# Abductor Deficiency in Total Hip Arthroplasty: Anatomy, Diagnosis, and Treatment Strategies

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Abductor deficiency following total hip arthroplasty is a rare and complex problem facing reconstructive surgeons today. The appropriate management is contingent on a thorough knowledge of the clinical anatomy of the abductor mechanism and its potential pathology during and after total hip arthroplasty. Throughout this review we will highlight the relevant clinical anatomy, diagnostic modalities, and various surgical options to aid in the appropriate management of this difficult problem.
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## Introduction

The abductor mechanism of the hip is composed of 3 primary muscles; Tensor Fascia Lata (TFL), gluteus medius, and gluteus minimus. These muscles have crucial roles in not only hip abduction but pelvis biomechanics, gait, and stability of the hip joint. Dysfunction of the abductor mechanism can contributes to pain, weakness, and decreased function of the hip joint.[1] Greater trochanter pain syndrome is a spectrum of disorders ranging from dysfunction of the abductor mechanism to frank tears of the abductor tendons without history of any prior surgery (Fig. 1).[2-5] It has been reported that abductor tendon tears have been found up to 25% incidentally in patients at time of primary THA and has been reported to be present in up to 70% in elderly women undergoing primary THA.[6]

Alternatively, the incidence of abductor deficiency following THA has been reported up to 22% in the literature regardless of approach.[7] Abductor dysfunction after total hip arthroplasty is a different clinical entity than preoperative or incidental tears and is usually caused iatrogenically either by inadvertent intraoperative damage to the superior gluteal nerve, failure of repair or rupture of the abductor tendon insertion from the greater trochanter, implant loosening, or lysis of the greater trochanter.[8-13]

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Regardless of etiology, the diagnosis and management of abductor dysfunction after arthroplasty is a difficult problem for the treating reconstructive surgeon and leads to poor functional outcomes for the patient. Thus, the following sections will give an overview of the pertinent clinical anatomy, diagnostic tools, and evolving
surgical treatment strategies for symptomatic abductor deficiency following total hip arthroplasty.

## Anatomy

The gluteus medius, gluteus minimus, and tensor fascia lata (TFL) are the main muscles that comprise the abductor mechanism of the hip. These muscles are the primary contributors to hip abduction and internal rotation. All 3 muscles are innervated by the superior gluteal nerve, composed of L4-S1 nerve roots of the sacral plexus. During normal gait, the abductor mechanism is responsible for maintaining a level pelvis during the single-leg stance phase of gait by counteracting the downward pull of the body's center of mass. Additionally, in the arthroplasty setting the abductor mechanism, specifically, the gluteus medius is a major contributor to stability of the implanted prosthesis. Dysfunction of these muscles postoperatively can lead to various complications ranging from pain to recurrent dislocations. The following sections will briefly discuss the relevant clinical anatomy of the abductor muscles as well as possible surgical approaches that may impact their integrity in the setting of hip arthroplasty.
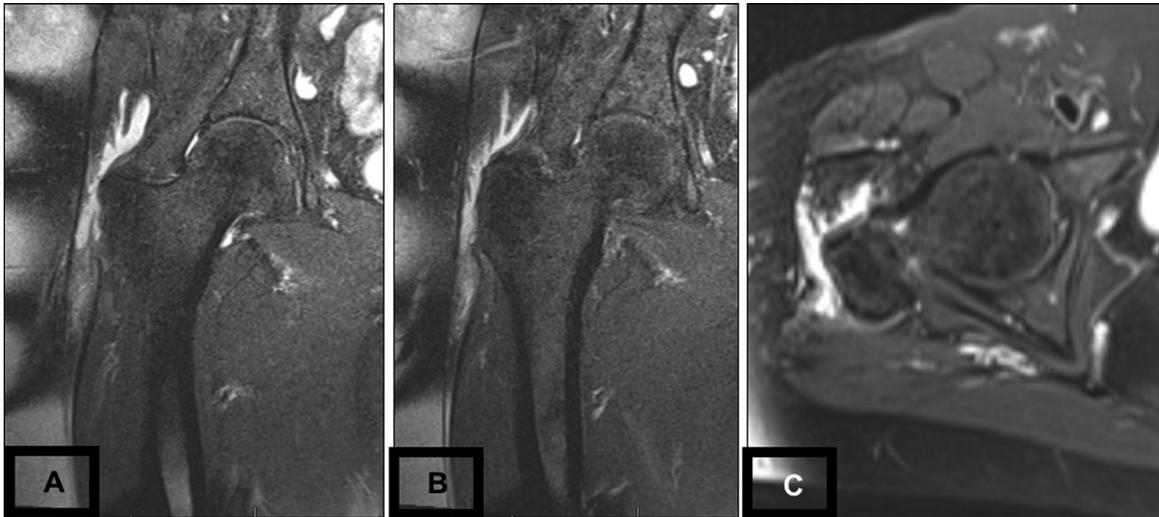
**Figure 1** Abductor deficiency in the native hip. Coronal and axial T2-weighted MRI of the native right hip in a symptomatic 72-year-old female presenting with chronic hip pain and weakness. Coronal and axial MR imaging demonstrate right gluteus medius and minimus insertional tears with substantial retraction and a fluid-filled gap extending to the subtrochanteric region.

## Gluteus Medius

The gluteus medius muscle is a large fan-shaped muscle that curves from the anterior superior iliac spine back to the posterior superior iliac spine with attachments on the outer edge of the iliac crest. The deep fascicles of the muscle originate from the gluteal fossa of the ilium, spanning from the posterior sacroiliac ligaments to the anterior superior iliac spine. The more superficial fibers of the gluteus medius originate from the gluteal aponeurosis which is a fibrous band of tissue that lies between the superior border of the gluteus maximus and confluent with the tensor fascia lata (TFL). Additionally, there is a smaller discrete origin that is limited to the posteroinferior lip of the iliac crest. The Gluteus medius is separated from the deep iliac periosteum by a loose fibrous layer.[14] The gluteus medius has distinct anterior, middle and posterior portions with associated fascicles, all of which are innervated by distinct branches of the superior gluteal nerve.[15] The anterior and middle portions of the muscle are vertically oriented and allow a vertical pull from the greater trochanter and are the primary muscles that initiate hip abduction.[15] The posterior portion of the gluteus medius runs more oblique and is parallel to the femoral neck, stabilizing the hip joint in gait from heel strike to full stance.[16]

The insertion of the gluteus medius tendon onto the greater trochanter is complex. Anatomic studies have shown that there are 2 distinct attachment sites on the greater trochanter. The posterior gluteus medius fibers attach to the superior-posterior facet. The central and anterior portions of the tendon insert into the lateral facet.[17] Anterior portions of the gluteus medius are thought to be more active during hip flexion and internal rotation, whereas posterior fibers have larger contributions to extension and external rotation. However, all of the portions of the gluteus medius contribute to hip abduction.[18]

## Gluteus Minimus

The gluteus minimus is also fan-shaped albeit smaller muscle that works in conjunction with the gluteus medius as a hip abductor. The minimus originates from the surface of the ilium between the anterior and interior gluteal lines inserting onto the anterosuperior border of the greater trochanter. The origin extends from the anterosuperior edge of the greater sciatic foramen posteriorly to the anterior ilium between the anterior supererior and inferior spines. A small portion of the fibers insert directly into the anterior and superior aspects of the hip capsule. Recent studies have shown that the minimus itself like the medius is segmented with specific superior gluteal nerve branches to each segment.[19] The posterior part of the minimus is also parallel to the femoral neck of the femur, and contributes more so to stabilization of the femoral head in the acetabulum during the gait cycle.[19,20] The anterior portion of the gluteus minimus is vertical and aids in initiation of hip abduction, which is then in turn completed by the TFL and gluteus medius.[19]

## Tensor Fascia Lata

The tensor fascaia lata (TFL) is the third hip abductor discussed and plays a vital role in native hip stability. Additionally, the TFL is often used in the surgical management of hip abductor deficiency following THA. The TFL lies superior to the anterior portion of the gluteus medius and is surrounded by a superficial and deep layer of fascia lata that encapsulates the TFL muscle belly. The TFL arises from the most anterior part of the outer lip of the iliac crest with its fibers running distally and inserting onto the fascia lata just below and anterior to the greater trochanter. The fascia lata ultimately becomes the iliotibial band and inserts distally on the proximal aspect of the tibia. The tensor fascia lata is one of the major muscles counter-balancing the forces of body weight

during the stance phase of the gait cycle,[19,21] and is a primary hip abductor.

## Superior Gluteal Nerve

The superior gluteal nerve innervates all 3 of the abductor muscles and knowledge of its specific anatomy is vital for reconstructive surgeons regardless of their preferred approach to the hip. The superior gluteal nerve is derived from the posterior branches of the fourth and fifth lumber and first sacral spinal nerves and comprises only motor fibers. It runs with the superior gluteal artery and vein forming a neurovascular bundle that exits the greater sciatic notch, exiting superior to the piriformis muscle, continuing anteriorly between gluteus medius and minimus.[22] There have been variable reports in the literature with regard to the specific anatomic patterns of the SGN.[23] However, the nerve is most commonly described as branching into a superior and inferior portion once it exits the greater sciatic foramen, with the superior rami innervating the gluteus medius (less commonly the minimus), and the inferior rami innervating the gluteus medius, minimus, and tensor fascia lata.[24]

Damage to the SGN during total hip arthroplasty is most frequently caused by traction or direct trauma to the nerve intraoperatively. Risk of damage to the SGN is variable depending on the specific approach utilized by the treating surgeon. The most commonly discussed risk is direct injury during the lateral or anterolateral approach to the hip thought to be due to the proximal extension of the gluteus medius split. Multiple authors have reported on the superior gluteal 'safe zone' and state that the proximal extension of the gluteus medius split during these approaches should be limited to 3-5 cm above the greater the trochanter.[23,25,26] However, patient specific anatomy including patient height needs to be taken into consideration when determining how far proximal to extend the transgluteal split as the 'safe zone' can differ depending on specific patient factors.[22] Furthermore, risk of damage to the SGN is not limited to lateral approaches to the hip. Although rare, damage of the SGN, specifically the innervation of the TFL, has been reported in the literature during the anterior approach. This can be seen with too proximal ligation or coagulation of the ascending branches of the lateral circumflex artery as it enters the TFL. Additionally, excessive femoral traction, inappropriate retractor placement or inappropriate proximal extension of the anterior approach has also caused SGN injury.[27] During the posterior approach the SGN becomes in danger as it exits the greater sciatic notch superior to the piriformis muscle. Overall, knowledge of the course of the SGN is vital as damage can occur in any approach to the hip if appropriate precaution is not taken.

## Diagnosis

Unsurprisingly, the most reliable method in diagnosis of abductor deficiency in the postoperative setting is a thorough history and physical examination. Generally, patients will endorse deep pain and soreness over the abductor musculature and greater trochanter, particularly after strenuous activity. If an acute abductor tendon rupture occurs, there usually is a history of minor trauma with or without a popping sensation, followed by immediate onset of symptoms. If there is any description of subluxation or history of frank dislocation events, abductor deficiency should always be on the differential. Alternatively, for chronic abductor deficiency patients may present with an asymptomatic period, followed by gradual onset of pain and progressive need of assistive devices. When examining the patient with suspected abductor deficiency, gait, limb length discrepancy, and range of motion should be thoroughly evaluated. Motor strength, specifically abduction strength lying on the contralateral hip needs to be evaluated. Overt weakness, gait abnormalities such as Trendelenburg gait, abductor lurch, and positive Trendelenburg testing should immediately raise concerns for abductor deficiency. Although rare, in some instances in complete disruption one can feel a palpable defect in the abductor tendons over the greater trochanter. This is usually seen in cases of traumatic rupture.[8]

Imaging studies should not be used in isolation, but rather to confirm your clinical suspicion and validate patient symptoms and physical exam as advanced imaging studies may over diagnose asymptomatic abductor deficiency. As always, plain radiographs should be obtained and can lend valuable information with regards to abductor dysfunction. Generally, radiography provides information regarding implant position, leg length, offset, and signs of loosening, which have all been associated with abductor weakness. Decreased offset specifically has been reported as a potential cause of abductor weakness.[28] Additionally, one needs to assess for lysis and/or periprosthetic greater trochanteric fracture.

The use of advanced imaging modalities has also been reported in the literature and may aid in diagnosis. Ultrasound has been experimentally used and in one study using the technique found that 20.6% of patients at 1 year out from THA had abductor tendon tears. Interestingly, only 11.7% of these patients were symptomatic and had positive Trendelenberg testing.[29] Arthrography has also been examined as a diagnostic tool in abductor deficiency and one study showed that 14 of 33 patients testing positively for abductor tear via arthrogram had confirmed intraoperative abductor tears. However, 9 of the 19 patients with negative arthrograms also had known intraoperative tears. Thus, this technique is not recommended as it is not specific and highly invasive.[30]

The most useful advanced imaging tool is metal subtraction MRI (Fig. 2) which has been shown to be a highly sensitive test for abductor tears. In a prospective study one year after THA, authors found a significantly higher incidence of tears in the gluteus medius and minimus in those patients which were symptomatic. They also found that signal change and fatty atrophy of the abductor muscle bellies were associated with more severe functional symptoms.[31] In another prospective study of patients undergoing an anterolateral or modified lateral approach, metal subtraction MRI at 1 year after THA showed a 50% incidence of abductor tendon tears and fatty atrophy, however, these were not consistent with patient physical exam, pain, and clinical outcome scores.[32] Thus, MRI may be an overly sensitive test and should be interpreted within the context of each individual patient.
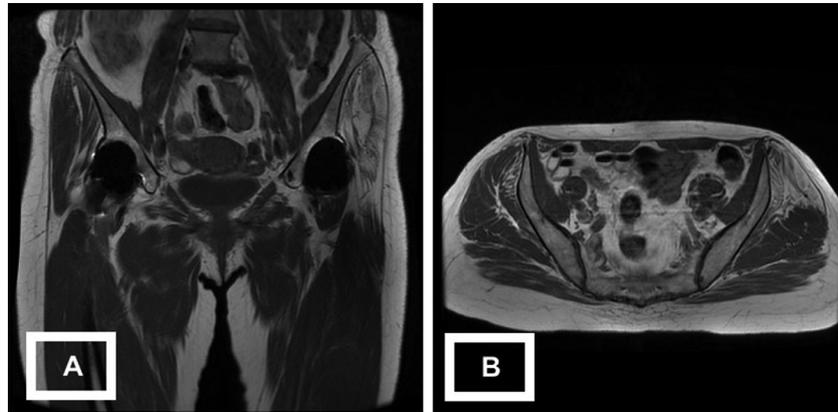
**Figure 2** Metal subtraction MRI 1-year after total hip arthroplasty. Coronal and axial cuts of a T1-weighted MRI with metal subtraction in a patient with significant clinical abductor deficiency 1-year status post total hip arthroplasty. Fatty degeneration within the muscle belly of the abductors is evident on the left.

Lastly, if a superior gluteal nerve palsy as the cause of abductor weakness is suspected, observation should be undertaken as 95% of patients will recover spontaneously by 2 years after THA.[33] Serial EMG may be warranted in these settings to assess recovery and extent of injury.

Overall, diagnosis of abductor deficiency should be based off clinical history and exam, Trendelenburg testing, and review of plain radiography. More advanced diagnostic tests may include MRI, which should be used as a confirmatory test. If the abductor mechanism is known to be intact but SGN palsy is expected than nerve conduction tests should be used through serial EMG to aid in diagnosis and assess recovery.[1]

## Treatment Strategies

The incidence of isolated symptomatic abductor deficiency as an indication for revision surgery is rare and consequently the literature regarding both its operative and non-operative management is lacking. Furthermore, there is no published reports of the natural history of abductor tears following THA in the literature. With that said, there are multiple surgical techniques that exist in the literature with varying results. Surgical treatment strategies include direct tendon repair through intraosseous tunnels, soft tissue muscle transfer, allograft reconstruction, and greater trochanter fixation.

### Direct Repair

To our knowledge there are 3 reports of direct repair of abductor tears or avulsions following THA published in the literature, and thus outcome data is sparse. In all of these reports, the technique for direct repair involved mobilizing the gluteus medius tendon as needed, limiting release to not damage the superior gluteal nerve. Then freshening up the insertion site to create an appropriate healing environment and directly repairing the torn tendons through multiple intraosseous tunnels with heavy nonabsorbable sutures. In all of the reports specific repair was dictated by patient anatomy and also included vastus lateralis and/or tensor fascia

lata advancement as needed. Postoperative protocols varied but largely consisted of a period of protected weight bearing and restricted hip abduction and external rotation. Additionally, some patients underwent acetabular component revision concomitantly at the time of abductor repair for various reasons. Results within these studies were inconsistent at best. In one study, the authors felt that 4 of the 9 patients treated had good or excellent results, whereas 3 of 9 had poor results.[8] In another study, 50% of patients had both a substantial improvement in pain and limp. They also found that results were superior in patients with low BMI (<30) or with early postoperative repair defined as within 1-14 months of index surgery.[11] Unfortunately, that means that the other half of the patients had either poor or fair results. The third study showed that the after direct repair the average pain, limp and strength scores improved significantly; however, re-rupture did occur in 4 of the 12 patients.[9] These authors also showed that preoperative fatty degenerative changes seen on preoperative MRI were associated with poor outcomes. After reviewing this literature, this approach seems to be best served in the physiologically well patient, with low BMI, and an acute traumatic postoperative rupture of the abductor tendons with otherwise stable and appropriately aligned components.

### Soft Tissue Muscle Transfer

Soft tissue advancement as a strategy for abductor deficiency usually involves either the advancement of vastus lateralis or the gluteus maximus (with or without the tensor fascia lata). The technique for vastus lateralis advancement for abductor defects has good results, although with limited numbers.[34,35] In this technique an extensile lateral incision is made above the greater trochanter down to the lateral patellar margin. The iliotibial band is split in line with its fibers, and the entire vastus lateralis is exposed. The vastus lateralis is then mobilized from the intermedius proximally to distally. The attachment to the quad is divided and mobilized while taking care not to damage the neurovascular pedicle found between the rectus femoris and vastus lateralis. Once appropriately

mobilized the proximal vastus is advanced and sutured to the remaining abductors with heavy braided suture in a side to side manner. The distal vastus is then attached to the greater trochanter via transosseous tunnels while the leg is abducted 20 degrees. Postoperative care again includes abduction bracing or splinting with protected weight bearing for a period of time based on surgeon preference. In the 14 patients treated with this technique, 100% of them had improvement in their limp and pain scores. However, one should be mindful of the potential for heterotopic ossification (HO) as one of the patients needed reoperation for scar tissue and HO removal.

Alternatively, soft tissue transfer with a gluteus maximus muscle flap has been described.[36] This technique is obviously reliant on a healthy, robust, functional gluteus maximus muscle, and is of particular use in those patients with chronic avulsions and/or inflammatory destruction of the abductor portions of the gluteus medius and minimus tendons. This technique utilizes a posterior approach to the hip, splitting the gluteus maximus muscle in line with its fibers along approximately half the muscle length. The incision is extended distally, splitting the fascia lata fibers, ending well below the greater trochanter. Next, the anterior gluteus maximus muscle is exposed and the fascia lata anterior the gluteus maximus is split from the top of the muscle to approximately 4 cm below the gluteus maximus attachment in to the fascia. This incision connects to the fascial incision made during initial exposure, leaving a distal isolated fascial flap. After the fascia is cut anteriorly the gluteus maximus muscle is then elevated to form a triangular muscle flap with the base proximally. The lateral cortex of the greater trochanter is then removed with an osteotomy roughly 2 × 3 cm to allow attachment of the anterior muscle flap directly onto the femur. Multiple holes are drilled in the bone. The vastus lateralis is then split and detached from the proximal femur. The hip is then abducted roughly 15 degrees and the muscle flap is sutured on to the greater trochanter with multiple heavy nonabsorbable sutures, pulling the flap distally. The gluteus maximus flap is secured under the vastus lateralis that was detached and once the gluteus maximus is tied down, the vastus lateralis is then sutured back to its original attachment site. The edges of the flaps are then closed using braided absorbable stitch. Postoperative care includes protected weight bearing, restricted abduction, with steady progression of strengthening and weight bearing based on surgeon preference. Overall 16 patients underwent repair with this technique, all but 2 patients were able to abduct against gravity and walked without a limp. One of the 2 patients with poor outcomes had a persistent limp and was unable to abduct against gravity. The other had a periprosthetic greater trochanter fracture after a fall.[37,38]

## Greater Trochanteric Fixation

Isolated greater trochanter fractures after total hip arthroplasty are a rare occurrence. However, when they do occur in the setting of THA they may cause significant pain and abductor dysfunction. The large majority of isolated greater trochanter fractures are treated nonoperatively if the prosthesis remains stable and the fracture is minimally displaced. However, patients with significant functional impairment (ie, abductor weakness), displacement, greater trochanter osteolytic fractures, and greater trochanter nonunions may require surgical intervention. Trochanteric fractures are most commonly seen in patients with poor bone quality, the revision setting, and in hip arthroplasty conversion from previous intertrochanteric fracture fixation. Historically, isolated Vancouver A fractures have been successfully treated conservatively if displaced less than 2.5 cm. Greater than 2.5 cm with associated pain and dysfunction may warrant surgery although results are varied.[39] A number of options exist for fixation of isolated greater trochanter fractures including wiring tension band techniques, cable fixation, claw plates, locking plates, and modular femoral stems with a trochanteric bolt. Technical consideration is usually dependent on whether the implant is loose, there is osteolysis of the greater trochanter, and how displaced the fracture is.

The surgical approach for all the above options is similar. Once the fascia is split in line with the trochanter and the femoral shaft, the trochanteric piece is identified. The trochanteric fracture piece and the fracture bed on the femur or freshened up until healthy bleeding bone is present. The leg is held in abduction between 20 and 30 degrees and the trochanteric piece is reduced and held in place with Kirschner wires for preliminary fixation. Wires are then passed through the trochanter piece, crossed, and passed through bone tunnels made in the femur, or placed around the calcar. The wires are then tensioned appropriately based on manufacturer.

Another option for fixation using the same approach is the laterally based trochanteric claw plate. This is a plate that sits laterally on the trochanter gripping the proximal fragments with claws and distally with teeth that resist proximal migration of the plate. The plates are then fixed with oblique wires allowing compression of the fracture site. In one study, 31 patients were treated with this technique and had promising results. Harris Hip scores increased, there was no evidence of postoperative abductor lurch, and abduction increased from 0 degrees preoperatively to 25 degrees postoperatively. However, there were 6 plate related complications of the 31 hips treated with this technique including infection, painful bursitis requiring plate removal, and nonunion.[40]

Another option for greater trochanter fixation is through revision modular components. Certain modular components allow the use of a trochanteric bolt for trochanteric fixation. Once the final implant has been impacted in place, a guide is secured to the proximal body of the femoral component. The fragment is compressed into place and the hole drilled. The bolt is then inserted to bring down and fix the trochanteric fragment. The surgeon may augment this technique with the use of an additional claw plate. The use of cables may be used if additional stability is desired (Fig. 3).

Abductor deficiency may also lead to significant instability and some authors have advocated for a trochanteric slide advancement for abductor deficiency related instability. These procedures are technically demanding and likely to fail if used in patients with component malpositioning as the cause of instability. Thus, if component positioning has
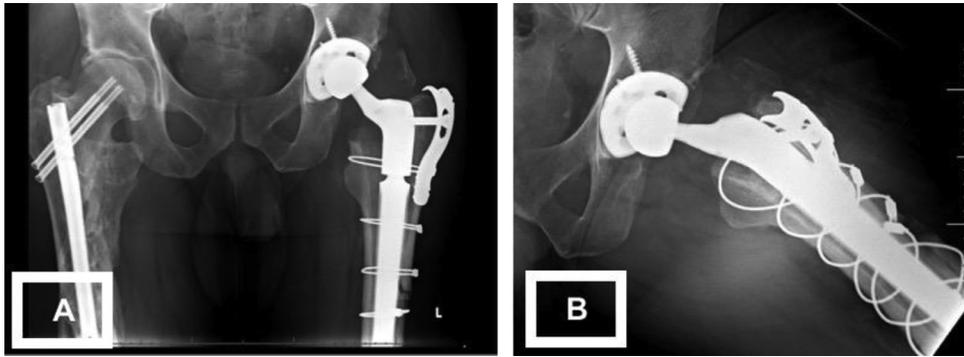
**Figure 3** Radiographs status postrevision left total hip arthroplasty. AP and lateral radiographs status post left revision arthroplasty in a patient who underwent an extended trochanteric osteotomy. The trochanteric segment was fixed with a trochanteric plate that was bolt secured to the revision stem (Arcos, Depuy) for added fixation. AP, anteroposterior.

absolutely been determined to be acceptable, and the patient continues to be unstable, then trochanteric advancement may be a suitable surgical option (Fig. 4). In this technique the insertion of the abductors are identified, and the greater trochanter is carefully marked to ensure no disruption of the abductors themselves. An osteotomy is then undertaken of the greater trochanter and advanced distally to tension the abductors appropriately, this is usually between 1 and 2 cm distally. The claw plate is then placed and fixed as earlier described over the advanced trochanteric piece. In one study, twenty-one patients were treated with this technique and followed for an average of 2.7 years. 17 of the 21 patients had successful outcomes with no further reported dislocations.[41] Therefore, this technique may be an option in patients with instability, intact abductors, and no evidence of component malposition or sources of impingement.

Another rare complication of THA leading to abductor deficiency can be seen with osteolytic fractures of the greater trochanter secondary to polyethylene wear debris after THA.[42] Treatment of these osteolytic related fractures are controversial,

however, some authors argue that fixation at the time of revision THA is necessary.[43] In this technique the fractured trochanter is carefully freed from its fibrous attachments, and the osteolytic lesions in the greater trochanter are debrided back to healthy bleeding cortex. The defects are then packed with morselized bone bank allograft, usually from the femoral head. The greater trochanter can then be reattached either with figure of 8 tension-band wires or a claw plate. The primary source of wear and osteolysis is addressed in the same procedure to maximize successful outcome. Postoperatively patients are usually treated with a period of nonweight bearing and an abduction brace to limit tension on the repair. Duration of protected weight bearing is dependent on size of the lytic lesion, signs of radiographic union, and symptoms. Patients are then monitored with serial radiographs and advancement in weight bearing is based off radiographic union and pain. In one study, at an average follow up of 4 years, 18 of the 19 fractures had gone on to successful union. Average time to healing was 5 months, with an overall improvement in Harris hip score of 60 points. One of the 19 patients had treatment failure but was
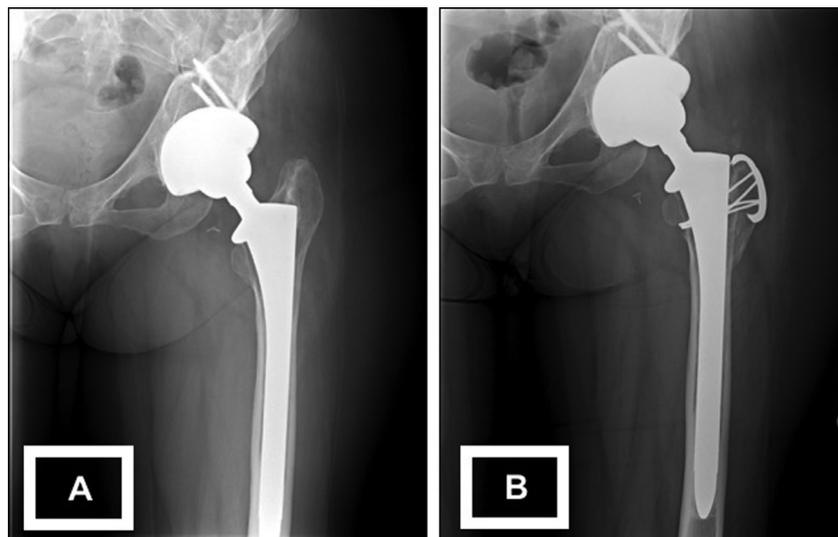


**Figure 4** Preoperative and postoperative radiographs of left greater trochanteric slide. Radiographs demonstrating greater trochanteric slide advancement. Slide advancement was utilized to tighten the abductors. Once advanced the trochanter was secured with a trochanteric plate and cables.

felt to be secondary to poor compliance with abduction bracing. Recurrent osteolysis of the greater trochanter was found in one hip at the 8-year follow-up examination.[42]

# Conclusion

Abductor deficiency in the setting of total hip arthroplasty is a difficult problem to manage for the treating reconstructive surgeon and can lead to pain, instability, and poor functional outcomes for the patient. For successful management one needs to have a thorough grasp of the underlying clinical anatomy, biomechanics, and varying surgical techniques to address this complex problem.

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