolated event is actually the accumulation of injury at the cellular and metabolic level. [3-5]

The common etiologic factor in tendon injury and the one that we can control is the conditioning strategy of the individual horse. Conditioning a horse for competition is viewed superficially as something simplistic and routine.

The reality is that this process is extremely complex and requires a high level of expertise and experience. All horses are individuals and respond to conditioning differently. A conditioning strategy that may be appropriate for one horse may either be too strenuous not strenuous enough for another (Figure 1 and 2).

Strategies that are too strenuous will invariably result in injury. In contrast, strategies that are not strenuous enough will not provide sufficient physiologic stimulation to cause up regulation of cardiovascular, thermoregulatory, endocrine, and musculoskeletal systems required to improve athletic performance.

Specific components of the conditioning strategy that are relevant to overall preparation for competition are: fitness level, volume of exercise, training surface, shoeing technique, and overall health.

Often when any of these factors is compromised the result is musculoskeletal injury – one of the most common is an overstrain tendon injury.

Figure 1. Conditioning a horse for competition requires a high level of expertise and experience.

Figure 2. Horses respond to conditioning differently which is why uniquely tailored conditioning strategies are necessary.

Let us examine each of these factors:

Fitness Level

The fitness level of a performance horse is a factor that must be foremost in the trainers mind. All training protocols or conditioning strategies must be designed around the fitness level of the individual horse. What is crucial here is the trainer's ability to judge the fitness of the horse. This can be done using heart rate monitors or blood lactate levels (VLa4 or La9). Heart rate monitors can indicate the level of exertion by determining the per cent of maximum heart rate at which a horse is performing. Also recovery after exercise can be determined by heart rate. VLa 4 is the velocity a horse is travelling when its blood lactate reaches 4 mmoles/liter. VLa 4 increases with fitness i.e. conditioning will raise the VLa 4. Also the La 9 is the lactate level at 9 m/sec. A fitter horse will have a lower blood lactate at 9 m/sec that an unfit horse because it will be functioning on more of an aerobic energy generating metabolism instead of an anaerobic energy generating metabolism – i.e. it will not have exceeded its anaerobic threshold. In the absence of these more technologically advanced monitoring techniques a trainer can evaluate fitness by the subjective assessment of fatigue resulting from various levels of exertion. Also it is reasonable to assume that fitness acquisition requires certain duration of training to achieve. Trainers learn how to predict how long an individual horse will require to achieve a desired level of fitness. Rushing a horse through a conditioning strategy will invariably result in a lower level of fitness than is required for a given discipline and an increased probability of injury.

The key to any conditioning strategy is establishing a foundation and then building on this for specific disciplines by advanced and specialized conditioning.

The trainer must realize that fitness is not a constant physiologic characteristic. The fitness of a horse varies and a high level of fitness requires a constant conditioning program. The trainer must judge the fitness of the horse before adjusting the fitness program for a given competition.

Volume of Exercise

Horses are most prone to injury when the volume of exercise is inappropriate for or exceeds the fitness level. There are two basic training errors that allow this to happen. The first is an acute increase in exertion that is referred to as "over reaching". The overall increase in the volume of exercise may result from an increase in duration, intensity, or frequency of training that exceeds the fitness level of the horse to the extent that the fatigue level is high. Over reaching can occur when a horse is asked to exceed its fitness level either in a single training exercise or in competition. The resulting high level of fatigue results in an increased probability of injury.

Over reaching is a common training error that is usually avoidable. A skilled trainer knows the limitations of a given horse and is careful not to exceed them. When overreaching is imposed on a conditioning strategy, it is imperative that sufficient rest is provided to allow complete recovery before subjecting the horse to further exertion and fatigue. After an episode of overreaching the trainer must be especially observant for signs of injury. Also special therapeutic measures such as massage, ice, or hydrotherapy are often helpful after an episode of overreaching.

The other more insidious conditioning error is overtraining. In this situation the volume of exercise is too great for an extended period of time. Over training results in chronic fatigue and a down regulation of the hypothalamic pituitary adrenal axis. In this situation both bone and soft tissue structures are not allowed time to adequately remodel and slowly begin to degenerate. In tendons the result is a series of microscopic tears that eventually accumulate and cause the tendon to weaken and rupture. All performance horses should be carefully observed for signs of overtraining. The most fundamental characteristic is a horse that just seems tired, lacks energy, and fails to improve performance in spite of continued attempts at conditioning. Often there is a change in the horse's personality and it seems unwilling or resentful to exert itself. There may be several subtle lameness problems that arise along with conditions affecting other systems like equine gastric ulcer syndrome (EGUS) and inflammatory airway disease (IAD). The onset of overtraining is insidious but once it has developed it is difficult to reverse without long periods of greatly reduced exercise and even possibly complete rest with discontinuation of conditioning.

Training Surface

The contribution of training surface has been contradictory in the literature. Both hard and soft surfaces can contribute to tendon injury. The important aspect is that a properly maintained and safe training surface does not by itself produce injury – fatigue resulting in hyperextension of the metacarpophalangeal joint causes injury. Training surfaces can produce fatigue in different ways. Surfaces that are deep and soft create fatigue by causing the horse to

utilize more energy at a given velocity. Hard surfaces require less energy to travel at a given speed and horses can therefore travel faster for longer distances on a hard surface. Increased speed creates more concussion on all the components of the musculoskeletal system and therefore increases the probability of injury. Also increased speed causes increased fetlock hyperextension and increased strain on the superficial digital flexor tendon. Therefore both hard and soft surfaces can indirectly contribute to tendon injury. Synthetic racing surfaces add consistency and a stable interface for the hoof that has been reported to reduce the overall injury rate. [2, 6, 7] Changes in the track surface that a horse has become accustomed to can result in injury. For example training on a synthetic track and racing on a hard track or training on a hard track can contribute to injury because the musculoskeletal system is subjected to forces (both concussive and strain) that it has not been previously accustomed to. Recently, synthetic surfaces have been associated with an increase in the rate of loading of all components of the musculoskeletal system including the tendons. This is because there is less, slide as the hoof makes contact with the surface and biomechanical forces are generated at a faster rate. This increases the probability of injury because the faster a structure is loaded the higher the probability of damage or failure. Therefore any change in training surface should be accompanied by an adjustment period of several weeks. Often this is not possible and the end result is an injury.

Shoeing Technique

Of all the management practices involved with athletic horses, shoeing is among the most important. The principals are simplistic but all too often ignored. The principles can be categorized into three major concepts 1) hoof balance, 2) shoe fit, and 3) shoe characteristics. Each of these will be discussed independently. *Hoof balance* – must be evaluated in the mediolateral and dorsopalmar perspective. When a hoof is balanced in the mediolateral perspective both the medial and lateral heel and hoof wall are the same length. This allows both heels to land simultaneously when the horse walking over level ground. When a foot is out of balance there is a tendency for the hoof to land on one side first before the other side. This obviously creates uneven biomechanical forces that are detrimental to all the bony and soft tissues components of the distal limb. When the hoof is out of balance in the dorsopalmar perspective the hoof pastern axis is broken either in the dorsal or palmar direction. Deviation in the palmar direction is the most common condition recognized in performance horses. Palmar deviation results in the condition recognized as under run heels or long toe low heel conformation. The long toe low heel conformation is especially dangerous for horses than move at speed when they are fatigued i.e. race horses and endurance competitors. This conformation increases the work of break over causing increased strain on the digital flexor tendons and has been associated with tendon injury and catastrophic breakdown.

Also this conformation predisposes to fatigue and hyperextension of the fetlock joint that can result in tendon and suspensory ligament damage. When the hoof pastern axis is broken in the dorsal direction the resulting conformational defect is a club foot with high hoof angle.

This conformation is not desirable because it subjects the hoof wall, navicular apparatus, and coffin bone to abnormal or exaggerated biomechanical forces that are detrimental to the longevity of the athletic career. *Shoe Fit* – Shoes that are too small do not provide enough heel support. This tends to create soreness in the heel area. Shoes that are too big create

abnormal braking or torsional forces on the distal limb that can result in injury. *Shoe Characteristics* - In spite of the large variety of disciplines and shoe characteristics some basic principles apply. The most important concepts to remember are to use the lightest shoe possible that will still afford sufficient protection and to avoid traction devices like toe grabs and heel calks. Heavy shoes are undesirable because weight causes fatigue and hyperextension of distal joints and increased tendon strain. Traction devices are undesirable because they produce severe breaking action on the hoof that increases concussion and overall strain of the hoof wall and distal boney and tendinous and ligamentous structures. Toe grabs are the traction device that has received the most attention and the consensus is that a toe grab should not exceed 2 millimeters in height and are only useful on the hind feet. Toe grabs on the front feet actually produce a breaking and jarring effect that results in fatigue, hyperextension, and injury of tendons and other musculoskeletal components of the distal limb.

Overall Health

Ideally horses that are competing in athletic endeavors should be in an anabolic state. They should be in a high plane of nutrition and have high energy levels. The condition that should be avoided is chronic fatigue. Health issues like exercise induced pulmonary hemorrhage (EIPH), IAD, EGUS, and musculoskeletal problems also have a negative impact on performance. These conditions are all directly or indirectly related to over training, chronic fatigue, and a deterioration of overall health. The major problem to avoid is overtraining because it is detrimental to all aspects of performance and will eventually lead to withdrawal from competition due to poor performance or injury [8, 9].

PHYSICAL CONDITIONING

The most important aspect of any conditioning program is developing a foundation. This concept can best be understood by thinking of foundation as getting in shape so you can get in shape. Foundation is critical to any conditioning strategy – without a solid foundation the probability of injury increases and the overall effectiveness of any conditioning strategy decreases. Without an appropriate foundation it will be very difficult to achieve a high level of fitness and in all probability the horse will sustain an injury. Foundation (referred to as "base" by some trainers) requires time, skill, and appropriate training facilities. There is no set formula for establishing a foundation other than it will take approximately 12 weeks and progresses through a period of walking, trotting, hill work, and galloping.

All of these phases are achieved without overly fatiguing the horse. Basically what we are trying to do is cause the horse to exert itself without depleting energy reserves or causing over use repetitive injury or degenerative injury to soft tissue structures. Another important consideration is to avoid excessive stress on subchondral bone resulting in sclerosis and loss of compressibility and shock absorption.

This increased stiffness leads to micro fracture of the subchondral bone and degeneration of articular cartilage and eventually osteoarthritis. Therefore the level of exertion must be

advanced gradually allowing time for physiologic and structural adaptation. As the horse advances into the foundation program it is able to perform increasingly more strenuous tasks with less physical effort. At the end of 8 weeks the horse should be able to trot 5 consecutive miles at approximately 4 m/sec (9 mph) without any noticeable fatigue. At the end of a 12 week program the horse should be able to do five consecutive repetitions of one half mile trot and slow gallop intervals without noticeable fatigue. Foundation varies little for different equine disciplines. Some individuals are able to advance through the foundation stage with ease; others have difficulty obtaining this basic level of fitness. It is actually in the foundation phase of conditioning that individuals that have athletic potential are identified. If an individual is unable to complete this phase of conditioning, it is unwise to continue on to the more intense specialized conditioning for specific disciplines.

After successfully completing the foundation phase of conditioning individuals are advanced into specialized strategies that will optimize performance in given disciplines. The three components of advanced conditioning are speed, strength, and stamina. Different disciplines require different specialized advanced conditioning; for example the western performance events emphasize speed and strength while Thoroughbred race horses require speed and stamina. Dressage competition requires strength. In order for a horse to achieve a high level of specialized conditioning it must have established a solid conditioning foundation or specialization will be unsuccessful.

PREVENTION

The key to prevention of tendon injuries is avoiding all the things that have been discussed as causative factors. The primary factors to avoid are overreaching, overtraining, inappropriate shoeing, and over exertion on surfaces that the horse is not accustomed. With regards to overreaching, this is a mistake that is often made but is easily avoided. A trainer that is familiar with the conditioning history of a horse knows what the horse can handle comfortably and without risk. Exceeding this limit runs the risk of injury. The problem comes when the trainer is not familiar with a given horse or when competition or owner pressure forces the trainer to impose an overreaching situation on a horse. If this is done often enough the probability of injury is high. The reality is that horses are often subjected to overreaching situations. This occurs because of the time and expense of preparing a horse for competition and the extreme nature of most equine disciplines that make overreaching situations almost inevitable. Overtraining is another conditioning error. In the process of conditioning a horse the trainer must walk a fine line between training and overtraining. The conditioning strategy must be intense enough to cause the physiologic capabilities to up regulate but not so severe that the overall health of the horse is compromised.

It is the skill and experience of the trainer that prevents a horse from being subjected to an overtraining situation. Even under the most cautious training regime overtraining can occur. The hallmark sign of a horse that is over trained is chronic fatigue and a failure to thrive. Basically the volume of exercise is too great and the horse is unable to adapt to it physiologically.

With continued training the performance does not improve and actually can deteriorate. This horse is very prone to injury because he cannot respond to conditioning and is in a

catabolic energy balance. Injuries will heal slowly if at all and a horse subjected to this kind of conditioning strategy will invariably have a shortened athletic career caused by musculoskeletal injury or other health problems. Appropriate shoeing is another critical factor in prevention of tendon injuries. This topic is discussed in the section on shoeing.

A change in track surface is another thing to avoid. A horse that is unaccustomed to a surface over which he is asked to perform strenuous or fast work is liable to misstep and become injured. Trainers should be aware that the horse should be given an adjustment period before being asked for strenuous effort on an unfamiliar surface.

The reality is that under current training methods and competition demands it is very difficult to prevent tendon injuries. The race horse particularly functions very close to the threshold of physiologic limits and biomechanical failure. This extreme level of physical activity is detrimental to the structural integrity of the overall musculoskeletal system. This is evidenced by bone injuries like sclerosis of subchondral bone along with chip and condylar fractures. Also degenerative joint disease results from this king of overuse. Tendon degeneration is an accumulation of micro damage that begins when the horse is put into race training and can result in mechanical failure. The trainer must not add any more stress on an already jeopardized biological system composed of interdependent structures. The key factor that relates to prevention of tendon injuries is to recognize additional stress factors. The most important of these factors is speed. Any fast work requires several days of recuperation. Also a horse's ability to function at speed without injury is something that must be developed slowly. Things like altered racing surfaces, erratic conditioning schedules, inappropriate shoeing, and inadequate recovery time are all stress risers that increase the probability of tendon injury.

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In: The Equine Tendon in Health and Disease
Editor: Henry Jann
ISBN: 978-1-62808-048-3

Chapter 8

EQUINE REHABILITATION

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ABSTRACT

Rehabilitation is crucial to an athlete's recovery from injury or illness. In humans, doctors prescribe physical therapy as an essential component of the patient's recovery after surgery or illness. Without adequate rehabilitation, science has shown that outcomes can be disappointing.

In equine veterinary medicine, rehabilitation plays a vital role in returning a horse to its occupation or, in extreme cases, to restore its quality of life after a career-ending injury. Recently, the philosophy of rehabilitation has evolved to emphasize "prehabilitation," a protocol that addresses potential problems before they compound to diminish performance.

KESMARC KY

In 2001, the Kentucky Equine Sports Medicine and Rehabilitation Center (KESMARC KY) became the world's first facility dedicated solely to the rehabilitation of the horse. Its origins were in Pilot Point, Texas, where, in 1992, I established a layup facility at my ranch that featured the first underwater treadmill in Texas and the first equine hyperbaric chamber outside a veterinary setting and only the second one in use in the horse industry. In 1997, Douglas Herthel, D.V.M., pioneered equine hyperbarics at his Alamo Pintado Equine Medical Center in Los Olivos, California. In 2000, he introduced a prototype equine hyperbaric chamber and adapted human hyperbaric medicine to benefit the horse. A collaboration with Dr. Herthel enabled us to bring hyperbarics to the horse industry.

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The Pilot Point facility was simple, but the philosophy that enabled me to achieve exceptional results in rehabilitating horses was unprecedented. Soon, in response to industry demand, I relocated my endeavor to Lexington, Kentucky, and founded KESMARC KY in nearby Versailles. Since then, rehabilitation centers all over the world have used my facility as a blueprint.

In 2013, KESMARC KY's facility includes an equine swimming pool, inground underwater treadmill (generically referred to as an aquatred), inground cold-saltwater spa, freestyle automatic walker, whole-body vibration platform, and indoor jogging track. The jewel of its program is a state-of-the-art equine hyperbaric chamber. With the exception of round pens and turnout paddocks, the entire facility is housed under one roof.

Safety and Biosecurity

Anyone can purchase a list of equipment, erect it on a venue, and advertise as an equine rehabilitation center. Without a team with an intimate knowledge of the horse combined with training and extensive experience in the modalities offered, the results will be ineffective and potentially disastrous. The key responsibility of every rehabilitation center must be the safety of the horse and handler—both physical safety and biosecurity. Injuries to horses and/or personnel because of poorly designed facilities or lack of horsemanship skills cannot be permitted. Any time you add the horse to any equation, everything changes, so a careful approach needs to be taken with the horse in mind.

Great thought was given to the planning and construction of every part of the KESMARC KY facility. I began by choosing a suitable, existing, training farm with a properly constructed pool and indoor jogging track and then retrofit it with additional equipment. My team and I discussed the worst things that potentially could happen when a horse was introduced to each situation and modality. Being experienced horsemen, we realized the likelihood these scenarios would occur, so we put every precaution in place to prevent them and to keep the horse safe.

Every piece of equipment, area of use, and building at KESMARC KY was designed with safety in mind. None of the modalities was built to fit a particular space for convenience. They were methodically looked at with the horse's behavior in mind and asking the questions: What will the horse do when put in this situation? What is the worst-case scenario or crisis that could arise? With these answers, each modality and its setting were designed to prevent a catastrophe. You can design and build the safest physical plant and have every precaution in place for all aspects of safety and biosecurity, but all that work is wasted if you do not have experienced horsemen as handlers. Every modality holds a unique set of fears for a horse. With improper handling, a fearful horse can panic, creating a dangerous situation for itself and its handler.

Biosecurity

Biosecurity is as important as anything we do to ensure the physical safety of horses and personnel. Breach of biosecurity that endangers horses and/or personnel can and should cause a facility to shut down.

Since KESMARC KY's inception, one outside veterinarian with a focus on infectious diseases and biosecurity has been in charge of monitoring the entire facility year round. This includes keeping abreast of disease outbreaks that occur anywhere in the world, designing biosecurity protocols for the facility, training personnel in their proper implementation, and safeguarding every horse in the facility against communicable disease.

This veterinarian oversees biosecurity for every case within the facility, but he does not take over a case from the attending veterinarian. He is kept in the loop on anything in the facility that is of question or concern. We have handled biosecurity successfully in this manner since the facility's inception.

Biosecurity pertains to situations outside the facility as well as in house.

KESMARC KY is vigilant in the awareness of outbreaks anywhere in the world and will not accept a horse from a problem area or facility until the outbreak is resolved or addressed. When we are aware an outbreak exists, we question vanners who are scheduled to deliver horses to our facility about any possible exposure to the outbreak. This includes other scheduled pickup locations for the same van and the provenance of other horses riding on that van.

Based on what we learn about an outbreak, we institute a heightened level of biosecurity to protect our client horses, personnel, and facility. At the same time, we do not divulge confidential information we learn about an outbreak until it is officially made public.

At KESMARC KY, all personnel are trained in the proper handling of everything that comes in contact with the horse—from the cleaning of water buckets to the disinfection of chains, leads, brushes, stalls, etc. Biosecurity measures are in place at all times, and stepped-up levels of biosecurity are instituted when we perceive a potential problem.

THE KESMARC KY APPROACH

The horse is the most compensating creature on the planet. To support its massive size and weight, the horse will employ every part of its body and mind to cope with anything that interferes with the delicate balance and symmetry of movement.

When the horse's body is in balance, muscles, tendons, ligaments, joints, and hooves share the load equally. This is called synchronization. When excess stress is placed on a particular component, synchronization is lost, stressed areas begin to fatigue, and something gets overloaded. If the balance of the horse's body is not restored, injury results.

Because horses are individuals, cookie-cutter protocols cannot be applied to every horse with the same injury. For example, just because a horse walks through the door with a bowed tendon does not mean that all we do is address the tendon. We treat the entire horse as a puzzle—figuring out all the factors that caused the injury—and put the puzzle back together simultaneously so that when we address that tendon, we also address why that tendon bowed.

Sick or injured horses come to KESMARC KY for special care that helps their veterinarians achieve better results. Horses with difficult or complex wounds, fire survivors, horses with challenging infections, ones recuperating from surgery, and horses with musculoskeletal or airway problems are just some of those we've helped.

At KESMARC KY, we take a team approach to healing, with the horse's attending veterinarian taking the lead.

The attending veterinarian has access to an array of highly experienced professionals in podiatry, dentistry, nutrition, acupuncture, chiropractic, sports therapy, radiology, surgery, internal medicine, and more. The best equine professionals and facilities—hospitals, laboratories, pharmacies, etc.—in the world are in the Blue Grass area and at the attending veterinarian's disposal. The attending veterinarian designates which professionals will evaluate the horse and report their findings. Based on these results and assisted by input from the KESMARC KY staff, the attending veterinarian then designs a program for the horse using the modalities that best will achieve the goal.

When a horse arrives at KESMARC KY with an unresolved condition, it most likely is a tough, complex case. With the team approach, fresh eyes look at these horses, making it easier to figure out the real problems and address them. Several specialists from different veterinary practices may work together to assist the attending veterinarian.

Horses arrive at KESMARC KY under a variety of dynamics. Some ship in with an attending veterinarian; some arrive with instructions that a specific local veterinarian will supervise the case and report back the client's farm veterinarian; other horses ship in directly from the owner or the trainer with a request that we select a local veterinarian with expertise in the horse's particular ailment to oversee the case; yet others arrive with a program already laid out by the horse's farm veterinarian.

When working with the attending veterinarian to design a rehabilitation program, I keep the client's budget in mind. Some owners send their horses to us with a virtual blank check and the directive to do whatever is in our repertoire that will help return the horse to its former level of health and performance. For other owners, I work closely with them and their veterinarians to put together an effective program that will be agreeable financially to all parties.

Outcomes are everything in equine rehabilitation. Horses come to KESMARC KY with high expectations by the owner and the attending veterinarian because we are held to a higher standard of care than a traditional layup facility. My responsibility is to explain the realistic outcome we expect based on the experience we have had rehabilitating horses with a similar condition.

KESMARC KY's success is due in large part to our dedicated staff. In addition to being the best and most experienced technicians in their fields, everyone at KESMARC KY knows horses and horsemanship. They forge a bond with each horse in their care, and this trust allows the horse to progress more confidently through its individual program.

The KESMARC KY Staff

Each staff member brings unique skills to the team and is respected for his or her expertise and opinion. Every employee at KESMARC KY has a voice and an important role in the care of the horses at the facility. Constant communication and group meetings address the needs of each horse. Any concern is reported to me and addressed. I always have the final say on how to proceed, but I respect the opinions of all involved.

Keeping a rehabilitation center running safely and smoothly is a complex endeavor. My staff is the most experienced in the field, but only because we have been at this work the longest of anyone in equine rehabilitation, and the members of my staff have been with me since the beginning.

KESMARC KY's staff is restricted to individuals with an intimate knowledge of horses and horsemanship, plus years of experience in their respective fields. Two key personnel top that list:

My current farm manager has been with me for 16 years. He is a consummate horseman, and his passion for the work we do is tangible. He never stops learning and studying the concepts of equine rehabilitation, nutrition, and horse husbandry at the highest level, including novel techniques and emerging science.

In addition to managing the entire facility, he is in charge of all water therapy because his expertise in horse handling is unmatched. He has put more horses in the water safely—whether in the swimming pool, on the aquatred, or in the cold-saltwater spa—than anyone in equine rehabilitation, based on the sheer volume of horses that have come through our program over the years. He also is an exceptional equine dentist, and he has worked with some of the best farriers in the country, giving him an extensive understanding of equine feet.

As manager of the aquatic team at KESMARC KY, he has personally handled a variety of horses—Dubai World Cup winners, Olympic competitors, multiple world champions in every discipline, and a wide range of breeds. His aquatic team personally handles every horse that comes to the facility to swim, exercise on the aquatred, or stand in the cold-saltwater spa. Each modality requires a unique set of skills. These people comprise the best and most experienced aquatic team in equine rehabilitation, having handled thousands of horses per year since KESMARC KY's inception.

My case manager oversees every horse in the facility. She is a certified hyperbaric veterinary technician, plus she has had advanced training in hyperbaric safety. She has personally treated more horses in the hyperbaric chamber than anyone in the field, making her a world leader in the technology.

As case manager, her duties include evaluating the progress of every case at the facility and reporting all the medical details at the farm to me—every temperature, every foot pulse, which horses are eating and which are not, the status of bandage changes on wounds and their proper dressing, scheduling therapists and farriers, and recordkeeping. Her extensive training and volume of experience in critical care and wound management gives her superior skills. She also is highly skilled at working and communicating with veterinarians from all specialties. As part of our biosecurity protocol, she is in charge of all incoming and outgoing horses and their records.

Logic behind Layout

KESMARC KY's complex is carefully designed to allow the horse to thrive in a consistent, relaxed, happy environment. With the exception of turnout paddocks, the entire complex is under one roof. The main room houses the pool, freestyle automatic walker, inground aquatred, and wash racks. Rubber pavers line the floor for added safety. All modalities are housed under one roof so the horse can be moved safely from the stall to the modalities.

The inground cold-saltwater spa, whole-body vibration platform, and hyperbaric oxygen chamber are housed in custom-designed annexes to the training barn.

Stallions, colts, and geldings are housed on one side of the training barn, and mares and fillies are on the opposite side.

Stalls are 14'x14' and deeply bedded in shavings-only to support healthy airways. Stalls have windows in each side partition, solid rear walls, and grilled fronts so horses can interact with those in adjoining stalls and across the aisle. I have found horses are much quieter when they can see each other in a controlled way.

Making sure a horse is happy in its environment but not overly stimulated is of real importance in rehabilitation. We observe each horse to determine the place in the barn where it will be happiest. Some horses are more content where there is less activity; others thrive with the bustle of activity near the center of the barn.

Most equine athletes have a genuine work ethic and enjoy their jobs. When a horse's job is taken away, it may become unhappy and sometimes dangerous because of pent-up energy, frustration, and anxiety. This is where the rehabilitation setting of controlled exercise is so beneficial. Consistent routine and work are crucial not only to the healing process, but also to the mental wellness of the horse.

There is no worse situation for an injured athlete than to remain in the normal training setting. When the acute stage of the injury is over, the horse may not understand why other horses in the barn are going to the arena, being turned out, or going to the track while it must stay in its stall. Real benefit is derived from moving the horse from the training environment to the rehabilitation setting.

This facility is all under roof for several reasons. Being under roof assures that no days are lost due to weather. Also, it is imperative that the horse is not exposed to outside stimulation that would excite it and necessitate sedation. Constant sedation is unhealthy and often the only option to keep a horse safe and under control in the normal training setting.

Nutrition

Traditionally, concentrate is withdrawn from stall-bound horses because, if they are maintained on their athletic level of nutrition, they become too fractious, difficult to handle, and a danger to themselves and others. The downside is that withdrawing concentrate deprives the horse of nutrients. Daily aerobic exercise enables the horse to be maintained on a good nutritional program to support healing without fear of the animal becoming too fractious. By keeping them active, we can continue to feed them the amount of nutrition needed to repair their bodies without worrying about them hurting themselves in the stall.

Great attention is given to a horse's specific nutritional needs. When our nutritionist devises a feeding program for an individual horse, he considers and addresses a variety of factors:

- What is the horse's energy level?
- Is the horse recuperating from surgery?
- Is the horse on anti-inflammatory drugs and/or antibiotics?
- Is the horse's immune system and digestive system being nurtured through this time?
- Is the feed easily digestible?
- Has the horse been under stress before its arrival that requires special attention?
- Does the horse have specific nutritional needs that require a specific supplement for optimum healing?

Feet

My experience in rehabilitation convinces me that the majority of the soundness problems I see in the horse start with the feet. If corrections are not made to ensure proper balance, the injury will have a poor prognosis for healing and will be more likely to recur when the horse returns to work. I cannot emphasize this point enough.

The rehabilitation setting is the perfect place to address foot issues. We provide the attending veterinarian with access to specialty farriers and veterinary farriers who can collaborate to develop the best plan for each individual case.

Controlled Exercise

Consistent, controlled exercise is the key to quality healing and a successful outcome. Horses sidelined by injury, illness, or surgery can come back to athletic form through a carefully designed, individualized program. The horse is monitored and reevaluated throughout the controlled-exercise program, and the type and amount of work is adjusted to achieve the optimal benefits.

When the attending veterinarian releases the horse from stall rest, a typical controlled-exercise program begins with hand walking and the use of the inground cold-saltwater spa to draw out inflammation and edema. (A post-surgical horse should not enter any water modality sooner than three days after the removal of sutures.)

At the point where a traditional program might advocate turnout, we initiate water aerobics (swimming or aquatred) because uncontrolled exercise too soon after injury is detrimental to healing. If appropriate, the horse also spends time on the freestyle automatic walker before proceeding to controlled, weightbearing exercise.

A rider is introduced to the daily program when the attending veterinarian feels controlled loading is appropriate. This begins with walking under tack on the indoor track. When diagnostic rechecks confirm the horse's satisfactory progress, the attending veterinarian may allow the horse to advance from tack-walking to jogging.

A horse's average day in rehabilitation is surprisingly active, which keeps it mentally fit, and the increase in controlled activity prepares the horse to return to training.

WHEN A HORSE ARRIVES

When a horse arrives at KESMARC KY, the results of any diagnostics provided by the referring veterinarian as part of the horse's history are retained in the horse's medical record to be used in designing a plan of rehabilitation. If the attending veterinarian requests a diagnostic workup, appropriate action is initiated.

The overall condition of the horse is assessed:

- Feet;
- Teeth;
- Weight and body condition;

- Deworming status; and
- Vaccination status.

A nutritionist reviews the horse's status and determines its specific dietary requirements to ensure an appropriate level of energy and to facilitate healing:

- Supplements it is receiving, if any, are carefully assessed;
- Every horse is placed on a prebiotic/probiotic regimen for its entire stay to support proper digestion and to protect the gastrointestinal tract from any antibiotics and/or anti-inflammatory drugs the horse is receiving;
- If the horse is on any antibiotics and/or anti-inflammatory drugs, its appetite is carefully monitored;
- Water intake is closely monitored. For this reason, our facility does not use automatic waterers;
- Consumption of concentrate and hay is closely monitored for appetite; and
- If the horse cannot consume hay, alternative roughage is provided. Steamed hay is beneficial for some horses with airway and digestive issues.

Each horse is monitored constantly by my staff, and any changes needed to ensure the horse's total health and happiness are made and documented. Every detail matters:

- The horse's temperature is taken daily for its entire stay;
- Temperament is evaluated to assure the horse is in the best location at the facility for its individual happiness so the horse will settle in properly and thrive; and
- Demeanor and response to pain is observed and documented.

Post-surgical horses receive specialized care:

- The horse is closely monitored, and pertinent information is documented for the attending veterinarian;
- Supportive bandages appropriate for the horse's incision are maintained, and attendees keep a daily log to ensure the details are addressed as the attending veterinarian prefers;
- Bandage-change schedules are rigidly followed; and
- Reevaluations are conducted according to the attending veterinarian's recommendations, and results are logged into the horse's file.

Outside therapists—podiatrist, dentist, nutritionist, acupuncturist, chiropractor, sports therapist, and more—are brought in upon the attending veterinarian's request. If the attending veterinarian believes blood work, cytologies, cultures, and sensitivities are indicated, we initiate them.

Once the attending veterinarian lays out a plan, my staff evaluates the horse to make sure it is physically and mentally able to handle the regimen. If the horse does not have the training we think necessary to be safe, we discuss that with the client and attending veterinarian and help them to make the necessary changes to their game plan.

Some horses begin their rehabilitation programs with stall rest. We take advantage of this time to start putting the horse together before the controlled movement begins. This is the best time to address the feet and make sure the balance and angle are correct. (Some therapeutic shoeing techniques can benefit the rehabilitation of an injury.) During stall rest also is a good time to schedule visits by a dentist, chiropractor, acupuncturist, and/or sports therapist.

SOFT-TISSUE INJURIES

Soft-tissue injuries are the most common ones we see at KESMARC KY. Rehabilitation begins with a solid diagnosis, most commonly through ultrasonography or, if the injury involves the foot, magnetic resonance imaging. A current ultrasound that confirms the proper amount of healing has taken place is crucial before the start of controlled exercise.

The discipline and age of the horse, how acute the injury is when the horse arrives at KESMARC KY, and what therapies the horse has received prior to its arrival are important factors to consider when devising an individualized rehabilitation plan. Hand walking, inground cold-saltwater spa, other therapeutic modalities, and hyperbaric oxygen therapy can be initiated to accelerate the healing process and produce a better quality of healing.

Rehabilitation of injuries to the body of the suspensory ligament has a much higher rate of success in returning the horse to training than injuries at the osseous junction.

Tendon and suspensory injuries—whether athletically induced or caused by traumatic injury and laceration, a concussive strike, or a bandage bow—all are very different. Therefore, our response to each situation is unique, and each individualized program must change appropriately based the progress or setbacks indicated by diagnostic rechecks.

Sometimes it is difficult to balance the beneficial break-up of adhesions against the concern for further deterioration of the tendon during the healing process. Only knowledge and years of experience rehabilitating soft-tissue injuries allow us to recognize how to move forward in a responsible way.

NOTE: Tendons are truly an anomaly in terms of what will hold up when the horse returns to performance. I have had horses with injured tendons that should be able to perform and do not, yet some horses with much worse tendon injuries go on to be competitive and very successful. Because of this unknown, many owners and their veterinarians attempt to rehabilitate horses with tendon injuries, but the reality is that most tendons do not hold up.

MODALITIES

Swimming Pool

Swimming is a non-weight bearing exercise that provides a great cardiovascular workout without the stress of concussion, which is especially detrimental during early stages of bone or soft-tissue healing and for horses with sore or problem feet.

Enhanced circulation that occurs in the lower limbs when concussion is removed from the equation only can be accomplished in the pool. Swimming is the perfect way to pump blood up and down the leg without the interruption of weight bearing.

Swimming also elevates the horse's heart rate, and the increased circulation greatly benefits the healing process and reduces inflammation.

Also, break-up of adhesions and promoting a good range of motion with much less pain are easier to accomplish in the water.

KESMARC KY utilizes a 16'-deep, 290,000-gallon pool in a circular design with bowed sides and an island in the middle. When no water is in the pool, it looks like a giant doughnut. Two bridges hang from the ceiling: one bridge enables the inside handler to get to the center island; the other bridge spans the deep end of the ramp where it meets the pool to enable the outside handler to parallel the horse as it swims laps around the pool. Both bridges are suspended high enough above the water to prevent the horse from hitting its head or striking its hooves, should it become fractious while swimming.

The circular design of the pool allows us to slowly increase aerobic conditioning until the horse is able to begin weight bearing exercise (Figure 1). Swimming alternating directions, starting with one lap and gradually increasing laps as the horse progresses, enables us to control the conditioning program as needed for the individual horse.

In contrast, a straight-line pool teaches a horse to rush from one end of the pool to the other, making it difficult to achieve the controlled conditioning possible in a circular pool.

The ramp into the pool is wide and fully padded on the bottom, sides, and over the edges. Water depth runs from shallow at the entrance to the ramp to deep enough to support a swimming horse where the ramp meets the pool.

For safety, the horse must be swimming before it leaves the ramp and enters the pool. Likewise, when the horse exits the pool, it must be able to swim onto the ramp.

If the pool end of the ramp is too shallow, a swimming horse can be injured if its front feet strike the bottom of the ramp as it exits the pool.

Figure 1. Aerobic conditioning in the swimming pool.

Sometimes it is necessary to back a horse down the ramp the first or second time it enters the pool. If a horse refuses to enter the pool, it is unsafe (and sometimes next to impossible) to force it in. Most horses can be persuaded to back down the ramp where they can turn around and swim into the pool. NEVER ALLOW A HORSE TO JUMP INTO A POOL.

The pool's filtration system includes a custom-designed, 8' x 6' sand filter with pumps large enough to handle the great volume of water necessary to sanitize and filter the water.

The island system enables two handlers to accompany the horse through the schooling process. For the horse's first session in the pool, it swims one lap and then is allowed to take a break on the ramp to recover mentally and physically from the new experience. After recovery, the horse swims a second lap before exiting the pool.

In subsequent sessions, the number of laps is increased as the horse's tolerance increases and condition improves. Typically, horses will start out slowly, swimming about two laps. As they become more fit, the swimming sessions are increased by one lap, if the horse can tolerate it. Becoming overly tired from too many laps can cause the horse physical and mental stress that makes it fear swimming and most likely will cause the horse to resist entering the pool.

The horse's attending veterinarian closely monitors its conditioning program throughout the healing process to ensure the horse is on target for its goal. Knowing how much conditioning is necessary for the individual horse is important. More is not necessarily better, depending on the injury and the time the horse will be off for rehabilitation. Rushing rehabilitation by doing too much too soon will not benefit the horse. This is important to remember with tendon and suspensory injuries.

When the attending veterinarian determines that adding riding to the horse's conditioning program is appropriate, the horse swims in the morning and is ridden in the early afternoon. This serves two purposes: It helps keep the horse's energy at a manageable level for a safer ride for both the horse and rider, and swimming achieves superior movement going into the important riding phase of the healing process.

For bone and soft-tissue injuries, loading planned for the correct stage of rehabilitation and prescribed for the proper duration is a crucial part of the healing process. Without it, proper healing sometimes does not occur.

Cross training with the pool and normal training can be very beneficial. Also, some horses greatly benefit from alternating sessions in the pool with aquatred work.

The handlers that swim every horse at KESMARC KY have a collective 40 years experience with swimming and aquatred work. Both modalities require an intimate understanding of the horse, superior horsemanship skills, and a special set of training skills. Making split decisions that are correct when schooling a horse in these modalities means the difference between a horse being safe and a horse being in real trouble.

Our safety record is impeccable only because of the great care and training of the staff that handles these horses. Their instincts and understanding instill confidence in the horse so the benefits of these therapies can go much further than just the physical. The confidence engendered in young horses carries on throughout their lives in many ways.

Swimming is contraindicated for horses with certain conditions, such as airway issues, exercise-induced pulmonary hemorrhage ("bleeding"), recovery from throat surgery, and some sore backs (depending on the root cause of the soreness).

Figure 2. Strength training in the inground underwater treadmill (aquatred).

Inground Underwater Treadmill (Aquatred)

The aquatred is a hydraulic treadmill submerged in water that reaches three-quarters of the way up the horse's sides. The aquatred provides buoyancy that reduces weightbearing and, as the horse walks on the treadmill, strength training through the resistance of the water (Figure 2).

Using the same muscle groups as on land with the added resistance of the water, horses achieve a superior resistance workout without the cardiovascular and respiratory demand placed on them in the pool. For this reason, horses with airway or throat issues benefit from the aquatred because it allows a normal breathing pattern. Resistance is a safe way to get the horse back to work more efficiently with less physical stress.

Horses truly enjoy being in the aquatred and get a great deal of physical as well as mental benefits from it. In my years of experience, I have never seen any modality that horses enjoy more than the aquatred.

The inground aquatred is one of the best tools in equine rehabilitation and conditioning—provided it is installed in a permanent inground site.

Our unit's safety is completely due to the inground design and the experience of the handlers. The inground system gives the handlers more safe options should a horse become fractious.

If you have a horse in an enclosed box at ground level, the leverage needed to control the horse is not the same as when the unit is inground. With the above-ground design, the only option to get a panicking horse out of the aquatred is to open the door to allow the horse to exit the unit before it jumps out—a dangerous scenario. If a horse becomes fractious in an inground unit, there are many ways to keep the situation safe, the simplest being walking the horse out of the unit.

Even highly schooled horses may be apprehensive of the aquatred. Not only are they asked to walk into a virtual trench filled with water, but the "ground" also begins to move under their feet. Understanding their fear and acknowledging it is a crucial part of successful schooling.

Once the horse is acclimated, the inground aquatred is the perfect place to "hand walk" a fractious or good-feeling horse, both while recuperating from injury and post surgery if the sutures have been removed and the surgical site is completely healed.

We make the inground aquatred a fun and non-frightening experience for the horse through proper handling, schooling, and experience. Just like anything else we, as professionals, do with a horse, it is crucial to know the individual's personality and how to work with it, not against it. With this approach, horses get a great deal of psychological benefits, in addition to the primary benefit of physical therapy.

Correct acclimation and training of the horse for the aquatred is crucial so the horse can derive optimum physical benefit. Early schooling on the aquatred is more about acclimation than therapy. A small amount of sedation always is administered for the horse's introduction to the aquatred. Mild sedation suppresses the horse's natural fear-and-flight response and helps to reassure the horse that it is safe.

The safety of the inground design and proper acclimation make sedation beyond the first or second session in the aquatred unnecessary. Any therapy or piece of equipment used over a long period of time that necessitates sedation with every use should be discarded. Sedated horses do not derive the optimum benefits from exercise that alert horses do, and frequent sedation can be counterproductive to the healing process as well as the overall health of the horse.

After properly acclimating the horse to the aquatred, we begin each workout with a steady warm-up at the walk. After the five-minute warm-up, we increase the speed to a power walk, the fastest steady walk a horse can do without breaking into a trot. In the beginning of the aquatred program, the duration is limited to prevent the horse from overwork in the initial stages of conditioning and training. As the horse's condition improves, the duration of the power-walk exercise is gradually increased, ultimately reaching 15 minutes. At the conclusion of each session, the speed is decreased over several minutes to cool down the horse to a stop. The horse is removed from the aquatred, rinsed off on the wash rack, and placed on the freestyle automatic walker to finish cooling out and dry.

The aquatred, which affords the horse 40%-60% buoyancy, increases flexibility and break-up of adhesions. After colic surgery, the aquatred is a perfect modality to break up adhesions at the surgical site while supporting the abdomen with the buoyancy of the water.

Buoyancy, and the significant reduction in concussion it provides, combines with the resistance of the water to create the optimum benefit of the inground aquatred. When a horse with a musculoskeletal ailment strikes the ground, the resulting concussion causes pain, and the horse responds by shortening its stride.

In the aquatred, concussion and the pain it causes are greatly reduced, allowing the horse to stride out and break up adhesions that restrict its movement.

The inground aquatred holds benefits for different classes of horses, including:

- Injured horses;
- Horses needing a break from the rigors of training but not wanting to lose condition; and
- Fit, older athletes that need to maintain their fitness and extend their longevity and careers.

Swimming Vs. Aquatred

Swimming and aquatred work are very different modalities that are not interchangeable. One is not better than the other. Deciding which modality is appropriate for the individual horse depends on its injury, temperament, and occupation or discipline. Some injuries, such as soft-tissue conditions of the lower hind end, do not benefit from aquatred work but respond more favorably to swimming.

Swimming has been long accepted as a way for a horse to exercise its whole body—back, neck, legs, forearms, and gaskins—and gain cardiovascular fitness while sparing its joints and cartilage from the impact of weightbearing. Additionally, the resistance of the water enhances the workout.

A recuperating horse typically exercises first in the aquatred. When the horse gains strength, it can progress to the swimming pool.

Freestyle Automatic Walker

The freestyle automatic walker allows horses to exercise untethered between panels in individual 35' x 8-1/2' compartments. It most commonly is used to facilitate warm up and cool down. The freestyle automatic walker at KESMARC KY sits on a woodchip surface, which is proven to reduce concussion (Figure 3).

The operator sets the speed and can stop the walker to reverse its direction to allow a horse to develop its muscles symmetrically.

Another advantage of the freestyle automatic walker is that it allows the horse to improve its balance. Typically, we maintain the unit's speed at a brisk walk, but for special cases we may increase the speed to a trot, as indicated.

The freestyle automatic walker at KESMARC KY was custom built for the enclosed main room, which also houses the pool and aquatred. Horses can do aquatherapy, no matter the weather, and go onto the walker to dry and cool out.

Enclosing this room also gives this walker its safety of use. If this walker were outside, probably 40% of the horses could not go safely into it because of the overstimulation of the outside surroundings.

We still hand walk many horses per the attending veterinarian's direction. We also hand walk the very few horses that become too excited on the walker.

Figure 3. Untethered exercise using the freestyle automatic walker.

Figure 4. Custom inground cold-saltwater spa at KESMARC KY.

Cold-Saltwater Spa

The inground cold-saltwater spa was custom designed by KESMARC KY for improved equine safety (Figure 4). It has softly sloping ramps at each end that allow the horse easy access to and exit from the water. The ramp is wider at each end and narrows in the center, where the water depth can be adjusted to the correct depth for the individual horse.

The saltwater is chilled to 33 degrees Fahrenheit, with salt and chlorine for sanitation added, as needed. The water is closely monitored for salt and chlorine levels and temperature. The filtration system is extensive to protect the chiller from debris. Horses using the cold-saltwater spa wear a tail bag to prevent manure from dropping into the water, should they defecate while standing in the spa. (This is the only water therapy where a tail bag can be worn because of the depth of water.)

The cold-saltwater spa is beneficial for tendon and suspensory injuries, post-surgical therapy for the lower limbs, healing of cuts or wounds on the lower limb provided the wound does not communicate with the joint, and drawing heat and soreness out of feet after training or competition.

This modality has great benefits throughout training, as well. With its use, injuries such as strains and beginning inflammatory responses can be greatly helped to keep them from becoming larger issues. The cold-saltwater spa keeps joints tight and takes swelling out so quickly it is visible immediately after the treatment.

This therapeutic tool is of such importance for so many conditions of the lower limb. All along, we at KESMARC KY agreed with the concept of this therapy but regarded the commercially available, above-ground spas to be unsafe. We would not put a horse in a box, close the door, and fill it with water. Even with a half door, the chances of a horse getting upset and jumping out was a concern, and I learned of many horses that tried to jump out of such a design. Some horses had done so even after several problem-free treatments. I recognized the danger of this the first time I saw the above-ground design used by many centers. I also learned that many horses are sedated every time they go into the spa, which is unacceptable.

I designed the inground, cold-saltwater spa at KESMARC KY with safety in mind. It has a soft slope into the center of the spa and completely padded edges, so if a horse gets upset, handlers can get the situation under control easily without injury to the horse.

After proper acclimation and training, horses walk into the inground cold-saltwater spa quietly and stand safely for their treatment. If a horse should get upset, the handler simply walks it out, allows it to settle down, and then quietly walks it back into the water. This requires a different approach from that used with the aquatred and swimming pool, so the handler must possess a unique set of skills. Being very patient and calm with a knowledgeable demeanor promotes trust and security in the horse.

Jogging Track

For the horse that has gone a long time without a rider—whether a racehorse, heavy sport horse, or athlete in another equine discipline—the idea is to safely control its return to work. The indoor jogging track at KESMARC KY is a simple yet unique application for controlled exercise and weight bearing.

The track is designed as an oblong chute with controlled turns and a synthetic surface of ground polymer and rubber that provides good footing that guards against concussion.

If a horse being ridden on the indoor track becomes fractious, it is much easier to control it in a confined area. If you give a horse access to an entire arena or an outside training track, the possibilities for error are too high. The horse can do more physically than is desired, making it prone to reinjury. With the controlled indoor setting, the horse and rider are given a safe environment in which to develop a solid foundation and level of overall body fitness for the horse. We begin the loading work (riding) when the horse has achieved a good base of conditioning via water aerobics so as to provide a continuation of healing and conditioning that leaves the horse closer to getting back to efficient training with less likelihood of reinjury.

Vibration Platform

Whole-body vibration is an emerging therapy in equine medicine. Although human and animal studies support its efficacy, controlled studies have not been completed in the horse.

Principal researcher Clinton Rubin, Ph.D., showed that the application of extremely low-level, vertical vibration to animals and humans increases bone formation and encourages stem cells away from fat and toward bone [1, 2]. One of Rubin's major discoveries is that low-level vibration therapy stimulates the honeycomb trabecular bone in condyles. [3]

Low-level vibration therapy causes rapid (30-50 per second) contractions of muscles. These contractions promote blood flow, which, in turn, promotes oxygenation of tissues. Connective tissue also is stimulated by these rapid muscle contractions.

Horses, like humans, are subjected to vibration therapy by standing on a vibration platform for 10-15 minutes, several times a week (Figure 5). The gentle vibration is thought to produce not only physical results, but horses exiting the vibration platform also appear calm and relaxed. Despite the lack of equine studies, I believe whole-body vibration therapy has a place in rehabilitation, conditioning, and maintenance of the equine athlete. Vibration therapy could be beneficial during layups to reduce loss of bone density and during rehabilitation to rebuild bone strength in preparation for return to training. But without proven study results, I refrain from using it in certain cases, such as young horses with growth issues, horses with recent fractures, those whose fractures have been fixed with screws, and similar conditions. I believe this therapy eventually will be supported by science and will prove beneficial for conditions of the foot by stimulating circulation for better growth, based on anecdotal reports from prominent horsemen who have used vibration therapy on their horses. Laminitic horses seem to get an analgesic effect from the vibration, as well as increased blood flow and stimulation of bone turnover, which is greatly lost in laminitic horses reluctant to stand and walk that often are recumbent. I also feel vibration therapy will prove beneficial for soft-tissue injuries through increased circulation.

Hyperbaric Oxygen Therapy

Adequate oxygen supply to body tissues is a basic requirement for healing. Hyperbaric oxygen therapy (HBOT) is the medical technology that delivers that oxygen.

Within the controlled environment of the chamber, an increased level of oxygen is applied under increased pressure, effectively and dramatically increasing the oxygen in the blood and plasma.

I have been involved with HBOT since 2000. It is one of the real passions of my life. When I began my journey into HBOT, I realized there were conditions and situations that either had a poor prognosis or the horse simply did not survive, despite due diligence and observing standard of care. When I began to expose horses to HBOT, I saw some of those outcomes change in a positive way.

Since 2000, I have introduced HBOT to the horse industry in Kentucky; helped found the Veterinary Hyperbaric Medical Society; facilitated the education of some of the top trainers and technicians in this field today, who began as members of the KESMARC KY staff; administered more veterinary-prescribed HBOT treatments than anyone in equine hyperbaric medicine; and helped found the company that manufactures the stationary hyperbaric chamber. This has been a journey of great importance and with many challenges from a standpoint of education and an understanding of HBOT's efficacy and safety.

All chamber operators at KESMARC KY are technicians trained in human hyperbaric medicine. We also have personnel certified in veterinary hyperbaric medicine.

As of 2013, KESMARC KY has administered more than 15,000 equine hyperbaric treatments and has developed a wide range of protocols based on our results (Figure 6).

HBOT is most effective when used as an adjunct therapy. It can be very cost effective when used by the veterinarian in conjunction with traditional medicine to enhance antibiotics and other treatments and to allow for greater penetration to the target area. Early use of HBOT greatly improves the prognosis for acute injuries and conditions by decreasing the likelihood they will develop into long-term issues.

Figure 5. Administration of whole-body vibration therapy.

Figure 6. KESMARC KY's state-of-the-art equine hyperbaric chamber.

In addition to treating acute injuries, HBOT can be employed for the treatment of chronic injuries and conditions, as well as utilized as a therapeutic aid in athletic recovery.

HBOT helps to stimulate the immune system, potentiates antibiotics, decreases scarring, and increases stem cell production up to eight fold in the body. Non-invasive, it successfully has been used in multiple conditions by veterinarians for more than a decade.

Indications include:

- Recovery following strenuous exercise
- Respiratory disorders, such as bleeders
- Infections
- Non-healing wounds
- Post-surgical recovery (including colic, orthopedic, and respiratory surgeries)
- Traumatic injury resulting in soft-tissue inflammation
- Acute nerve trauma
- Post-injection reactions
- Before and after stem-cell and plasma-rich platelet (PRP) therapy
- Reproductive issues
- Laminitis
- Birth asphyxia (dummy foals)
- Thrombosis
- Gastric ulcers
- Colitis
- Tissue necrosis due to snake or spider bites
- Rhabdomyolysis (tying-up)

During HBOT, a horse is placed in a pressurized chamber for approximately 45-60 minutes, where it breathes concentrated oxygen. This enables the blood to carry the increased amount of oxygen to the tissues. Benefits include reduction of inflammation, enhanced effectiveness of antibiotics, and regeneration of connective tissue (tendons and ligaments).

In human medicine, HBOT is a well-accepted treatment—approved by most health insurance providers for treatment of 12 specific conditions—for certain patients, including burn victims and those with difficult-to-heal wounds. Even some equine underwriters have approved HBOT for insured horses in special cases.

HBOT was used primarily as a rehabilitation tool when it was first adopted by the horse industry. Although many injured or ill horses benefit from it, HBOT is now a vital component in many training programs as a means to help sound athletes recover from the rigors of intense competition.

Turnout

Turnout is an important part of the overall health of the horse, but it must be appropriate for the horse's condition and the rehabilitation goal set for it. Stall rest often is required for the early stage of healing, especially for bone and soft-tissue injuries. Once the veterinarian approves controlled movement, the horse can progress through the various modalities until it is able to be turned out safely.

If a horse needs an extended time off, turnout is important, but only if the horse's temperament can safely allow it. Common sense plays a crucial role in knowing the individual horse's personality.

Depending on that assessment and the individual horse's situation, turnout can be detrimental to the healing process, especially for a horse allowed to run and buck around a round pen or paddock too soon after surgery or an injury.

Turnout is introduced in increments as part of the horse's controlled exercise program. It begins with short sessions in a small, restrictive round pen and progresses to longer sessions. Eventually, the horse graduates to a small paddock then a larger paddock, as tolerated.

PREHABILITATION

A natural evolution has occurred in equine rehabilitation to expand it from solely rehabilitation of injured and ill horses to include prehabilitation, which focuses on wellness and conditioning of sound horses to prevent injury. More and more, I hear horsemen refer to it as "quality time off."

Active equine athletes come to KESMARC KY during a hard campaign, or while the horse is on a timely break from competition and training or after the season closes to recover via HBOT, cold-saltwater spa therapy, and turnout, along with enough swimming or aquatred to keep a good level of conditioning while still enjoying a break.

If this practice becomes more widely adopted in the horse industry, I believe horses will remain sounder longer than they presently do, and they will enjoy longer, more productive careers, no matter what the discipline.

CONCLUSION

The strongest take-home message is that, due to the complexity of this work, it is difficult to offer absolute protocols. Rehabilitation must start with solid diagnostics and allow the individual horse and the dynamics of its injuries tell you how to proceed.

When I started rehabilitating horses more than 25 years ago, no one taught equine rehabilitation. I was very fortunate to be able to work with and learn from some of the best veterinary specialists in their respective fields. It is the volume of cases over many years that sets KESMARC KY apart. Every case has taught us something, and we continue to learn from each and every horse.

In 2013, many short courses and academic programs in equine rehabilitation exist. I am sure that many do a good job of teaching this field, but be warned: Some have no clue how to properly rehabilitate a horse, let alone teach others how to do it. These schools send out improperly educated graduates as formally trained rehabilitation specialists who are hired by rehabilitation facilities (other than KESMARC KY) or set up their own centers. With this improper training so widespread, I fully understand why veterinarians are apprehensive about sending their clients' horses to a rehabilitation facility.

Proven results and reputation are the most important factors when choosing a rehabilitation facility. Keep in mind: One window of opportunity exists to achieve the best possible outcome for an injury or an illness. Don't waste it.

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