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Chapter

Contemporary Non-Surgical Considerations in the Management of People with Extra- and Intra-Articular Hip Pathologies

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Abstract

The hip joint can often be affected by extra- and intra-articular pathologies including gluteal tendinopathy, femoroacetabular impingement syndrome and hip osteoarthritis. Understanding alterations associated with these pathologies will provide greater insight into developing and optimising patient-specific treatments. A number of biomechanical and neuromuscular impairment are associated with Femoracetabular impingement (FAI), gluteal tendinopathy (GT) and hip osteoarthritis (OA) conditions including but not limited to muscle weakness, altered postural control, restricted range of motion and altered tendon/joint loading. These alterations can present differently in sub-groups of patients and result directly from the pathological process and/or indirectly from pain and its consequences (e.g. reduced activity). These impairments are often targets for conservative interventions but there is currently little clinical trial evidence to show that treatments can modify these impairments. Clinical trial evidence does, however, support conservative treatment options for each of the pathologies reviewed. Clinical outcome tools used to evaluate the effects of treatment and track change over time are recommended.

Keywords: hip osteoarthritis, femoroacetabular impingement, gluteal tendinopathy, exercise, biomechanics, outcome measurement

1. Introduction

This chapter will present contemporary conservative considerations for the management of extra- and intra-articular hip pathologies including gluteal tendinopathy (GT), femoroacetabular impingement (FAI) syndrome and hip osteoarthritis (OA). The clinical presentation of hip pathology is frequent and can be complex. Over the past decade research has uncovered new insights into biomechanical alterations associations with GT, FAI and hip OA that enables clinicians to better understand the condition and management options. We provide an overview of the most significant discoveries as well as unpack the evidence for effective conservative management. Clinical outcome tools used to evaluate the effectiveness of treatments and track change over time in these hip conditions are reviewed.
2. Gluteal tendinopathy

Gluteal tendinopathy, also referred to as “greater trochanteric pain syndrome”, is a chronic, debilitating musculoskeletal condition affecting the tendinous insertion of the gluteus medius and/or minimus muscles at or above their attachments into the greater trochanter of the femur [1]. The hallmark features of this extra-articular hip condition are pain and tenderness to palpation at or around the region of the greater trochanter [1–3]. Prevalence rates of GT have been reported at 18% of those aged 50–79 years presenting to general practitioners [3]. Individuals with GT are most frequently over the age of 40 years [4] and typically experience pain during walking, stair climbing and/or lying on the affected side [1–3].

2.1 Biomechanical considerations in gluteal tendinopathy

2.1.1 Important anatomical and biomechanical considerations

The trochanteric bursae were previously considered the primary structure implicated in greater trochanteric pain [5]. However, new evidence from magnetic resonance imaging (MRI) [6, 7], ultrasound [8, 9] and surgical case series’ [7, 10] has led to a contemporary understanding of the pathological mechanisms of the gluteal tendons underpinning greater trochanter pain. This progressive understanding of tendon involvement has necessitated important advances regarding biomechanical considerations associated with GT.

The gluteal tendons are vulnerable to anatomical compression against the (i) underlying greater trochanter, as they wrap over the borders of its bony facets into their respective insertions [11], and (ii) from the overlying iliotibial band (ITB), particularly as the hip moves into adduction. With increasing adduction of the femur relative to the pelvis, the insertion of the gluteus minimus and medius muscles on the greater trochanter are moved away from their respective origins on the ilium, placing longitudinal tensile and transverse tensile strain through the tendon fibres passing over the greater trochanter. In addition, the ITB exerts progressively higher compressive forces at the greater trochanter as the hip moves into hip adduction (4 N at 0°, increased by nine-fold to 36 N at 10° and 106 N at 40°) [12], which has direct consequences for gluteal tendon loading. Excessive tensile and compressive loads are accepted to be detrimental for tendon health and particularly relevant for the development and perpetuation of tendinopathy [13]. Thus, dynamic control of hip adduction is pertinent in the assessment and management of GT [14].

2.1.2 Hip abductor muscle weakness and clinical relevance to loading biomechanics

Like other tendinopathies, muscle weakness is a feature of GT [15]. Strength deficiencies of 32% of the hip abductor muscles on the symptomatic hip and 23% on the asymptomatic hip have been identified in individuals with clinically and MRI diagnosed GT compared to age- and sex-comparable controls [15]. The primary functional role of the hip abductor muscles is to maintain alignment of the pelvis in the frontal plane during gait, to eccentrically control the provocative position of hip adduction [16]. The relationship between hip adduction angle and hip abductor tendon loading in GT highlights the importance of abductor muscle strength for adequate eccentric control of hip adduction in this patient group [16]. Clinicians often evaluate hip abductor function by visually evaluating a patient’s ability to maintain and control position of the pelvis in single leg stance (SLS) [17]. Further, SLS kinematics are considered relevant for control of single leg loading during gait.
Data from three-dimensional motion capture analysis identified that individuals with GT exhibit greater lateral pelvic shift and hip adduction in preparation for SLS, and more hip adduction and less contralateral pelvic elevation during SLS in the frontal plane when compared to age and sex matched controls [18] (Figure 1). Though these findings may be, in part, explained by hip abductor muscle weakness [18], they also provide important insight into why single leg stance is provocative for many individuals with GT. Specifically, the increased potential tensile and compressive load through the gluteal tendons as the muscles work to control the position of the pelvis on the femur, is a likely relevant mechanism for tendon overload and pain.

2.1.3 Gait biomechanics

To date, only one study has evaluated walking kinematics and kinetics in individuals with GT compared to healthy controls. In contrast to pain-free controls, individuals with GT exhibit a significantly greater external hip adduction moment during the stance phase of walking [19] and during stair climbing [20]. These observations are thought to have distinct clinical relevance, given the external hip adduction moment represents an internal hip abductor moment contributed to by active and passive tension in the primary hip abductor muscles (i.e. the gluteus minimus and medius) [21]. Of importance to clinicians who use visual observation as part of their assessment in GT, contralateral pelvic drop is associated with a greater magnitude of the external hip adduction moment [19]. While data has shown that individuals with GT exhibit greater contralateral pelvic drop during late stance compared to controls, with implications for hip adduction angles and tendon loading, this between-group difference during walking was small on average (1.4 degrees), with questionable clinical relevance [19]. This small mean difference may be explained by variation in walking strategies utilised by participants in the GT group. A secondary analysis identified distinct subgroups in those with GT [19]. This novel and clinically relevant observation highlights that people with GT can compensate for hip abductor weakness in different ways, which coincide with compensations reported in individuals with intra-articular hip pain [22].
with extra-articular hip pathology, such as GT [19]. Specifically, two subgroups were identified in those with GT: (1) individuals demonstrating an uncompensated Trendelenburg (contralateral pelvic drop and associated contralateral trunk lean where no compensation is made for hip abductor weakness and the position of the pelvis cannot be maintained in the frontal plane); and (2) individuals demonstrating a compensated Trendelenburg (ipsilateral trunk lean in an attempt to bring the centre of mass closer to the base of support, resulting in reduced hip abductor muscle requirements and maintenance of the position of the pelvis in the frontal plane) (Figure 2).

2.2 Non-surgical management for gluteal tendinopathy

Evidence for the management of gluteal tendinopathy is continuing to emerge. Historically, as a result of limited understanding of the pathology and associated impairments in GT, treatment had been simplistic, targeting symptoms or the presumed pathological involvement of the trochanteric bursae. More recently, drawing from contemporary evidence in other tendinopathies and an understanding of tendon structure and function, exercise interventions for GT have been refined and are beginning to be tested in randomised controlled trials with promising results. The most recent systematic review at the time of print concluded that poor quality and insufficient data prevented any conclusions to be drawn regarding optimal treatment for greater trochanteric pain syndrome including GT [23]. Studies in this review and others describe interventions of surgical tendon repair, ITB release and bursectomy, corticosteroid injection, home exercise, shock wave therapy and dry needling [23]. Issues arise when interpreting the collective results of these studies with respect to GT, as the samples are diverse with respect to co-morbidities (e.g. hip OA, lumbar pathology), symptom duration, and most importantly, clinical and

![Figure 2.](image)
imaging diagnosis specific to GT. Further, very few interventions have been evaluated in randomised controlled trials.

The strong focus on corticosteroid injection in GT arises from the original theory that trochanteric pain was due to an inflammatory process within the trochanteric bursae. However, the effectiveness and safety of the use of corticosteroid injection in tendinopathy is debatable. Evidence from a high quality systematic review pooling 41 studies evaluating the effect of corticosteroid injection on upper limb, patella and Achilles tendinopathies suggests that while cortisone improves symptoms in the short term, there are no long term effects at 13–26 weeks or ≥ 52 weeks [24]. While these findings cannot be directly inferred to GT, a similar attenuation effect of symptom relief in response to corticosteroid injection has been demonstrated in three clinical trials in greater trochanteric pain syndrome [25–27], questioning the efficacy of corticosteroid use in GT.

Given that tendon is a metabolically active tissue that maintains its integrity in response to tensile loading, exercise and load modification appear to be important aspects of effective treatment in management of tendinopathy [28]. In order to modify tendon load in the lower limb, addressing lower limb biomechanics and neuromuscular control is considered an effective clinical strategy [14]. Specific to GT, modifying compressive load at the greater trochanter is thought to be particularly relevant [5, 27]. Load modification can be achieved by reducing time spent in sustained positions of hip adduction where the gluteal tendons are vulnerable to compressive loading against the greater trochanter below and iliotibial band above (e.g. sitting cross legged, standing ‘hanging on one hip’, sleeping on the affected side or the unaffected side with the affected limb crossing into hip adduction) or dynamic adduction during gait [14]. The latter is thought to be best achieved by including (1) functional weight bearing hip abductor muscle exercises (e.g. bridging, squat, side-stepping) focusing on pelvic alignment control in the frontal and transverse planes double to single leg loading and by focusing on (2) hip abductor strengthening exercises to address muscle weakness and increase loading capacity of the gluteal tendons [15] (e.g. side-stepping with band, reformer based sliders). A fundamental principle in tendinopathy management which must be applied in this exercise prescription context is that of progressive graduated overload to enable tendon remodelling and adaptation [14]. It is essential that exercise difficulty is gradually increased as tolerated to ensure optimal muscle activation to enable gains in muscle strength and function without significant aggravation of pain. Finally specific to the context of GT, (3) motor control of the entire hip abductor muscle mechanism thought to be important to reduce overactivity of tensor fascia lata (and subsequent ITB tension) relative to the deeper segments of the gluteus minimus and medius muscles [29] to facilitate gluteal tendon tensile loading and avoid compressive loads, known to be detrimental to tendon health. Patient tactile feedback over the tensor fascia lata and gluteal muscles is thought to be a useful clinical strategy to address this goal [14].

A recent clinical trial demonstrated that a progressive exercise program incorporating functional training, targeted strengthening and dynamic motor control of the pelvis, delivered with patient education over 8-weeks under supervision of a physiotherapist, was superior to a wait-and-see approach or corticosteroid injection [27]. These results are promising and contribute to the body of evidence for treatment of GT. Importantly, they also add to the contemporary conversation that emphasises the need for patient education in management. As outlined, it is evident that hip abductor muscle strength, biomechanical and neuromuscular patterns be considered in the assessment and management of GT. However, data from individuals with GT highlights that the kinematic presentation of GT is heterogeneous [19, 20]. Thus a ‘one size fits all’ approach to assessment and management
is unlikely to be effective. Clinicians should evaluate patients who present with GT with respect to specific biomechanical and neuromuscular impairments, and tailor treatment and load modification based on the principles of tendinopathy treatment.

3. Femoroacetabular impingement syndrome

FAI syndrome is a motion related condition of the hip joint and is associated with hip pain and impaired function in younger active adults [30]. FAI is characterised by abnormally shaped hip bones (i.e. head of femur and/or acetabulum), which can lead to mechanical impingement during movement [30]. Repetitive mechanical impingement is thought to lead to chondral stresses that cause irreversible structural pathology [31]. FAI syndrome is considered a principal determinant of future development of hip osteoarthritis [32].

3.1 Biomechanical considerations in femoroacetabular impingement syndrome

3.1.1 Hip joint biomechanics

Evidence for altered hip joint biomechanics during movement in individuals with FAI syndrome is mounting [33]. Gait has been well studied in this population. Findings from systematic reviews [31, 33] and empirical studies provide moderate evidence for less sagittal plane hip range of motion (ROM) [34], primarily driven by a lower peak hip extension angle [35], during gait in individuals with FAI syndrome compared to healthy controls. Lower peak hip internal rotation angle [35, 36] and lower peak hip external rotation joint torque [35] have also be reported during stance in FAI syndrome compared to healthy controls. However, the biomechanical adaptations exhibited by individuals with FAI syndrome during gait are generally small on average, and consequently of uncertain clinical significance.

Hip joint biomechanics during squatting [37–39] in FAI syndrome also differs only subtly from individuals without pain or FAI morphology. Though some studies report that individuals with FAI syndrome are unable to squat as deep as controls [37, 39], hip flexion range is not significantly reduced during task completion [37–39]. Individuals with FAI syndrome place the hip in a more adducted position during squatting [38] and step ascent [40], which may be secondary to hip abductor muscle weakness commonly reported in FAI syndrome cohorts [41]. Biomechanical comparisons during these more demanding tasks targeting positions of impingement (i.e. squatting and step ascent) have extended knowledge regarding altered hip joint biomechanics in individuals with FAI syndrome. Nevertheless, the implications of these alterations, including any relationship with pain and/or function and/or joint structure remain unclear.

3.1.2 Biomechanics of adjacent segments

Individual variation in movement strategy and interaction between adjacent body segments (i.e. pelvis, trunk) may account for the small between-group differences observed in hip joint biomechanics when comparing individuals with FAI syndrome to healthy controls. Failure to consider such factors may explain the modest effects of conservative treatment [42] and the unrestored hip function observed post-operatively [36]. Reduced sagittal plane pelvic range of motion has been identified during squatting in FAI syndrome compared to healthy controls.
[39], and has been proposed as a risk factor for symptom presentation [43]. Greater anterior pelvic tilt has also been reported in FAI syndrome during squatting [37] and step descent [44] compared to healthy controls. This biomechanical alteration may be counterproductive for pathology since an increase in anterior pelvic position will promote hip flexion and thus impingement.

Few studies have considered pelvic and trunk control in the frontal plane despite the implications for hip joint loading [45]. Control of frontal plane pelvic alignment during single leg support is necessary to prevent movement into impingement. Pain and/or hip abductor muscle weakness, both features of FAI syndrome, could hinder control of the pelvis in the frontal plane. On the other hand, altered frontal plane control of the trunk may moderate provocative hip joint contact forces (i.e. reduced demand on hip abductor muscles), and has been observed in cohorts with hip osteoarthritis [22, 46]. Recent findings from a step ascent task corroborate that control of adjacent segments may play an important role in symptom management in FAI syndrome [40]. When individuals with FAI syndrome were sub-grouped based on trunk and pelvis dominant strategies, those who exhibited lateral trunk lean and maintained neutral pelvis alignment reported no pain and prevented the hip from moving towards an impinging position. It is reasonable to suggest that this strategy may alleviate load on the abnormal hip joint structures. In direct contrast to this, 86% of participants who exhibited poor pelvis control, inherently moving the hip into an impinging position, reported moderate levels of pain [40]. Control of—and interaction between—adjacent body segments may play an important role in symptomatic and structural preservation or deterioration in FAI syndrome. Further, altered hip joint function remains unresolved post-operatively [36], suggesting that a hip-only treatment focus may be misguided. Functional biomechanics is modifiable, and could be changed by conservative interventions and rehabilitation programs [47].

3.1.3 Patient subgrouping

FAI syndrome is a complex condition [48] with no common pathological pathway [30]. Patient presentation is heterogeneous, which may explain the modest treatment effects [42]. Different biomechanical strategies are used by separate subgroups of participants to perform a task [38, 49], albeit some more advantageous than others for symptoms and function. As with established hip OA [50], no conservative treatment is likely to be effective for all individuals with FAI syndrome. Maximum efficacy will only be attained with interventions catered to the individual. More research must be done to improve understanding of the patient-specific biomechanical alterations associated with FAI syndrome in order to better manage the disease and its consequences.

3.1.4 Implications for joint structure

Biomechanical alterations in individuals with FAI syndrome are subtle but may relate to enhanced protection for the hip, albeit with possible long-term consequences. It comes as no surprise that individuals with FAI syndrome exhibit less prominent biomechanical alterations than individuals with structural damage and hip OA [51]. Individuals with FAI syndrome have less severe morphological deformities and accordingly, exhibit more subtle biomechanical modifications. The absence of longitudinal studies means that it is not known whether these small biomechanical alterations are precursors to the larger deviations observed in those with established hip OA.
3.2 Non-surgical management for femoroacetabular impingement syndrome

Arthroscopic hip surgery is the most common treatment for FAI syndrome [52]. Despite a dramatic upsurge in the number of surgeries performed over the past decade [52], surgical intervention for FAI syndrome does not completely restore hip joint function to that of healthy controls [36] or uniformly improve pain [53], despite correction of the hip's bony abnormalities. This may be because surgery corrects the local mechanical issue (i.e. correction of the bony abnormalities until impingement free motion is obtained), but without resolution of the altered movement strategies adopted pre-operatively.

Findings from the only large randomised controlled trial comparing hip arthroscopy and best conservative care for the treatment of FAI syndrome support the short-term efficacy of arthroscopic hip surgery [54]. However, patients in both groups reported significant improvements in hip-related quality of life at 12-months, and the costs associated with surgery were higher than with conservative care [54]. Non-surgical treatments for FAI syndrome, such as exercise, activity adaptation and education, are globally recommended [48], and attractive given the relatively low harmful risks and associated costs. Identification of non-surgical interventions to reduce the burden of hip OA in its early stages, including FAI syndrome, is an important public health priority [55]. At present, conservative treatment effects for FAI syndrome are also modest [42, 56], likely due, in part, to a lack of understanding regarding the underlying mechanisms associated with clinical and structural decline.

3.2.1 Conservative care

Theoretically, an adequately designed, evidence-based, appropriately administered conservative management program may have the potential to alleviate symptoms, and in turn prevent disease progression, thus postponing or negating the need for surgery [56]. Current clinical practice entails combinations of physiotherapist-led rehabilitation, education, and activity modification for the management of FAI syndrome [48, 56]. There is little evidence from randomised control trials to guide conservative care for FAI syndrome, which means that conservative treatments are largely based on clinical theory and/or extrapolation of evidence from other clinical conditions.

Potential targets for conservative treatment include the abnormal movement patterns and hip muscle weakness seen in patients with FAI syndrome [31]. Gait assessment alone is unlikely to provide clear information to guide treatment of FAI syndrome. However, the biomechanical alterations at the hip joint and adjacent segments apparent during more demanding tasks (e.g. squatting, step ascent and descent) may be relevant in the clinical management of this patient population. Altered movement patterns in the form of altered hip joint biomechanics have been identified during tasks with similar demands in these patients post-operatively [36]. Pre-operative treatments addressing these biomechanical abnormalities may also have scope to improve surgical outcomes.

Retraining of deep hip muscle function (e.g. quadratus femoris, obturator internus) is a common objective of non-operative management [57] and post-operative rehabilitation [58] for FAI syndrome. Conservative care commonly targets deep hip external rotator muscle strengthening and neuromuscular retraining with the aim of improving dynamic hip joint stability [57]. Although experimental evidence suggests that activation of these deep muscles may contribute to dynamic stability in a healthy hip [59], it is less clear if adaptations in neuromuscular control are associated with FAI syndrome. Cross-sectional data acquired during gait provide
preliminary evidence of the extent and nature of FAI-related changes to deep hip muscle activation [49]. However, an improved understanding of deep hip muscle function during more demanding, provocative tasks is needed to provide a comprehensive recommendation for retraining.

Hip strength assessment may be important in the clinical management of FAI syndrome. Evaluation of agonist/antagonist and/or between-limb strength ratios could be particularly beneficial clinically, as body size normalisation and control normative data for individual movement directions are not required. Reduced abduction strength in FAI patients [41] may have important implications as the abductor muscles control the position of the pelvis relative to the femur [60]. This is critical to prevent movement (i.e. contralateral pelvic drop) into a position that impinges the hip joint during single leg weight bearing tasks, such as those commonly required in sport where FAI syndrome has been identified (e.g. soccer, dancing, football) [61]. Treatment programs targeting the primary abductor muscles may improve pelvic-femoral stability during single leg task performance in individuals with FAI syndrome, though any implications of such treatments for symptoms and joint structure are not yet clear.

3.2.2 Optimising treatment

Clinical interventions to restore normal musculoskeletal function around the hip joint may be beneficial, but future research is needed to determine whether these features can and should be changed, and whether this improves outcomes. Cam impingement has been proposed as a modifiable risk factor for hip OA [32]. Optimising treatments relies on the identification of novel treatment targets to slow femoral lesion progression and prolong the development of structural damage and early hip OA.

A critical step in the clinical management of individuals with FAI syndrome is to identify which biomechanical and neuromuscular features are: (i) positive and should be encouraged; (ii) negative and should be discouraged; and (iii) potentially positive prior to surgery to compensate for the abnormal morphology but should be a target for treatment following surgery to prevent further impairments. It would be precipitous to categorise these features without the support of longitudinal data. Nevertheless, it is abundantly clear that the widespread implications for FAI-related clinical practice depend on the appropriate classification of any modifiable targets for treatment.

The evaluation of conservative management programs, that include a range of techniques to modify joint motion/loading/function such as joint mobilisation techniques, hip bracing, and targeted exercise programs (including range of motion, strengthening, and/or neuromuscular retraining) are required on a range of outcomes in FAI syndrome (including any modifiable risk factors). Though the evidence underpinning these treatments is still in its infancy, the development of conservative treatments, including post-operative rehabilitation strategies and pre-operative training programs that aim to improve surgical outcomes, is a critical component as we move towards improving treatment outcomes.

4. Hip osteoarthritis

Hip OA is a major public health problem and affects one in four adults over their lifetime [62]. The condition substantially impairs quality of life and causes pain and physical dysfunction. Around the world, hip replacement surgery for hip OA is on the rise, and the burden of OA on society and health care cost will continue to rise.
due to the ageing population and escalation in obesity rates [63]. Therefore, treatments that reduce symptoms and delay the need for joint replacement are critical.

4.1 Biomechanical considerations in hip osteoarthritis

Kinematic and kinetic alterations are reported in people with hip OA compared to healthy controls. There is marked interest in hip joint loading as a culprit for disease progression, arguably due to the evidence in knee OA. Higher knee joint loading has been implicated in structural joint degeneration in middle-aged people at risk of early knee OA [64] and in individuals with established with knee OA [65]. However, few longitudinal studies have evaluated the association between hip joint biomechanics during gait and alterations in hip joint structure [66, 67]. A 12-month longitudinal study of women concluded that higher cumulative hip joint loading assessed as the number of steps per day, in the frontal plane, was associated with joint space narrowing at the hip joint [67]. However, there is insufficient evidence regarding which direction of loading magnitude change is detrimental for joint health (i.e. under- or over-loading). Recent investigations have highlighted the effect of sex, stage of disease and symptom severity on measures of joint loading, as well as the intricate relationships between these measures and hip joint load. Similar to other hip pathologies, hip OA is a heterogeneous disease, and exploration of patient and disease characteristics are needed to better understand moderators of hip joint load. For the clinician, ‘joint loading’ is not examinable or visible in the clinical setting. However, the trunk and pelvis, together are major contributors to the centre of mass, the position of which (relative to the hip joint centre) influences hip joint loading. Thus, visual examination of trunk and pelvic kinematics during functional tasks is an important part of assessment.

4.1.1 Sex and joint loading

Measures of frontal plane loading appear to be dependent on sex. For example, in disease-free individuals the external hip adduction moment is typically greater in females as compared to males [68]. Between-sex differences in anatomy may explain these differences, at least in part. Females typically have a wider pelvis than males [69], which inherently increases the lateral distance of the centre of mass from the hip joint centre, and thus increases the hip adduction moment. However, any underlying anatomical differences appear secondary to disease stage when explaining difference in frontal plane moments. A series of cross-sectional studies indicate that between-sex differences in frontal plane loading are apparent in those with unilateral mild-to-moderate hip OA [70], while measures of hip joint loading are not different between men and women with end-stage hip OA [68]. The indirect effect of sex on hip joint loading earlier in the disease process was also detected in meta-regression analysis of a systematic review. Studies with a greater proportion of men demonstrated a greater average standardised mean difference for reduced frontal plane loading between people with hip OA compared to controls [71]. Given that loading may be relevant for disease progression, it may be clinically pertinent to consider sex-specific interventions for hip OA.

4.1.2 Stage of disease and joint loading

Measures of hip joint loading are also dependent on disease severity. A recent systematic review and meta-analysis of 13 studies suggests that people with hip OA appear to underload compared to controls [72]. Moreover, the sub-group analysis indicates that people awaiting total hip replacement (i.e. greater disease
severity) underload the hip joint compared to controls; whereas people with less severe disease have comparable measures of joint loading in the sagittal and frontal plane [73]. These observations are consistent with empirical investigations determining the influence of disease severity on measures of hip joint loading [71]. Understanding the effect of joint loading on joint structure and symptoms is imperative to guide conservative hip OA management.

4.1.3 Relevance of pain and symptoms

Slow walking speed is a risk factor for mortality [74] and chronic functional limitation in older adults [75]. A systematic review of 17 studies estimated that people with hip OA have a self-selected walking speed of 0.95 m/s, a markedly 26% slower than controls [76]. In light of critical walking speed estimates of 1.0 m/s [72], the observation that people with hip OA walk slower than critical walking speed estimates is alarming. Slower walking speed can be attributable to symptoms and a reduction in stride length in people with hip OA [76]. However, a recent cross-sectional study [73] investigating people with moderate radiographic hip OA with and without symptoms found that irrespective of symptoms, people with radiographic hip OA walk slower than disease-free individuals. These data question symptoms as a cause for reduced walking speed and instead, appear to reflect a longer-term adaptation hip joint degeneration. In addition to being an important marker of function [74, 75], walking speed also influences measures of hip joint loading. Investigators grapple with understanding whether alteration in measures of joint loading are predominately reflections of alterations in walking speed [77]. It appears that in addition to slower walking speed, neuromuscular adaptations are likely to underpin the reduction in hip joint loading in individuals with hip OA.

Evidence regarding pain during walking and how it influences movement strategies is emerging in OA literature [78]. In hip OA, the overall evidence supports the contention that people with hip OA, particularly at end-stage of the disease, underload during walking compared to controls [79]. The premise being that symptoms potentially cause people to walk slower. However, recent cross-sectional findings refute this logic [72], highlighting the complexities between symptoms and joint loading. In a study of people with unilateral mild-to-moderate radiographic hip OA, those who reported moderate pain during walking had higher frontal plane joint loading compared to people who reported less pain during walking [72]. These data suggest that people with mild or no pain during walking modified their gait biomechanics to exert lower frontal hip joint loading. Evidently, the relationship between symptoms and joint loading is intricate.

4.2 Conservative management for hip osteoarthritis

“What can I do myself to decrease OA symptoms and prevent the OA from getting worse?” These were prioritised as the most important questions by patients and health professionals in relation to hip and knee OA [80]. Treatments to reduce hip OA symptoms and delay the need for joint replacement are critical. Joint replacement is costly and is only reserved for end-stage disease when non-surgical treatments are no longer effective. Current clinical guidelines [55, 81, 82], including the recent update by the Royal Australian College of General Practitioners [83], emphasise that a healthy lifestyle consisting of regular exercise and weight management are the core management strategies for hip OA. Interestingly, there are no clinical trials for weight management in people with hip OA [83], and consequently the subsequent overview explores evidence for exercise in these individuals.
Exercise is advised for all people with hip OA irrespective of age, disease severity, symptoms and co-morbidities [81]. A recent meta-analyses in people with hip OA identified 12 RCTs and showed small-to-modest beneficial effects of exercise on pain (standardised mean difference [SMD] \(-0.28, 95\% \text{ CI}: -0.45 \text{ to } -0.10\)) and physical function \((-0.34 \text{ SMD}, 95\% \text{ CI}: -0.50 \text{ to } -0.18\)) compared to no exercise [84]. Notably, two trials including 154 people scheduled for total hip replacement [85, 86], had large improvements in pain \((-0.63 \text{ SMD}, 95\% \text{ CI}: -0.95 \text{ to } -0.30\)) and physical function \((-0.71 \text{ SMD}, 95\% \text{ CI}: -1.04 \text{ to } -0.39\) following 8–10 weeks of exercise. In addition to beneficial effects on symptoms, exercise can potentially delay total hip replacement. A long-term follow-up of a clinical trial found that exercise combined with patient education can potentially reduce the need for total hip replacement by 44% in people with hip OA [87].

Evidence strongly supports the use of exercise as treatment for hip OA symptoms and can potentially prevent disease progression. In line with high quality evidence and clinical guidelines, physiotherapists in the UK [88] and Australia [89] typically recommend exercise in the management of hip OA. However, knowledge on the specifics of exercise prescription is a recognised barrier to exercise uptake [90]. Reintahl [91] eloquently likens exercise prescription to drug prescription. For example, the physician determines the type of medication, the amount or intensity, the frequency of intake and the duration of use. Exercise prescription typically follows the frequency, intensity, type, time, volume and progression (FITT-VP) principles [92], but evidence on best exercise prescription is lacking for treatment of hip OA symptoms. Below, we provide an update on the current evidence for dosage and type of exercise.

### 4.2.1 Exercise dosage

Meta-analyses from trials with high compliance to the American College of Sports Medicine (ACSM) exercise guidelines with respect to dosage was \(-0.42 \text{ SMD} (95\% \text{ CI}: -0.58 \text{ to } -0.26)\) for pain, and studies with uncertain compliance to ACSM dosage was \(-0.05 \text{ SMD} (95\% \text{ CI}: -0.35 \text{ to } -0.25)\) for pain. Improvement in physical function of \(-0.41 \text{ SMD} (95\% \text{ CI}: -0.58, -0.24)\) was comparable to pain in trials with high compliance to the ACSM dosage guidelines while effect from trials with uncertain compliance was \(-0.23 \text{ SMD} (95\% \text{ CI}: -0.52, 0.06)\) [84]. These data support the prescription of exercise in accordance with ACSM guidelines, particularly in relation to pain. A Cochrane review revealed that patients with OA are confused about their cause of pain, and they do not know what they should and should not do, and as a consequence, they avoid activity for fear of causing harm [93]. Collectively, health professionals can use existing evidence to reassure patients about the value of exercise to safely manage symptoms.

### 4.2.2 Exercise type

All clinical trials to date include lower-limb strengthening [85, 86, 94–103], which is unsurprising given that hip and knee muscle weakness is widely established in people with hip OA [104]. However, only a few clinical trials in people with hip OA include aerobic exercise [96, 101, 103]. People with hip OA often present with co-morbidities, such as poor cardiovascular fitness and low psychological well-being, and these are associated with greater hip OA symptom severity [105, 106]. Aerobic exercise and muscle strengthening exercise address different impairments associated with hip OA symptoms and the adaptations people experience are distinctly different for each exercise type. Aerobic exercise may enhance the effects of strengthening exercise on hip OA symptoms by targeting...
cardiovascular fitness and psychological well-being [107]. In our own analysis, pain and physical function scores before and after exercise interventions in people with mild-to-moderate hip OA were sourced through publications and direct author contact. Changes in pain and physical function in studies that used a combination of aerobic and strengthening exercise are compared to those studies that used strengthening exercise only (Figure 3). This preliminary comparison provides support that greater effects on hip OA-related pain and physical dysfunction occur when a combination of aerobic and strengthening exercise is prescribed rather than strengthening exercise alone (Figure 3). Despite the clear rationale to support the premise that a combination of aerobic and muscle strengthening exercise could be more beneficial for hip OA symptoms than either exercise on its own, no clinical trials have directly tested this hypothesis.

5. Outcome measures

Measuring patient-specific outcomes following an intervention or over a course of care is important for clinical research and best evidence-based practice. Outcomes that are most meaningful from the patient’s perspective, such as those that measure symptoms of pain and physical function during activities of daily living, are imperative [108, 109]. Other outcomes of impairments, such as strength, flexibility, range of motion are also important for clinicians and researchers to assess and monitor, but are more often used for clinical differential diagnosis or
prognosis and are usually secondary outcome measures to pain and physical function [109–111].

Measurement of pain and physical function are complex and cover multiple dimensions. For example, pain can be measured in multiple contexts including intensity, duration, type and location. Physical functioning can not only be measured in many contexts but it also crosses multiple domains. According to the International Classification of Functioning, Disability and Health (ICF), physical function spans body functions and structure, activity and participation domains [112].

Many outcome tools for pain and physical function have been described for hip conditions and a selection of tools with the best level of measurement evidence is recommended [109]. Ideally, measure outcomes should be suitably valid, reliable and responsive to change. Known values of the minimum important difference (MID) are important for interpreting meaningful change and are useful to help set individual targets and goals with patients [113].

Patient outcomes can be measured using patient-reported outcome measures (PROMs) and performance-based tests measured by the clinician/researcher. Pain is usually measured with PROMs, such as pain scales and questionnaires, however physical functioning can be measured with both PROMs and performance-based tests. Performance-based tests reflect what patients can do rather than what they think they can do, which is usually captured with PROMs. When assessing physical function, it is recommended that both PROMs and performance-based tests are used as they can encapsulate different information as they test different constructs of function [114].

Patient outcomes can be measured using individual-specific, condition-specific and/or generic outcome tools. There are several condition-generic, individual-specific PROMs that are useful in assessing and monitoring symptoms and function in people with a variety of hip conditions.

5.1 Condition-generic, individual-specific patient-reported outcome measures

The 11-point Numerical Pain Rating Scale (NPRS) can be used to track pain symptoms and can be customised to individual dimensions of pain. For example, average, current or greatest pain in the previous 24-hours or week can be measured ranging from 0 (no pain) to 10 (worst possible pain). Similarly, pain during an activity such as walking can be measured ranging from 0 (no pain on walking) to 10 (severe pain on walking). The MID for the NPRS (scale 0–10) in musculoskeletal conditions ranges from 1.5 points (small change) to 3.5 points (large change) [115] and in hip OA is defined as a change in pain during walking of 1.8 points [116].

The Patient-reported functional scale (PRFS) [117] assesses current level of difficulty associated with 3–5 activities that the individual identifies as being important, each measured on an 11-point scale, where 0 is unable to perform the activity and 10 is able to perform the activity as normal. The MID for the PSFS ranges from 1.3 points (small change) to 2.7 points (large change [115].

Patient-perceived change following an intervention over time can be measured on a Global Rating of Change (GROC) scale, customised to the outcome to be measured, and used by the patient to rate their perceived overall change as worse, no change or better. If worse, the patient is asked to indicate how much worse, from very much worse to slightly worse. If better, then they are asked how much better, from slightly better to very much better. An example is the 11-point GROC [118] with a change scale ranging from −5 to +5. The GROC scale can be very useful to set individual levels of acceptable change over a stated time frame and to set individual treatment goals [119].
There are also several condition-generic PROMs useful for assessing quality of life in a variety of hip conditions. These include the Medical Outcome Study 36 questions short form (SF-36) [120], the EuroQol (EQ-5D) [121] and the Assessment of quality of life (AQOL) [122]. Patient-specific quality of life questionnaires have also been developed for hip OA such as the Osteoarthritis Knee and Hip Quality of Life questionnaire (OAKHQOL) [123].

5.2 Condition-specific outcomes

The following sections will outline condition-specific PROMs and performance-based tests used to measure pain and physical function in the hip conditions outlined previously in this chapter. Outcomes are selected based on available clinical practice guideline recommendations, measurement property evidence and reported use within clinical trials. A summary of the outcomes presented across the three hip conditions including the outcome domains, scoring method, and where known, MID values are provided in Table 1.

5.2.1 Gluteal tendinopathy

A number of valid and reliable measures used in recent clinical trials to measure change in pain and function in patients following corticosteroid injections and exercise [129] and recommended in a systematic review [130] are promising suitable outcomes for people with gluteal tendinopathies. These include the Victorian Institute of Sport Assessment-Gluteal tendon (VISA-G) questionnaire [131] that evaluates the severity of disability using 8 items about current pain and function. Regarding performance-based tests, the single-leg stance test with light fingertip support is useful to assess provocation of pain during a 30-second period. A report of pain over the greater trochanteric region indicates a positive test. This test has excellent sensitivity (100%) and specificity (97.3%), making it an ideal screening out test when pain is negative [2]. Additionally, the pain-free time and the time the patient can maintain a level pelvis in single-leg stance can also be recorded to measure change over time. Other performance-based tests include the single leg squat test where the ability to single leg squat as far as possible 5 times with the non-support leg out front and arms folded across the chest is rated on 5 criteria as good, fair or poor [132] and the star-excursion balance test that evaluates the ability to stand on one leg and reach the other leg into eight directions as far as possible [133].

5.2.2 Femoroacetabular impingement

A number of specifically designed, reliable and well-validated PROMs are recommended for measuring outcomes in people with FAI by an international, multidisciplinary consensus statement endorsed by 25 clinical societies worldwide [48]. The International Hip Outcome Tool (iHOT-33) is a patient-derived questionnaire designed to measure hip-related quality of life in young adults with non-arthritic hip pain over four domains: symptoms and functional limitations; sports and recreational physical activities; job related concerns; and social, emotional and lifestyle concerns [124]. The hip and groin outcome score (HAGOS) was developed for physically active young to middle-aged adults [134] and contains 37 questions, covering six domains of pain; symptoms; physical function in daily living, sport and recreation; participation in physical function, sports and recreation, and hip and/or groin related QOL. The hip outcome score (HOS) [126] was developed to assess treatment outcomes of hip arthroscopy in young to middle-aged individuals and contains 28 questions, covering activities of daily living, and sport.
### Hip Surgeries

<table>
<thead>
<tr>
<th>Condition</th>
<th>Outcome</th>
<th>Items</th>
<th>Scoring</th>
<th>MID</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Patient-reported outcomes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Numeric Pain Rating Scale (NPRS)</td>
<td>All Pain</td>
<td>1</td>
<td>0–10 scale</td>
<td>1.5–3.5 points in musculoskeletal conditions [115]; 1.8 points for hip OA [116]</td>
</tr>
<tr>
<td>Patient-Specific Functional Scale (PSFS)</td>
<td>All Physical function</td>
<td>3–5</td>
<td>0–10 scale</td>
<td>1.3–2.7 points in musculoskeletal conditions [115]</td>
</tr>
<tr>
<td>Global Rating of Change (GROC) Scale</td>
<td>All Change in condition</td>
<td>1</td>
<td>Variable scales e.g. −5 to +5, higher scores indicate improvement</td>
<td>Individualised e.g. moderately better/ somewhat worse</td>
</tr>
<tr>
<td>Victorian Institute of Sport Assessment- Gluteal tendon (VISA-G)</td>
<td>GT Pain, physical function</td>
<td>8</td>
<td>0–100 mm VAS</td>
<td>Between 6 mm [124] and 10 mm [125] in young adults after hip arthroscopy</td>
</tr>
<tr>
<td>International Hip Outcome Tool (iHOT-33)</td>
<td>FAI Pain, physical function, quality of life</td>
<td>33</td>
<td>0–100 mm VAS, where 100 indicates better quality of life score</td>
<td>Less than 10 mm (10%) on each subscale in young adults after hip arthroscopy [125]</td>
</tr>
<tr>
<td>Hip and Groin Outcome Score (HAGOS)</td>
<td>FAI Pain, physical function, quality of life</td>
<td>37</td>
<td>0–100 mm VAS where 100 indicates no problems</td>
<td>5–9 mm for ADL subscale; 6 mm for sports subscale in young adults after arthroscopic surgery [125, 126]</td>
</tr>
<tr>
<td>Hip Outcome Score (HOS)</td>
<td>FAI Physical function</td>
<td>28</td>
<td>0–100 mm VAS</td>
<td>6/68 points on the physical function subscale in people with hip OA [127]</td>
</tr>
<tr>
<td>Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC)</td>
<td>Hip OA Pain, stiffness, physical function</td>
<td>32</td>
<td>0–4 point scale</td>
<td></td>
</tr>
<tr>
<td>Hip disability and Osteoarthritis Outcome Score (HOOS)</td>
<td>Hip OA Pain, physical function, quality of life</td>
<td>40</td>
<td>0–4 point scale</td>
<td></td>
</tr>
</tbody>
</table>
A clear recommendation of which performance-based tests should be used for this condition is yet to be made, however tests that are reliable and best discriminate between individuals with FAI and those without have been described [135].

<table>
<thead>
<tr>
<th>Condition</th>
<th>Outcome</th>
<th>Items</th>
<th>Scoring</th>
<th>MID</th>
</tr>
</thead>
<tbody>
<tr>
<td>30-sec single leg stance</td>
<td>GT Pain, physical function</td>
<td>1 Pain free; hold pelvis level up to 30 s</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Single leg squat test</td>
<td>GT Physical function</td>
<td>1 Rated as good, fair, poor</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Star-exursion balance test</td>
<td>GT Physical function, balance</td>
<td>1 Distance reached in centimetres normalised to leg length with larger distances indicating greater balance and higher physical function</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stair Climb test</td>
<td>All Physical function</td>
<td>1 Faster time in seconds indicates higher level of physical function</td>
<td></td>
<td></td>
</tr>
<tr>
<td>30-sec chair stand test</td>
<td>Hip OA Physical function</td>
<td>1 Great number of repetitions indicates higher level of physical function</td>
<td>2–3 repetitions in people with hip OA [128]</td>
<td></td>
</tr>
<tr>
<td>40-m fast paced walk test</td>
<td>Hip OA Physical function</td>
<td>1 Faster time in seconds or greater speed in metres/second indicates higher level of physical function</td>
<td>0.2–0.3 m/s in people with hip OA [128]</td>
<td></td>
</tr>
<tr>
<td>Timed Up and Go test</td>
<td>Hip OA</td>
<td>1 Faster time in seconds indicates higher level of physical function</td>
<td>0.8–1.4 s in people with hip OA [128]</td>
<td></td>
</tr>
<tr>
<td>6-minute walk test</td>
<td>Hip OA Physical function, aerobic capacity</td>
<td>1 Greater distance covered in metres indicates higher level of physical function and aerobic capacity</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

ADL, activities of daily living; FAI, femoroacetabular impingement; MID, minimum important difference; OA, osteoarthritis; VAS, visual analogue scale.

Table 1. Patient-reported outcome measures and performance-based tests for hip conditions.
includes the 5-times sit-to-stand test where the time taken to transition from sitting to standing from a standard chair five times is recorded in seconds; and the stair ascend test where the time taken to ascend a flight of stairs as quickly as possible without using a handrail is recorded in seconds.

5.2.3 Hip osteoarthritis

Numerous clinical practice guidelines, for example [83, 108, 111], and recommendations, for example [110, 136, 137] informed from high level measurement property evidence and expert consensus strongly recommend a number of condition-specific PROMs. The Western Ontario and McMaster Universities Osteoarthritis (WOMAC) Index [138] measures pain, stiffness and physical function The Hip disability and Osteoarthritis Outcome Score (HOOS) [139] consists of five subscales; pain, other symptoms, function in daily living, function in sport and recreation, and hip related quality of life. This scale incorporates items from the WOMAC scale so can also be extracted from this questionnaire.

The Osteoarthritis Research Society International (OARSI) recommend performance-based measures of physical function representing typical activities relevant to individuals diagnosed with hip or knee OA [136, 137]. Comprehensive descriptions, including set up, equipment, preparation (environment, participant, and tester), procedures, verbal instructions and scoring are available on the OARSI website: http://oarsi.org/research/physical-performance-measures along with videos of each recommended test. The full set includes five tests and the first three were recommended as the minimum core set: (i) 30 s chair stand test where the number of full stands a person can perform in a 30 s period is recorded in seconds; (ii) 40 m fast-paced walk test where the time taken to walk 4 × 10 m as quickly but as safely as possible is recorded in seconds which can be converted to speed recorded in metres per second; (iii) stair climb test where the time taken to ascend and descend a flight of stairs (with optional use of handrail) is recorded in seconds; (iv) timed up and go where the time taken to stand up from a standard chair with arm-rests, walk at regular pace to a line 3 m away, turn around and return to the seated position is recorded in seconds; and (v) six-minute walk test where the maximum possible distance walked in 6 min is recorded in metres covered.

6. Conclusion

Evidence supports exercise as a promising solution to the most important questions asked by patients with extra- and intra-articular hip pathologies and health professionals. Exercise can reduce hip symptoms and potentially prevent disease progression. Stakeholders, including but not limited to, health care professionals, research communities, consumer organisations, and local and national policy makers must make a deliberate effort to translate the positive message of exercise as a treatment for hip conditions. Research is ongoing to further empower patients and clinicians with evidence around best-prescription for exercise.
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