Review Article

Incidence, Injury Mechanisms, and Recovery of Iatrogenic Nerve Injuries During Hip and Knee Arthroplasty

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ABSTRACT

Iatrogenic nerve injury is a rare but potentially devastating complication in total joint arthroplasty of the hip and the knee. Multiple previous studies have evaluated the incidence, mechanisms of injury, recovery, and potential treatments for this complication. Injury in total hip arthroplasty generally involves direct injury of sensory nerves from the incision, direct or traction injury of during exposure, or limb lengthening. Injury in total knee arthroplasty generally involves direct injury of sensory nerves from incision, injury due to errant placement of retractors, during balancing, or from traction because of deformity correction. Treatment of iatrogenic nerve injuries has ranged from observation, intraoperative prevention by nerve monitoring, limb shortening postoperatively, medications, and decompression. The orthopaedic surgeon should be versed in these etiologies to advise their patients on the incidence of injury, to prevent occurrence by understanding risky intraoperative maneuvers, and to select appropriate interventions when nerve injuries occur.

The incidence of notable peripheral nerve injury has been reported to be 0.2% to 3.7% in total hip arthroplasty (THA)[1] and 0.16% to 1.5% in total knee arthroplasty (TKA).[2] However, depending on the specific nerve injured, the rates of recovery and the deficit experienced by the patient can be variable because of the innervation pattern and the degree of motor or sensory loss.[3-7] The purpose of this review was to identify the incidence of the various nerve injuries that can occur during TKA and THA, describe common mechanisms of injury, and discuss the rates of recovery with potential treatment options.

Total Hip Arthroplasty

The summary of nerve injuries, their potential mechanisms, and recovery is shown in Supplemental Digital Content 1 and 2 (http://links.lww.com/JAAOS/A658; http://links.lww.com/JAAOS/A659).

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Lateral Femoral Cutaneous Nerve

The lateral femoral cutaneous nerve (LFCN) is a sensory nerve arising from the L2 and L3 nerve roots. It courses along the psoas muscle and exits the pelvis superficial to the sartorius and tensor fascia lata (TFL), where it can be damaged during the anterior approach to the hip.[8] LFCN injury causes anterolateral thigh numbness and potentially neuropathic pain (meralgia paresthetica) in that region.

The risk of injury to the LFCN is mainly related to its path in the anterolateral thigh because it travels in the subcutaneous tissue in the muscular interval of the sartorius and the TFL, which is the main internervous interval used for direct anterior (DA) THA.[9,10] Both Bartlett et al and Ruden et al described the branching pattern of the nerve. Bartlett et al described the following four patterns: a classic pattern, a femoral dominant pattern, a late branching pattern, and a trifurcate branching pattern (Figure 1). They noted that 44% of the time, the nerve crossed the DA incision line.[9] Conversely, Ruden et al discussed the following three types: a sartorius type, a posterior type, and a fan type. They noted that particularly when the fan type was present (32% of cases), the nerve is at risk for crossing the DA incision.[10]

The incidence of LFCN injury in DA THA has ranged from 14.8% to 81% in various series.[8,11,12] The treatment options reported mainly consist of observation because most improve with time.[12,13] Both Ozaki et al and Patton et al showed that with observation alone, the incidence of neuropathic-related symptoms reduced to 10% to 11.2% of patients, and Ozaki et al showed that 96% of patients with symptoms related to LFCN injury improved after 26 months of follow-up.[12,13] A review by Vajapey et al found that after a minimum of 2 years of follow-up, only 4% to 11% of patients had residual symptoms,[8] although it should be noted that Western Ontario and McMaster Universities Osteoarthritis Index scores were no different based on LFCN symptoms in a case series by Gala et al.[14]

Femoral Nerve

The femoral nerve (FN) originates from the L2 to L4 nerve roots and then runs in the psoas muscle through the abdomen and the pelvis. The nerve exits just under the inguinal ligament and runs medial to the acetabulum. It then proceeds lateral to the femoral artery and nerve in the femoral triangle.[3,8] Third, the reported incidence of FN palsy after THA is low at 0.01% to 2.3%.[3,5,6,8,15,16]

Injury leads to anterior thigh numbness, numbness in the saphenous distribution, and loss of quadriceps function.

Multiple anatomic studies have evaluated the position of the anterior acetabular retractor in both DA and posterior THA.[14-19] These have generally confirmed a few technical tips for placing this retractor. Careful intracapsular placement should be stressed (under either direct vision or palpation of the anterior wall). Extracapsular placement can cause injury to the psoas muscle and direct compression of the nerve[18] (Figure 2). Three cadaveric studies have evaluated various positions from superior to inferior on the acetabular rim for ideal placement of the retractor.[19-21] The nerve is also more at risk, with retractor placement being more inferior. More superior placement closer to the anterior inferior iliac spine is safer because this increases the distance from the retractor to the nerve. Appropriate retractor placement can be assisted by lifting the leg and releasing tension on the femur to facilitate the retractor sitting between the labrum and the capsule. The reported incidence of FN palsy after THA is low at 0.01% to 2.3%.[3,5,6,8,15,16] The FN is also at risk during retractor placement in the DA approach, particularly during femoral preparation. Excessive medial placement can cause direct injury to the nerve. In addition, hyperextension of the leg for femoral exposure can result in traction injury to the FN.

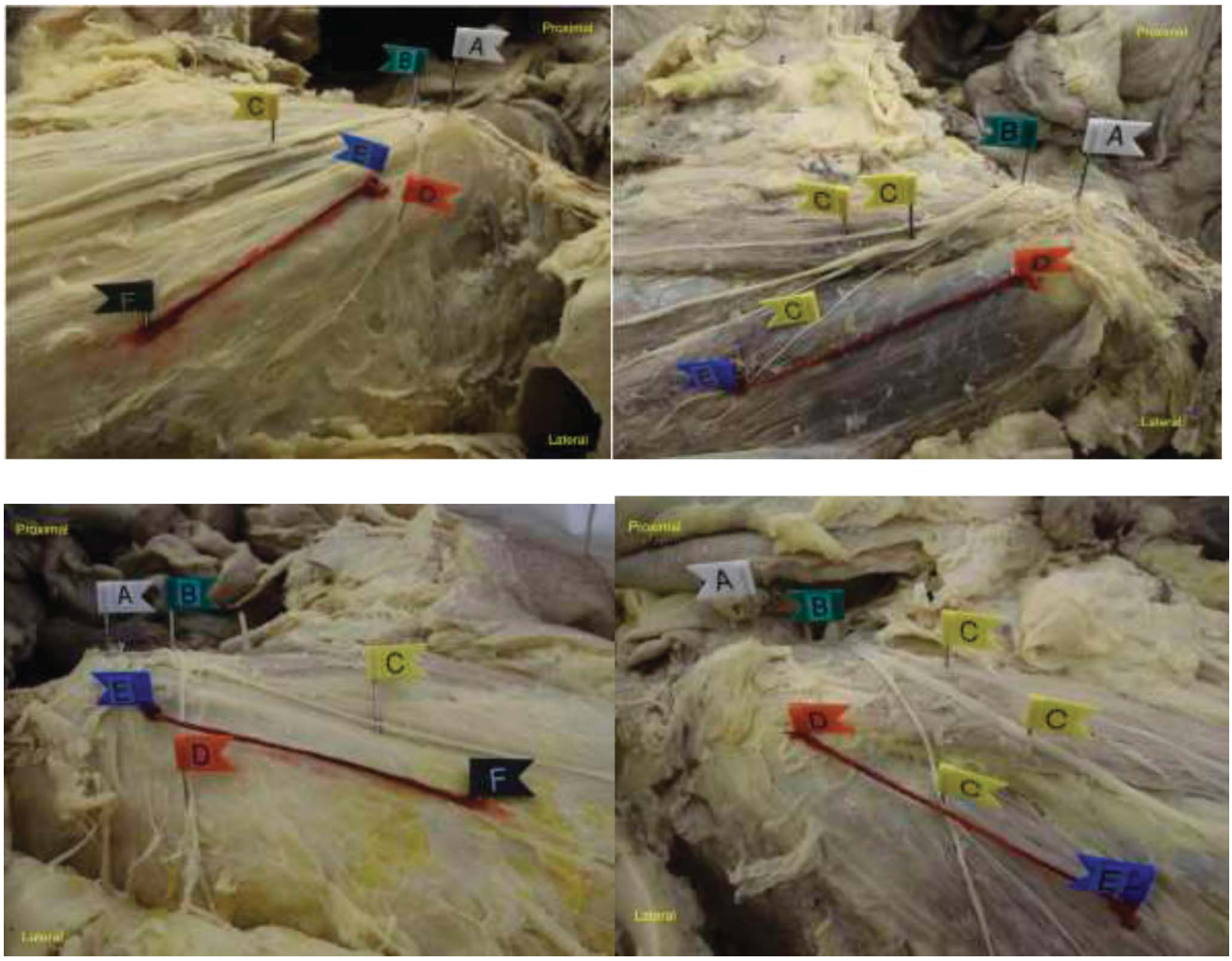
Treatment of FN injuries has generally involved observation alone because most obtain complete or partial recovery of function.[5,8,15,16] In the case series by Fleischman et al, 17 patients underwent objective motor testing. Of these patients, 64.7% (11 patients) did not require the use of an assistive device, 82.7% retained some permanent sensory loss, and 50% had complete resolution of the motor deficits, with two patients (5.9%) having an extension force less than 75% of the contralateral limb.[15]

Sciatic Nerve

The sciatic nerve (SN) begins from the lumbosacral plexus and comprises L4 to S3 nerve roots. It exits the pelvis through the greater sciatic notch, with a variable course of anterior, posterior, or through the piriformis muscle. It then runs posterior to the quadratus femoris and enters the posterior compartment of the thigh, where it eventually splits into the common peroneal and tibial branches.[3,8] Injury can result in sensory or motor loss, most commonly affecting the peroneal division. This may be due to intraneural anatomy and increased tolerance to the stretch of the tibial implant, as compared with the common peroneal implant.[22]

Preventing injury to the SN consists of careful attention to five separate steps of the THA procedure. First, carefully making the posterior cut in the capsule as to not injure the nerve. Second, when exposing the acetabulum, if placing a retractor posteriorly, be sure to place this

Figure 1



Photograph showing lateral femoral cutaneous branching patterns from Bartlett et al.[9] Top left, classic branching pattern; top right, femoral branching pattern; bottom left, late branching pattern; and bottom right, trifurcation pattern. The red line marks the direct anterior incision plane, A marks the anterior superior iliac spine, B marks the nerve at the inguinal ligament, and C marks the branches of the nerve. Bartlett JD, Lawrence JE, Khanduja V: What is the risk posed to the lateral femoral cutaneous nerve during the use of the anterior portal of supine hip arthroscopy and the minimally invasive anterior approach for total hip arthroplasty? *Arthroscopy* 2018;34:1833-1840.

intracapsular. Third, when exposing the acetabulum, monitoring the placement of any self-retaining retractors. Fourth, monitoring the trajectory of acetabular screws progressing toward the sciatic notch. Fifth, monitoring for lengthening during the trialing of implants.[17,19,23] The posterior acetabular retractor can be within 2 cm of the SN in some anatomic studies (Figure 3) and even within 1.54 cm of the SN in smaller female patients.[19,23] Lengthening of more than 2 cm is a risk factor of palsy especially without neural monitoring, although no acceptable lengthening limit has been definitively defined.[17,24,25] Lengthening has also been reported as a percentage of lengthening, with experimental studies documenting injury with greater than a 6% increase in length.[25] Direct injury from screw laceration, self-retaining retractor pressure (eg, Charnley), electrocautery or bone cement heat, scalpel damage (Figure 4), local analgesic injection, wiring of the trochanter with clamping, and suture ligation have also been reported, although they are rare.[3]

The incidence of SN palsy is 0.068% to 1.9%,[3,5,6,16,17,24-26] with this number increasing when notable lengthening (>2 cm) is a part of the surgical plan to 1.4% to 52%.[17,24,25] There are specific patients where a preoperative discussion on the risk of nerve injury should be done because they are at higher risk.

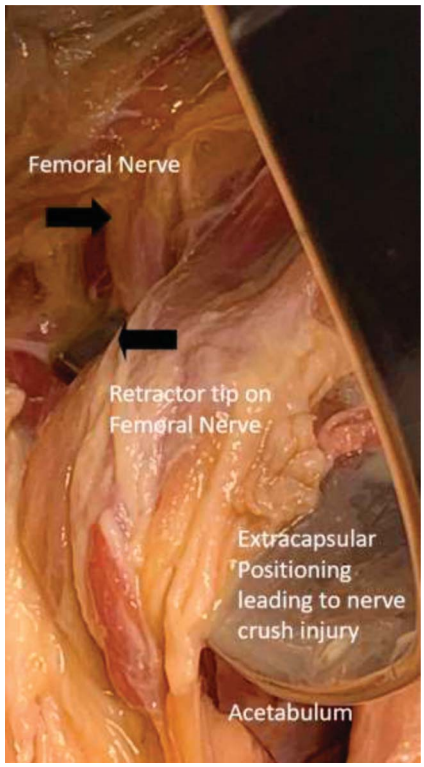
These patients include patients receiving THA for post-traumatic arthritis, developmental dysplasia, or notable shortening of the hip.[5] Patients should also be counseled of increased risk when doing a posterior-based approach rather than an anterior-based approach.[5] Treatment options include observation, acute shortening when lengthening was a part of the surgical plan, and decompression especially if a hematoma is noted to be present on physical examination or advanced imaging, such as CT or MRI. Decompression of the common peroneal nerve (CPN) distally as it wraps around the fibular neck has also been reported with some success, though this is controversial.[16,24,26-29] Pritchett evaluated many of these treatment methods. He noted that "meaningful recovery" of the nerve occurred in 40% of the patients with direct repair in cases of transection, 61% with neurolysis and an intact nerve, and 71% with acute limb shortening when lengthening was thought to be the cause.[27] Chughtai et al[26] showed that decompression of the SN in the thigh when clinically indicated improved the recovery rate: 75% had neurologic improvement with a second surgery compared with 33% who underwent observation alone. Finally, Wilson et al attempted decompression of the CPN more distally in the fibular tunnel at the knee to assist in nerve recovery. They found that 65% of patients had a motor grade 3 or higher after this procedure.[29] Because SN injury is an infrequent complication of THA and no randomized studies exist, the optimal method of treatment is unclear.

Superior Gluteal Nerve

The superior gluteal nerve (SGN) begins from the L4 to S1 nerve roots, exits the pelvis through the greater sciatic notch where it innervates the TFL, gluteus medius, and gluteus minimus.[3] Deficit of this nerve leads to abductor weakness and limp.

Injury to the SGN generally occurs during the lateral (eg, Hardinge) or anterolateral (eg, Watson-Jones) approaches to the hip because the nerve is 4.7 to 6.4 cm proximal to the greater trochanter[30] (Figure 5) because of excessive proximal muscle splitting or retraction. However, SGN injury has also been described with utilization of the posterior (eg, Southwick) approach during placement of the superior acetabular retractor when it is placed 1.74 to 2.5 cm from the location of the nerve, causing traction injury.[19,23] Using a laterally based approach, this number can increase to 77%.[6] There is very little chance of injury to the SGN when using a DA approach because it wraps around the gluteus medius. However, it can be damaged because it enters the TFL (Figure 6). A study has also evaluated injury to the distal branches of the SGN because it enters

Figure 2



Photograph showing the femoral nerve proximity and danger of piercing the anterior capsule when placing the anterior retractor.

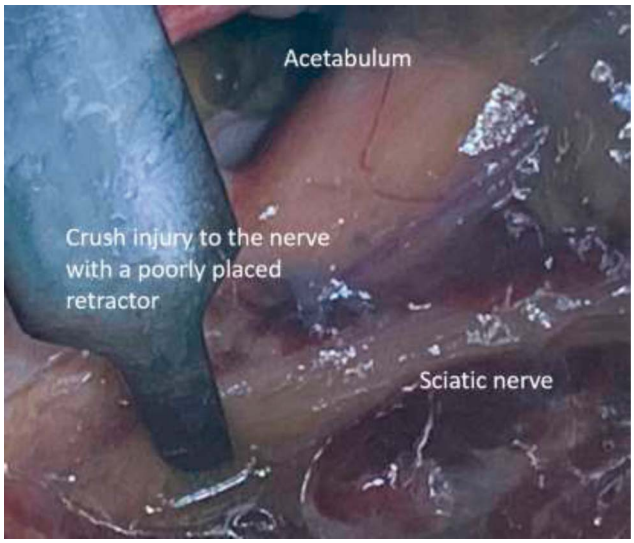
the TFL during the DA approach. This has been noted to be a cause of postoperative atrophy of the TFL around the incision site, although the functional significance of this remains unclear.[31] During the posterior approach, injury to the SGN is relatively rare at 0.3%.[16] However, it has been recently noted that the SGN can be injured during the positioning for augments in revision surgery, especially those which are larger than 5 cm.[32] Treatment is generally nonsurgical because it presents as partial weakness of the abductors because the posterior portion of the abductors remains innervated. Most patients regain function and have better prognosis, as compared with SN injuries.[6]

Obturator Nerve

The obturator nerve (ON) is a mixed nerve that arises from the L2 to L4 nerve roots. It runs in the pelvis on the posterior aspect of the psoas muscle, exits the pelvis, and enters the medial compartment of the thigh through the obturator foramen.[3] Injury causes weakness with hip adduction and medial thigh numbness. These are less detrimental muscular losses for function of the limb, as compared with the loss of the FN or SN.

There are two mechanisms of injury that are described for the ON. Inferior acetabular retractor placement has

Figure 3



Photograph showing the sciatic nerve proximity and potential injury from a posterior acetabular retractor.

been noted as a potential factor (Figure 7). McConaghie et al showed that the retractor can contact the nerve when it is levered during removal[18] (Figure 6). Case reports have also shown that the nerve can be damaged by extruded cement when a cemented acetabular implant is placed.[33]

The incidence of ON nerve injury is very low at 0.01%.[3] Treatment involves observation because the deficit is not as notably detrimental as injury to the previous nerves discussed.[3]

Total Knee Arthroplasty

The summary of nerve injuries, their potential mechanisms, and recovery is shown in Supplemental Digital Content 3 and 4 (http://links.lww.com/JAAOS/A660; http://links.lww.com/JAAOS/A661).

Common Peroneal Nerve

The CPN is one of the two branches of the SN when it splits with the tibial nerve (TN) in the posterior thigh. The CPN runs lateral and just posterior to the biceps femoris tendon because it inserts into the fibula and then traces around the fibular neck, whereafter it branches into the superficial and deep divisions that innervate the lateral and anterior compartments of the leg, respectively.[3]

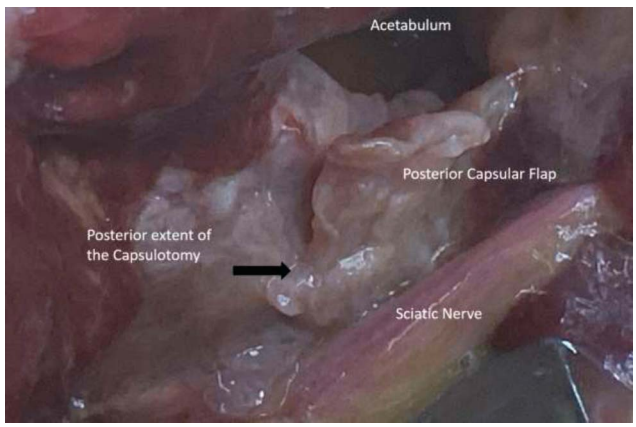
Injury to the CPN is the most common motor nerve injury after TKA, and it is the most studied.[7,22,34-36] The most common proposed mechanisms are stretch from leg lengthening or retractor placement and injury during a posterolateral corner release (Figure 8). Stretch injury seems to be a risk especially when doing a TKA in a patient with a valgus deformity, a flexion contracture, or a combined valgus deformity with a flexion contracture.[36] Lengthening of an average of 3.5 mm, however, caused no injury in 91 cases.[37] Multiple studies have evaluated the safety of the Ranawat inside-out release of the posterolateral structures.[34,35] During this portion of TKA balancing, a scalpel, an electrocautery, or needle is used to "pie-crust" the tight lateral structures in a valgus knee. The location of the CPN is 7.8 to 26.2 mm from the capsule, regardless of the amount of deformity.[34,35]

The incidence of CPN injury has been reported to be between 0.3% and 4.3%.[7,36] Treatment of this complication includes observation, loosening of constricting bandages, and flexing the knee. Alternately, the injured nerve can undergo neurolysis and decompression. Prophylactic decompression has also been advocated for severe deformity.[7,36] Most patients in a recent systematic review were treated with observation alone.[36] Patients tend to improve after CPN injury although they may have persistent weakness, particularly of the extensor hallucis longus. Importantly, if the patient has an incomplete palsy at the time of diagnosis, they have a greater chance of full recovery compared with those with complete deficit (66% versus 39%).[36]

Tibial Nerve

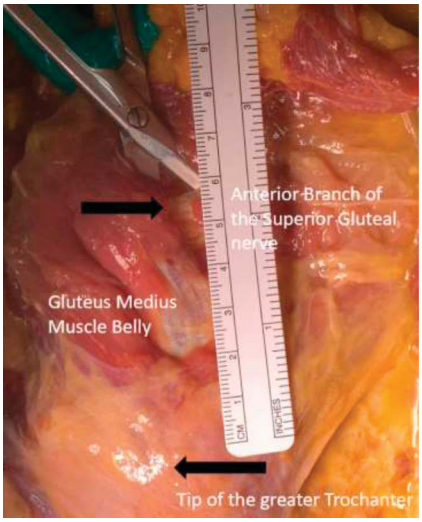
The TN branch is less frequently injured than the CPN in TKA (and THA). This is partly because of its tissue consistency[22] being less prone to stretch as well as its anatomic location in the leg. The TN splits with the

Figure 4



Photograph showing the sciatic nerve proximity and chance of injury from extended posterior capsulotomy in a posterior approach.

Figure 5



Photograph showing the superior gluteal nerve distance from the greater trochanter in the Hardinge approach.

CPN in the posterior thigh from the SN and travels superficial and lateral to the popliteal artery and the vein in the popliteal fossa. It innervates the posterior superficial and deep compartments of the leg and splits into the plantar nerves of the foot.[3]

The main mechanism for injury to the TN during TKA is due to stretch, and it generally occurs in conjunction with a CPN injury[22,36] because of the resistance to stretch, as compared with the CPN. The nerve is relatively distant from the bony structures and injury to the popliteal vasculature will occur before injury to the nerve given their locations in the popliteal space.

The incidence of TN injury in TKA is 0.27% according to a case series by Schinsky et al.[7] All patients who developed TN palsy were treated with observation. They all recovered full motor and sensory function of the TN by 1 year.[7]

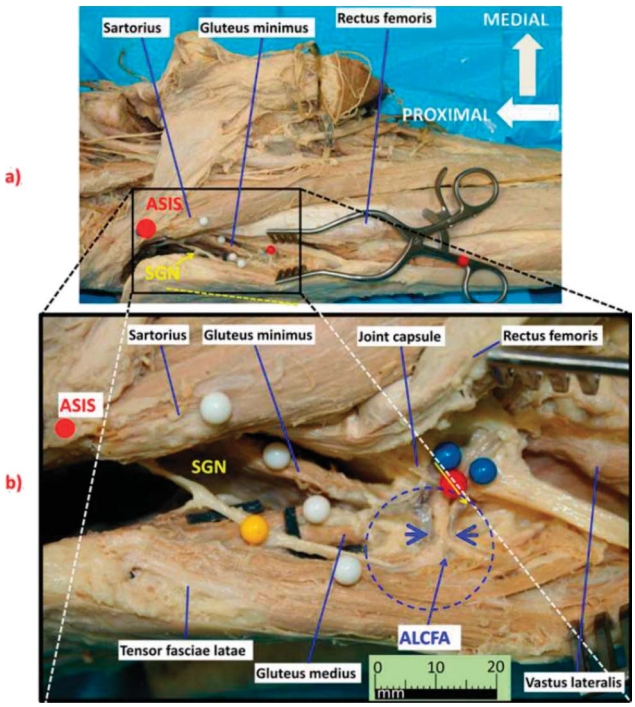
Inferior Branch of the Saphenous Nerve

The saphenous nerve is a sensory extension of the FN in the thigh. It travels down the medial thigh, and because it gets to the knee, it sends branches from medial to lateral, providing sensibility to the skin anterior to the knee. A particular branch known as the inferior branch of the saphenous nerve (IBSN) has been implicated as the branch that is cut, leaving patients with inferolateral numbness in relation to the patella.[38] This can be an issue for pain with kneeling, painful neuromas, or neuropathic pain.[39-41]

Anatomic studies have evaluated the position of the IBSN because it relates to the patellar tendon and the tibial tubercle. The nerve is located at an average distance of 16.82 mm superior to the tibial tubercle, and it is crossing the approach[38] (Figure 9). Another study evaluated the difference in relative risk to the IBSN using a standard midline approach versus a minimally invasive type approach. Tanavalee et al[41] found no difference in the ability to avoid the nerve, given its position inferior to the patella and superior to the tibial tubercle, which is cut regardless of the approach type.

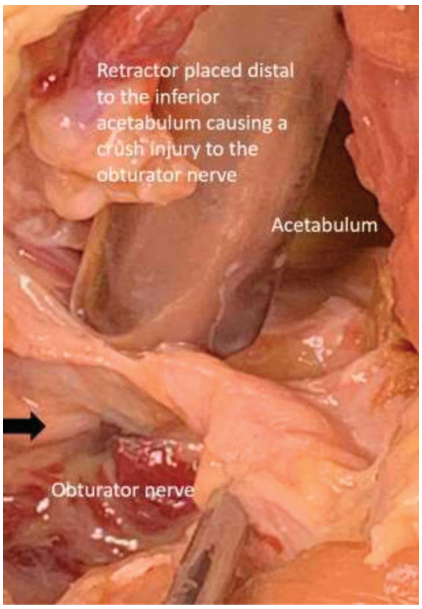
Anterolateral numbness has been reported in 31% to 100% of patients undergoing various exposures for TKA.[39-41] This has been treated with observation alone in most cases. The area of skin numbness recovers in 70% to 99% of patients[39,41] over a 1-year period. This seems to take longer to occur in patients with previous systemic nerve issues, such as diabetes mellitus, although the overall rate of recovery is the same.[39] In patients who did have persistent sensibility loss, the overall area decreases to around 3 to 10 cm^2, with this area being smaller if the incision is concentrated more on the anterolateral aspect of the knee as opposed to the

Figure 6



From Grob et al. Photograph showing the SGN branch to the tensor fascia lata that can be injured during a direct anterior approach. ALCFA = ascending branch of the lateral circumflex artery, ASIS = anterior superior iliac spine, SGN = superior gluteal nerve. Grob K, Manestar M, Ackland T, Filgueira L, Kuster M: Potential risk to the superior gluteal nerve during the anterior approach to the hip joint. *J Bone Joint Surg* 2015;97:1426-1431.[31]

Figure 7



Photograph showing the obturator nerve proximity and potential injury from the inferior retractor.

anteromedial aspect.[40] This is likely secondary to the nerve fibers traversing the knee from medial to lateral.

Our Approach to Preventing and Treating Nerve Injuries

Preoperative Planning

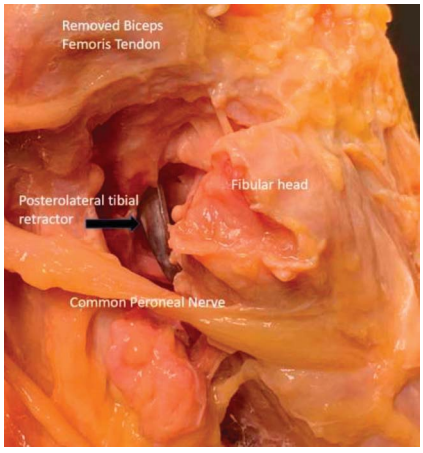
Preoperative planning is the initial step we take to avoid a potential nerve injury. Every joint is a template, and thus, when lengthening of a notable degree in a total hip or deformity correct of a notable degree is to be undertaken in the total knee, it is recognized preoperatively. For the THA with notable dysplasia where lengthening is necessary, we would consider doing a subtrochanteric shortening osteotomy to avoid SN palsy. We also consider using intraoperative monitoring to avoid injury to the SN.[17] In the knee with a notable flexion contracture and valgus deformity together (greater than combined 20 degrees), we consider prophylactic CPN release to prevent stretch injury to the nerve. Preoperatively, we have a discussion with the patient concerning the approach to the total hip or TKA. For the TKA, we advise the patient that it is common for anterior lateral numbness after the procedure. For the anterior THA, we discuss the risk of having lateral thigh numbness after the procedure. We also discuss any patient who is undergoing notable lengthening or undergoing revision THA, the potential for SN dysfunction postoperatively.

Intraoperative

For a posterior THA, we are careful in the proximal extent of the gluteus maximus split because this can damage the inferior gluteal nerves. We then feel posterior when placing the Charnley retractor to avoid direct injury to the nerve during retractor placement. For the anterior THA, we work lateral through the TFL fascia to avoid damage to the LFCN and FN during the exposure. Next, we are careful with retractor placement during the acetabular exposure. Placement of the anterior retractor is scrutinized throughout the case and repositioned when it appears too inferior. We are careful to place the retractor between the labrum and the hip capsule.

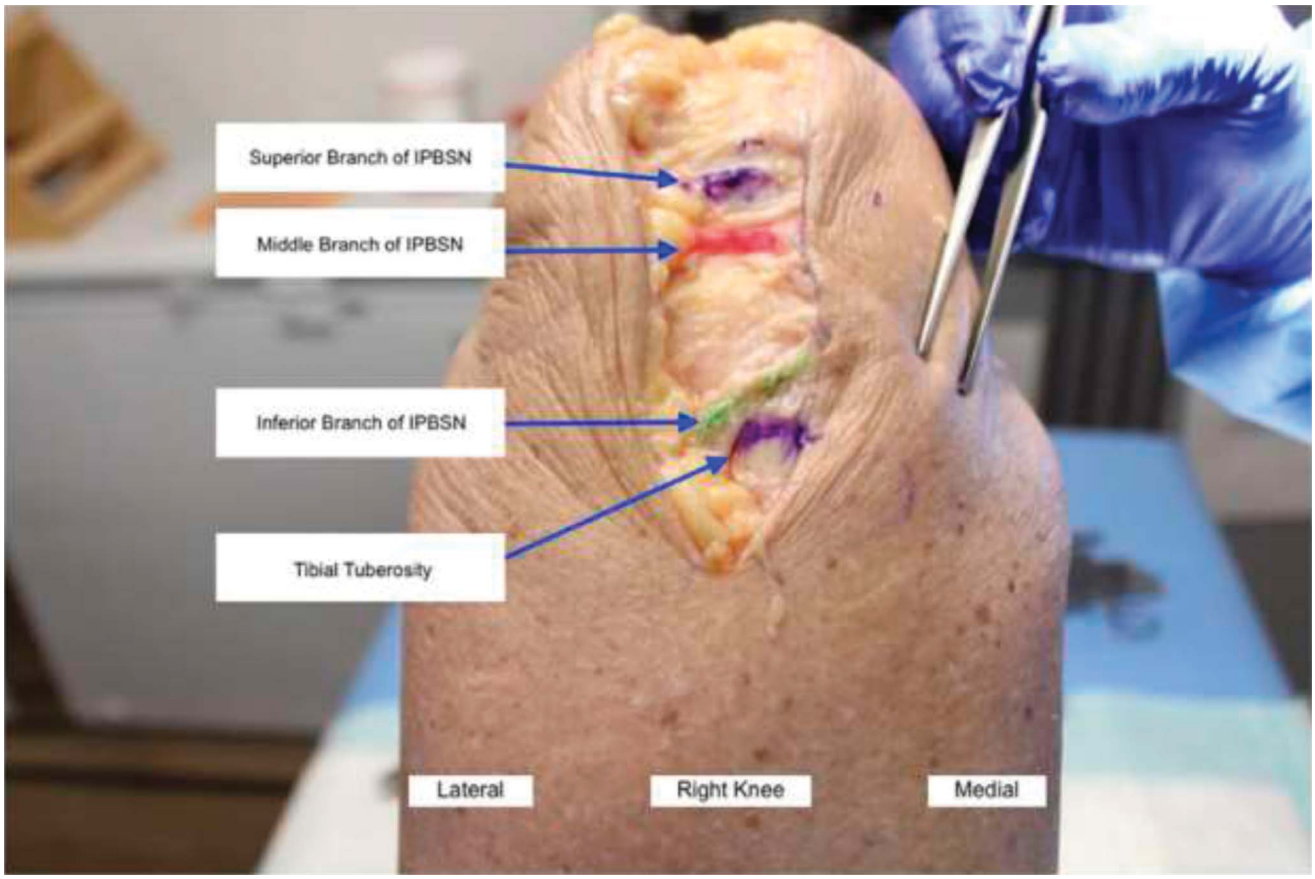
For the TKA, we provide no specific protection to the IBSN, it is generally cut in most procedures. We tend to avoid the use of the tourniquet during routine TKA or only use it during cementation. Although complications are rare, Tarwala et al saw no complications in patients where tourniquets were used only during cementation versus tourniquets used routinely.[40] Nerve complications are noted to specifically occur during prolonged tourniquet use, so we limit the time to 120 minutes when the tourniquet is used.[42] We are careful during the exposure concerning the placement of the lateral retractor because posterior and lateral placement can place the CPN at risk. When a posterolateral release is necessary, we use laminar spreaders and a 15 blade to carefully release the posterolateral structures, under direct vision during balancing. When using a periarticular injection for pain control, care should be taken to avoid placement near the CPM because this can cause a transient nerve palsy. This can specifically occur

Figure 8



Photograph showing the common peroneal nerve proximity and potential injury with a posterolaterally placed knee retractor.

Figure 9



Photograph showing the branching patterns of the infrapatellar branch of the saphenous nerve (IPBSN). Lee SR, Dahlgren NJP, Staggers JR, et al: Cadaveric study of the infrapatellar branch of the saphenous nerve: Can damage be prevented in total knee arthroplasty? *J Clin Orthop Trauma* 2019;10:274-277.[38]

when posterolateral capsule requires a release in a valgus knee.[43]

Postoperative

We do early nerve checks on all arthroplasty patients the day of surgery after neuraxial anesthesia is worn off. We do this to identify nerve palsies early in the perioperative period. Nonsurgical treatment of the SN and CPN palsies involves extension of the hip and flexion of the knee to take tension off the nerve.[44] Medications can be considered. There has been evidence to suggest that corticosteroids and erythropoietin may be neuroprotective and enhance recovery, although no large trials exist.[45,46] Erythropoietin also has a favorable adverse effect profile.

Surgically, treatment has not been well defined for timing and intervention.[44] We will do early exploration and neurolysis of the SN to address palsies in these nerves when complete deficits occur in the immediate postoperative period. For SN palsy after a THA with a clear cause, we do neurolysis of the SN at the hip and decompression of any hematoma, address impingement by cages or screws, and do shortening of the limb if lengthening occurred during the initial surgical procedure.[27] Should there be no specific notable cause of SN injury, we monitor the patients for a trial period of 6 months, and if they continue to have persistent deficits, we offer them distal decompression around the fibular head and neurolysis at the hip to improve their symptoms.[28,29] After a TKA, we explore and do neurolysis around the CPN for a CPN palsy, although evidence is limited.[36] This involves a nonsurgical observational period of 1 month, followed by decompression as described by Zywiel et al[47] because this can improve their motion and function even if the issue is primarily sensory in nature. There is also limited evidence that late neurolysis after this period is effective in assisting with recovery.[48] We generally observe ON, FN, TN, LFCN, and IBSN deficits because these tend to improve with time although again, if there has been no improvement in 6 months, we tend to offer exploration.[27,44]

Summary

Nerve injuries in THA and TKA can be a cause of persistent symptoms and dysfunction after these procedures. Sensory nerve injuries are common and can be expected for each approach, including the LFCN in the DA THA and the IBSN in all approaches for the TKA. Other injuries are very rare, such as ON injury, which have few cases reported in the literature. Careful study of the anatomy can provide the surgeon with techniques to avoid injury by meticulous placement of retractors or care when doing releases when needed. Nerve injuries in arthroplasty are often treated with observation alone with a likelihood of at least partial improvement of most injuries. Occasionally, decompression is recommended when a compressive force such as a hematoma can be identified. Shortening of the limb in the case of SN injury in THA should be considered if the cause of the neuropathy is thought to be acute lengthening. Further research is required to better elicit improved techniques to avoid nerve injury and strategies to treat these complications when they occur.

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