

Neurologic Injuries After Total Hip Arthroplasty

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Abstract

Neurologic injuries are a potentially devastating complication of total hip arthroplasty (THA). Review of the literature reveals that these injuries are uncommon. The reported incidence ranges from 0.08% to 7.6%. The incidence in primary THA ranges from 0.09% to 3.7% and in revision THA from 0% to 7.6%. Reported etiologies include intraoperative direct nerve injury, significant leg lengthening, improper retractor placement, cement extravasation, cement-related thermal damage, patient positioning, manipulation, and postoperative hematoma. Risk factors include developmental dysplasia of the hip, the female sex, posttraumatic arthritis, and revision surgery. However, no single risk factor has been consistently reported to be significant, and many patients with no known risk factors incur neurologic injuries.

Development of a peripheral nerve palsy after total hip arthroplasty (THA) is an uncommon but potentially devastating complication. The incidence in primary THA ranges from 0.09% to 3.7% and in revision THA from 0% to 7.6%¹⁻¹⁷ (Table). An understanding of this complication remains elusive. Identification of patients at increased risk, and meticulous operative technique, can reduce the occurrence of this frustrating complication.

Seddon¹⁸ categorized the extent of peripheral nerve injury on the basis of degree of anatomical and functional disruption. *Neuropraxia* is local myelin damage with preserved axon continuity, usually secondary to compression. *Axonotmesis* is loss of continuity of axons with variable

preservation of the connective-tissue elements of the nerve. *Neurotmesis* is complete disruption of the nerve resulting in permanent loss of function in the absence of operative repair.¹⁹

ANATOMY Sciatic Nerve

The sciatic nerve arises from nerve roots L4 to S3 and is composed of the peroneal and tibial divisions.²⁰ These divisions typically travel together in a single sheath.²¹ The sciatic nerve exits the pelvis inferior to the piriformis muscle. It travels superficial to the external rotators and deep to the gluteus maximus muscle at the level of the hip joint. Distal to the ischial tuberosity, it courses medial to the gluteal sling between the long head of the biceps femoris and the adductor magnus muscles. Branches from the tibial division innervate the hamstring muscles, except for the short head of the biceps, which is innervated by the

“Edwards and colleagues³⁸ reported that leg lengthening is a significant risk factor for neurologic injury [of the sciatic nerve].”

peroneal division. This is the only muscle innervated by the peroneal division above the level of the knee. At the level of the popliteal fossa, the sciatic nerve divides into the tibial and common peroneal nerves.

The common peroneal nerve innervates the anterior and lateral compartments of the leg through the deep and superficial peroneal nerves, respectively. The deep peroneal nerve innervates the tibialis anterior, extensor digitorum longus, extensor hallucis longus, and peroneus tertius muscles. The sensory branch innervates the interspace between the first and second toes. The superficial peroneal nerve innervates the peroneus longus and brevis muscles. The sensory branches innervate the dorsum of the foot. The tibial nerve innervates the superficial and deep posterior compartment muscles and terminates as the medial and lateral plantar nerves. The tibial division of the sciatic nerve is larger than the peroneal division and runs posterior to that division at the level of the hip. The nerve gradually rotates until the tibial division runs medial to the peroneal division at the posterior aspect of the thigh.

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Table. Incidence of Reported Nerve Palsies in Primary and Revision Total Hip Arthroplasty^a

Study	No. of Patients	Total No. of Palsies (%)	No. of Primary Cases	No. of Palsies in Primary Cases (%)	No. of Revision Cases (%)	No. of Palsies in Revision Cases (%)
Amstutz & colleagues ¹	88	6 (7.6%)	—	—	88	6 (7.6%)
Beckenbaugh & Ilstrup ²	300	4 (1.3%)	253	—	47	—
Buchholz & Noack ³	3948	75 (1.9%)	3855	—	93	—
Eftekhar & Stinchfield ⁴	700	9 (1.3%)	556	—	144	—
Farrell & colleagues ⁵	27,004	47 (0.17%)	27,004	47 (0.17%)	—	—
Johanson & colleagues ⁶	5667	34 (0.6%)	—	—	—	—
Lazansky ⁷	501	3 (0.6%)	403	—	98	—
Lubinus ⁸	1350	28 (2.1%)	—	—	—	—
Moczynski & colleagues ⁹	237	4 (1.3%)	155	—	82	—
Murray ¹⁰	808	15 (1.9%)	557	10 (1.8%)	251	5 (2.0%)
Navarro & colleagues ¹¹	1000	8 (0.8%)	630	3 (0.5%)	370	5 (1.4%)
Nercessian & colleagues ¹²	7133	45 (0.5%)	—	—	—	—
Nercessian & colleagues ¹³	1287	1 (0.08%)	1152	1 (0.09%)	135	0 (0%)
Robinson & colleagues ¹⁴	316	2 (0.63%)	—	—	—	—
Schmalzried & colleagues ¹⁵	2355	43 (1.8%)	1661	21 (1.3%)	694	22 (3.2%)
Weber & colleagues ¹⁶	2012	14 (0.7%)	2012	14 (0.7%)	—	—
Wilson & Scales ¹⁷	108	4 (3.7%)	108	4 (3.7%)	—	—

^aDash (—) indicates data not available.

These divisions do not exchange any neural tissue, which accounts for the numerous anatomical variants noted in the literature.

The reported incidence of anatomical variation in the sciatic nerve is 15% to 30%.^{20,22-24} Pokorny and colleagues²⁵ found an atypical anatomical relationship between the sciatic nerve and the piriformis muscle in 20.9% of cadaveric dissections. The most common variation was division of the sciatic nerve into tibial and peroneal nerves above the piriformis muscle, with one branch traveling through and the other superior to the muscle.

The common peroneal division is injured more often than the tibial division is, because of the distal tethering of the terminal branches¹⁵ and the superficial fibular arcade at the level of the knee. In addition, the peroneal division is more vulnerable to compression because of fewer but larger-caliber nerve bundles with less interposed connective tissue. The result is more tightly packed fascicles, in comparison with the tibial division, which has smaller-caliber bundles with abundant connective tissue.²⁶⁻²⁸ Finally, in accord with Laplace's law,²⁹ the larger-caliber nerve bundles of the peroneal division are at higher risk for compression.

Femoral Nerve

The femoral nerve arises from nerve roots L2 to L4, passes through the psoas muscle, and then runs between the iliacus and psoas muscles. It enters the thigh as the most lateral structure in the femoral triangle. The femoral nerve innervates the quadriceps mechanism and provides sensory innervation to the anteromedial aspect of the thigh and calf.

Obturator Nerve

The obturator nerve arises from nerve roots L2 to L4, runs within the posterior aspect of the psoas muscle, and emerges at the sacral ala. It continues along the iliopectineal line and exits

the pelvis through the obturator canal at the superolateral aspect of the obturator foramen. The obturator nerve innervates the adductor muscles and provides sensory innervation to a small region on the medial aspect of the thigh. Along its course, it traverses the quadrilateral surface of the acetabulum.

Superior Gluteal Nerve

The superior gluteal nerve arises from nerve roots L4 to S1, exits the pelvis through the greater sciatic notch, and innervates the tensor fascia lata, gluteus minimus, and gluteus medius muscles. It travels with the superior gluteal artery deep to the gluteus maximus and medius but superficial to the minimus. There is a 3- to 5-cm "safe zone" proximal to the greater trochanter.^{30,31} The superior gluteal nerve travels with the sciatic nerve opposite the posterior superior acetabular quadrant. The acetabular thickness is 25 mm or more in this region, permitting safe placement of screws.³²

INJURY INCIDENCE

Sciatic Nerve

The reported incidence of sciatic nerve palsy associated with THA ranges from 0.05% to 1.9%.^{5,9-17,33} In a series of 27,000 cases of primary THA, Farrell and colleagues⁵ reported a 0.05% incidence of sciatic nerve palsy. Murray¹⁰ reported an incidence of 0.1% in 808 cases, with the event occurring in a revision case, while Navarro and colleagues¹¹ reported an incidence of 0.1% in 1000 cases, with the event occurring in a primary case. Nercessian and colleagues¹² evaluated 7133 patients and reported a sciatic nerve palsy rate of 0.08%, with 1 case occurring in a revision procedure and 5 cases occurring in primary procedures. Schmalzried and colleagues¹⁵ reported an incidence of 1.5% in 2355 primary and revision cases, and Wilson and Scales¹⁷ reported an incidence of 1.9% in 108 primary cases. Hurd and colleagues³⁴

reported no cases of sciatic nerve injury in 752 patients in whom the gluteus maximus insertion was released, compared with 3 (0.4%) of 804 patients in whom the gluteus maximus tendon was left intact. Weber and colleagues¹⁶ prospectively evaluated 30 THA cases, 70% of which showed electromyographic (EMG) evidence of neurologic injury after surgery, though motor weakness was detected in only 2 cases. The sciatic nerve was involved in 12 of these cases. Weale and colleagues³⁵ confirmed these findings—that clinical examination underestimates the incidence of neurologic injury after THA.

“...clinical examination underestimates the incidence of neurologic injury after THA.”

Peroneal Nerve

The reported incidence of peroneal nerve injury in THA ranges from 0.3% to 2.1%.^{4,5,7-12,17} Farrell and colleagues⁵ reported an incidence of 1.1% in 27,004 primary cases. Lazansky⁷ reported a 0.4% incidence of ipsilateral peroneal nerve injuries and a 0.2% incidence (1 case) of contralateral peroneal nerve injury. Navarro and colleagues¹¹ reported a 0.6% incidence of peroneal nerve injury in 6 of 1000 cases, 5 of which were revision cases associated with concomitant ipsilateral femoral nerve injuries. The reported incidence of isolated peroneal nerve injury may be confounded by unrecognized injury to the entire sciatic nerve.

Femoral Nerve

The reported incidence of femoral neuropathy associated with THA is 0.01% to 2.3%.^{5,7,9-12,15,16,33,36} Farrell and colleagues⁵ reported 1 case of femoral neuropathy in 27,004 primary THA cases. Navarro and colleagues¹¹ reported 3 cases of femoral neuropathy in 1000 hip replacements—1 in a primary case and 2 in revision surgeries, both of which were associated with concurrent injury to the ipsilateral peroneal nerve. Schmalzried and colleagues¹⁵ reported 11 cases of femoral nerve palsy associated with 3126 primary and revision cases—5 isolated injuries and 6 associated with concomitant ipsilateral sciatic nerve injuries. Weber and colleagues¹⁶ reported 6 cases of femoral nerve injuries in primary THAs, 3 of which were associated with concurrent ipsilateral sciatic nerve injuries. Simmons and colleagues,³⁶ in a study dedicated exclusively to the study of femoral neuropathy associated with THA, reported 10 cases. The 7 primary cases were associated with the Hardinge anterolateral approach, while the 3 revision cases occurred with the transtrochanteric approach.

Obturator Nerve

The reported incidence of obturator nerve injury is 0.01%.^{12,16} Nercessian and colleagues¹² reported 1 obturator nerve injury in 7133 cases. This injury occurred in a revision surgery for a patient with rheumatoid arthritis.

Weber and colleagues¹⁶ in their series of 2012 cases reported 1 incident of obturator nerve injury, in a primary case.

Superior Gluteal Nerve

To our knowledge, no cases of injury to the superior gluteal nerve have been reported in the literature. However, the common postoperative pain and weakness of the abductor mechanism associated with THA may mask injury to this nerve. EMG studies performed by Abitbol and colleagues³⁷ revealed subclinical injury to the gluteal nerves in both the lateral and posterior approaches to the hip, indicating that injury is likely to occur, but the clinical incidence remains unknown.

Lateral Femoral Cutaneous Nerve

Nercessian and colleagues¹² reported 1 case of injury to the lateral femoral cutaneous nerve, in a primary THA. The incidence in this report was 0.01% (1 of 7133 cases).

ETIOLOGY OF INJURIES

Sciatic Nerve

Edwards and colleagues³⁸ reported that lengthening is a significant risk factor for neurologic injury; lengthening of 2.7 cm increased the risk for injury to the peroneal division, whereas lengthening of 4.4 cm increased risk for the entire sciatic nerve. Weber and colleagues¹⁶ similarly found that significant leg lengthening increased the risk for sciatic nerve palsy. Schmalzried and colleagues¹⁵ suggested that leg lengthening does not directly cause neurologic injury but that tension placed on the nerve results in indirect compression by prominent prosthetic or osseous structures. Egli and colleagues³⁹ concurred that lengthening increases susceptibility to injury but is not the primary cause of sciatic nerve palsy. Nercessian and colleagues¹³ reported leg lengthening of 2 to 5.8 cm in 66 of 1284 patients after THA. Although the incidence of neurologic injury in this study was 0.08%, patients with leg lengthening of more than 2 cm did not experience neurologic deficits.

Lundborg and Rydevik⁴⁰ demonstrated the effect of increased vascular tension on blood flow in a rabbit model. Venous slowing was seen with a mean of 8% lengthening, and complete cessation of flow with 15% lengthening of vessels. Lundborg and Rydevik⁴⁰ and Lundborg⁴¹ studied the effect of ischemia time on neurologic function and found complete deterioration of nerve function after 30 to 90 minutes. However, rapid recovery of function was possible after fewer than 5 to 6 hours of ischemia. Eight hours of ischemia caused irreversible damage.

Fleming and colleagues⁴² described development of sciatic nerve palsy secondary to hematoma formation in 5 patients, 4 of whom had been receiving anticoagulation therapy. The presentation was intense postoperative buttock and leg pain, swelling, and tension about the wound. Recovery was optimized by decompression within 24 hours of presentation.

Birch and colleagues⁴³ reported a case of polymethylmethacrylate (PMMA)-induced thermal damage to the sci-

atic nerve. They demonstrated that thermal damage extends less than 1 cm along the nerve, and they recommended excising the nerve 1 cm proximally and distally and following up with nerve grafting to treat this complication.

Neural impingement resulting from PMMA cement extrusion has been reported in the sciatic, femoral, and obturator nerves.⁴⁴⁻⁴⁷ Oleksak and Edge⁴⁷ described a case of sciatic nerve compression by PMMA cement. The cement was excised 6 years after the initial surgery, with complete resolution of symptoms.

Complete intraoperative laceration of nerves is uncommon.⁴⁸ Laceration may be caused by scalpel, by penetration of the femoral cortex during reaming, or by constriction from suture or wire.³⁸ Osteophyte impingement, acetabular bone graft, and prosthetic prominences have been proposed as additional mechanisms of neurologic injury during THA. Sciatic nerve palsy has also been described as resulting from migration of fractured trochanteric wire and nerve entrapment by trochanteric wire.^{49,50}

Femoral Nerve

Femoral nerve injuries result primarily from improper placement of the anterior acetabular retractor as the tip of the Hohmann retractor is placed near the femoral nerve.^{32,51} Simmons and colleagues³⁶ concluded that all femoral neuropathies in their series were secondary to retractor placement with direct compression of the nerve. Several reports have implicated cement extrusion as causing femoral neuropathy by neural impingement or complete encasement.^{16,33,36,44,52} Bleeding with subsequent hematoma formation has also been reported to cause femoral nerve palsy.^{33,52}

Obturator Nerve

Injuries to the obturator nerve result from penetration of the anterior quadrant of the acetabulum.³² This is related to the close proximity of the obturator nerve to the acetabular floor.

“The ‘safe zone’ for the superior gluteal nerve extends 3 to 5 cm proximal to the tip of the greater trochanter...”

Superior Gluteal Nerve

The “safe zone” for the superior gluteal nerve extends 3 to 5 cm proximal to the tip of the greater trochanter; splitting of the gluteus medius more proximally places the nerve at risk.^{30,48}

Common Peroneal Nerve

Injury to the common peroneal nerve may occur at the level of the fibular head secondary to perioperative compression or may be a manifestation of more proximal injury to the

peroneal division of the sciatic nerve.^{16,38} Edwards and colleagues³⁸ indicated that stretch-induced diffuse nerve damage may occur between the hip and the knee, with the primary injury site at the neck of the fibula. The injury level must be determined for prognostic purposes.

Lateral Femoral Cutaneous Nerve

Injury to the lateral femoral cutaneous nerve results from pressure secondary to the hip rests and supports that are used to position the patient during THA.¹⁹

“Injuries to the obturator nerve result from penetration of the anterior quadrant of the acetabulum.”³²

RISK FACTORS

In a study of 2012 THAs, Weber and colleagues¹⁶ reported that female sex is the only factor that correlated with development of neuropathy. Johanson and colleagues⁶ noted that 79% of the patients who sustained nerve injuries in their study were female—a finding consistent with that of several other authors who attributed increased risk to female sex.^{33,38} Edwards and colleagues³⁸ reported that 74% of their patients with nerve palsies were female, 22% of whom had developmental dysplasia of the hip (DDH). This relatively high percentage of female patients with DDH was thought to be a confounding variable. Higher incidence of neurologic injuries in females versus males has been postulated to result from females’ reduced muscle mass, different local vascular anatomy, and shorter limbs.^{16,19,53}

Schmalzreid and colleagues¹⁵ reported that patients with DDH and patients undergoing revision surgery were at significantly increased risk for neurologic injury. In a series of 27,000 patients, Farrell and colleagues⁵ reported that DDH was a significant risk factor in those undergoing primary THA; concluded that excessive limb lengthening, post-traumatic arthritis, and cementless femoral fixation were risk factors; and proposed that the increased risk associated with cementless femoral fixation was related to the need for forceful impaction of the femoral component.

Navarro and colleagues,¹¹ evaluating the role of surgical approach in development of neurologic injury after THA, concluded that anatomical variation and the complexity of the reconstruction, but not the surgical approach, were associated with neurologic injury. Johanson and colleagues⁶ and Weale and colleagues³⁵ concurred that incidence of neurologic injury is unrelated to surgical approach.

Anatomical variance is a risk factor associated with development of nerve palsy. Pokorny and colleagues²⁵ reported a 20.9% incidence of an atypical relationship between the sciatic nerve and the piriformis muscle in cadaveric specimens. They described an increased likeli-

hood of neurologic injury during THA as a result of this anatomical variance.

Sculco⁵⁴ described nerve injury as a potential risk in minimally invasive surgery and recommended minimization of posterior compression on the sciatic nerve with retraction, avoidance of excessive levering on the soft tissues, and extension of the incision if visualization is inadequate.

DIAGNOSIS

Most neurologic injuries are diagnosed during the immediate postoperative period, either in the recovery room or when the patient arrives on the floor. It is assumed that the palsy is caused by neuropraxia, and intervention is seldom indicated.

Sciatic Nerve

Sciatic nerve injury may manifest as isolated peroneal nerve dysfunction caused by injury of the peroneal division of the sciatic nerve alone. The distinction is important for prognostic purposes. However, physical examination alone is often insufficient to make this distinction. A careful neurologic examination of both divisions of the sciatic nerve is critical when a patient presents with a footdrop after surgery. Hurd and colleagues³⁴ reported 2 cases in which the location of sciatic nerve compression was localized by magnetic resonance imaging to between the ischial tuberosity and the femoral insertion of the gluteus maximus tendon.

Femoral Nerve

Femoral nerve injury presents with thigh pain, anteromedial and medial paresthesias of the leg, and quadriceps weakness. Quadriceps weakness, which manifests as difficulty with stair climbing, may be demonstrated in a bed-confined patient by having the patient press down on the popliteal space while the physician looks for quadriceps contraction.⁴⁸ Loss of the patellar tendon reflex may also indicate femoral nerve dysfunction. Inability to contract the quadriceps may be secondary to postoperative pain or to continued femoral nerve block administered for postoperative pain control.

Obturator Nerve

Obturator nerve injury presents with persistent pain in the groin or thigh and adductor weakness. Suspicion of obturator nerve injury is suggested if these symptoms follow intrapelvic screw or allograft placement or if intrapelvic cement extrusion is visible on x-rays.^{45,48} An obturator nerve block may confirm the diagnosis.⁴⁸

Superior Gluteal Nerve

Damage to the superior gluteal nerve manifests clinically as a positive Trendelenburg sign or gait with weak abduction. Although this is commonly present after surgery secondary to pain, persistent weakness is a sign of possible neurologic injury.

Lateral Femoral Cutaneous Nerve

Damage to the lateral femoral cutaneous nerve presents with numbness over the lateral aspect of the thigh.

PROGNOSIS

Various rates of recovery, either partial or complete, are reported in the literature. Nercessian and colleagues¹² reported partial or no recovery of combined upper and lower extremity neurologic injuries in 14 (33%) of 42 patients. Permanent injury occurred in 27% of the primary cases and in 43% of the revision cases. Overall incidence of permanent peripheral neuropathy in their 7133 patients was 0.21%.

Farrell and colleagues⁵ reported that the majority of patients with nerve palsies after THA, whether complete or incomplete, never fully regained preoperative strength. Pekkarinen and colleagues⁵⁵ indicated that the ability to predict outcome after sciatic nerve injury was particularly difficult in the early stages after the injury. Clawson and Seddon^{56,57} described variability in useful postrepair sciatic nerve motor recovery, with return of function in 79% of tibial division injuries and in 36% of peroneal division injuries.

Femoral nerve injuries related to cement compression respond well to surgical intervention; those with complete femoral nerve entrapment have a more guarded prognosis.^{16,44} Simmons and colleagues³⁶ reported that all patients with direct retractor-related injuries to the femoral nerve had a full recovery.

Damage to the lateral femoral cutaneous nerve may be permanent, but patients typically accommodate to the paresthesias after several years.⁵⁸

TREATMENT

Surgical intervention may be considered for acute loss of sciatic nerve function in cases of documented limb lengthening,^{5,59,60} for hematoma evacuation,^{5,26,42} or for suspicion of other mechanical causes of neuropathy.²⁶ Sakai and colleagues⁵⁹ and Silbey and Callaghan⁶⁰ reported independently on sciatic nerve palsy in patients who had significant limb lengthening after THA and recovered after modular neck size reduction and calcar shortening. Unwin and Scott⁶¹ suggested that wound exploration be considered when there is any suggestion of intraneural or extraneural hematoma, the possibility of cement extrusion, or damage by knife or suture. They considered pain in the distribution of the affected nerve to be the most accurate indication for wound exploration.

PREVENTION

Stone and colleagues,⁶² using somatosensory evoked potentials (SSEPs) to intraoperatively monitor 50 patients, reported that appropriate measures taken in response to SSEP changes resulted in no postoperative neurologic injuries. The authors suggested that SSEP monitoring during revision THA may be useful. Nercessian and colleagues⁶³ described use of SSEPs in 25 revision THAs. Signs of transient intraoperative neurologic compromise were detected in 12 instances in 8 patients (32%)—7 caused by retraction and 5 caused by limb positioning. The authors suggested that intraoperative SSEP monitoring be considered during difficult revision cases to reduce the risk for permanent neurologic injuries.

Perelles and colleagues⁶⁴ used SSEP monitoring to monitor sciatic nerve function in 52 THAs. They advocated minimizing lateral and/or anterior retraction of the proximal femur to reduce the incidence of neurologic injury. They also recommended against SSEP monitoring during routine THA but suggested that this monitoring technique be considered during revision and DDH cases. Several other authors have agreed that use of SSEP monitoring during primary THA is not indicated. However, SSEP monitoring may be useful in revision and DDH cases and when significant leg lengthening is expected.^{62,63,65,66}

Brown and colleagues⁶⁷ described use of spontaneously elicited EMG (sEMG) and near nerve action potentials (NAPs) in a series of 63 revision THAs. They reported that neurologic monitoring with sEMG and NAPs provided instant notification of potential nerve damage, and they recommended considering intraoperative neurophysiologic monitoring during revision THA.

Gentle maneuvering and careful positioning of the limb during THA reduce the risk for neurologic injury.^{68,69} Shiramizu and colleagues⁶⁸ used intraoperative motor evoked potentials (MEPs) to evaluate sciatic nerve function in patients undergoing THA with the posterolateral approach. They reported abnormal MEPs after hip dislocation in all positions other than 60° of flexion and 60° of internal rotation with maximal knee flexion. Satcher and colleagues⁶⁹ combined MEPs with EMG to identify specific intraoperative maneuvers that increase the risk for sciatic nerve injury during revision THA. They recommended avoiding hip flexion during posterior acetabular retraction when using the posterior approach with posterior dislocation. In addition, they recommended clear identification of the sciatic nerve in cases that require complex structural acetabular augmentation with allograft.

Navarro and colleagues¹¹ reported that increased awareness in the operating room led to a more than 50% reduction in incidence of sciatic nerve injury. Heller and colleagues⁵¹ highlighted the importance of avoiding compression of the femoral nerve with anterior acetabular retractors in patients with reduced muscle mass, as they lack a robust psoas tendon to protect the nerve. Obturator nerve injury may be reduced by avoiding penetration of the anterior acetabular quadrant with screws and reamers. Injury to the superior gluteal nerve may be minimized by blunt dissection of the gluteus medius muscle with the anterolateral and direct lateral approaches to the hip.⁵⁸

Unwin and Scott⁶¹ recommended meticulous retractor placement, visualization and protection of the sciatic nerve in revision cases, special care while supporting the limb throughout the procedure, avoidance of excessive traction during hip dislocation, and neurophysiologic monitoring of the sciatic nerve in high-risk cases.

CONCLUSIONS

Although there is a low reported incidence of neurologic injury associated with THA, there is a high subclinical nerve palsy rate, and the true incidence is likely higher than reported because of unrecognized partial palsies and lack of an effective mechanism for recording complications in

hospital databases. Despite identification of numerous risk factors and etiologies, 50% of neurologic injuries are not associated with an identifiable cause.

We suggest that all patients be given preoperative counseling about the risk for neurologic injury. Patients with known risk factors—including females, patients with DDH, patients undergoing revision surgery, and patients in whom leg lengthening of more than 4 cm is expected—may require further education about the risks for injury. Intraoperative monitoring is not recommended for routine primary THAs but may be considered in patients with known risk factors. Physician awareness and meticulous operative technique have been shown to reduce the risk. Extremes of position should not be maintained over extended periods, and great care should be taken when placing retractors. Although implicated as a cause of neurologic injury by several authors, neurolysis may be considered as indicated.^{48,64,67-70}

We recommend that neurologic function be assessed after surgery, in the recovery room, and documented.⁶¹ For patients who received regional or spinal anesthesia, we suggest that they be examined after the anesthesia has become inactive. Exploration of painful palsies and hematoma evacuation is recommended as indicated. Calcar/neck shortening may be considered in patients with significant leg lengthening. Nonpainful palsies are difficult to manage, as surgical exploration is not as likely to resolve them as it is to resolve painful palsies. Risks for additional surgery must be weighed against the likelihood of improvement in neurologic function on an individual patient basis. Research is needed in this area to further define the risk factors and etiologies of neurologic injury associated with THA.

AUTHORS' DISCLOSURE STATEMENT

The authors report no actual or potential conflict of interest in relation to this article.

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This paper will be judged for the Resident Writer's Award.
