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EXERCISE INTERVENTIONS FOR THE MANAGEMENT OF TENDINOPATHY: AN OVERVIEW

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ABSTRACT

Objective: This article provides an overview of the efficacy of current exercise interventions for managing tendinopathy, and reviews the more up-to-date exercise prescriptions, such as the use of prolonged isometric contractions and tempo-driven strength training to suppress intracortical inhibition.

Discussion: The term tendinopathy is a widely accepted generic term that encompasses any abnormal condition affecting a tendon. While various medical conditions such as diabetes, prior use of fluoroquinolone antibiotics and/or hypercholesterolemia greatly increase the risk of developing a tendon injury, these injuries are most frequently the result of overuse, explaining the high prevalence in people participating in sports, particularly explosive sports such as basketball, soccer, and/or volleyball. Older athletes are especially prone to tendinopathy because of age-related decreases in tendon resiliency. Recent research shows that cortical inhibition develops following tendon injury and frequently results in impaired motor output to the damaged tendon that perpetuates chronicity. Performing prolonged isometric contractions and exercising to the beat of a metronome have been shown to suppress cortical inhibition, and these interventions should be considered in the management of all tendon injuries.

Conclusion: Rehabilitative exercises are discussed, including the importance of strengthening synergistic muscles in an attempt to offload the damaged tendon, the need to move through a full range of triplanar motion while performing rehabilitative exercises, and the benefits of strengthening tendons and muscles while they are in their lengthened positions. A detailed exercise protocol for managing non-insertional Achilles tendinopathy is reviewed, and this exercise routine can be modified to treat any tendon in the body. *(J Contemporary Chiropr 2025;8:39-49)*

Key Indexing Terms: Tendinopathy; Prolonged Isometric Contractions; Tempo-Driven Strength Training; Metronome; Intracortical Inhibition; Interfascicular Gliding.

INTRODUCTION

Tendons, particularly the long tendons of the foot and ankle, are amazing structures in that they are strong enough to tolerate up to 12 times body weight without failure, yet flexible enough to store and return more than 90% of the energy used to stretch them (1) (Fig. 1). Because they connect muscle to bone, the natural elasticity of tendons reduces tensile strains that would otherwise damage neighboring muscle-tendon junctions. Additionally, the subsequent return of stored energy greatly improves our metabolic efficiency while walking and running, which is in large part responsible for our success as a species. Despite their impressive strength and resilience, tendons are injured with surprising regularity, accounting for approximately 30% of all overuse injuries. (2) The Achilles tendon is especially prone to injury because it operates close to its rupture point during high load activities. (3)

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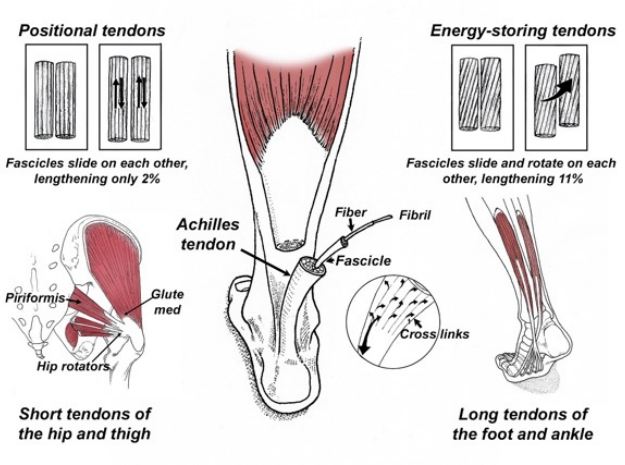

Figure 1. Tendon anatomy. Tendons are divided into fascicles, fibers, and fibrils, which blend with the corresponding connective tissue layers of muscles. Tendons derive their strength from the parallel arrangement of type 1 collagen fibers that are connected through a series of cross-links that distribute pressure evenly throughout the tendon. Note that while tendons in the proximal muscles, such as the glutes and quads, are short and relatively inelastic (they lengthen less than 2% when fully stretched), tendons below the knee are extremely long and flexible, lengthening as much as 11% when fully loaded. Due to their functional differences, tendons above the knee are referred to as positional tendons, while tendons below the knee are referred to as energy-storing tendons.

DISCUSSION

Microstructural Tendon Changes

Unfortunately, once injured, tendons become overly flexible and often develop long-term strength deficits that increase the risk of future injury. (4) At a cellular level, tendinopathic tendons present with increased cell numbers and ground substance, vascular hyperplasia, increased concentration of neurochemicals, and disorganized and immature collagen. (5) In a particularly detailed study in which rat tendons were loaded to failure while analyzing microstructural changes within the tendon, researchers from Mount Sinai School of Medicine noted that at higher levels of tensile force, there was "dissociation among fibers, transversely oriented fiber discontinuities, and isolated rupture patterns with fibers that appeared distorted in alignment and deformed into a curl formation." (6) The disrupted and torn fibers alter the structural integrity of the tendon, making it significantly more compliant as the weakened tendon stretches when loaded. Over time, the increased compliance in tendinopathic tendons results in long-term strength deficits as the attached muscular structures are forced to take up the slack from the more compliant tendon, which forces the muscles to work in their shortened positions and produces long-term weakness by compromising the length/tension relationship of the involved muscle fibers.

Heavy Load Eccentric Protocols

Because tendons are living tissue that respond to mechanical forces by altering their molecular structure (a process known as mechanotransduction), tendinopathies have been treated with a range of therapeutic exercises designed to improve tendon strength and function. By far, the most frequently prescribed exercise prescription for managing tendinopathy is the heavy-load eccentric exercise program described by Alfredson et al. more than 25 years ago. (7) Because eccentric muscular contraction produces a subtle vibration in the muscle that gets transferred into the tendon, it has been suggested that the vibration associated with eccentric contractions somehow stimulates tendon remodeling. (8) Despite the popularity of heavy-load eccentric training, these protocols have recently been proven to be relatively ineffective, as 60% of people treated with this intervention report continued pain and discomfort 5 years later. (9) Because the heavy-load protocols tend to be uncomfortable, there is often low patient compliance due to increased pain, and some studies show these protocols actually worsen pain. (10) Additionally, the theory that the subtle vibrations associated with eccentric contractions somehow stimulates tendon repair has been disproven, as heavy-load eccentric protocols in no way alter tendon structure or function. (11)

Recent Advances in Exercise Prescription

In an attempt to improve outcomes associated with exercise therapy, researchers are looking beyond heavy-load eccentric protocols to determine if there are specific exercise interventions that can actually stimulate tendon remodeling. To that end, some interesting new research out of Australia shows that fluid flow dynamics within the tendon may play a key role in accelerating recovery. (12) These authors cite numerous studies showing when a healthy tendon is exercised, there is a significant flow of fluid away from the core of the tendon, and the shear stress from this fluid flow creates a tensile strain that stimulates tendon remodeling (13-15) (Fig. 2). This outflow of fluid is absent in tendinopathic tendons.

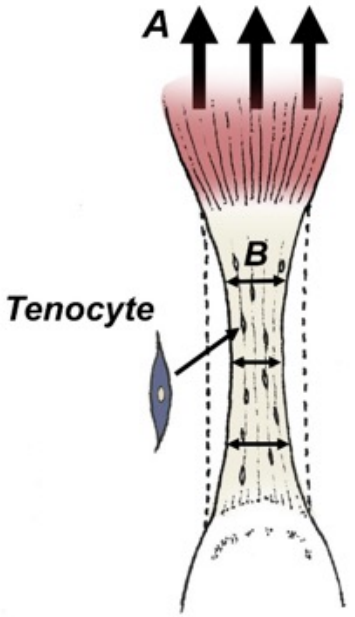

Fig. 2. Because tendons are made of nearly 70% water, muscle contraction (A) creates an internal force that squeezes fluid from the tendon (B), comparable to twisting a wet towel. Movement of the fluid stimulates specialized cells called tenocytes to accelerate tendon remodeling.

To identify which exercises most effectively stimulate intratendinous fluid flow, the authors used 3-dimensional ultrasonography to monitor flow dynamics as participants performed isometric contractions at intensities that varied from 35 to 75% full effort, with load durations ranging from 2 to 8 seconds. Upon completion of the study, the authors determined the subjects performing the heavy-load, long-duration exercises had a 13% reduction in tendon volume, which was far and away greater than any other treatment group. As a result, the authors state that in order to accelerate tendon repair, "the applied load must be heavy and sustained for a long-duration."

Additional research is showing that exercises that improve interfascicular gliding between tendon substructures may play an important role in the management of tendinopathy. Researchers from Finland show that when people with healthy Achilles tendons exercise, different portions of the gastrocnemius and soleus muscles pull on their corresponding tendon fibers, creating a nonuniform pattern of interfascicular sliding within the Achilles tendon. (16) The sliding of one tendon fiber against another mechanically stimulates tenocytes to accelerate tendon remodeling (Fig. 3).

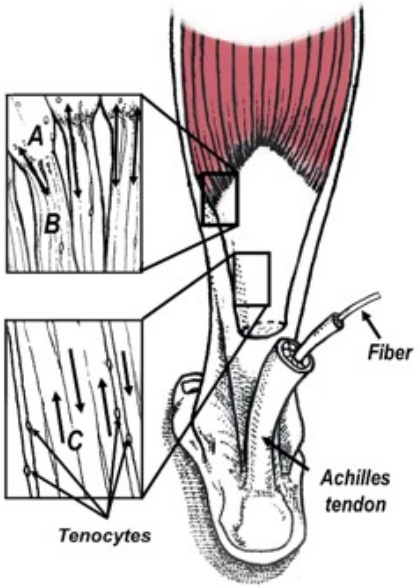

Fig. 3. Because muscle fibers (A) attach to corresponding tendon fibers (B), when individual muscle fibers contract, their respective tendon fibers slide over one another (C), generating a mechanical shear force that stimulates tendon remodeling. In a healthy tendon, all available muscle fibers pull on their corresponding tendon fibers, which in turn stimulates remodeling by creating widespread interfascicular sliding throughout the tendon.

In contrast to healthy tendons, individuals with damaged tendons show a more limited variation in interfascicular sliding. The limited variation is due to the fact that many of the sub-tendon structures have been ruptured or mechanically compromised and they are unable to transfer tensile load. Referred to as stress deprivation or stress shielding, these small, damaged sections of the tendon are not exposed to tensile strain, and hence are incapable of remodeling. In fact, Cook et al. (17) claim the damaged portions of the tendon are beyond repair, and rehabilitative exercises should focus on strengthening the healthy unaffected fibers. The authors make the interesting comment that you should "treat the doughnut, not the hole." In contrast, Keith Baar (18) emphasizes that prolonged isometric contractions actually do stimulate repair of the damaged sections. Baar states that during prolonged isometric contractions, the healthy portions of the tendon stretch under the applied load, exposing the damaged section to tensile strain. Baar sites several

case histories in which the "hole" in a tendinopathic tendon gradually disappears in response to prolonged isometric contraction protocols. Additional research by Kubo and others (19,20) confirm that prolonged isometric contractions, especially when performed with the tendon in a lengthened position, can improve tendon compliance by as much as 50%. (21) These studies all emphasize that the load should be approximately 70% of maximum voluntary contraction, and the length of time under tension usually varies between 30 and 45 seconds. Because the enzymes responsible for tendon repair become quiescent after the first 10 minutes of exercise and then become active again 6 hours later (22), a typical exercise regimen is to perform 4, 30-second isometric contractions at near full effort, preferably with the tendon in a lengthened position, and repeat that routine every 6 hours.

To identify exactly which exercises increase the amount of interfascicular sliding, Handsfield et al. (23) evaluated tendon fiber sliding patterns as people performed different calf exercises. Of all the exercises studied, the bent knee heel drop exercise that specifically targets the soleus muscle produced the greatest increase in interfascicular sliding while simultaneously placing the least stress on the tendon. The authors claim that this exercise will have "a major role in the future of tendon rehabilitation." The outcome of this paper is consistent with recent research showing that weakness of the soleus is the single best predictor of non-insertional Achilles injury. (24) Because the soleus possesses 50% more muscle mass than the neighboring gastrocnemius muscle, failure of the soleus to generate force would significantly impair interfascicular tendon sliding, which could be corrected with bent knee heel drop exercises. Additional research shows that weakness of the lateral gastrocnemius also contributes to Achilles tendinopathy (25), as fibers from that portion of the muscle fail to pull on their corresponding tendon fascicles, which causes them to weaken over time. Exercises that target the lateral gastrocnemius, like straight leg toe-in heel raises, can improve tendon function by enhancing interfascicular gliding in the corresponding component of the Achilles tendon. These studies confirm that the best way to treat a tendinopathy is to prescribe an exercise that stimulates the entire muscle, not just portions of it. In some cases, this may require prolonged, high force isometrics and/or the use of blood flow restriction training, which creates a preferential shift towards fast twitch muscle fibers and has the added benefit of increasing the production of growth hormone. Several studies have shown that blood flow restriction training accelerates tendon remodeling. (26,27)

The Role of the Central Nervous System in Chronic Tendinopathy

Perhaps the most interesting research on exercise intervention for the management of tendinopathy is showing that in order to get the best outcomes, the prescribed exercises must improve strength and resilience at the cellular level of the tendon itself, and also improve sensory/motor processing in the central nervous system. Over the last few years, a series of important papers have shown that the brain plays a huge role in perpetuating tendon injuries by amplifying sensory information from damaged tendons and creating inhibitory signals in the motor cortex that reduce and/or delay motor output to the damaged tendon. (28-31) Although the cortical inhibition is initially protective in nature as it reduces the muscular force that can be passed into the damaged tendon, over time, the cortical changes become permanent, resulting in long-term strength deficits that gradually weaken the tendon and impair athletic performance. Cortical inhibition explains why most people possess significant endurance deficits that persist years after the tendon has healed. The current theory is that inhibitory neurons form between the motor cortex and the midbrain, and these newly formed neurons release the neurotransmitter GABA, which is a powerful inhibitor of motor function. Rio et al. (31) compare the newly formed cortical inhibition to driving a car while keeping one foot on the brake and the other foot on the accelerator: the car cannot accelerate while the brake is being pressed down.

Involvement of the central nervous system explains why some people with relatively healthy tendons complain of significant pain, yet 35% of an asymptomatic population present with cellular changes consistent with tendinopathy yet have no discomfort (32). Coombes et al. (33) came up with an interesting model in which they propose tendinopathy should be conceptualized as 3 interrelated components: tendon pathology, sensory changes in the pain system, and impairment in the motor system (Fig. 4). The authors support this statement by noting the sensory system can overreact to tendinopathy due to the increased presence of substance P and calcitonin gene-related peptide reactive nerve fibers that form in damaged tendons. These neurochemicals are potent pain modulators and explain why patients with tendinopathy have approximately 50% reductions in pressure pain thresholds and are often hypersensitive in response to noxious stimulation. (33) Recent research shows that reducing pain in a tendinopathic tendon can improve

motor performance and reduce pain catastrophizing (34). Coombes et al. (33) claim that by perceptualizing tendinopathy as having separate sub components, it may be possible to improve outcomes by matching individual patient presentations to the most effective treatment approaches.

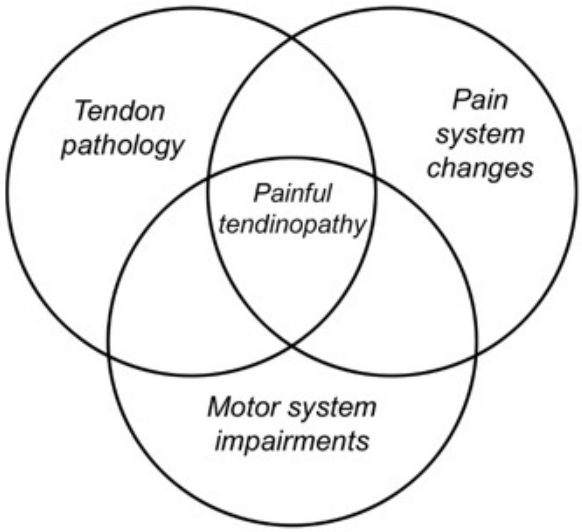

Fig. 4. Pathophysiological model of tendon loading described by Coombes et al. (33). This model addresses the sensory, motor, and local cellular changes associated with tendinopathy.

To study the effects of cortical inhibition on motor output, researchers use a device called transcranial magnetic stimulation. This technique involves the application of an electromagnetic field to specific areas of the motor cortex, and then measuring the muscle's reaction to the applied stimulus. In 2015, in 1 of the first studies to evaluate the effect of intracortical inhibition in injured athletes, researchers from Australia (31) used transcranial magnetic stimulation and determined that volleyball players with patellar tendinopathy had markedly higher levels of cortical inhibition compared with their noninjured counterparts, and the degree of cortical inhibition correlated strongly with the degree of pain. In 2022, Orssatto et al. (28) used transcranial magnetic stimulation on runners with and without Achilles tendinopathy and demonstrated that compared to the uninjured runners, runners with Achilles tendinopathy had 14.3% greater amounts of cortical inhibition, which was associated with significant reductions in calf endurance. The authors bring up an important point by noting that when the central nervous system inhibition is involved, the resultant strength deficits associated with the tendon injury are present bilaterally, not just on the injured side. This is because cortical inhibition has been shown to affect both sides of the body, even when only one side is injured. (28) The injured athletes in their study had a 38% decrease in calf endurance on the symptomatic side, and a 34% decrease in calf endurance on the asymptomatic side. There is no way that bilateral weakness would be present unless that weakness was driven by the central nervous system.

Exercise Interventions to Suppress Intracortical Inhibition

Because of the strong correlation between cortical inhibition and tendinopathy, researchers are attempting to figure out ways to reduce cortical inhibition with various exercise interventions. In a particularly interesting study, Rio et al. (36) compared the effect of a single session of prolonged isometric contractions versus a heavy-load eccentric/concentric training protocol. Subsequent evaluation with transcranial magnetic stimulation confirmed that only the isometric group had significant reductions in intracortical inhibition, along with immediate reductions in pain and importantly, a nearly 20% improvement in strength. Note that neither cortical inhibition, pain, nor strength was affected by the heavy-load strength training program.

What makes this research so fascinating is that it explains why conventional heavy load eccentric protocols have fared so poorly: they produce pain and pain produces cortical inhibition. Surprisingly, the vast majority of heavy-load tendon strengthening protocols recommend these exercises be performed even if they produce significant discomfort. In fact, the original protocol described by Alfredson et al. (7) recommends performing these exercises just short of the point that the pain becomes "disabling." Rio et al. (36) state the poor outcomes associated with heavy load programs may result from the fact that experiencing pain while exercising may negatively alter motor control and cause cortical reorganization, "as pain itself is known to alter cortical representation."

The most fascinating research evaluating various ways to incorporate exercise to reduce intracortical inhibition relates to the use of metronomes while strength training. In a detailed 2024 systematic review of corticospinal inhibition, Gordon et al. (29) demonstrate that tempo-driven strength training, in which a

metronome is used to time eccentric and concentric components of an exercise, can effectively suppress cortical inhibition and improve corticospinal plasticity, physical performance, and strength. Interestingly, the rate of force development improved significantly with metronome training. The authors state the mechanism responsible for improved corticomotor function following metronome training relates to the fact that forcing an individual to exercise at a specific cadence increases the precision necessary to maintain the specific timing of the movement, "thus strengthening existing neural connections and potentially the formation of new connections via the removal of local inhibition." The authors reference research showing that even isometric contractions are more effective at reducing cortical inhibition when done to the beat of a metronome. (37)

Exercise Intervention

The exercises described in figure 6 incorporate prolonged isometric contractions and metronome training. The prolonged isometric contractions act as a natural analgesic, enhance fluid outflow from within the tendon, and improve motor function by suppressing intracortical inhibition. All of these factors address all 3 subcomponents of tendinopathy described by Coombes et al. (33) While several studies have incorporated metronomes performed at 20 beats per minute (31,38), this protocol incorporates a much higher frequency of 80 beats per minute, which is similar to single limb cadence present while running (each foot hits the ground approximately 80 to 90 times per minute while running). Research shows that exercising at a higher metronome cadence increases the rate of force production and improves neuromotor agility. (39)

The Importance of Strengthening Synergists

To strengthen the synergists of the Achilles tendon, particularly peroneus longus and flexor hallucis longus, a warm up is performed by doing 25 repetitions on a ToePro exercise platform at a rate of 80 beats per minute. Although these exercises can be done on a standard slant board, this particular device places the long and short digital flexors and the superficial and deep posterior calf muscles in their lengthened positions while they are being exercised, which has been proven to result in significantly greater muscle and tendon remodeling than conventional exercises. (19,20) Exercising muscles in their lengthened positions also allows for a greater transfer of strength gains to dynamic performance. (20) Strengthening the flexor hallucis longus muscle is particularly important for offloading a damaged Achilles tendon. In a recent MRI study of 120 people, half with and half without Achilles tendinopathy, Wirth et al. (40) show that patients with Achilles tendinopathy present with significant hypertrophy of the flexor hallucis longus muscle, which the authors attribute to compensatory overactivity of this muscle as it attempts to offload the damaged Achilles tendon. Another extremely important component of this exercise protocol is that the rearfoot is forced to move through a full range of inversion and eversion. Bojsen-Møller and Magnusson (3) state that moving the rearfoot through a full range of frontal plane motion is essential for Achilles recovery as calcaneal eversion stresses the medial side of the tendon while calcaneal inversion stresses the lateral side of the tendon. The authors cite several studies showing that varying the degree of calcaneal inversion/eversion can increase intratendinous strain by as much as 15%. Lastly, the low load/high cadence was chosen to strengthen synergists because this particular exercise prescription was shown to accelerate muscle repair and remodeling better than conventional heavy load/low repetition routines. (41) The low loads are essential when managing painful tendons as they are less likely to produce pain.

The prolonged isometric contractions, which are the cornerstone of this program, are maintained for 30 seconds, which is the protocol recommended by Kubo and others. (19-21) The isometric contraction routine is slightly modified from most other protocols, in that the person is performing slight pulsing actions in synergistic muscles while maintaining the isometric contraction, which in addition to decreasing intracortical inhibition, may help create a motor engram to the synergists.

Personalized Exercise Prescription

While most studies recommend the isometric contractions be performed at about 70% full effort, research by Domroes et al. (42) suggest that this load may be too light for some and too heavy for others. These authors note that there has to be a specific "sweet spot" of resistance, which is the amount of resistance that causes the tendon fascicles to lengthen anywhere between 4.5 and 6.5% when loaded. While exercising in this range has been shown to improve tendon mechanical properties, exercising with weights that produce strain changes above this range may damage tendons. Conversely, using light weights that produce less than 3% interfascicular lengthening may be insufficient to produce tendon remodeling. In their study, the amount of weight necessary to produce the

ideal 6.2% tendon strain varied between 47% and 90% full effort. The authors state that by personalizing an exercise prescription by modifying the weight to keep tendon elongation in that 6.2% sweet spot, clinical outcomes should significantly improve. Although these authors used ultrasonography and special force sensors, it may be possible to determine the ideal tendon load simply by looking at the thumb radius index (Fig. 5).

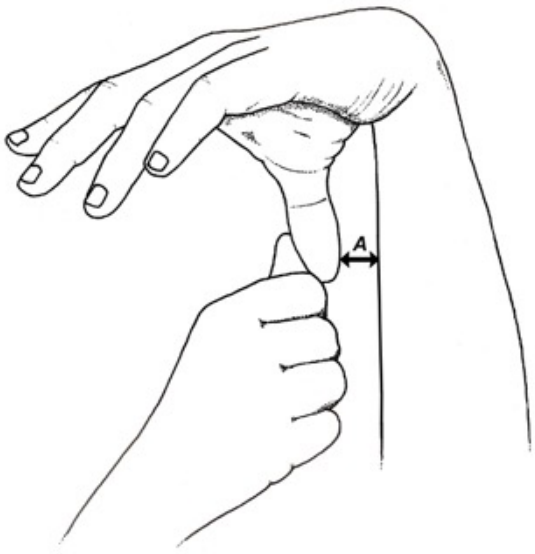

Fig. 5. The thumb to radius index (A) is measured with the wrist flexed and radially deviated. Hypermobility is present when the thumb can be positioned within 2 cm of the radius (45).

This index is a sensitive marker for global laxity, which is an indicator of collagen strength. Individuals with the thumb radius index of less than 2 cm should use less resistance during the prolonged isometric contractions, pushing themselves to about 50% full effort. In contrast, people with thumb radius indexes of 12 cm or more should use heavier weights, approaching 90% full effort. Although unproven, the connection between ligamentous laxity and interfascicular sliding is not as far-fetched as it may sound. Several studies have shown a strong correlation between poor collagen content in skin (as measured via excessive skin elasticity) and reduced collagen in bone. (43,44) Because excessive skin elasticity correlates strongly with reduced bone mineral density, it is possible that a similar correlation may exist between ligamentous laxity and increased tendon compliance.

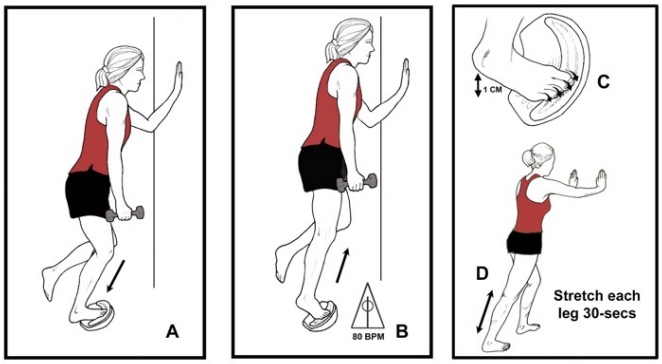

Fig. 6. Exercise intervention for managing non-insertional Achilles tendinopathy using a ToePro. See text for discussion.

Non-Insertional Achilles Tendinopathy Exercise Intervention

The exercise routine begins by placing a ToePro near a wall while holding a weight in one hand and contacting the wall with the opposite hand for balance (A). You decide how much weight to hold based on your level of fitness. Ideally, you should be able to perform between 20 to 25 repetitions before you fatigue. If you are unable to do 20 repetitions, use less weight. If you can do more than 25 repetitions, increase the weight so you are fatigued after performing the 25th repetition. Extremely fit athletes can often do this exercise while holding 40 to 50 pounds, while beginners often need to lean against a supportive surface, like a table, so they are able to do a minimum of 20 repetitions. You start the exercise by positioning your foot so the tips of your toes are in the top groove of the ToePro. Next, set a metronome to 80 bpm and do 25 single-leg heel raises in time with the metronome (B). Your knee should be slightly flexed at the low point of the exercise and straight at the high point. Once finished with your 25th single-leg heel raise, lower your heel so that it is 1 cm from the floor and hold a 30-second isometric contraction. Importantly, while holding this isometric contraction, you should be lightly tapping the tips of your toes into the ToePro in time with the beat of the metronome (arrows in C). Note that people with cortical inhibition are often unable to tap their toes in time with the metronome, and if this is the case, reduce the beat frequency of the metronome until you find a tempo you can keep up with. Over time, gradually increase to 80 bpm as you improve. After completing the isometric contraction with toe pulses, perform 25 single-leg repetitions on the opposite leg, and finish with another 30-second isometric contraction with toe taps. When finished with your first set, take a 60-second rest. During this rest, hold a straight-leg calf stretch for 30-seconds on each leg (D). Repeat the same

routine two more times, making sure to take 60-second stretch breaks between sets.

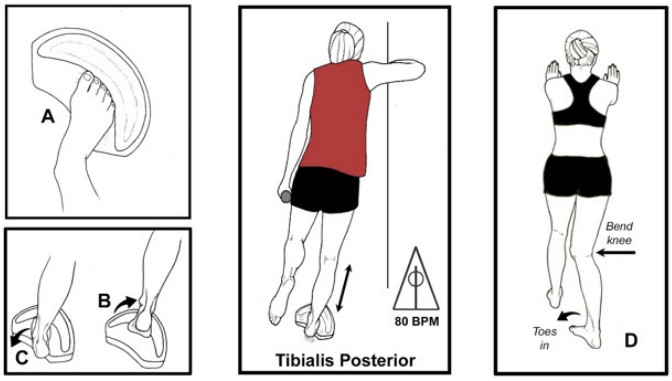

Figure 7. Tibialis posterior exercise.

The next 2 exercises target the tibialis posterior and peroneus longus muscles. To exercise tibialis posterior (Figure 7), rotate the ToePro slightly so your 2nd through 5th toes line up with the top crest (A), and then lean sidewards into a wall holding a weight in the hand opposite the wall. Next, repeatedly raise and lower your heel in this position, making sure you actively raise your arch as much as possible while going up (B), and lower your arch as you get towards the low point of the exercise (C). Keep your knee slightly bent the entire time while doing this exercise and time your heel-raises to the beat of the metronome. After your 25th repetition, hold a 30-second isometric contraction with your heel close to the floor (as in C) and your knee slightly bent. This duplicates the exercise described by Handsfield et al. (23) that maximizes interfascicular sliding while minimizing tendon strain. Your foot should be fully everted while performing this isometric contraction, which increases interfascicular sliding along the medial side of the tendon (3). You should perform subtle pulses where you slightly raise your arch in time with the metronome during the isometric hold. Note that these movements are subtle and should not cause discomfort. This routine is repeated on the opposite leg and when finished, perform a 30-second stretch on each tibialis posterior (D).

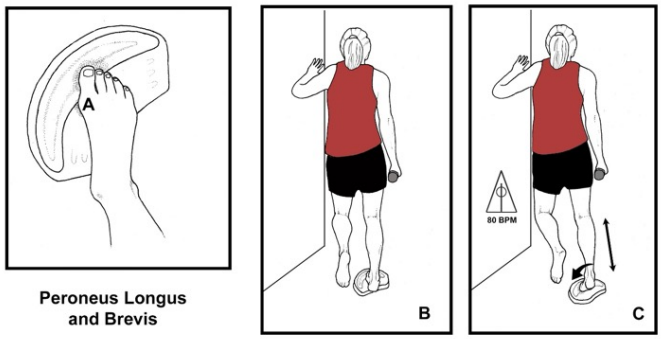

Figure 8. Exercise for the peroneal muscles.

To exercise peroneus longus (Figure 8), rotate the ToePro so your big toe fits into the upper crest and your inner forefoot is near the center base of the crest (A). Next, lean sidewards into the wall with a weight held in the opposite hand (B). Once stable in this position, repeat single-leg heel raises driving your inner forefoot and great toe into the ToePro (C). This is an excellent exercise, and you will feel your peroneal muscles fire vigorously while performing 25 repetitions. As with the other exercises, finish by lowering your heel near the floor and maintain a 30-second isometric contraction, attempting to pulse your big toe and inner forefoot into the foam in time with the metronome. Throughout the isometric contraction, your knee should be slightly flexed and your heel should be fully inverted to maintain the peroneals in their lengthened positions, which increases interfascicular sliding along the lateral side of the Achilles tendon (3). The exercise routine is complete when you perform the same routine on the opposite side and finish with a 60-second rest. While resting, you should perform 30-second bent-knee calf stretches on each leg (as in the tibialis posterior routine). Note the 30-second stretching durations used in this protocol were chosen because stretching a muscle for this length of time temporarily lengthens the muscle without impairing performance (18).

CONCLUSION

While future research will help define the ideal exercise prescription (e.g., intensities, sets, reps, frequencies, and durations), current evidence strongly suggests that a combination of metronome-based isotonic exercises coupled with prolonged isometric contractions can effectively rewire the central nervous system to reduce harmful cortical inhibition, while simultaneously stimulating tendon repair. Better outcomes may be achieved by performing the prolonged isometric contractions with muscles and tendons in

their lengthened positions, and when synergists are strengthened through a full range of triplanar motion. These interventions may be more effective when they are customized to the individual by modifying the intensity based on pain and/or joint laxity. While the later stages of tendon rehab should focus on ways to increase strength and agility through the use of sport-specific plyometric drills, the initial stages of rehabilitation should focus on ways to reduce pain, improve tendon strength and just as importantly, correct the faulty cortical inhibition that is perpetuating tendon dysfunction by inhibiting motor output.

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