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# Gluteal Tendinopathy: Integrating Pathomechanics and Clinical Features in Its Management

**G**luteal tendinopathy is thought to be the primary cause of lateral hip pain,<sup>4,6,18,44,55,57</sup> and has the potential to affect a person's quality of life, earning potential, and activity level.<sup>15,28,36,71</sup> This condition presents as pain over the greater trochanter that may extend down the lateral thigh. It is most commonly reported in people over the age of 40,<sup>56,76,105</sup> with women outnumbering men by between 2.4 and 4 to 1.<sup>73,79</sup> People with gluteal tendinopathy have

been reported to have difficulty with lying on their side at night, standing, walking, climbing up or down stairs, and sitting.<sup>105</sup>

As with other pain conditions, effective treatment of gluteal tendinopathy relies on the clinician making the correct diagnosis; understanding the etiology and pathology; recognizing, understanding, and addressing the modifiable risk factors and comorbidities; identifying and evaluating the contribution of bio-

mechanical deficiencies and likewise pain; and then prescribing, modifying, and progressing the most appropriate interventions, based on clinical reasoning and changes in the condition over time.

## PATHOANATOMY

**L**ATERAL HIP PAIN HAS BEEN LIKENED to shoulder rotator cuff disease, with its contiguous bone, tendon, and

bursal anatomy and associated pathologies.<sup>14,45</sup> In people with lateral hip pain, thickening and thinning of and tears in the gluteus medius and/or gluteus minimus tendons have been observed, and changes in bursal structure have been documented on ultrasound<sup>18,29,47,60</sup> and magnetic resonance imaging (MRI).<sup>4,6,9,14,45-47,69</sup> While this condition has traditionally been referred to as trochanteric bursitis,<sup>12,59,83</sup> gluteus medius and/or minimus tendinopathy is now accepted as the most prevalent pathology in those with pain and tenderness over the greater trochanter.<sup>6,46,47,60</sup> In an ultrasound study<sup>18</sup> of 75 individuals with symptoms of pain and point tenderness over the greater trochanter, only 8 had bursal enlargement; the predominant pathology, gluteus medius tendinopathy and, in more severe cases, tendon tears, occurred most commonly in the deep and anterior portions of the tendon. Another recent imaging study<sup>60</sup> of 877 individuals with greater trochanteric pain demonstrated a similar low incidence of bursal change, with only 20% exhibiting bursal thickening on ultrasound. When present, bursal pathology most commonly occurs in the trochanteric bursa or sub-gluteus maximus bursa, but has also been occasionally identified in the sub-gluteus medius or sub-gluteus minimus bursae.<sup>105</sup>

● **SYNOPSIS:** Gluteal tendinopathy is now believed to be the primary local source of lateral hip pain, or greater trochanteric pain syndrome, previously referred to as trochanteric bursitis. This condition is prevalent, particularly among postmenopausal women, and has a considerable negative influence on quality of life. Improved prognosis and outcomes in the future for those with gluteal tendinopathy will be underpinned by advances in diagnostic testing, a clearer understanding of risk factors and comorbidities, and evidence-based management programs. High-quality studies that

meet these requirements are still lacking. This clinical commentary provides direction to assist the clinician with assessment and management of the patient with gluteal tendinopathy, based on currently limited available evidence on this condition and the wider tendon literature and on the combined clinical experience of the authors. *J Orthop Sports Phys Ther* 2015;45(11):910-922. Epub 17 Sep 2015. doi:10.2519/jospt.2015.5829

● **KEY WORDS:** greater trochanteric pain syndrome, hip, lateral hip pain, trochanteric bursitis

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The histopathological changes seen in the gluteal tendons and bursae<sup>31</sup> in those with lateral hip pain are consistent with degenerative changes seen in other tendinopathies.<sup>18,29,31</sup> The signal substance (a chemical messaging molecule), substance P, was found in higher frequencies in both the tendon and the bursa in people who had undergone tendon reconstruction surgery compared with matched specimens from a population who had undergone hip arthroplasty surgery.<sup>31</sup>

For the purposes of this review, gluteus medius and/or minimus tendinopathy, with or without associated bursal pathology, will be referred to as gluteal tendinopathy. All of these structures are likely to be influenced similarly by the pathomechanics discussed in this review, and management strategies should therefore be similarly beneficial for all involved local structures.

## RISK FACTORS

**W**HILE A NUMBER OF RISK FACTORS for the development of gluteal tendinopathy have been proposed, few have been validated. Being female and over 40 years of age have been frequently recognized as risk factors for developing lateral hip pain.<sup>17,81</sup> In addition, the prevalence of lateral hip pain (likely gluteal tendinopathy) in people with low back pain has been reported to be as high as 35%,<sup>17,95</sup> with increased duration of low back pain associated with increased incidence of lateral hip pain.<sup>17,81</sup> The relationship between these 2 conditions may relate to possible gluteal dysfunction associated with back or sacroiliac joint pain,<sup>40,43</sup> or increased stress through the back as a result of poor lateral stability of the pelvis. In either case, the relationship warrants further investigation. Importantly, treating the tendon-related pain has been shown to improve the function of those with low back pain,<sup>79,95</sup> suggesting an interaction if not a causal relationship.

The morphology of the female pelvis has been hypothesized as a possible risk

factor for the development of gluteal tendinopathy,<sup>85</sup> with coxa vara and greater trochanteric offset both potentially predisposing to greater compressive loading of the gluteal tendons, which will be explored further in the Pathomechanics section. In an all-female prospective study, no bony differences were found in a number of radiographic indices of pelvic width and trochanteric offset between those with gluteal tendon-related pain, asymptomatic age- and sex-matched controls, and participants with hip osteoarthritis only.<sup>27</sup> These findings conflict with a larger but less controlled retrospective study by Viradia et al,<sup>97</sup> who reported on males and females and found that individuals with lateral hip pain had a greater trochanteric offset. Trochanteric offset was determined on an anteroposterior radiograph by subtracting the width of the pelvis (the linear distance between the most lateral aspects of both iliac wings) from the distance between the most lateral aspects of the greater trochanters. This suggests that trochanteric offset may be a risk factor for developing local soft tissue pathology at the greater trochanter, which is primarily gluteal tendinopathy. This is the first study that appears to identify a risk factor in men.

In a separate study also looking at pelvic bony anatomy, Fearon et al<sup>27</sup> identified that a femoral neck-shaft angle of less than 134° was more commonly seen in women who failed conservative treatment for gluteal tendinopathy and were scheduled for tendon reconstruction surgery. These findings suggest a risk of greater severity of the condition, although not a role in its development. While proposed as a risk factor for developing lateral hip pain and therefore gluteal tendinopathy, an association with leg-length discrepancy has not been demonstrated.<sup>44,64,82,103</sup>

Other anthropometric measures, such as body mass index and waist, hip, and trochanteric girth, have been assessed in this population.<sup>30,81</sup> Although body mass index does not dissociate those with lateral hip pain,<sup>30,81</sup> gynoid adiposity, mea-

sured by tape measure as the pelvic girth at the level of the greater trochanters, was larger in those with lateral hip pain compared with asymptomatic controls and those with hip osteoarthritis only.<sup>27</sup> Searches of major databases failed to identify studies that examined the role of other potentially modifiable risk factors or impairments, such as strength or flexibility deficits, in the development of gluteal tendinopathy.

## DIAGNOSIS

### Clinical Tests

**M**AKING THE DIAGNOSIS OF GLUTEAL tendinopathy can be difficult. A thorough examination of the hip, back, and pelvis should be undertaken to determine if the primary cause of the trochanteric pain lies at, or is distant to, the greater trochanter. Symptomatic local pathology may coexist with more distant sources. Key indicators of comorbidities arising from the back and hip joints and other important differential diagnoses are outlined in **TABLE 1**.<sup>2,3,13,16,30,33,58,67,70,72,81,86,88,93-95,105</sup>

A number of hip evaluation tests have been proposed for the differential diagnosis of hip pain. It is important to note that the site of any reproduced pain provides the clinician with valuable information, increasing the diagnostic accuracy of the test.<sup>30</sup> Many orthopaedic hip tests can be used for diagnostic purposes for more than 1 condition. The site of pain reproduction allows site-specific evaluation.

In a meta-analysis of orthopaedic tests, Reiman et al<sup>74</sup> provided some clarity regarding the value of a number of tests used for diagnosis of gluteal tendinopathy, including the single-leg stance test and resisted medial and lateral rotation and abduction, as reported by Lequesne et al,<sup>57</sup> Bird et al,<sup>6</sup> and Woodley et al<sup>105</sup> (**TABLE 2**). These studies all had imaging evidence of local pathology at the greater trochanter as the reference test, with a predominance of findings indicating gluteal tendinopathy. A fourth paper specifically evaluated orthopaedic

**TABLE 1**

**DIFFERENTIAL DIAGNOSIS IN RELATION TO LATERAL HIP PAIN**

Differential Diagnosis	Possible Past History	Key Current Symptoms and Signs
Bony metastasis, most commonly breast, prostate, kidney, lung, and thyroid <sup>16</sup>	A history of cancer—but not necessarily	A deep unrelenting pain is characteristic of metastatic bone pain May be worse at night May be aggravated by mechanical stress of the bone May appear like an insufficiency (stress or osteoporotic) fracture <sup>16</sup> Unexplained weight loss The clinical picture is unclear, even when comorbidities are considered
Neck-of-femur fracture <sup>13</sup>	Known osteoporosis with a history of a fall or rapid increase in activity	Pain around the hip (groin, buttocks, anterior and/or lateral thigh) that is aggravated with weight bearing. Range of movement may be normal
Hip joint pathology (intra-articular: eg, osteoarthritis, femoral acetabular impingement, avascular necrosis)	Family or personal current history of osteoarthritis in other joints Past history of hip trauma (osteoarthritis) Known femoral acetabular impingement <sup>70</sup> Difficulty with putting on/taking off shoes and socks <sup>30</sup> History of cortisone use or alcohol abuse (avascular necrosis) <sup>108</sup>	Pain is reported to be in 1 or more of groin, deep buttock, anterior thigh, and/or knee region <sup>68</sup> Hip passive medial rotation range of movement reproduces groin pain, deep buttock pain, and/or lateral hip pain <sup>2,58</sup> Loss of joint range of movement <sup>2,70,93</sup> Hip locking, giving way, clicking in groin <sup>96</sup> FADDIR positive <sup>74</sup>
Lumbar spine referral	Patient reports low back pain in addition to lateral thigh pain <sup>93,95</sup>	Dermatome and sclerotome distribution of pain. As ITB-related pain and tenderness have been reported in association with lateral hip pain, <sup>81,105</sup> it is more likely that a pain distribution that follows the ITB, rather than a dermatomal distribution, is emanating from local pathology rather than from spinal pathology
Inflammatory diseases (eg, rheumatoid arthritis)	A known history of inflammatory disease or multiple synovial sites of pain <sup>72</sup>	Frank clinical inflammation (heat, erythema, edema) in multiple areas, morning stiffness greater than 1 hour, symmetrical signs, hand involvement, and gastrointestinal dysfunction <sup>3,72</sup>
Alternative extra-articular pathology • Ischiofemoral impingement/quadratus femoris tear • Piriformis and related sciatic nerve entrapment syndromes	Possible morphological issues identified on imaging	Ischiofemoral impingement/quadratus femoris tear <sup>67,88,94</sup> Pain over quadratus femoris/ischiofemoral region rather than laterally over greater trochanter, with or without groin pain Snapping sensation in ischiofemoral interval with walking/running may occur May experience pain with a variety of postures and activities Piriformis and related syndromes <sup>33</sup> Pain location in greater sciatic notch region, mid buttock, or posterior hip rather than laterally over greater trochanter Pain with sitting or actions that repetitively load the hip external rotators May have sciatic-like symptoms Consider if muscle spasm is secondary to other issues

*Abbreviations: FADDIR, flexion, adduction, internal rotation; ITB, iliotibial band.*

special tests in relation to the differential diagnosis between hip osteoarthritis and gluteal tendinopathy.<sup>30</sup> This study used clinical diagnosis of a local soft tissue pathology at the greater trochanter and radiographs, with additional confirmation of gluteal tendon pathology made at surgery for half the group. The flexion, abduction, external rotation and Ober tests (**TABLE 2**) were evaluated in addition to the above tests. The studies included in the meta-analysis, as noted by Reiman et al<sup>74</sup> and the article by Fearon et al,<sup>30</sup> all

have methodological limitations that impact on the generalizability of the results. This means that all these articles are likely reporting diagnostic values higher than would be seen in the general population.

We would like to comment on some features of these tests. First, the diagnostic value of a pain-provocation test has been shown to be improved by simply asking if the patient can identify the specific site of pain reproduction.<sup>30</sup> Second, tests that rely on the assessor applying resistance, for example, resisted medial

rotation and abduction, are subject to assessor bias due to possible assessor variation in response to the patient's presentation, or simply due to day-to-day variation; so, while valuable, these variables need to be considered.

Finally, we note that the single-leg stance tests reported in the above studies have not been performed in a consistent manner. Fearon et al<sup>30</sup> used a method originally designed as a balance test, assessing the time (up to 30 seconds) for which participants could maintain sin-

TABLE 2

## SUMMARY OF SENSITIVITY AND SPECIFICITY OF DIAGNOSTIC TESTS FOR LATERAL HIP PAIN

Test	Number of Studies	Sensitivity	Specificity	Reference Standard
Single-leg stance*				MRI <sup>6,57,105</sup>
Duration	1 <sup>105</sup>	23	94	
Pelvic tilt	1 <sup>6</sup>	72.7	76.9	
Pain provocation <sup>†</sup>	1 <sup>57</sup>	100	97.3	
Resisted medial rotation				MRI, <sup>6</sup> clinical assessment <sup>30</sup>
Pain provocation <sup>†</sup>	2 <sup>6,30</sup>	55-61	69-90	
Resisted lateral derotation				MRI <sup>57</sup>
Pain provocation <sup>†</sup>	1 <sup>57</sup>	88	97.3	
Resisted abduction				MRI, <sup>6,57</sup> clinical assessment <sup>30</sup>
Pain provocation <sup>†</sup>	3 <sup>6,30,57</sup>	58.5-71	46-85.0	
FABER				Clinical assessment <sup>30</sup>
Pain provocation <sup>†</sup>	1 <sup>30†</sup>	82.9	90.0	
Ober				Clinical assessment <sup>30</sup>
Pain provocation <sup>†</sup>	1 <sup>30†</sup>	41.0	95.0	

Abbreviations: FABER, flexion, abduction, external rotation; MRI, magnetic resonance imaging.

\*Sensitivity and specificity were not provided by 1 paper, as the timed version of the single-leg stance test did not differentiate between lateral hip pain and hip osteoarthritis.<sup>30</sup>

<sup>†</sup>The reproduction of the individual's pain over the greater trochanter. Unless otherwise stated, reproduction of pain over the gluteal tendons was considered a positive result. The reproduction of groin, sacroiliac joint, or buttock pain suggests dysfunction of structures other than the gluteal tendons.<sup>25,57</sup>

<sup>‡</sup>Sensitivity and specificity were not provided by 1 paper, as the authors reported that these tests were not useful for diagnosis.<sup>105</sup>

gle-leg stance without upper-limb support.<sup>34</sup> The duration of single-leg stance did not differentiate between 2 groups of people with hip-related pathology (gluteal tendinopathy and hip osteoarthritis), with both groups performing more poorly than an asymptomatic control group.<sup>30</sup> This version of the test therefore establishes that those with hip pathology have poorer balance than a normal control group, but its usefulness in differential diagnosis of hip pain is limited. Woodley et al<sup>105</sup> performed the test as per Hardcastle and Nade's<sup>39</sup> version of the Trendelenburg test, where a normal response was recorded if the individual was able to elevate his or her pelvis on the nonstance side and hold this position for at least 30 seconds, with light support provided by the examiner. If the contralateral pelvis dropped below a horizontal position, the test was recorded as abnormal. This ver-

sion of the test is therefore not limited by balance and does directly test hip abductor muscle function. Keeping the pelvis above the horizontal, however, will eliminate or minimize hip adduction, which reduces gluteal tendon compression and may lessen its value as a diagnostic test.

Lequesne et al's<sup>57</sup> version of the single-leg stance test controls for balance by allowing light fingertip support. It does not dictate pelvic position, only that the trunk be maintained in a vertical position. The position is maintained for 30 seconds or to the onset of greater trochanteric pain. Maintaining the trunk upright necessitates some amount of hip adduction, even in a normal population. In those who potentially have poorer hip abductor function and endurance, the hip may reach a position of adduction earlier, resulting in tendon compression under active tensile load and possibly reproducing the per-

son's pain. Patients with other hip and lumbopelvic pathologies may also sink into hip adduction without trochanteric pain. While this test is still to be compared to a group symptomatic of other pathologies, anecdotally it appears to be valuable. At this point, therefore, we recommend sustained single-leg stance for 30 seconds or to the onset of pain over the greater trochanter, as per Lequesne et al.<sup>57</sup> Clinicians should note that, although ability to control pelvic position is not measured as part of this diagnostic test, noting the patient's quality of pelvic control may provide treatment direction.

### Imaging

Radiography, MRI, ultrasound, and scintigraphic imaging have all been reported in the literature as helpful adjuncts in clarifying the diagnosis of gluteal tendinopathy. However, signs of local soft tissue pathology at the greater trochanter are common in imaging of those without lateral hip pain<sup>9,102</sup>; thus, diagnosis should not rely solely on imaging studies. Radiology should be employed when the diagnosis is unclear; when other lesions need to be excluded; and/or the condition is long-standing, unremitting, or not responding to an appropriate management program. Radiographs, often the first line of imaging, are useful in excluding occult lesions but not in demonstrating soft tissue lesions. Furthermore, they have the negative effect of radiation exposure.

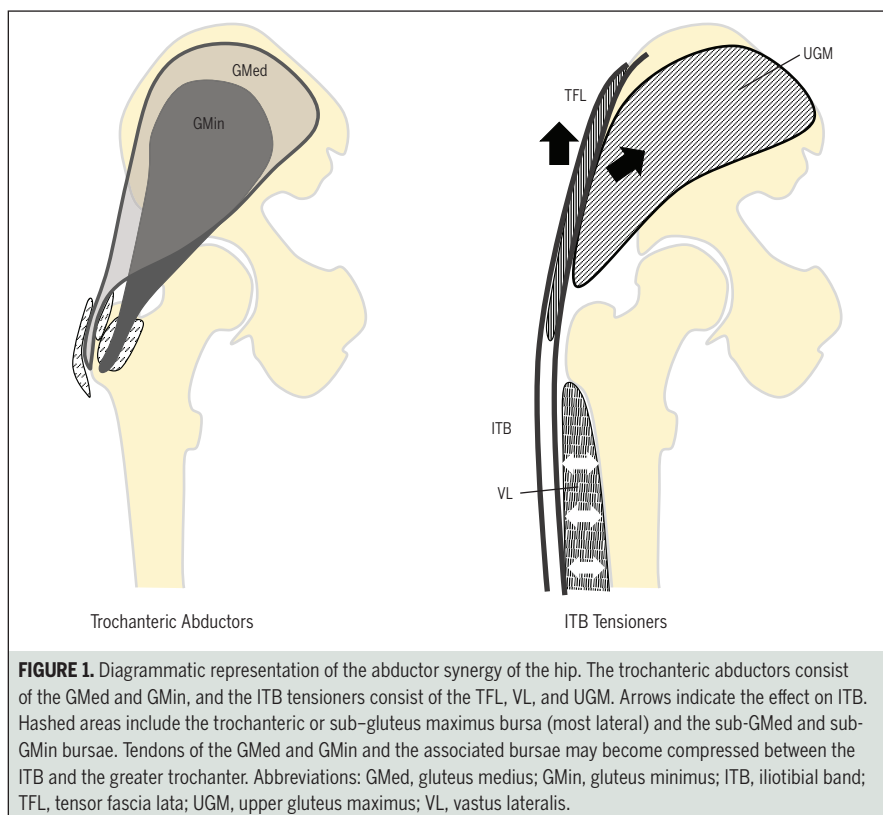
High-quality prospective imaging studies that include surgical and histological confirmation of tendon pathology status are lacking. A recent systematic review of 7 MRI studies and 2 ultrasound studies for diagnosing gluteal tendon tears (with surgical confirmation)<sup>102</sup> found that MRI had a sensitivity of between 33% and 100%, a specificity of between 92% and 100%, and a positive predictive value of between 71% and 100%. Ultrasound was found to have a higher sensitivity (79%-100%) and positive predictive value (95%-100%). In this small systematic review,<sup>102</sup> the authors suggested that ultrasound was likely a

better choice, as there were fewer false positives. Woodley et al,<sup>105</sup> however, pointed out that MRI provides considerable information regarding adjacent structures. A later narrative review<sup>63</sup> concluded that while ultrasound is cheaper and more readily available than MRI, MRI should be the imaging modality of choice. Occasionally, scintigraphic studies are used to augment the differential diagnosis.<sup>100</sup>

## PATHOMECHANICS

IT IS REASONABLE TO ASSUME THAT THE pathomechanics underlying the development of gluteal tendinopathy are similar to those proposed for other insertional tendinopathies: relatively increased (overload<sup>21,80</sup>) or decreased (stress/load shielding<sup>1,68</sup>) tensile load applied longitudinally along the tendon, excessive transverse load applied across the tendon (compression, mostly at or near the bony insertion<sup>1,19</sup>), and most often a combination of these factors.<sup>1,19</sup> The combination of tensile and compressive overload appears to be particularly damaging.<sup>87</sup> Matrix degradation associated with any of these adverse loading scenarios can reduce the tensile load-bearing capacity of the tendon and predispose it to tearing at relatively lower tensile load.<sup>1</sup>

Excessive tensile load alone would not explain the most common pattern of pathology that develops within the gluteal tendons. A close analogy has been drawn between pathology of the supraspinatus tendon and that of the gluteus medius tendon, both structures more commonly developing deep, undersurface tears.<sup>29</sup> While similar evidence is not yet available at the hip, the deep fibers of the supraspinatus tendon carry the least tensile load and are therefore relatively shielded from tensile stress in lower ranges of shoulder abduction.<sup>5</sup> In these ranges, the deep fibers of the supraspinatus tendon are also exposed to high compressive loads against the bony insertion. As the shoulder abducts into higher ranges, compressive load reduces



**FIGURE 1.** Diagrammatic representation of the abductor synergy of the hip. The trochanteric abductors consist of the GMed and GMin, and the ITB tensioners consist of the TFL, VL, and UGM. Arrows indicate the effect on ITB. Hashed areas include the trochanteric or sub-gluteus maximus bursa (most lateral) and the sub-GMed and sub-GMin bursae. Tendons of the GMed and GMin and the associated bursae may become compressed between the ITB and the greater trochanter. Abbreviations: GMed, gluteus medius; GMin, gluteus minimus; ITB, iliotibial band; TFL, tensor fascia lata; UGM, upper gluteus maximus; VL, vastus lateralis.

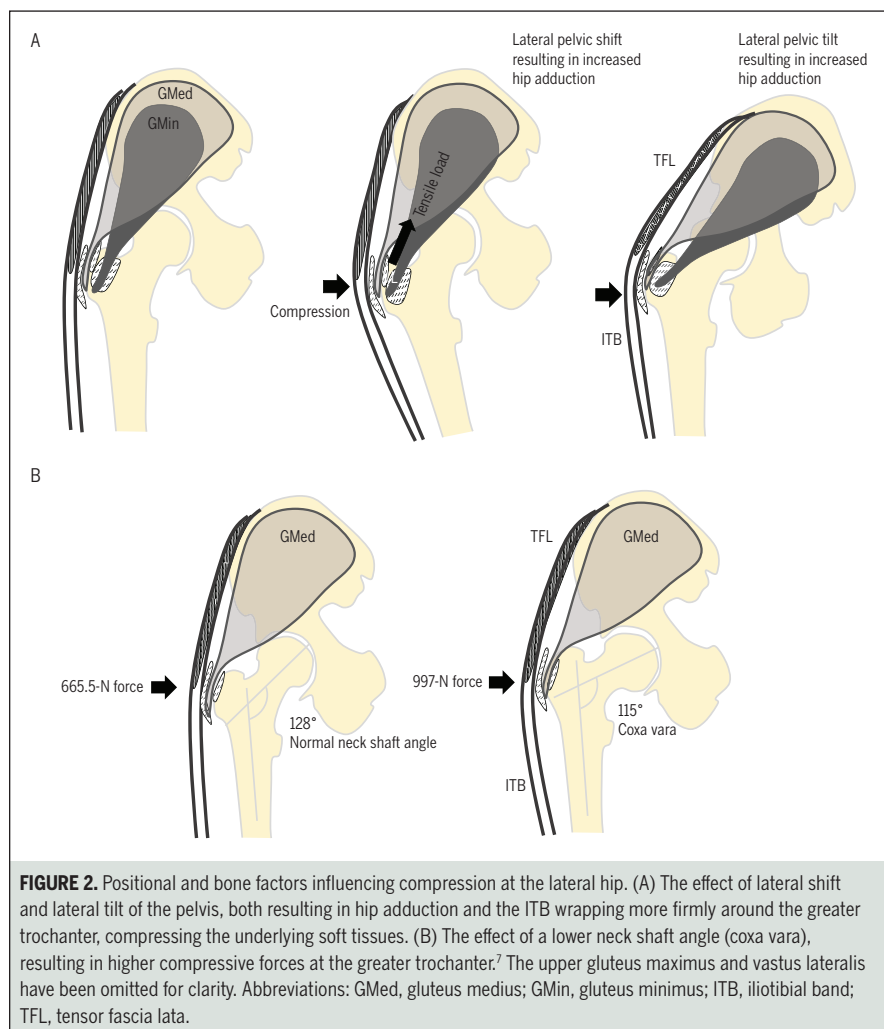
and tensile load increases in this region of the tendon.<sup>5</sup> The ensuing argument suggests that if tensile load were the primary pathomechanical factor, pathology would present first and most commonly in the superficial fibers of the tendon, which is often not the case.<sup>1</sup>

Compressive loads and relative shielding from tensile loads were consequently offered as alternative explanations for the development of pathological change in the supraspinatus tendon, which then becomes intolerant of tensile load and vulnerable to secondary damage when the arm is raised into higher ranges of abduction.<sup>1</sup> During normal daily weight-bearing function, the hip is used in low ranges of abduction, with single-leg function normally performed in slight hip adduction.<sup>24,107</sup> The deep fibers of the gluteus medius and minimus tendons are likely to carry less tensile load in these ranges than the more superficial fibers. At the shoulder, the highest concentrations of aggrecan, a matrix proteoglycan known to be prevalent in areas of com-

pression, have been demonstrated in the deep, joint side regions of the supraspinatus tendon as it wraps around the humeral head.<sup>62</sup>

A recent study<sup>106</sup> of the anatomy of the gluteus medius tendon insertion and mechanics aimed to determine why pathology of this tendon is more common in females than in males. The authors found that the gluteus medius in females has a smaller insertion on the femur across which to dissipate tensile load and a shorter moment arm, resulting in reduced mechanical efficiency.<sup>106</sup> This mechanical disadvantage is further heightened in those with a smaller femoral neck-shaft angle.<sup>27,106</sup> This may lead to higher tensile loads in female gluteal tendons.

It is also possible that women who have less efficient gluteus medius muscles more regularly use increased adduction during function to provide a mechanical advantage for their abductors. The hip abductors have been shown to generate the highest forces from an adducted hip position,<sup>52</sup> likely associated with



**FIGURE 2.** Positional and bone factors influencing compression at the lateral hip. (A) The effect of lateral shift and lateral tilt of the pelvis, both resulting in hip adduction and the ITB wrapping more firmly around the greater trochanter, compressing the underlying soft tissues. (B) The effect of a lower neck shaft angle (coxa vara), resulting in higher compressive forces at the greater trochanter.<sup>7</sup> The upper gluteus maximus and vastus lateralis have been omitted for clarity. Abbreviations: GMed, gluteus medius; GMin, gluteus minimus; ITB, iliotibial band; TFL, tensor fascia lata.

length-tension relationships. In addition, pre-tensioning the iliotibial band in adduction provides an advantage for the superficial abductor system, exerting its force via the iliotibial band (iliotibial band tensioners) (FIGURE 1).<sup>38,98</sup> These strategies may reduce tensile load and increase compressive load on the deeper regions of the tendons of the gluteus medius and minimus (trochanteric abductors) (FIGURE 1).

Compression of the distal portion of the gluteus medius and minimus tendons occurs against the bone into which they insert, the greater trochanter. It is amplified at the hip by the effect of the overlying iliotibial band in positions of hip adduction (FIGURE 2A)<sup>8</sup> and influenced by femoral neck shaft angle (FIGURE 2B).<sup>7</sup> The

iliotibial band exerts progressively higher compressive load at the greater trochanter as the hip is adducted (4 N at 0°, rising to 36 N at 10°, and 106 N at 40° of hip adduction).<sup>8</sup> This study was performed with the hip in a neutral flexion/extension posture; however, the compressive nature of the iliotibial band may persist in positions of adduction throughout the sagittal plane, due to the strong relationships between the iliotibial band, the fascia lata, the gluteal muscles and fascia.<sup>89,91,96</sup>

Activity of the iliotibial band tensioners in a position of hip adduction may result in higher levels of compressive loading at the greater trochanter than a passively adopted position of adduction. Abductor muscle force and lateral pelvic

stability are contributed to by both the iliotibial band tensioners, which supply 30% of the abductor force required to sustain a level pelvis in single-leg stance, and the trochanteric abductors, which supply the remaining and predominant 70% of required force.<sup>53</sup> The iliotibial band tensioners are an integral part of this system, as the gluteus medius alone has been demonstrated to be mechanically insufficient to generate adequate force to resist the hip adduction torque in single-leg weight bearing.<sup>78</sup> Weakness and atrophy of the trochanteric abductors may result in a relatively greater level of contribution to force production from the iliotibial band tensioners, or an increase in hip adduction, leading to higher compressive forces.

In those with symptomatic gluteal tendon pathology, significant fatty atrophy of the gluteus medius and minimus has been demonstrated.<sup>69</sup> In studies of patients with clinical symptoms of lateral hip pain, a study<sup>105</sup> of 40 symptomatic hips and 40 asymptomatic hips found atrophic changes in the gluteus minimus and medius in 40% of the hips, with changes almost exclusively occurring in the symptomatic group. On imaging, it was established that 53% of the symptomatic group had pathology of the gluteus medius and/or minimus tendons.<sup>105</sup> Another smaller study<sup>35</sup> of 10 individuals with unilateral lateral hip pain and 10 controls reported that mean muscle volumes for the gluteus medius and minimus were smaller for the symptomatic hips of the group with lateral hip pain compared to the matched hips of the control group, but differences were not significant when data were collapsed across sides and compared between groups. The study was likely underpowered, with only 3 of 20 hips demonstrating gluteal tendon pathology on imaging.<sup>35</sup> While further research on larger groups with established gluteal tendon pathology is warranted, from the information available it would appear that in groups with a high prevalence of symptomatic gluteal tendon pathology, atrophy of the gluteus minimus and/or medius is common.

Less information is available on changes in the more superficial abductor muscles. Gluteus maximus atrophy was observed in only 1 hip in the larger lateral hip pain study discussed above,<sup>105</sup> and the tensor fascia lata was shown to be hypertrophied compared to the healthy side in those with unilateral tendon pathology.<sup>92</sup> Causation cannot be established with such cross-sectional data; however, the information available suggests that changes within the abductor muscle synergy may be associated with tendon pathology.

Functional lower-limb movement patterns may be disturbed in those with gluteal tendinopathy. In the absence of scientifically confirmed movement aberrations in this patient group, the following clinical observations are offered: excessive amounts of hip adduction are often employed during bilateral (squatting, lunging, sit-to-stand) and single-leg (stair climbing/descending, single-leg stance and squat, hop/landing) loading tasks. In the less painful or more conditioned patient, deficits may only be clinically observable during higher-load tasks. Deficits present as excessive lateral pelvic tilt and/or lateral pelvic shift, often accompanied by excessive hip internal rotation. These patterns may be a consequence of hip abductor muscle insufficiencies and/or an altered motor control strategy. The combination of trochanteric abductor insufficiency, increased contribution from the iliotibial band tensioners, and excessive use of functional adduction may represent a mechanical risk factor for the gluteal tendons that are exposed to combined compressive and tensile load in these scenarios.

## FINDINGS FROM CLINICAL TRIALS

**T**HE BEST APPROACH FOR CLINICAL management of gluteal tendinopathy has yet to be elucidated, with few studies and limited availability of high-quality evidence.<sup>23</sup> Interventions that have been studied include exercise,



FIGURE 3. Positions of compression for the gluteal tendons.

shockwave therapy, corticosteroid injection, and surgery.

Only 1 study has examined the effect of an exercise intervention for patients with pain and tenderness over the greater trochanter and positive findings on clinical tests for a local soft tissue pathology.<sup>76</sup> This nonrandomized trial compared home exercise with shockwave therapy and corticosteroid injection. The exercise intervention resulted in a poor early outcome, with only 7% of participants reporting an improvement at 4 weeks. However, positive outcomes had risen to 40% at 4 months and 80% at 15 months. The exercise program included piriformis (hip flexion/adduction) and iliotibial band (hip adduction) stretches that potentially expose the gluteal tendons to compression, sagittal plane strengthening such as straight leg raise, wall squats, and prone hip extension, but no direct hip abductor exercises.<sup>76</sup> Minimizing compressive loading by avoiding stretching and adding frontal plane abductor strengthening may deliver enhanced outcomes.

Participants in the shockwave intervention arm also fared poorly at 4

weeks, with only 13% of participants reporting improvement, subsequently rising to 68% by 4 months and 74% by 15 months.<sup>76</sup> A further study by the same group compared shockwave against various other traditional nonoperative measures that were not described. The results of this study suggested a single treatment of shockwave therapy to be more effective than other conservative measures at a 12-month follow-up.<sup>36</sup>

Corticosteroid injection provided moderate pain relief (average reduction of 55%<sup>54</sup>) within 4 weeks for 72% to 75% of those with lateral hip pain,<sup>54,76</sup> dropping to 41% to 55% by 3 to 4 months,<sup>54,76,90</sup> and at 12 months there was no difference in outcomes between groups that received corticosteroid injection and those that received usual care (analgesics as needed).<sup>11</sup>

Surgical interventions are reserved for severe or chronic pathologies with tendon tears and/or failure of conservative rehabilitation. Case series suggest that iliotibial band decompression, bursectomy, and/or gluteal tendon reconstruction reduce pain and improve function in those with recalcitrant problems.<sup>22,25,26,29,56,99,101</sup>





#### Sidelying

Lowermost hip: weight directly over greater trochanter.  
Uppermost hip: flexed and adducted

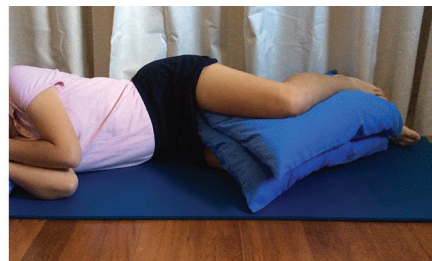
High Compression



#### Supine

Hips slightly abducted

No Compression



#### Modified Sidelying

Pillows between legs and eggshell mattress overlay

Reduced Compression

**FIGURE 4.** Sleeping positions: high, reduced, and no compression at the lateral hip.

## PROPOSED PHYSICAL THERAPY MANAGEMENT STRATEGIES

**T**HE PROPOSED STRATEGIES INCORPORATE aforementioned pathoetiology, general information on tendon pain management, and principles and concepts of optimization of hip abductor muscle function, hip movement, and lower-limb alignment.

### Load Management

**Reducing Compression** For insertional tendinopathies, minimizing positions or activities that involve sustained or repetitive compression of the tendon has been recommended, particularly when compressive forces are applied in combination with high tensile loads.<sup>19,20</sup> It is our clinical observation that there is benefit in advising patients to avoid hip-adducted positions, such as standing “hanging on 1 hip,” standing with legs crossed, and sitting with knees crossed or with knees together (**FIGURE 3**).

Nighttime postures should also be considered. In sidelying, the gluteal tendons on both sides are compressed: the underlying side against the bed, the uppermost side due to the adducted hip position (**FIGURE 4**). Alternative or modified positions would include lying supine with a pillow under the knees if necessary (to unload the anterolateral hips and lumbar

spine). Sidelying is difficult to eliminate, so an eggshell mattress overlay may reduce the compression for the underlying hip, with pillows between the knees and shins reducing adduction of the uppermost hip (**FIGURE 4**). Some patients may also gain relief in a position that is one quarter from prone, in which the body weight rests on the anterolateral thigh (removing compressive load from the greater trochanter), with the uppermost hip in relative abduction.

Hip adduction stretches, in hip flexion or extension (**FIGURE 3**), combine compressive and strong passive tensile loads and should be avoided. This is consistent with advice to avoid stretching in the management of other insertional tendinopathies such as insertional Achilles tendinopathy and proximal hamstring tendinopathy.<sup>19,20</sup> As per common clinical practice, massage and needling techniques may be used in place of stretches, although strong “iliotibial band releases” (massage of the lateral thigh) may be provocative, as the iliotibial band is often tender.<sup>81</sup> Movement patterns employed during functional weight-bearing tasks should be evaluated,<sup>38</sup> and deficiencies noted and used to direct treatment. In particular, femoropelvic control may require optimization, particularly in the frontal plane (as discussed below).

**Controlling High Tensile Loads** Controlling high tensile loads, particularly

rapid increases in activities that involve a stretch-shortening cycle or added compression, is thought to be critical to optimal outcomes of those with tendon pain.<sup>19,20</sup> Education of the patient regarding avoidance of potentially aggravating activities and careful titration of exercise volume are key components of a load-management strategy.

Recreational or sporting activity can usually be maintained in some form, provided the most provocative aspects of those activities are avoided or minimized. Load management during activity for the older or physically deconditioned patient may involve minimization of hill and stair climbing and titrating walking distance as required to control symptoms. For the athlete, temporary suspension of long-distance running, tempo running, hill running, and plyometric drills could be required.<sup>20</sup> Alternative activities such as water-based exercise and cycling could be explored.

### Exercise Therapy

While controlling provocative tensile and compressive loads is likely to be a key component of early recovery, instituting restorative loading through an early and gradually progressive tensile loading program (in positions of minimal hip adduction) aims to reduce pain and improve the tendon’s tensile load-bearing capacity.<sup>20</sup> In addition, strengthening exercises coupled with specific exercises to incorporate

# [ CLINICAL COMMENTARY ]

strength gains into functional movement and to re-educate movement and postures under graduating levels of difficulty appropriate for the individual are likely key to the rehabilitation.

**Isometric Exercises** Sustained isometric muscle contractions are now commonly employed clinically for management of tendon pain<sup>20,77</sup> due to the known analgesic effects.<sup>65</sup> Isometric contractions activate segmental and extrasegmental descending pain inhibitory pathways,<sup>50,51</sup> and sustained low-intensity contractions (25% maximum voluntary isometric contraction) are more effective in raising pain pressure thresholds than are high-intensity contractions (80% maximum voluntary isometric contraction) in the normal population.<sup>41</sup> For patellar tendinopathy, a clinical recommendation for isometric knee extensor loading has been made: 70% maximum voluntary isometric contraction held for 45 to 60 seconds, repeated 4 times, several times a day.<sup>77</sup> The authors of a recent article<sup>75</sup> have demonstrated that five 45-second isometric quadriceps contractions held at 70% of a maximum contraction provided almost complete relief of patellar tendon pain, immediately and for at least 45 minutes, whereas isotonic exercise had only a small and transient effect on pain. In addition, following this isometric training protocol, maximum voluntary isometric contraction was increased and cortical inhibition of quadriceps contraction, detected pre-intervention with transcranial electromagnetic stimulation, was reduced. This is the only study to date to assess the effect of isometric exercise on tendon pain.

The optimal isometric loading dose is yet to be determined for tendon pain and may vary with the patient population and with the particular tendon and its anatomical relationships. For example, higher isometric loads may be better tolerated by younger, more conditioned patients who develop patellar tendinopathy compared with the relatively older and generally less conditioned individual with gluteal tendinopathy. Furthermore, the anatomical structure of the tendons and



**FIGURE 5.** Hip abductor exercises for management of gluteal tendinopathy. Low-load isometric abduction in supine, sidelying, or standing, all performed with focused attention on gentle “trochanteric abductor” activation (gluteus medius and minimus) while keeping the iliotibial band tensioners relaxed (tensor fascia lata, upper gluteus maximus, and vastus lateralis). Low-load abduction may be cued with visualizations such as, “Imagine doing the side splits” in supine and standing and preparing to lift the top leg into abduction (shin horizontal) when in sidelying. High-load, low-velocity weight-bearing abduction performed on a sliding platform with spring resistance in both upright and squat positions to vary stimulus to the abductors; side stepping with the emphasis on pushing into abduction with the stance leg and maintaining optimal pelvic and trunk alignment; band side slides represent a weight-bearing home version of the exercise performed on the sliding platform, except that the weight remains centered on the stationary side, with the sliding leg moving into abduction and with optimal control maintained around the stationary hip and trunk.

relationships between adjacent structures differ considerably between the anterior knee and lateral hip regions. At this stage, a low-intensity effort focused on trochanteric abductor recruitment, and therefore loading these tendons in a non-pain-provocative manner, is recommended for patients with painful gluteal tendinopathy. Higher isometric loads, in at least slight hip abduction to avoid compression, may be possible once patient response is carefully assessed.

Low-load, low-velocity isometric hip abduction may be performed in sidelying, with the affected side uppermost and pillows used to maintain the hip in

neutral or in slight abduction to avoid tendon compression (FIGURE 5). For bilateral pathologies, a supine, slightly abducted position can be substituted, with a belt or an elastic band around the distal thighs for light resistance (FIGURE 5). Low-load isometric abduction can also be performed in standing with slight abduction, and even in leaning with the back against a wall or the hands on a bench in front, if the patient is unable to achieve relaxation of the iliotibial band tensioners in the start position. Instructing the patient to slowly ramp the intensity of the contraction and to minimize pain is suggested in the early stages, until therapist

and patient have determined how reactive the tendon is.

**Low-Velocity, High-Tensile Load Exercise** To achieve muscle hypertrophy of the gluteus medius and minimus and to improve the tensile load-bearing capacity of the gluteal tendons, higher-level tensile loading is required. Low-velocity, high-tensile load exercise, typical of muscle hypertrophy programs, has been shown to also produce beneficial effects on tendon structure that are not provided by eccentric-only programs.<sup>48,49</sup> Targeted strengthening of the trochanteric abductors is perhaps best achieved in those with lateral hip pain through low-velocity, high-tensile load abduction, which minimizes tendon compression. Spring-resisted sliding platforms such as Pilates reformers provide an excellent opportunity for high-load concentric/eccentric hip abductor exercise, due to their ability to provide weight-bearing stimulus and a method of easily titrating the tensile-loading dose, while minimizing tendon compression by allowing exercise in the mid- to inner-range positions of abduction (**FIGURE 5**).

Weight-bearing exercise has been demonstrated to promote higher levels of gluteus medius activation than non-weight-bearing exercise.<sup>10</sup> By moving into inner-range abduction, compressive load of the gluteal tendons is minimized and the iliotibial band tensioners will be mechanically disadvantaged, shifting greater relative stimulus to the trochanteric abductors. In contrast, single-leg sagittal plane tasks such as weighted single-leg squats are naturally performed in some hip adduction,<sup>24</sup> so tendon compression cannot be avoided and the opportunity to bias the deeper abductors is potentially reduced. The spring resistance also allows the therapist to largely eliminate floor friction and be more specific with quantification and therefore graduated progression of tensile tendon loads applied in the frontal plane. If spring-resisted equipment is not available, however, single-leg, band-resisted abduction can be performed with 1 foot on a slide mat or

slippery surface. This allows maintenance of the proprioceptive input of semi-weight bearing (**FIGURE 5**), although an equivalent level of resistance cannot be applied in the same controlled manner as a spring-resisted sliding platform. For the older or deconditioned patient, even side stepping, with the emphasis on the trail leg to push into abduction, can be useful for weight-bearing abductor loading (**FIGURE 5**).

High-tensile load exercise should only be performed 3 times per week, as per a standard strengthening program, allowing adequate time for soft tissue recovery and adaptation.<sup>61</sup> To achieve muscle hypertrophy, the patient must work at an adequate intensity, although there is considerable potential for pain exacerbation and even disruption of a weakened degenerative tendon if tensile loading is initiated at an excessive level or the loading is progressed too rapidly. It is safest to start with a moderate level of effort and low repetitions, until tendon response to tensile loading is established. A 24-hour load-monitoring approach to tendon-based exercise is recommended.<sup>20,84</sup> For gluteal tendinopathy, change in night pain is often a good indicator of response to the exercise program. Increases in night pain may indicate that the load has been too high and needs to be adjusted. Once each level of tensile load is well tolerated, the load should be slowly increased and the response monitored to maximize structural change in the musculotendinous unit, while avoiding or minimizing pain exacerbation.

**Movement Retraining and Functional Loading** While targeted strengthening of the hip abductors should help address muscle atrophy and provide a graded exposure for the tendon to tensile loading, this may be insufficient to engender changes in frontal plane femoropelvic control. Evidence suggests that gross hip abductor strength is not strongly correlated with hip adduction angle during functional tasks such as a single-leg squat,<sup>24</sup> and improving abductor strength in a group with patellofemoral pain did not improve the knee valgus angle.<sup>32</sup> Hip abductor

strengthening together with movement retraining (training control of pelvic and femoral alignment during single-leg squat variations) was successful in improving single-leg squat mechanics but did not alter abnormal running mechanics.<sup>104</sup> Focused attention on reducing hip adduction during running, with real-time kinematic feedback, significantly reduced hip adduction and contralateral pelvic drop during running, but changes in hip adduction during single-leg squat were not quite significant,<sup>66</sup> suggesting that movement retraining needs to be specific to the task.

For those with gluteal tendinopathy, targeted hip abductor strengthening should therefore be accompanied by movement retraining from basic through to higher-level functions, as required by the individual. Depending on the patient's level of pain, physical conditioning, and occupational and sporting requirements, this may involve control of hip adduction during everyday body-weight tasks such as moving between sitting and standing, performing a half-squat, standing on 1 leg, and ascending a standard step height. As pain eases and as appropriate for the particular patient, control of hip adduction under higher loads, at faster speeds, and during more complex actions such as running, landing, and change of direction can be retrained.

### Management of Modifiable Risk Factors and Comorbidities

Management of associated modifiable risk factors and comorbidities is often a feature of rehabilitation for gluteal tendinopathy. While bony morphology cannot be modified, interventions to improve function of the lumbar spine, hip, and knee may be necessary to optimize movement control of the hip and pelvis, and therefore the loading environment of the gluteal tendons. Coexisting degenerative joint disease of the lumbar spine,<sup>17,95</sup> hip,<sup>42</sup> and knee<sup>81</sup> may result in associated weakness of the hip and knee extensors. Functional exercises, such as bridging, squatting, and step-type exercises, can serve multiple purposes in optimizing

control of functional hip adduction, improving function of the lower-limb extensors, and improving muscular support of the lumbopelvic region, hip, and knee. Manual therapy and other specific exercise for the lumbar spine, hip, or knee joint may be required to address the co-existing joint disease, but it is important that the principles of respecting gluteal tendon load, particularly the control of compression, are preserved. Exercise and general increases in activity may also reduce weight and gynoid adiposity.

## CONCLUSION

**G**LUTEAL TENDINOPATHY IS THE most common local source of lateral hip pain. From the evidence available, excessive compressive loading of these tendons and their adjacent bursae in a position of hip adduction is a highly likely driver for pathology and pain in this condition. A substantial amount of additional work is required to establish a clinical test battery with high diagnostic utility. Similarly, there is poor evidence as to what constitutes best management for lateral hip pain. Following the proposed guidelines for load management and exercise in rehabilitation of tendinopathic conditions,<sup>19,20,37</sup> those with lateral hip pain should minimize sustained, repetitive, or loaded hip adduction due to the high compressive forces at the greater trochanter. Exercise should include sustained isometric abduction to assist with early pain relief. Gradual progression in tensile loading moving toward low-velocity, heavy-load abduction will improve conditioning of the abductor musculotendinous complex and load-bearing capacity during function. Functional retraining, such as double- and single-leg weight-bearing tasks with emphasis on actively minimizing adduction during dynamic loading, should assist in transferring strength gains into functional gains. High-quality trials are required to clarify which diagnostic tests and treatment strategies are most effective in the management of lateral hip pain. ●

## REFERENCES

1. Almekinders LC, Weinhold PS, Maffulli N. Compression etiology in tendinopathy. *Clin Sports Med*. 2003;22:703-710. [http://dx.doi.org/10.1016/S0278-5919\(03\)00067-X](http://dx.doi.org/10.1016/S0278-5919(03)00067-X)
2. Altman R, Alarcón G, Appelrouth D, et al. The American College of Rheumatology criteria for the classification and reporting of osteoarthritis of the hip. *Arthritis Rheum*. 1991;34:505-514.
3. Baeten D, De Keyser F. The histopathology of spondyloarthropathy. *Curr Mol Med*. 2004;4:1-12.
4. Bancroft LW, Blankenbaker DG. Imaging of the tendons about the pelvis. *AJR Am J Roentgenol*. 2010;195:605-617. <http://dx.doi.org/10.2214/AJR.10.4682>
5. Bey MJ, Song HK, Wehrli FW, Soslosky LJ. Intratendinous strain fields of the intact supraspinatus tendon: the effect of glenohumeral joint position and tendon region. *J Orthop Res*. 2002;20:869-874. [http://dx.doi.org/10.1016/S0736-0266\(01\)00177-2](http://dx.doi.org/10.1016/S0736-0266(01)00177-2)
6. Bird PA, Oakley SP, Shnier R, Kirkham BW. Prospective evaluation of magnetic resonance imaging and physical examination findings in patients with greater trochanteric pain syndrome. *Arthritis Rheum*. 2001;44:2138-2145. [http://dx.doi.org/10.1002/1529-0131\(200109\)44:9<2138::AID-ART367>3.0.CO;2-M](http://dx.doi.org/10.1002/1529-0131(200109)44:9<2138::AID-ART367>3.0.CO;2-M)
7. Birnbaum K, Pandorf T. Finite element model of the proximal femur under consideration of the hip centralizing forces of the iliotalib tract. *Clin Biomech (Bristol, Avon)*. 2011;26:58-64. <http://dx.doi.org/10.1016/j.clinbiomech.2010.09.005>
8. Birnbaum K, Siebert CH, Pandorf T, Schopphoff E, Prescher A, Niethard FU. Anatomical and biomechanical investigations of the iliotalib tract. *Surg Radiol Anat*. 2004;26:433-446. <http://dx.doi.org/10.1007/s00276-004-0265-8>
9. Blankenbaker DG, Ullrick SR, Davis KW, De Smet AA, Haaland B, Fine JP. Correlation of MRI findings with clinical findings of trochanteric pain syndrome. *Skeletal Radiol*. 2008;37:903-909. <http://dx.doi.org/10.1007/s00256-008-0514-8>
10. Bolgia LA, Uhl TL. Electromyographic analysis of hip rehabilitation exercises in a group of healthy subjects. *J Orthop Sports Phys Ther*. 2005;35:487-494. <http://dx.doi.org/10.2519/jospt.2005.35.8.487>
11. Brinks A, van Rijn RM, Willemsen SP, et al. Corticosteroid injections for greater trochanteric pain syndrome: a randomized controlled trial in primary care. *Ann Fam Med*. 2011;9:226-234. <http://dx.doi.org/10.1370/afm.1232>
12. Brooker AF, Jr. The surgical approach to refractory trochanteric bursitis. *Johns Hopkins Med J*. 1979;145:98-100.
13. Brukner P, Khan K. *Brukner & Khan's Clinical Sports Medicine*. 4th ed. New York, NY: McGraw-Hill; 2011.
14. Bunker TD, Esler CN, Leach WJ. Rotator cuff tear of the hip. *J Bone Joint Surg Br*. 1997;79:618-620.
15. Cohen SP, Strassels SA, Foster L, et al. Comparison of fluoroscopically guided and blind corticosteroid injections for greater trochanteric pain syndrome: multicentre randomised controlled trial. *BMJ*. 2009;338:b1088. <http://dx.doi.org/10.1136/bmj.b1088>
16. Coleman RE. Clinical features of metastatic bone disease and risk of skeletal morbidity. *Clin Cancer Res*. 2006;12:6243s-6249s. <http://dx.doi.org/10.1158/1078-0432.CCR-06-0931>
17. Collee G, Dijkmans BA, Vandenbroucke JP, Cats A. Greater trochanteric pain syndrome (trochanteric bursitis) in low back pain. *Scand J Rheumatol*. 1991;20:262-266.
18. Connell DA, Bass C, Sykes CA, Young D, Edwards E. Sonographic evaluation of gluteus medius and minimus tendinopathy. *Eur Radiol*. 2003;13:1339-1347. <http://dx.doi.org/10.1007/s00330-002-1740-4>
19. Cook JL, Purdam C. Is compressive load a factor in the development of tendinopathy? *Br J Sports Med*. 2012;46:163-168. <http://dx.doi.org/10.1136/bjsports-2011-090414>
20. Cook JL, Purdam CR. The challenge of managing tendinopathy in competing athletes. *Br J Sports Med*. 2014;48:506-509. <http://dx.doi.org/10.1136/bjsports-2012-092078>
21. Cook JL, Purdam CR. Is tendon pathology a continuum? A pathology model to explain the clinical presentation of load-induced tendinopathy. *Br J Sports Med*. 2009;43:409-416. <http://dx.doi.org/10.1136/bjsm.2008.051193>
22. Craig RA, Jones DP, Oakley AP, Dunbar JD. Iliotibial band Z-lengthening for refractory trochanteric bursitis (greater trochanteric pain syndrome). *ANZ J Surg*. 2007;77:996-998. <http://dx.doi.org/10.1111/j.1445-2197.2007.04298.x>
23. Del Buono A, Papalia R, Khanduja V, Denaro V, Maffulli N. Management of the greater trochanteric pain syndrome: a systematic review. *Br Med Bull*. 2012;102:115-131. <http://dx.doi.org/10.1093/bmb/ldr038>
24. DiMattia MA, Livengood AL, Uhl TL, Mattacola CG, Malone TR. What are the validity of the single-leg-squat test and its relationship to hip abduction strength? *J Sport Rehabil*. 2005;14:108-123.
25. Domb BG, Carreira DS. Endoscopic repair of full-thickness gluteus medius tears. *Arthrosc Tech*. 2013;2:e77-e81. <http://dx.doi.org/10.1016/j.eats.2012.11.005>
26. Farr D, Selesnick H, Janecki C, Cordas D. Arthroscopic bursectomy with concomitant iliotalibial band release for the treatment of recalcitrant trochanteric bursitis. *Arthroscopy*. 2007;23:905.e1-905.e5. <http://dx.doi.org/10.1016/j.arthro.2006.10.021>
27. Fearon A, Stephens S, Cook J, et al. The relationship of femoral neck shaft angle and adiposity to greater trochanteric pain syndrome in women. A case control morphology and anthropometric study. *Br J Sports Med*. 2012;46:888-892. <http://dx.doi.org/10.1136/bjsports-2011-090744>
28. Fearon AM, Cook JL, Scarvell JM, Neeman T,

- Cormick W, Smith PN. Greater trochanteric pain syndrome negatively affects work, physical activity and quality of life: a case control study. *J Arthroplasty*. 2014;29:383-386. <http://dx.doi.org/10.1016/j.arth.2012.10.016>
29. Fearon AM, Scarvell JM, Cook JL, Smith PN. Does ultrasound correlate with surgical or histologic findings in greater trochanteric pain syndrome? A pilot study. *Clin Orthop Relat Res*. 2010;468:1838-1844. <http://dx.doi.org/10.1007/s11999-009-1174-2>
30. Fearon AM, Scarvell JM, Neeman T, Cook JL, Cormick W, Smith PN. Greater trochanteric pain syndrome: defining the clinical syndrome. *Br J Sports Med*. 2013;47:649-653. <http://dx.doi.org/10.1136/bjsports-2012-091565>
31. Fearon AM, Twin J, Dahlstrom JE, et al. Increased substance P expression in the trochanteric bursa of patients with greater trochanteric pain syndrome. *Rheumatol Int*. 2014;34:1441-1448. <http://dx.doi.org/10.1007/s00296-014-2957-7>
32. Ferber R, Kendall KD, Farr L. Changes in knee biomechanics after a hip-abductor strengthening protocol for runners with patellofemoral pain syndrome. *J Athl Train*. 2011;46:142-149.
33. Filler AG. Piriformis and related entrapment syndromes: diagnosis & management. *Neurosurg Clin N Am*. 2008;19:609-622. <http://dx.doi.org/10.1016/j.nec.2008.07.029>
34. Finlay V, Phillips M, Wood F, Edgar D. A reliable and valid outcome battery for measuring recovery of lower limb function and balance after burn injury. *Burns*. 2010;36:780-786. <http://dx.doi.org/10.1016/j.burns.2009.10.019>
35. Flack NA, Meikle GR, Reddy M, Nicholson HD, Woodley SJ. Hip abductor muscle volume in women with lateral hip pain: a case-controlled study. *Surg Radiol Anat*. 2012;34:847-855. <http://dx.doi.org/10.1007/s00276-012-0970-7>
36. Furia JP, Rompe JD, Maffulli N. Low-energy extracorporeal shock wave therapy as a treatment for greater trochanteric pain syndrome. *Am J Sports Med*. 2009;37:1806-1813. <http://dx.doi.org/10.1177/0363546509333014>
37. Glasgow P, Phillips N, Bleakley C. Optimal loading: key variables and mechanisms. *Br J Sports Med*. 2015;49:278-279. <http://dx.doi.org/10.1136/bjsports-2014-094443>
38. Grimaldi A. Assessing lateral stability of the hip and pelvis. *Man Ther*. 2011;16:26-32. <http://dx.doi.org/10.1016/j.math.2010.08.005>
39. Hardcastle P, Nade S. The significance of the Trendelenburg test. *J Bone Joint Surg Br*. 1985;67:741-746.
40. Himmelreich H, Vogt L, Banzer W. Gluteal muscle recruitment during level, incline and stair ambulation in healthy subjects and chronic low back pain patients. *J Back Musculoskelet Rehabil*. 2008;21:193-199.
41. Hoeger Bement MK, Dicapo J, Rasiarmos R, Hunter SK. Dose response of isometric contractions on pain perception in healthy adults. *Med Sci Sports Exerc*. 2008;40:1880-1889. <http://dx.doi.org/10.1249/MSS.0b013e31817eeec>
42. Howell GE, Biggs RE, Bourne RB. Prevalence of abductor mechanism tears of the hips in patients with osteoarthritis. *J Arthroplasty*. 2001;16:121-123. <http://dx.doi.org/10.1054/arth.2001.19158>
43. Hungerford B, Gilleard W, Hodges P. Evidence of altered lumbopelvic muscle recruitment in the presence of sacroiliac joint pain. *Spine (Phila Pa 1976)*. 2003;28:1593-1600.
44. Iorio R, Healy WL, Warren PD, Appleby D. Lateral trochanteric pain following primary total hip arthroplasty. *J Arthroplasty*. 2006;21:233-236. <http://dx.doi.org/10.1016/j.arth.2005.03.041>
45. Kagan A, 2nd. Rotator cuff tears of the hip. *Clin Orthop Relat Res*. 1999:135-140.
46. Kingzett-Taylor A, Tirman PF, Feller J, et al. Tendinosis and tears of gluteus medius and minimus muscles as a cause of hip pain: MR imaging findings. *AJR Am J Roentgenol*. 1999;173:1123-1126. <http://dx.doi.org/10.2214/ajr.173.4.10511191>
47. Kong A, Van der Vliet A, Zadow S. MRI and US of gluteal tendinopathy in greater trochanteric pain syndrome. *Eur Radiol*. 2007;17:1772-1783. <http://dx.doi.org/10.1007/s00330-006-0485-x>
48. Kongsgaard M, Kovanan V, Aagaard P, et al. Corticosteroid injections, eccentric decline squat training and heavy slow resistance training in patellar tendinopathy. *Scand J Med Sci Sports*. 2009;19:790-802. <http://dx.doi.org/10.1111/j.1600-0838.2009.00949.x>
49. Kongsgaard M, Qvortrup K, Larsen J, et al. Fibril morphology and tendon mechanical properties in patellar tendinopathy: effects of heavy slow resistance training. *Am J Sports Med*. 2010;38:749-756. <http://dx.doi.org/10.1177/0363546509350915>
50. Kosek E, Ekholm J. Modulation of pressure pain thresholds during and following isometric contraction. *Pain*. 1995;61:481-486. [http://dx.doi.org/10.1016/0304-3959\(95\)00112-3](http://dx.doi.org/10.1016/0304-3959(95)00112-3)
51. Kosek E, Lundberg L. Segmental and plurisegmental modulation of pressure pain thresholds during static muscle contractions in healthy individuals. *Eur J Pain*. 2003;7:251-258. [http://dx.doi.org/10.1016/S1090-3801\(02\)00124-6](http://dx.doi.org/10.1016/S1090-3801(02)00124-6)
52. Kumagai M, Shiba N, Higuchi F, Nishimura H, Inoue A. Functional evaluation of hip abductor muscles with use of magnetic resonance imaging. *J Orthop Res*. 1997;15:888-893. <http://dx.doi.org/10.1002/jor.1100150615>
53. Kummer B. Is the Pauwels' theory of hip biomechanics still valid? A critical analysis, based on modern methods. *Ann Anat*. 1993;175:203-210.
54. Labrosse JM, Cardinal É, Leduc BE, et al. Effectiveness of ultrasound-guided corticosteroid injection for the treatment of gluteus medius tendinopathy. *AJR Am J Roentgenol*. 2010;194:202-206. <http://dx.doi.org/10.2214/AJR.08.1215>
55. Lequesne M. From "peri-arthritis" to hip "rotator cuff" tears. Trochanteric tendinobursitis. *Joint Bone Spine*. 2006;73:344-348. <http://dx.doi.org/10.1016/j.jbspin.2006.04.002>
56. Lequesne M, Dijan P, Vuillemin V, Mathieu P. Prospective study of refractory greater trochanter pain syndrome. MRI findings of gluteal tendon tears seen at surgery. Clinical and MRI results of tendon repair. *Joint Bone Spine*. 2008;75:458-464. <http://dx.doi.org/10.1016/j.jbspin.2007.12.004>
57. Lequesne M, Mathieu P, Vuillemin-Bodaghi V, Bard H, Dijan P. Gluteal tendinopathy in refractory greater trochanter pain syndrome: diagnostic value of two clinical tests. *Arthritis Rheum*. 2008;59:241-246. <http://dx.doi.org/10.1002/art.23354>
58. Leshner JM, Dreyfuss P, Hager N, Kaplan M, Furman M. Hip joint pain referral patterns: a descriptive study. *Pain Med*. 2008;9:22-25. <http://dx.doi.org/10.1111/j.1526-4637.2006.00153.x>
59. Little H. Trochanteric bursitis: a common cause of pelvic girdle pain. *Can Med Assoc J*. 1979;120:456-458.
60. Long SS, Surrey DE, Nazarian LN. Sonography of greater trochanteric pain syndrome and the rarity of primary bursitis. *AJR Am J Roentgenol*. 2013;201:1083-1086. <http://dx.doi.org/10.2214/AJR.12.10038>
61. Magnusson SP, Langberg H, Kjaer M. The pathogenesis of tendinopathy: balancing the response to loading. *Nat Rev Rheumatol*. 2010;6:262-268. <http://dx.doi.org/10.1038/nrrheum.2010.43>
62. Matuszewski PE, Chen YL, Szczesny SE, et al. Regional variation in human supraspinatus tendon proteoglycans: decorin, biglycan, and aggrecan. *Connect Tissue Res*. 2012;53:343-348. <http://dx.doi.org/10.3109/03008207.2012.654866>
63. McMahon SE, Smith TO, Hing CB. A systematic review of imaging modalities in the diagnosis of greater trochanteric pain syndrome. *Musculoskeletal Care*. 2012;10:232-239. <http://dx.doi.org/10.1002/msc.1024>
64. Meknas K, Johansen O, Kartus J. Retro-trochanteric sciatica-like pain: current concept. *Knee Surg Sports Traumatol Arthrosc*. 2011;19:1971-1985. <http://dx.doi.org/10.1007/s00167-011-1573-2>
65. Naugle KM, Fillingim RB, Riley JL, 3rd. A meta-analytic review of the hypoalgesic effects of exercise. *J Pain*. 2012;13:1139-1150. <http://dx.doi.org/10.1016/j.jpain.2012.09.006>
66. Noehren B, Scholz J, Davis I. The effect of real-time gait retraining on hip kinematics, pain and function in subjects with patellofemoral pain syndrome. *Br J Sports Med*. 2011;45:691-696. <http://dx.doi.org/10.1136/bjsm.2009.069112>
67. O'Brien SD, Bui-Mansfield LT. MRI of quadratus femoris muscle tear: another cause of hip pain. *AJR Am J Roentgenol*. 2007;189:1185-1189. <http://dx.doi.org/10.2214/AJR.07.2408>
68. Orchard JW, Cook JL, Halpin N. Stress-shielding as a cause of insertional tendinopathy: the operative technique of limited adductor tenotomy supports this theory. *J Sci Med Sport*. 2004;7:424-428.

69. Pffirrmann CW, Notzli HP, Dora C, Hodler J, Zanetti M. Abductor tendons and muscles assessed at MR imaging after total hip arthroplasty in asymptomatic and symptomatic patients. *Radiology*. 2005;235:969-976. <http://dx.doi.org/10.1148/radiol.2353040403>
70. Philippon MJ, Maxwell RB, Johnston TL, Schenker M, Briggs KK. Clinical presentation of femoroacetabular impingement. *Knee Surg Sports Traumatol Arthrosc*. 2007;15:1041-1047. <http://dx.doi.org/10.1007/s00167-007-0348-2>
71. Pretell J, Ortega J, García-Rayó R, Resines C. Distal fascia lata lengthening: an alternative surgical technique for recalcitrant trochanteric bursitis. *Int Orthop*. 2009;33:1223-1227. <http://dx.doi.org/10.1007/s00264-009-0727-z>
72. Rahman N, Penm E, Bhatia K. Arthritis and Musculoskeletal Conditions in Australia 2005: With a Focus on Osteoarthritis, Rheumatoid Arthritis and Osteoporosis. Canberra, Australia: Australian Institute of Health and Welfare; 2005.
73. Raman D, Haslock I. Trochanteric bursitis—a frequent cause of ‘hip’ pain in rheumatoid arthritis. *Ann Rheum Dis*. 1982;41:602-603. <http://dx.doi.org/10.1136/ard.41.6.602>
74. Reiman MP, Goode AP, Hegedus EJ, Cook CE, Wright AA. Diagnostic accuracy of clinical tests of the hip: a systematic review with meta-analysis. *Br J Sports Med*. 2013;47:893-902. <http://dx.doi.org/10.1136/bjsports-2012-091035>
75. Rio E, Kidgell D, Purdam C, et al. Isometric exercise induces analgesia and reduces inhibition in patellar tendinopathy. *Br J Sports Med*. 2015;49:1277-1283. <http://dx.doi.org/10.1136/bjsports-2014-094386>
76. Rompe JD, Segal NA, Cacchio A, Furia JP, Morrall A, Maffulli N. Home training, local corticosteroid injection, or radial shock wave therapy for greater trochanter pain syndrome. *Am J Sports Med*. 2009;37:1981-1990. <http://dx.doi.org/10.1177/0363546509334374>
77. Rudavsky A, Cook J. Physiotherapy management of patellar tendinopathy (jumper’s knee). *J Physiother*. 2014;60:122-129. <http://dx.doi.org/10.1016/j.jphys.2014.06.022>
78. Rybicki EF, Simonen FA, Weis EB, Jr. On the mathematical analysis of stress in the human femur. *J Biomech*. 1972;5:203-215.
79. Sayegh F, Potoupnis M, Kapetanios G. Greater trochanter bursitis pain syndrome in females with chronic low back pain and sciatica. *Acta Orthop Belg*. 2004;70:423-428.
80. Scott A, Cook JL, Hart DA, Walker DC, Duronio V, Khan KM. Tenocyte responses to mechanical loading in vivo: a role for local insulin-like growth factor 1 signaling in early tendinosis in rats. *Arthritis Rheum*. 2007;56:871-881. <http://dx.doi.org/10.1002/art.22426>
81. Segal NA, Felson DT, Torner JC, et al. Greater trochanteric pain syndrome: epidemiology and associated factors. *Arch Phys Med Rehabil*. 2007;88:988-992. <http://dx.doi.org/10.1016/j.apmr.2007.04.014>
82. Segal NA, Harvey W, Felson DT, et al. Leg-length inequality is not associated with greater trochanteric pain syndrome. *Arthritis Res Ther*. 2008;10:R62. <http://dx.doi.org/10.1186/ar2433>
83. Shbeeb MI, Matteson EL. Trochanteric bursitis (greater trochanter pain syndrome). *Mayo Clin Proc*. 1996;71:565-569.
84. Silbernagel KG, Thoméé R, Eriksson BI, Karlsson J. Continued sports activity, using a pain-monitoring model, during rehabilitation in patients with Achilles tendinopathy: a randomized controlled study. *Am J Sports Med*. 2007;35:897-906. <http://dx.doi.org/10.1177/0363546506298279>
85. Sim FH, Scott SG. Injuries of the pelvis and hip in athletes: anatomy and function. In: Nicholas JA, Hershman EB, eds. *The Lower Extremity and Spine in Sports Medicine*. St Louis, MO: Mosby; 1986:1119-1169.
86. Sims K. Assessment and treatment of hip osteoarthritis. *Man Ther*. 1999;4:136-144. <http://dx.doi.org/10.1054/math.1999.0192>
87. Soslowsky LJ, Thomopoulos S, Esmail A, et al. Rotator cuff tendinosis in an animal model: role of extrinsic and overuse factors. *Ann Biomed Eng*. 2002;30:1057-1063.
88. Stafford GH, Villar RN. Ischiofemoral impingement. *J Bone Joint Surg Br*. 2011;93:1300-1302. <http://dx.doi.org/10.1302/0301-620X.93B10.26714>
89. Stecco A, Gilliar W, Hill R, Fullerton B, Stecco C. The anatomical and functional relation between gluteus maximus and fascia lata. *J Bodyw Mov Ther*. 2013;17:512-517. <http://dx.doi.org/10.1016/j.jbmt.2013.04.004>
90. Stephens MB, Beutler AI, O’Connor FG. Musculoskeletal injections: a review of the evidence. *Am Fam Physician*. 2008;78:971-976.
91. Stern JT, Jr. Anatomical and functional specializations of the human gluteus maximus. *Am J Phys Anthropol*. 1972;36:315-339. <http://dx.doi.org/10.1002/ajpa.1330360303>
92. Sutter R, Kalberer F, Binkert CA, Graf N, Pffirrmann CW, Gutzeit A. Abductor tendon tears are associated with hypertrophy of the tensor fasciae latae muscle. *Skeletal Radiol*. 2013;42:627-633. <http://dx.doi.org/10.1007/s00256-012-1514-2>
93. Swezey RL. Pseudo-radiculopathy in subacute trochanteric bursitis of the subgluteus maximus bursa. *Arch Phys Med Rehabil*. 1976;57:387-390. [http://dx.doi.org/10.1016/0304-3959\(77\)90107-5](http://dx.doi.org/10.1016/0304-3959(77)90107-5)
94. Taneja AK, Bredella MA, Torriani M. Ischiofemoral impingement. *Magn Reson Imaging Clin N Am*. 2013;21:65-73. <http://dx.doi.org/10.1016/j.mric.2012.08.005>
95. Tortolani PJ, Carbone JJ, Quartararo LG. Greater trochanteric pain syndrome in patients referred to orthopedic spine specialists. *Spine J*. 2002;2:251-254.
96. Vieira EL, Vieira EA, da Silva RT, Berfein PA, Abdalla RJ, Cohen M. An anatomic study of the iliotibial tract. *Arthroscopy*. 2007;23:269-274. <http://dx.doi.org/10.1016/j.arthro.2006.11.019>
97. Viradia NK, Berger AA, Dahners LE. Relationship between width of greater trochanters and width of iliac wings in trochanteric bursitis. *Am J Orthop (Belle Mead NJ)*. 2011;40:E159-E162.
98. Vleeming A. *Movement, Stability and Low Back Pain: The Essential Role of the Pelvis*. New York, NY: Churchill Livingstone; 1997.
99. Voos JE, Shindle MK, Pruett A, Asnis PD, Kelly BT. Endoscopic repair of gluteus medius tendon tears of the hip. *Am J Sports Med*. 2009;37:743-747. <http://dx.doi.org/10.1177/0363546508328412>
100. Walker P, Kannangara S, Bruce WJ, Michael D, Van der Wall H. Lateral hip pain: does imaging predict response to localized injection? *Clin Orthop Relat Res*. 2007;457:144-149. <http://dx.doi.org/10.1097/BLO.0b013e318029199a>
101. Walsh MJ, Walton JR, Walsh NA. Surgical repair of the gluteal tendons: a report of 72 cases. *J Arthroplasty*. 2011;26:1514-1519. <http://dx.doi.org/10.1016/j.arth.2011.03.004>
102. Westacott DJ, Minns JI, Foguet P. The diagnostic accuracy of magnetic resonance imaging and ultrasonography in gluteal tendon tears - a systematic review. *Hip Int*. 2011;21:637-645.
103. Williams BS, Cohen SP. Greater trochanteric pain syndrome: a review of anatomy, diagnosis and treatment. *Anesth Analg*. 2009;108:1662-1670. <http://dx.doi.org/10.1213/ane.0b013e31819d6562>
104. Willy RW, Davis IS. The effect of a hip-strengthening program on mechanics during running and during a single-leg squat. *J Orthop Sports Phys Ther*. 2011;41:625-632. <http://dx.doi.org/10.2519/jospt.2011.3470>
105. Woodley SJ, Nicholson HD, Livingstone V, et al. Lateral hip pain: findings from magnetic resonance imaging and clinical examination. *J Orthop Sports Phys Ther*. 2008;38:313-328. <http://dx.doi.org/10.2519/jospt.2008.2685>
106. Woyski D, Olinger A, Wright B. Smaller insertion area and inefficient mechanics of the gluteus medius in females. *Surg Radiol Anat*. 2013;35:713-719. <http://dx.doi.org/10.1007/s00276-013-1096-2>
107. Youdas JW, Mrasz ST, Norstad BJ, Schinke JJ, Hollman JH. Determining meaningful changes in pelvic-on-femoral position during the Trendelenburg test. *J Sport Rehabil*. 2007;16:326-335.
108. Zalavras CG, Lieberman JR. Osteonecrosis of the femoral head: evaluation and treatment. *J Am Acad Orthop Surg*. 2014;22:455-464. <http://dx.doi.org/10.5435/JAAOS-22-07-455>

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