Tissue Tolerances of the Muscle-Tendon Unit

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ABSTRACT

CONTENT: This monograph discusses the wide ranging variables that affect the capacity of the muscle-tendon unit to facilitate human movement. A spectrum of clinically relevant content is covered, from anatomy and biomechanical considerations of normal tissue to pathologic changes with disease or injury. Current evidence regarding contemporary physical therapy and medical management of the pathologic muscle-tendon unit is presented and evaluated. Clinical reasoning related to intervention selection is also discussed. This content is intended to be useful for clinicians, researchers, educators, students, and all other stakeholders in orthopedic and sports settings. **CASE ANALYSES**: Four case studies are presented to assist in the application of knowledge in clinical practice. Specific tissue tolerance considerations are highlighted and management priorities are detailed to guide the reader through patient presentations similar to those encountered every day in the clinical setting.

Key Words: capacity, musculotendinous injury, tendinopathy

LEARNING OBJECTIVES

Upon completion of this monograph, the course participant will be able to:

1. Understand clinically relevant normal muscle and tendon tissue anatomy and biomechanics.
2. Understand physiological mechanisms and processes associated with pathologic muscle and tendon tissue.
3. Describe clinical and diagnostic tools used in identifying muscle-tendon abnormality.
4. Discuss the evidence surrounding the physical therapy management of dysfunctional muscle-tendon units.
5. Discuss the evidence related to medical and alternative management of the dysfunctional muscle-tendon unit.
6. Cite evidence related to improving muscle-tendon tissue tolerance to load, including injury prevention strategies.
7. Apply acquired information to case-based patient scenarios to enhance understanding and clinical reasoning in physical therapy management of muscle-tendon pathology.

INTRODUCTION

Human motion is a smooth coordinated process requiring a series of complex interactions of numerous body systems. An intricate relationship between muscles, tendons, and bones, and the seamless way that tissues work together so coherently allow us to perform our functional, recreational, and sport-related activities. When any part of the neuromusculoskeletal system becomes dysfunctional, clinicians are tasked with identifying and optimizing movement dysfunction to return the patient/client/athlete to their prior level of function. In order to identify dysfunction rapidly and effectively, it is imperative that we understand how typical movement occurs.

We could not have normal movement without a functional muscle and tendon unit. When a muscle contracts, it creates pull on the tendon that anchors the muscle to a bone, allowing the bone to move as needed for a task. Impairment to the muscle-tendon unit (MTU, for the purposes of this monograph) and subsequent alteration in force production and voluntary movement can contribute to substantial disability. In 2016, the Global Burden of Disease study reported that between 20% and 33% of people across the globe live with a painful musculoskeletal condition.[1] In the United States, that number is as high as 1 in 2 adults, contributing (with indirect costs) to an economic burden of $874 billion in 2015, or 5.7% of the gross domestic product.[2] While these data are not specific to MTU pathology, musculoskeletal disorders typically are accompanied by alterations in movement, which is commonly associated with MTU impairment. Therefore, it is essential for clinicians, researchers, students, and all other orthopedic and sports medicine stakeholders to understand the tissue tolerances of muscles and tendons, so they can evaluate and treat MTU dysfunction effectively.

The purpose of this monograph is to describe normal MTU architecture and function, pathologic MTU, and clinical management options for treating MTU dysfunction.

NORMAL MUSCLE-TENDON UNIT

Understanding normal muscle and tendon architecture and tissue development can be helpful in optimizing management of dysfunction. This section describes clinically relevant anatomical considerations, biomechanical principles, and clinical methods of measuring tissue capacity with the aim of allowing for more targeted treatment and enhancement.

Anatomy

Skeletal muscle tissue

Muscle is abundant in the human body, and can be responsible for up to 50% of our total body weight.[3] Skeletal muscle allows the human body to achieve and sustain positioning and movement. During any functional activity, muscles shorten, elongate, or maintain activity without changing length, in order to ensure efficient, safe, and appropriate task performance. To understand normal muscle activity and capacity, it may be helpful to first review basic muscle structure.

Skeletal muscle architecture is intricate.[4] Contractile proteins, actin and myosin, and noncontractile proteins such as titin and desmin combine to create myofilaments. Many myofilaments combine to create myofibrils. A group of myofibrils create a muscle fiber, the structural unit of a muscle, which is surrounded by a thin layer of connective tissue called the endomysium. Numerous muscle fibers, grouped together in a parallel alignment, form a muscle fascicle, which is surrounded by a thicker connective tissue called perimysium. Finally, a muscle belly (readily observed on visual observation during cadaver dissection) is a collection of muscle fascicles, and is surrounded by an epimysium. The endo, peri, and epimysiums enhance structural organization and improve mobility against surrounding structures. The hierarchical structure is shown in Figure 1.1.

While the muscle fiber is the structural unit of a muscle, its shortening and elongation is dependent on sarcomeres, which are components of the myofibril. Each sarcomere consists of the previously mentioned contractile and non-contractile proteins. Sarcomeres are serially organized with overlapping thin (actin) and thick (myosin) filaments contributing to a banded appearance on histopathological examination.

Vascular supply to skeletal muscle is essential to performance, because tissue oxygenation is needed for various repetitive activities such as gait or exercise. The presence of a functioning vascular supply also enhances the capacity of a muscle tissue to heal and recover from injury, unlike in other types of tissue, such as collagenous tissue. Primary arteries provide vascular inflow to skeletal muscles, and penetrate the epimysium and perimysium through feed arteries and branching arterioles to reach the muscle fibers. Capillaries surround each muscle fiber in a sweeping non-uniform and variable distribution.[5] Veins and venules are arranged in a similar fashion to arteries and arterioles.

Innervation of skeletal muscle comes from branches of peripheral nerves. A motor unit consists of a single motor neuron and all of its innervated muscle fibers. Muscles that require fine motor skills, such as those in the hand, have a smaller number of muscle fibers per motor neuron. Larger muscles responsible for gross movement, such as the quadriceps, have more muscle fibers per motor neuron.

Tendon tissue

Tendons perform the important task of transmitting tensile load from muscle to bone, providing joint movement and stability. Dense connective tissue allows tendons to absorb and transmit substantial loads without rupture. Cellular material makes up about 20% of tendons, and the extracellular matrix (ECM) accounts for about 80% of the total tissue volume. The ECM is 55% to 70% water. The remaining percentage of the ECM is primarily solids: mostly collagen, but also proteoglycans, elastin, and other proteins.[6]

Tenocytes are the cells primarily responsible for tendon metabolism. Arranged in parallel alignment along the line of stress, tenocytes balance the production and destruction of the ECM. The ECM's water content, collagenous tissue, and additional proteins contribute to the structure and adaptation to mechanical loading. The collagenous tissue contributes to the considerable ability of a tendon to withstand large tensile forces while elastin allows for some mechanical deformation and compliance, to avoid disruption. Collagen type I is the predominant fiber type in tendons, and is synthesized in response to mechanical loading by tenocyte activity. The structural hierarchy of a tendon (from smallest to largest unit) begins with collagen molecules, which aggregate in a heads (positive charge) to tails (negative charge) alignment to create a microfibril. A collection of microfibrils creates a fibril. A combination of fibrils creates a bundle, which is surrounded by an endotenon. A tendon is a combination of bundles (Figure 1.2). The epitenon, a loose connective tissue, surrounds the collection of bundles and transmits lymphatics, blood vessels, and neural tissue to the tendon. The densely packed and parallel aligned tissues contribute to the mechanical stability, primarily in response to tensile loading.

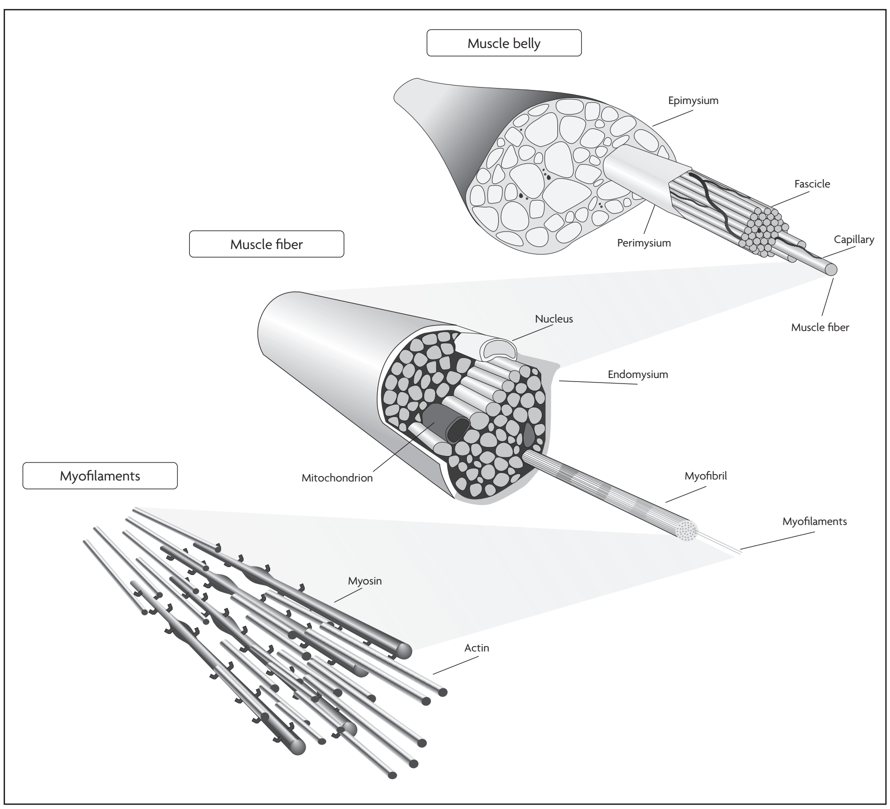

Figure 1.1. Skeletal muscle architecture.

The vascular supply of tendons is vital in maintaining healthy tissue, and has significant implications in tissue injury and repair. During cadaveric observation, tendon tissue is a shiny white color, as compared to muscle tissue which is red. This is because tendon tissue has a limited vascular supply: blood vessels represent only 1% to 2% of the ECM.[6] Tendon vascular nutrition is supplied by blood vessels from the perimysium, periosteal insertion, and paratenon or tendon sheath. Within the tendon sheath, synovial fluid flushes the tendon providing lubrication and enhanced gliding. The amount of blood supply depends on a number of factors, such as anatomical location, morphology, previous injury, and activity levels.[7] Neural innervation is limited, but is provided by small branches of nerve fibers that penetrate the epitenon, rarely terminating within the tendon itself.

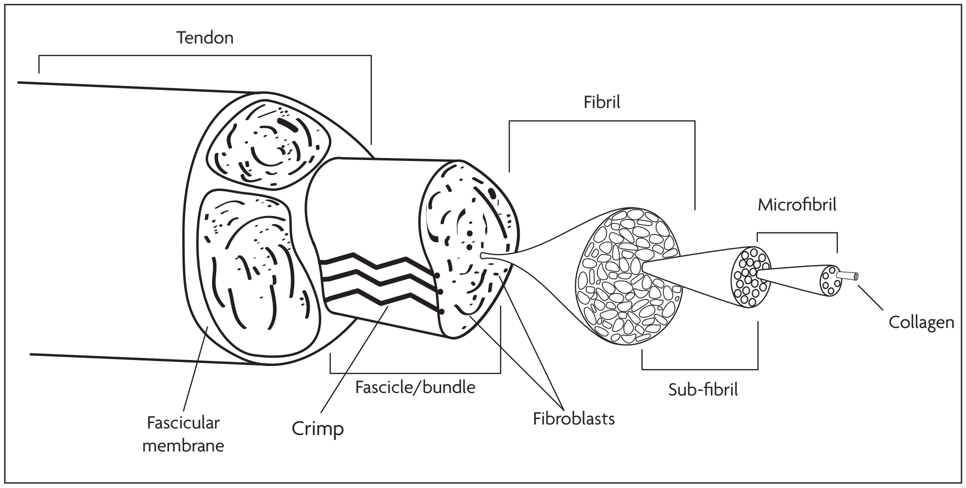

Figure 1.2. Tendon tissue architecture.

Myotendinous junction

The myotendinous or musculotendinous junction (MTJ) is where skeletal muscle and collagenous tendon tissue meet to create an MTU (Figure 1.3). The MTJ potentially can be a failure point in injury because of the high amount of stress that is transmitted from the tendon to the muscle and the muscle to the tendon. Because of its specialized purpose, the MTJ anatomy is unique. Generally, tendon tissue is oriented longitudinally to allow a uniform directional force transmission. At the MTJ, collagenous projections of the terminal tendon appear to be multidirectional in nature. Similarly, rather than a smooth orientation, a 3D analysis study of the MTJ found that collagen fibrils appear ridge-like and muscles furrow-like, with myofilaments terminating in the ridge-like protrusions.[8] The increased contact area at the MTJ almost creates a locking mechanism, making the muscle-tendon interface inherently stronger to meet the demands of movement.

Biomechanical Considerations of the Muscle-Tendon Unit

Force production in muscle

The physiology of a muscular contraction is complex.[9] A number of factors play a role in the ability of a muscle to contract, relax, and perform the requisite force production for a given task. For example, the physiologic cross-sectional area (CSA) and pennation angle of a muscle affect the amount of force that can be transmitted through the tendon to the bone.[10] The larger the CSA of a muscle, the greater the capability of a muscle to generate force. Pennation angle describes the orientation between the muscle fibers and the tendon. Generally,

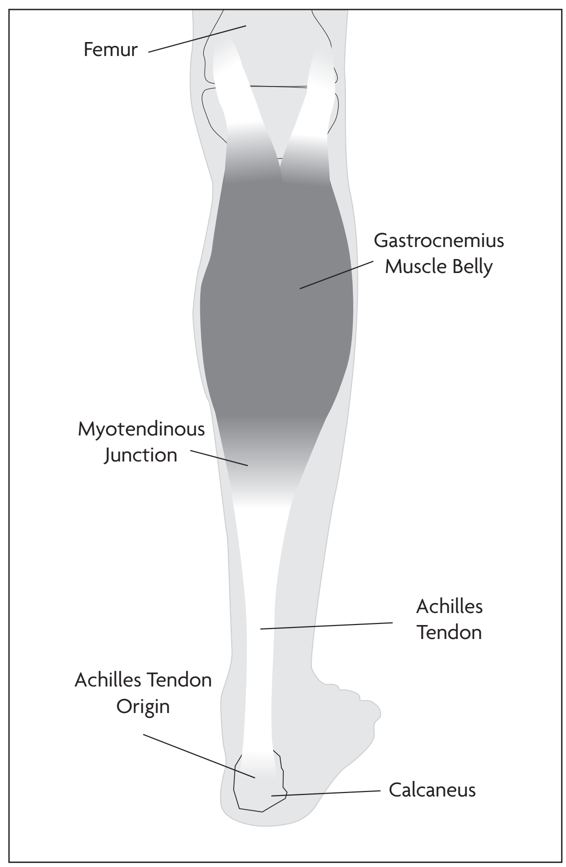

Figure 1.3. Myotendinous junction.

pennated muscles can create greater force than fusiform muscles with similar volume.[10]

A key relationship in muscle force capacity is the length-tension relationship (Figure 1.4). Force production is linked to active and passive tension of a muscle. Muscles tend to generate the most active force when in a mid-range position. When muscles are shortened or elongated from neutral, alterations in actin-myosin overlap at the sarcomere are thought to reduce the creation of active tension. With elongation, particularly of two joint muscles, active tension may decrease, yet passive tension of the muscle actually increases, to create a greater total tension than when in mid-range or shortened positions.[10] This may help explain why eccentric muscle contractions allow control of greater loads.

The load-velocity relationship is another concept in muscle activity. There appears to be an inverse relationship between the external load applied and the speed of shortening of a concentrically contracting muscle. This makes sound clinical sense, as we expect patients will be able to perform a task more rapidly when they have less resistance. If an external load equals the maximal force a muscle can apply, there will be no movement, and the muscle will be working isometrically. When the load increases beyond force exertion, the muscle will elongate, or eccentrically contract. The muscle will elongate faster as the load is increased. This will be important to consider when determining the amount of load and resistance needed to assure safe task performance.

The force-time relationship suggests that as the time of contraction increases, so will the amount of force generated. Slower muscle contractions may allow appropriate contractile protein positioning, in turn enhancing the capacity to reach maximal tension. This is relevant to clinicians creating rehabilitation programs. For a patient recovering from a hamstring strain who is attempting to improve muscle performance impairments, performing knee flexion rapidly against minimal resistance will not develop as much strength as performing more controlled contractions.

Power affects the speed at which tasks are performed. Power is defined as the rate of work (force x distance) performed over time, and is usually described in units of joules. When a task is performed in a shorter duration of time, more power can be generated. Plyometric and sport tasks are often considered to be powerful movements, yet slow movements also can require significant power. For example, even if performed at a slower speed, exerting force over a large distance can create substantial work. The role of power can be essential in the rehabilitation or training of the patient/athlete, to allow a given MTU to develop appropriate capacity to complete a given task.

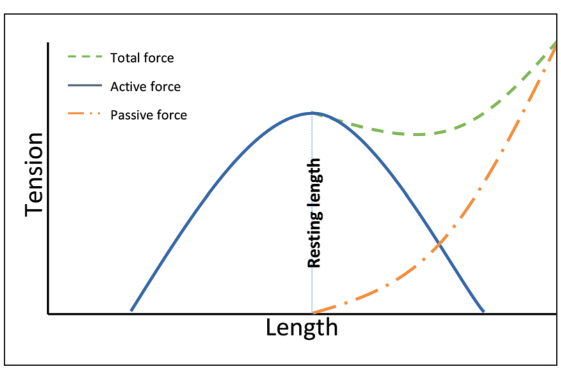

Figure 1.4. Length-tension relationship.

Muscle fatigue is an additional concept to consider in understanding force production. In contrast to the fatigue we see in clinical settings, muscular fatigue is defined as an exercise-induced reduction in the ability of muscle to produce force or power.[11] A wide variety of mechanisms have been hypothesized for the development of fatigue, including reduced excitability at the neuromuscular junction or sarcolemma, changes in excitation-contraction coupling, alterations in contractile mechanics, reduced blood flow or oxygenation supply.[10] Whether involved muscles are primarily slow-twitch (Type I aerobic) or fast-twitch (Type IIx anaerobic or IIa mixed), when the muscle is no longer able to meet the demands of a task, performance will decline.

Tendon tolerance to tensile load

Basic science studies allow researchers to investigate the mechanical properties of tissues in a number of different environments. The parallel orientation of tendon and longitudinal alignment from the bone to muscle interface suggests a primary ability to tolerate tensile load. By creating and plotting load elongation or stress-strain curves, various physiological properties of tendon tissue can be elucidated. Understanding the design principles and purpose of various biomechanical concepts serves numerous purposes. For example, extrapolation can produce essential clinical data in the design of plans of care.

A load elongation curve provides information regarding a tendon's capacity to tolerate tensile force, through loading the tendon to failure. On a load elongation curve plot, the x-axis represents the amount of elongation or deformation, while the y-axis represents the amount of load or force applied (Figure 1.5). The slope of the graph identifies the stiffness of the tissue, and the point at which continued elongation is coupled with a downslope is considered the tissue failure point. These curves can provide quantitative data regarding the ultimate load and elongation allowed prior to failure, as well as the total energy the tissue was able to absorb (the area under the curve).[12]

Similar to load elongation curves, stress-strain curves measure the capacity of a tissue under tensile load. Rather than elongation being a discrete measure of distance, strain (x-axis) is considered the tissue's percentage of elongation. The y-axis, or stress, is the load per unit of area. The slope of the stress-strain curve reveals the tendon's modulus of elasticity. In the majority of human tendons, the modulus of elasticity is linear, and increased loads create greater elongation. This is relevant for the

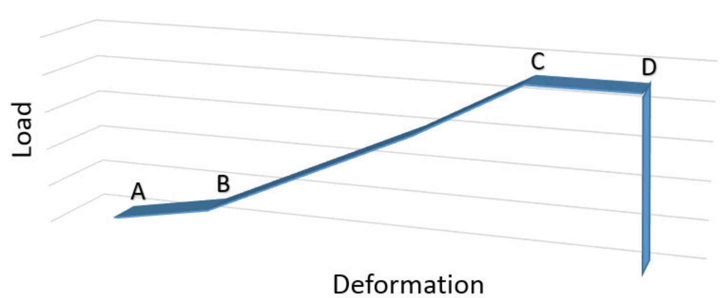

Figure Key. A – initiation of deformation; B – end of toe region, beginning of linear region; C – yield point; D – failure point, disruption of the tendon occurs. A to B – this period tensions the inherent tendon crimp, straightening out wavy collagen fibers; B to C – the slope of this linear period is described as the stiffness of the tissue; C to D – continued deformation occurs despite constant load, irreversible changes are made to the tendon tensile strength; Area under the curve – total amount of energy absorbed by the tissue

Figure 1.5. Load elongation curve.

orthopedic and sports clinician, in understanding how to stress a healing tissue without reaching the failure point.

While tendons can tolerate substantial tensile loading, compressive and torsional forces are not as well accepted. Compression, friction, and shearing of the tendon is frequently seen at the tendon's bony insertion. Care should be taken to consider appropriate and inappropriate forces applied (exposed) to the tendon during rehabilitation and performance enhancement programs.

Stretch-shortening cycle

While muscle function and human movement are often described in terms of isometric, concentric, and eccentric contractions, the human body actually uses stored energy and momentum to accomplish many functional repetitive movements. The stretch-shortening cycle (SSC) is a natural type of muscle function derived from the combination of eccentric lengthening and concentric shortening of muscles, to enhance the efficiency and performance of movement. The SSC is typically rapid and is integrated somewhat seamlessly into movement, yet it relies on a sequence of complex tissue interactions with mechanical, metabolic, and neural elements of involvement.[13]

Generally, an SSC occurs in two main conditions: (1) pre-activation and (2) variable activation of muscles preceding the functional movement.[14] The SSC is incorporated in activities involving both the upper and lower extremities. Jumping is a good example. Just before landing from a jump, the individual will have preparatory muscle activity to resist impact (pre-activation), followed by an active braking phase, stretching, and then immediate shortening of various lower extremity muscles such as the gastrocnemius muscle to propel the individual back off the ground (variable task specific muscle activation). Similar to compressing a spring and letting it recoil and jump off your hand, the MTU serves as a spring for the human body, using elastic strain energy storage to subsequently move the body part. It should be noted, however, that the overall MTU elongation is relatively small, somewhere in the range of 6% to 8% of the muscle-tendon length.[14] While the elastin and viscoelasticity of the tendon allow for some compliance and passive elongation, the relative MTU stiffness is important and necessary to avoid a loss of reflexive propulsion.

Clinical Methods of Identifying Tissue Tolerance and Capacity

A wide array of factors contribute to a person's capacity to perform normal movement. Some factors are modifiable while others are not. To estimate an individual's capacity to perform a task, clinicians should start by assessing the baseline function (or lack thereof) of the MTU. This should be completed at multiple levels of the International Classification of Functioning, Disability and Health model, the body functions and structure, activity and participation levels.

Muscle-tendon mobility

As noted previously, muscles can provide greater active force in mid-range positions, while passive tension increases with lengthening. It stands to reason that if a muscle is adaptively shortened, the muscle's passive and resultant total tension capacity would be limited, although conflicting evidence surrounds mobility impairments as a risk factor for injury.[15] If a muscle or joint system has mobility restrictions, tasks can still be functionally completed, but likely with compensation. Therefore, to estimate a MTU's capacity to generate and accept force, the MTU's mobility should be assessed, particularly in cases when muscles cross two joints.

In the lower extremity, restrictions are common in the hamstrings, rectus femoris muscle, and gastrocnemius muscle. These muscles crossing two joints consistently complete the SSC during daily activities and sports. To assess if the muscles can accommodate frequent fluctuations of position and load demands, therapists can use clinical tests to evaluate the length of the tissues, such as the Thomas test for the rectus femoris muscle (Figure 1.6). In tensioning two joint muscles, therapists should also consider the impact on mobility of additional tissues, such as the nerves. For example, to assess the functional mobility of the hamstrings in a patient with proximal hamstring tendinopathy ("opathy" is a suffix used to denote disease, derived from the Greek *pathos* or suffering), the clinician performs a straight leg raise (Figure 1.7). When the clinician reaches tissue resistance, the patient complains of posterior thigh tightness,