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Proximal Hamstring Tendinopathy: Clinical Aspects of Assessment and Management

Proximal hamstring tendinopathy (PHT) is common among distance runners and athletes performing either primarily sagittal plane (eg, sprinting, hurdling) or change-of-direction activities (eg, various football codes and hockey).[34,49] The condition may also affect people who do not participate in sport[27,49,68] and, not uncommonly, presents bilaterally in this demographic. Characteristics of PHT include deep, localized pain in the region of the ischial tuberosity that often worsens during or after running, lunging, squatting, and sitting. Diagnosis can be challenging, as tendinopathy of the hamstring origin is one of several potential sources of symptoms in this region. To date, there is very limited evidence to guide management. The aim of this article is to review clinical aspects of PHT assessment and management, including differential diagnosis and exercise prescription. Recommendations will be based on current evidence and understanding of pathology and pain in tendinopathy.

SYNOPSIS: Proximal hamstring tendinopathy (PHT) typically manifests as deep buttock pain at the hamstring common origin. Both athletic and nonathletic populations are affected by PHT. Pain and dysfunction are often long-standing and limit sporting and daily functions. There is limited evidence regarding diagnosis, assessment, and management; for example, there are no randomized controlled trials investigating rehabilitation of PHT. Some of the principles of management established in, for example, Achilles and patellar tendinopathy would appear to apply to PHT but are not as well documented. This narrative review and commentary will highlight clinical aspects of assessment and management of PHT, drawing on the available evidence and current principles of managing painful tendinopathy. The management outline presented aims to guide clinicians as well as future research. *J Orthop Sports Phys Ther 2016;46(6):483-493. Epub 15 Apr 2016. doi:10.2519/jospt.2016.5986*

KEY WORDS: *evaluation, tendon, treatment*

Anatomy

The hamstring muscles have a common origin on the lateral aspect of the ischial tuberosity. Semitendinosus and the long head of the biceps femoris share a conjoined tendon originating from the lateral facet, whereas the semimembranosus origin is deeper,[67] although anatomical variation is reported.[33]

The location of hamstring tendon pathology may vary; Lempainen et al[49] reported semimembranosus pathology in all cases, whereas Benazzo et al[10] found considerable variability (common hamstring tendon, 23%; biceps femoris, 41%; semimembranosus, 29%; and semitendinosus, 6%). Midportion tendinopathy in PHT has not been specifically reported in the literature, although this may comprise a part of the cohort described by Lempainen et al.[49] Midportion pathology more typically involves the semimembranosus and may be distinct from the acute partial or complete tears in this region described by Askling et al.[6,7]

Proximal hamstring tendinopathy is considered to be an insertional tendinopathy, and compression of the tendon at its attachment during hip flexion/adduction is thought to be a key etiological factor.[23] Evidence supporting this theory is limited, as pathoanatomical studies of the proximal hamstring tendons are sparse.[33] Shear force between the hamstring attachment and ischial tuberosity has been reported when replicating in vivo loading,[36] as well as increased displacement of the proximal hamstring tendon with increased hip flexion angle.[38] Further research is required to better define the nature and degree of tendon compression in functional activities.

Etiology

Etiology of tendinopathy is multifactorial, involving load-related extrinsic and

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intrinsic factors. Extrinsic factors include training errors, such as increasing volume or intensity too quickly, particularly the sudden introduction of sprinting, lunging, hurdles, or hills. Several case series[42,95] have been identified that report these training errors to precede PHT. These activities require the hamstring to contract or lengthen while in hip flexion and may result in provocative tensile and compressive load at the tendon insertion.[29,86] Symptoms may also occur due to excessive use of static stretches, for example, yoga and Pilates postures that involve sustained end-range hip flexion. In some patients, compressive load simply from sitting is the main load-inciting factor.[48]

Systemic factors may also influence risk of PHT. These are suggested to include genetic polymorphisms (eg, COL5A1 that encodes for collagen type V), age, body mass index, metabolic issues (eg, lipid-level imbalance, glucose intolerance, insulin resistance), hormonal changes, and, rarely, medication (eg, fluoroquinolone antibiotics),[9,50,92] all of which may increase the risk of developing tendinopathy.[1] Perimenopausal women with PHT are likely to have a systemic predisposition to their tendon pain, as loss of estrogen at menopause is thought to negatively influence tendon homeostasis.[37] Systemic factors are thought to reduce the threshold for tendon pain and pathology from load-related factors.

Hamstring Function

An understanding of hamstring muscle function is important in PHT rehabilitation. In upright running, which can be a key aggravating factor in athletic populations, the hamstrings eccentrically decelerate knee extension in the terminal swing phase. Peak force occurs in late swing, with a second peak reported in early stance.[18,35]

Energy storage in the late swing to early stance stretch/shorten cycle is likely to be a major contributor to hamstring origin overuse injury, and eccentric/concentric transition is associated with higher hamstring loads.[79] Elastic energy storage in the tendons and aponeuroses increases efficiency of locomotion at higher speeds in animals.[2] The hamstring origin may be subject to higher energy storage loads in greater hip or trunk flexion, for example, when running with forward trunk lean, overstriding, and during uphill running.

Given these functional requirements, eccentric bias and stretch/shorten cycle exercise have been recommended by some authors for hamstring muscle injury to facilitate muscle hypertrophy, strength, and length-tension changes (greater strength nearer to end range),[55] as well as return to sport.[8,88]

TABLE — Differential Diagnosis in Proximal Hamstring Tendinopathy

- Sciatic nerve irritation at the piriformis muscle or near the ischial tuberosity
- Ischiofemoral impingement
- Unfused ischial growth plate in a postadolescent athlete
- Apophysitis or avulsion among adolescents
- Deep gluteal muscle tear
- Posterior pubic or ischial ramus stress fracture
- Partial or complete rupture of the proximal hamstring tendon

Pathology and Pain

The pathological features in PHT are similar to those seen in common tendinopathies such as the Achilles and patellar tendons.[49] Tissue samples from pathological proximal hamstring tendons show increased cellularity, ground substance accumulation, collagen disorganization, and neurovascular ingrowth.[49] Cook and Purdam[25] recently proposed the continuum model of tendon pathology, where diffuse increased cellularity and ground substance (reactive tendinopathy) precede focal areas of collagen disorganization and neurovascular ingrowth, with progression over time to a morphology with discrete islands of degenerative tendinopathy. Malliaras et al[54] demonstrated an ordinal progression of patellar tendon ultrasound imaging pathology from diffuse (reactive) to localized (degenerative) changes among volleyball athletes. There is no evidence of similar structural groups on imaging in PHT. Most studies report localized tendon pathology at the enthesis, bone edema, and insertional tendon clefts.[10,28,48] As with Achilles, patellar, and other tendinopathies, asymptomatic tendon pathology is not uncommon. Alternatively, a clinical diagnosis of PHT without significant demonstrable pathology is also possible.[28] The focus of treatment in early presentation should therefore be on managing pain. The exact source of pain generation in tendinopathy is yet to be identified. The reader is directed elsewhere[75,80] for a comprehensive discussion of potential tendon pain mechanisms.

Subjective Assessment and Screening of Other Potential Pathology

Diagnosis of PHT is complex, requiring careful elucidation of subjective history, screening of other potential pathology, and utilization of commonly advocated diagnostic tests. Subjective assessment of typical tendon pain behavior should confirm well-localized ischial tuberosity pain that becomes less symptomatic after a few minutes of activity (eg, "warm-ups" when running) but is worse afterward.

Activities requiring deeper hip flexion, such as squatting or lunging and sitting for long periods, especially on harder surfaces, are often provocative. Proximal hamstring tendinopathy is rarely painful during activities that do not involve

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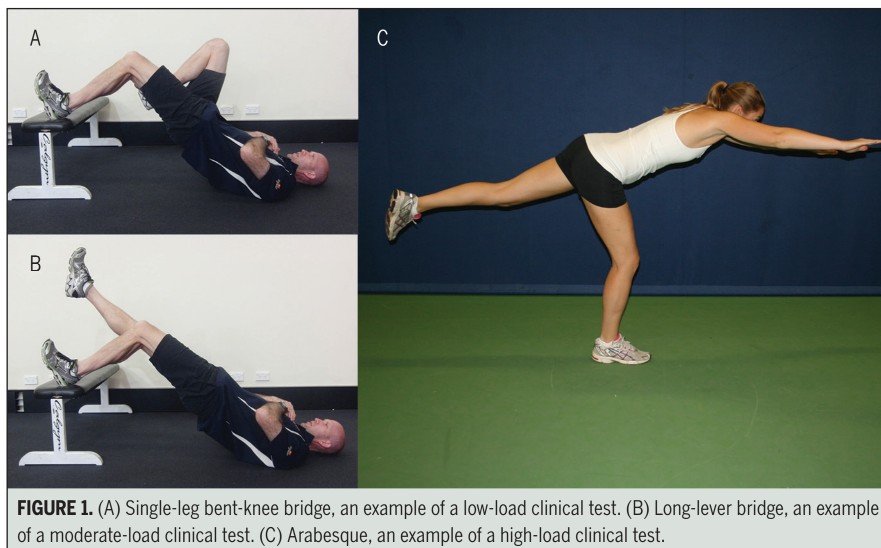

FIGURE 1. (A) Single-leg bent-knee bridge, an example of a low-load clinical test. (B) Long-lever bridge, an example of a moderate-load clinical test. (C) Arabesque, an example of a high-load clinical test.

energy storage or compression, such as slow walking on a level surface, standing, and lying. There may be stiffness in the morning or when starting to move after prolonged rest.

Some pain provocation after energy storage activities may be acceptable during rehabilitation, providing it lasts no longer than 24 hours.[44,83] Pain provocation of greater than 24 hours may be defined as "irritable," while pain that settles within 24 hours of energy storage loading can be defined as "stable."[53] After an initial warm-up, stable tendon pain may return toward the end of activity.

More diffuse symptoms may indicate lumbar, hip, or sacroiliac joint somatic referral; radiculopathy; or sciatic nerve compromise in the buttock, which is a common comorbidity given its proximity to the hamstring origin.[31,60,68] Multiple pathologies that may also cause buttock symptoms should be considered in differential diagnosis (**TABLE**). These pathologies often present with atypical tendon pain behavior (eg, more diffuse pain, aggravating factors are less specific to high hamstring load in hip flexion) and can be screened with provocative tests (eg, sacroiliac joint provocation tests, repeated motions of lumbar spine, straight leg raise test, slump test, provocation tests for sciatic nerve entrapment, and hip impingement testing),[13,30,46,56,70,71,87,94] specific differential palpation, and imaging. Not uncommonly in more chronic presentations, apparent isolated hamstring pathology may coexist with other pathologies, adding further complexity to both diagnosis and management.

Partial or complete proximal hamstring ruptures may also cause buttock symptoms. Importantly, partial or complete proximal hamstring ruptures are characterized by an acute onset, with a mechanism of extreme hip flexion combined with knee extension and often accompanied with an audible pop.[7] The management of partial proximal hamstring ruptures may be similar to that of PHT, with a graded loading program based on symptoms, but is associated with a prolonged recovery time.[7] Askling et al[6] describes a comprehensive review of the management of complete and partial proximal hamstring ruptures.

Clinicians should be aware of the psychosocial factors that may influence pain and management of PHT and adopt a biopsychosocial approach where appropriate. Patients may have erroneous beliefs (eg, pain is damaging, pathology is serious and will limit improvement) associated with heightened attention, anxiety, and fear avoidance.[5] Central sensitization, an amplification of neural signaling within the central nervous system that elicits pain hypersensitivity,[97] may also be a factor. Bilateral sensory changes that may reflect medium-term central nervous system modulation have been reported in tendinopathy, potentially indicating central sensitization,[75] although this has not been investigated in PHT. This highlights that pain is not simply tendon nociception but a complex output that is influenced by many factors. Symptoms that would raise the suspicion of central sensitization include diffuse pain without a clear stimulus-response relationship,[85] secondary hyperalgesia,[75] and pain that is disproportionate to the nature or extent of the injury.[65] Questionnaires, such as the Central Sensitization Inventory and Pain Sensitivity Questionnaire, may assist in the diagnosis and assessment of central sensitization.[58,64,77] The role of psychosocial factors and central sensitization has not been studied extensively in tendinopathy and is a key area for further research to complement recent work in this field by Woolf[97] and Nijs et al.[65]

Diagnostic Tests

Pain with provocative loading tests may assist in the diagnosis of PHT, but further research is required to confirm the diagnostic accuracy of such tests. The principle is to reproduce pain by placing the proximal hamstring tendons under progressively increasing compressive and tensile load (by increasing hip flexion angle). An example of a load test assessment for these patients may consist of progression from the single-leg bent-knee bridge (a low-load clinical test; **FIGURE 1A**) to the long-lever bridge (moderate-load test; **FIGURE 1B**) to arabesque (**FIGURE 1C**) movements and the single-leg deadlift (high-load clinical tests).[24] These motions may be initiated slowly, adding speed if asymptomatic. The pain score should increase with load across these tests.

Three passive stretch tests (bent-knee stretch, modified bent-knee stretch, and Puranen-Orava test) have moderate to high validity and high sensitivity and specificity for diagnosis of PHT.[14] However, in the authors' experience, these

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tests may be negative in less symptomatic cases. Other clinical conditions may be responsible for reported symptoms, and the use of magnetic resonance imaging is recommended.[14] In addition, clinically, pain response to palpation appears to vary and may have low specificity for diagnosing tendinopathy.[22]

A recent systematic review suggests that higher-quality studies are warranted to investigate the clinical utilization of special tests for the diagnosis of hamstring injuries.[72] This reinforces the importance of combining test results with a detailed history to determine tendon pain behavior in diagnosing PHT.

Identifying Impairments, Activity Limitations, and Participation Restrictions for Determination of Treatment Approach

Further assessment to determine possible musculoskeletal intrinsic factors that may contribute to increased provocative load on the proximal hamstring tendons is pertinent. Broadly, this may include assessment of joint range of motion, strength, coordination, and functional tasks and should be related to individuals' sport and activities of daily living. While impairment findings in the hip have demonstrated limited diagnostic value,[70,71,73] they can be valuable in guiding treatment.[73]

Single-leg squatting is assessed in regard to pain and function, including coronal/frontal and sagittal plane movement patterns. Excessive lumbopelvic sagittal plane movement (ie, anterior tilt and hip flexion) has been linked to hamstring muscle injury,[40,41,57] with suggestion of increased stress at the hamstring origin.[66] This reasoning may support the inclusion of trunk stabilization/strengthening exercises, which have been utilized in a multimodal management approach to PHT.[26,42] However, there are challenges in the accurate measurement of lumbopelvic movement in a clinical setting.[78] In principle, a more posteriorly tilted pelvis position will reduce hamstring stretch during function[35]; hence, interventions directed at reducing anterior pelvic tilt (in standing, sitting, or running) have the potential to reduce provocative hamstring load.

Running or walking gait analysis, or sports-specific movement-pattern assessment, is critical in the management of PHT. Overstriding, excessive forward trunk lean, and increased anterior pelvic tilt are clinical findings that may increase provocative load on the hamstring origin tendons. Increasing running step rate reduces stride length and hip flexion at foot strike[39] and increases gluteal activity in terminal swing.[19] While this intervention has the potential to reduce provocative hamstring tendon load, the role of running-gait retraining has not been studied in the treatment of PHT.

Patients may present with hamstring weakness and atrophy, often in relation to long-standing symptoms. Case series have reported hamstring weakness on manual testing[42] and reduced knee flexion and hip extension strength[59] in athletes with PHT. Handheld or fixed dynamometry may be used to assess hamstring strength.[43,90,95,96] In the absence of these instruments, the authors suggest testing strength in a leg-curl machine to preferentially isolate the hamstring muscles, as a loaded hamstring curl achieves high levels of biceps femoris and semitendinosus activity with minimal gluteus maximus (GMax) recruitment.[4]

Other kinetic-chain deficits have the potential to increase hamstring origin stress concentration. Gluteus maximus atrophy has been reported in PHT and is thought to contribute to proximal hamstring overload.[27] Gluteus maximus weakness may be measured with a handheld dynamometer, although this may be a challenge in stronger athletes.[90] Weakness of the gluteus medius (GMed) has been associated with PHT[42,95] potentially as a result of an increase in hip adduction and/or contralateral pelvic drop during squatting or lunging.

The adductor magnus is a significant hip extensor through a large range of hip flexion,[89] and its muscle fibers are intimately related to the origin of the semimembranosus,[67] hence assessment of adductor function may also be warranted. Distal kinetic-chain weakness or restriction and quadriceps dysfunctions are less common but should be considered.

Hamstring flexibility (range of motion) appears to vary considerably. Greater hamstring tensile stress and absorbed energy have been reported at end range among flexible compared with less flexible people,[51] which may increase entheseal compression and injury risk in populations that stretch already flexible hamstrings repetitively (yoga).

The current status of limited research into tendon loads, muscle actions, and synergies relating to the hamstring origin region, as well as a deeper understanding and robust assessment of kinetic-chain dysfunction, are also worthy of mention.

Patient-Reported Outcome Measures

Recently, the Victorian Institute of Sport Assessment pain and function outcome questionnaire has been adapted for the hamstring origin.[15] This is an appropriate outcome measure with high reliability and validity.[15] However, where within-session assessment of pain is required, a visual analog scale (VAS) rating of provocative functional tests (eg, long- or short-lever bridge, single-leg bent-knee bridge, or, later in rehabilitation, the arabesque and/or single-leg deadlift) is preferable for immediate symptom response to exercise or other interventions. A Patient-Specific Functional Scale rating of key functional deficits can also be used for within- and between-session assessment.

Symptom Management: Load Modification

Training-load modification is critical in managing pain in patients with irritable symptoms. In practice, abusive compressive (hip flexion) and energy storage loads are limited until pain irritability settles to a stable level. Stable pain should be mild during activity (eg, VAS score of 0 to 3 out of 10) and settle within 24 hours of an

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intervention with a moderate to high tendon load, such as repeated lunging. Therefore, a key subjective question in determining irritability and effective load management is, "What activities increase your symptoms and for how long?" Not uncommonly, the patient may be able to continue some steady-state running within pain/aggravation guidelines; however, hills, starts, and hurdles should be avoided until later stages.

Silbernagel et al[82] found that continuing sport activities did not lead to worse Achilles tendinopathy rehabilitation outcomes compared to stopping sport activities in the first 6 weeks. This seems to be in contrast to the patellar tendon.[93] Runners will often report distances they are able to run symptom free, or where pain is only present during certain types of training (eg, change of direction or squats). If, despite partial load management, symptoms are still present for greater than 24 hours, then cessation of the identified provocative elements within the sport may be advisable. In truly reactive or irritable patients, all painful compression and energy storage activity will need to be ceased until symptoms settle and become stable.

Provocative sporting activities can be temporarily replaced with cross-training to maintain cardiovascular fitness or adapted to reduce compressive loading. For example, cycling may be better tolerated if performed in a standing position. Swimming and water running are viable alternatives to painful activities. Posture modification should involve reducing hamstring origin compression (ie, reducing anterior pelvic tilt and hip flexion in standing, sleeping, and sitting). Shaped cushions can be useful to reduce compression in sitting, and so can encouraging more weight bearing on the posterior thighs rather than on the ischium. Repeated stretching of the hamstrings and hip flexion–dominant movement patterns, such as repetitive lifting and trunk flexion, should be avoided in the early, reactive phase.

REHABILITATION

The key to management of all tendinopathies is progressive loading, performed within a pain-monitoring framework, to reduce pain and restore function. Rehabilitation should be directed across the kinetic chain and can be progressed to include energy storage and release to normalize load capacity in the entire lower limb.[21]

At present, there is no clear guidance from the literature regarding PHT rehabilitation. Loading exercises for PHT have not been investigated in randomized controlled trials. Limited case series and case presentations have been published that demonstrate improvements in pain and function with conservative management, including hamstring strengthening.[26,34,42,59,95] Results from these studies cannot be generalized, however, due to small patient numbers, varying diagnostic methods, and the use of adjunct interventions alongside exercise.

Exercise Prescription

Rehabilitation stages in PHT, based on the authors' synthesis of available evidence of hamstring function, muscle recruitment during rehabilitation, and PHT pathoetiology, will be outlined below. Resistance training principles should be applied to ensure optimal loading, contraction speed, and time under tension.[3]

The authors recommend monitoring pain, at the same time daily, with a load test (eg, short- or long-lever bridge, arabesque) during rehabilitation. Some pain is acceptable during and after exercise (VAS, 0 to 3 out of 10),[44] but symptoms should settle within 24 hours and should not progressively worsen over the course of the loading program.[82] Every patient presents individually, and this may necessitate a focus on particular phases of rehabilitation or kinetic-chain factors.

Progression through the stages described below is based on symptoms and response when progressing the exercise load rather than on specific time frames. The 4-stage program is expected to take 3 to 6 months to complete, but is likely to show considerable individual variation depending on pain and functional deficits.

Stage 1: Isometric Hamstring Load Resisted isometric exercise in positions without tendon compression is advocated as an effective means to load the muscle-tendon unit and reduce pain in PHT with irritable symptoms.[24] Isometric exercise has been shown to have a generalized pain inhibitory response.[63] Rio et al[75] found that 5 sets of 45-second holds of moderate-resistance isometric exercise performed at 70% maximal voluntary isometric contraction reduced patellar tendon pain for at least 45 minutes and provided longer relief than isotonic exercise. Cook and Purdam[25] recommend repeating the isometric exercise several times per day. Dosage should be based on symptom severity and irritability, with shorter/less intense contractions used as necessary. For early-stage PHT, it is suggested that the hip be in a near neutral flexion/extension position or in minimal flexion (eg, 20° to 30° for straight-leg pull-downs). A good prognostic sign for isometrics is an immediate reduction in pain with hamstring loading tests postexercise.

Examples of appropriate exercises for this stage include isometric leg curl, bridge holds with hip in neutral, isometric straight-leg pull-down, and trunk extensions (**FIGURE 2**). Isometric long-leg bridging on 2 legs, progressing to 1-leg holds, is a useful alternative if access to gym equipment is limited.

Stage 2: Isotonic Hamstring Load With Minimal Hip Flexion Isotonic load may be introduced when there is minimal or no pain (VAS, 0-3) encountered during exercise loading through early ranges of hip flexion. The aim is to restore hamstring strength, bulk, and capacity in a functional range of motion, all important aspects in rehabilitation of tendinopathy.[81]

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Eccentric exercise has been widely accepted as the treatment of choice for tendinopathy[11]; however, there is little evidence for isolating the eccentric component.[52] Heavy slow resistance (HSR) training, which includes both concentric and eccentric components, has been found to compare favorably with isolated eccentric loading in the Achilles and patellar tendons. In Achilles tendinopathy, Beyer et al[11] found that both loading strategies resulted in improvement in pain and function but that HSR took considerably less time to complete and had better patient compliance and satisfaction. Load magnitude determines the effect on tendon (and muscle) adaptation, rather than contraction type,[12] and HSR was shown to achieve greater collagen turnover than submaximal eccentric loading.[44] As a result, HSR is preferred over isolated eccentric loading, although it should be noted that neither has been studied extensively in PHT.

The focus, with HSR, is on slow, fatiguing, resisted isotonic exercise, commencing at 15-repetition maximum (the maximum load that can be lifted 15 times in a single set) and progressing to 8-repetition maximum, with 3 to 4 sets performed every other day.[3] A contraction duration of 3 seconds for each phase (concentric and eccentric; 6 seconds total) of the exercise is recommended.[44,45] A metronome may be used to externally pace the exercise, introducing a skill-based element that may improve motor control.[74]

Loaded hip flexion is minimized in early stages to protect the enthesis against compressive stimulus.[23] Single-leg work is important to address asymmetrical strength loss. It is recommended that stage 1 exercises be continued on off-days, particularly if symptoms are still present and isometrics have an immediate positive effect on symptoms.

Suitable exercises in this stage include the single-leg bridge, prone hip extension, prone leg curl, Nordic hamstring exercise, bridging progressions, and supine leg curl (**FIGURE 3**). The single-leg bridge achieves moderate to high levels of activation of the hamstrings, GMax, and GMed[32] and can be progressed by adding load (eg, a weight plate over the pelvis) or bridging with a more extended knee. The supine leg curl, prone leg curl, and Nordic hamstring exercise all achieve very high levels of hamstring recruitment, with peak activity typically occurring at between 0° and 20° of hip flexion,[4,98] making them well suited to this stage.

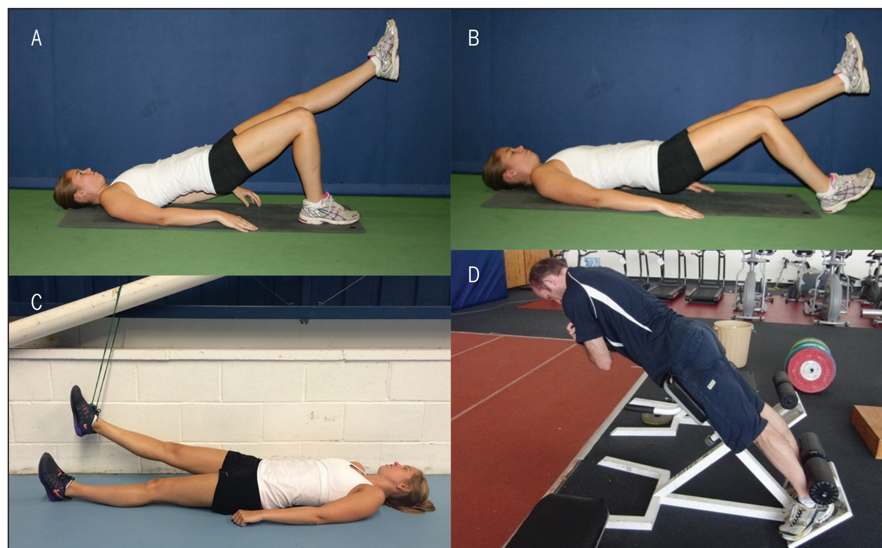

FIGURE 2. Examples of stage 1 exercises: (A) single-leg bridge hold, (B) long-lever bridge hold, (C) straight-leg pull-down, and (D) trunk extension.

FIGURE 3. Examples of stage 2 exercises: (A) Nordic hamstring exercise, (B) prone leg curl, (C) supine leg curl, and (D) bridging progressions (eg, adding weight).

Stage 3: Isotonic Exercises in Positions of Increased Hip Flexion (70°-90°) The goal of stage 3 is to continue hamstring muscle strength, hypertrophy, and functional position training while progressing into greater hip flexion. This training can be commenced when there is minimal pain (VAS, 0-3) with higher-loading hip flexion tests (eg, lunge or arabesque through the ranges utilized in the athlete's sport). Dosage and frequency of loading exercises are the same as stage 2 exercises, that is, every second day. A slow and controlled technique is important. Addition of loaded hip flexion may often be

FIGURE 4. Examples of stage 3 exercises: (A) Romanian deadlift, (B) step-ups, (C) walking lunges, (D) hip thrusts (weights can be added to the bar to increase resistance), and (E) single-leg deadlift.

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provocative, and pain with loading should be monitored 24 hours postexercise with hamstring load tests into hip flexion (eg, hamstring bridge, deadlift, or single-leg arabesque).[24] Progressing to 70° to 80° of loaded hip flexion is generally sufficient, but this will depend on individual pain presentation and functional goals.

Progression of exercises in this stage may include slow hip thrusts, forward step-ups, walking lunges, deadlifts, Romanian deadlifts, and "the diver" (**FIGURE 4**). Some of these are potentially not suitable for less active demographics. Hip thrusts are similar to bridges but involve a greater emphasis on hip flexion range, with feet flat on the floor and shoulders supported on a low bench (**FIGURE 4D**). Electromyographic data suggest high levels of activation in the upper and lower GMax, which exceed those of the biceps femoris during this exercise.[20] Forward step-up using a progressively higher step is a useful progression and can produce very high GMax activation levels.[69] Adding load recruits the biceps femoris and the GMed and significantly increases GMax activation,[84] suggesting that this exercise is a significant muscular challenge to the entire posterior kinetic chain. Deadlifts and lunges are a further progression, as they generally involve greater hip flexion than hip thrusts and step-ups. Lunges have been found to selectively recruit the proximal regions of the biceps femoris (long head) and adductor magnus on functional magnetic resonance imaging analysis.[61] Single-leg deadlifts increase lateral hip stabilization demands and have been shown to achieve high levels of activation in the GMax and GMed.[69] The diver is a single-leg functional progression of the deadlift that has been advocated in hamstring muscle injury rehabilitation.[8] Walking lunges (**FIGURE 4C**) also introduce a dynamic movement-control element and can be considered when strength and control with prior exercise are adequate. At the end of this phase, loading of the hamstring origin through sports-specific ranges should be comfortably achieved with little latent provocation.

Stage 4: Energy Storage Loading Stage 4 is only required for those returning to sports involving lower-limb energy storage or impact loading. Reintroduction of power/elastic stimulus for the myotendinous unit can be commenced when there is minimal pain (VAS, 0 to 3 during load tests, such as the arabesque) and adequate bilateral strength in single-leg stage 2 and 3 exercises. There should also be adequate execution and control of energy storage activity. Early in this phase, the amount of hip flexion during exercise may be limited to minimize tendon compression as the higher elastic loading is added. As this is the most provocative stage, a conservative approach is recommended, with exercises being performed every third day. A stage 1 day would then follow to settle the tendon, with the following day being a strengthening (stage 2 to 3) day, to form a 3-day, high/low/medium tendon load cycle twice a week, with a rest day allowed between cycles.[76]

A potential progression of stage 4 exercises may include sprinter leg curl, A-skips, fast sled push or pull, alternate-leg split squats, bounding, stair or hill bounding, kettlebell swings (**FIGURE 5**), and gradual reintroduction of sport-specific squat and lunge activities. For multidirection sports (eg, football, rugby), progression should include lateral, rotational, or cutting movements to improve strength and control and graduate entheseal loads in multiple planes of movement. Sessions would include a maximum of 3 to 4 of these activities (typically graduating to 15-20 repetitions or steps, 3 sets), usually starting with 1 and adding 1 to 2 per week, depending on pain response. The exercises chosen should reflect individual functional and sport demands (eg, running, sprinting, jumping, lifting, etc). For example, the sprinter leg curl is performed in the open chain, with the hip

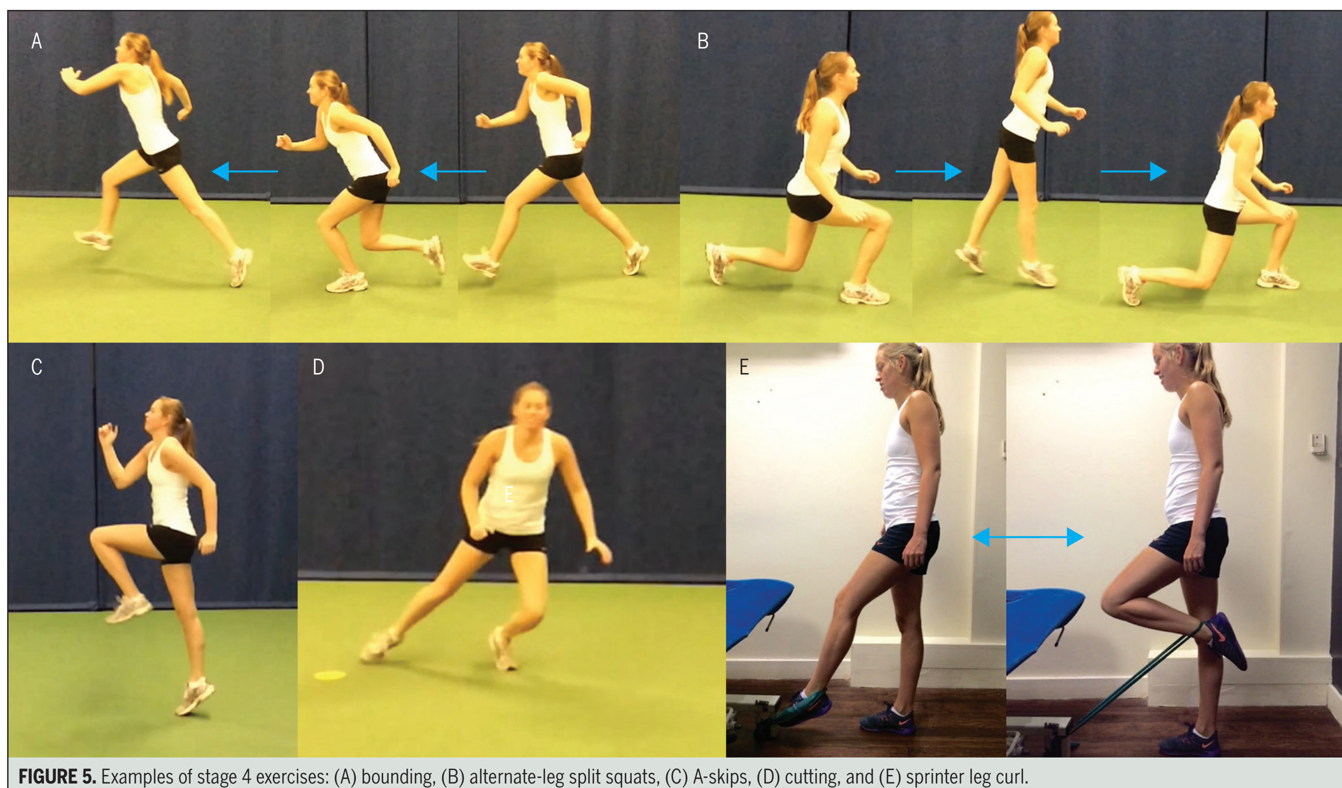

FIGURE 5. Examples of stage 4 exercises: (A) bounding, (B) alternate-leg split squats, (C) A-skips, (D) cutting, and (E) sprinter leg curl.

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in 80° and knee flexion movement from 130° to 20° (**FIGURE 5E**), and replicates the running cycle. Hip flexion range and symptom response should be considered during progression.

These exercise progressions form stepping stones for return to sport, yet it is also important that these activities not be simply added to the normal training volume on resumption, in order to avoid overload and exacerbation of symptoms.[93] A graded return to sport can be introduced when the athlete can tolerate the loading requirements of the sport with minimal symptom provocation (ie, VAS of 0 to 3, with pain settling within 24 hours on load tests). Care should be taken with the introduction of hill- and speed-training sessions, as these can be quite provocative. Team sports may have more complex loading requirements and will require more detailed assessment. A graded return in these cases usually involves gradual exposure to provocative activity in training prior to return to full competition.

Other Considerations Dry needling and soft tissue techniques have been utilized in the treatment of PHT, but supporting evidence is limited to case series.[42,59,95] In the authors' experience, massage and manual therapy addressing tone and restriction in the kinetic chain (eg, hamstring, tensor fascia latae) can be useful adjuncts in the management of PHT. However, such passive interventions are unlikely to improve tissue load capacity, which is a key element of tendinopathy rehabilitation.[21]

Anti-inflammatory medication (eg, ibuprofen) has been suggested for settling irritable tendon pain and may also inhibit tenocyte overstimulation and signaling.[91] Whether nonsteroidal anti-inflammatory drugs compromise tendon healing is not fully understood, although ibuprofen administration does not seem to impair normal tendon adaptation in response to resistance training.[17]

Other adjuncts that can be considered include extracorporeal shockwave therapy (ESWT) and injections. In the authors' experience, ESWT may modulate pain in less reactive tendinopathies but is much less effective and can even flare early-stage reactive symptoms. Cacchio et al[16] reported a superior result with ESWT compared to traditional conservative treatment; however, the mechanism of effect for ESWT remains unclear.

Peritendinous corticosteroid injection may provide short-term pain relief, but symptoms have a tendency to recur.[47,48] Platelet-rich therapies, autologous blood, and other agents may be utilized in longer-standing lesions; however, there is currently insufficient evidence to support the use of platelet-rich therapies for treating musculoskeletal soft tissue injuries.[62]

Surgical treatment of PHT may be an option for recalcitrant cases where symptoms have failed to improve with conservative management. A full description of this approach is beyond the scope of this paper, but the reader is directed to a recent review by Lempainen et al.[47]

CONCLUSION

This narrative review and commentary has highlighted key differential diagnoses, tendon-specific and kinetic-chain assessment approaches, and intervention strategies for patients presenting with symptomatic PHT. An outline for exercise progression of PHT is proposed, supported by contemporary evidence and principles of tendinopathy management. There are a number of limitations to the recommendations. There is presently a paucity of evidence relating to pathoetiology, biomechanical analyses, and clinical approaches relating to PHT as an entity. As there are presently no randomized controlled trials investigating rehabilitation, recommendations in this paper are based on current understanding of tendinopathy pathoetiology, as well as on hamstring muscle function and rehabilitation principles. These recommendations aim to provide a reference for both clinicians treating PHT and future research into management of this overuse injury. Future research should (1) evaluate the role of hip flexion angle and its effect on tendon compression and pain, (2) identify extrinsic and intrinsic risk factors for the development of PHT, and (3) study the effectiveness of loading programs for PHT using validated pain and functional outcome measures. ◉

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