■ HIP ARTHROPLASTY: AVOIDING AND MANAGING PROBLEMS

# Retraction: Post-operative neuropathy after total hip arthroplasty

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**Nerve palsy is a well-described complication following total hip arthroplasty, but is highly distressing and disabling. A nerve palsy may cause difficulty with the post-operative rehabilitation, and overall mobility of the patient. Nerve palsy may result from compression and tension to the affected nerve(s) during the course of the operation via surgical manipulation and retractor placement, tension from limb lengthening or compression from post-operative hematoma. In the literature, hip dysplasia, lengthening of the leg, the use of an uncemented femoral component, and female gender are associated with a greater risk of nerve palsy. We examined our experience at a high-volume, tertiary care referral centre, and found an overall incidence of 0.3% out of 39 056 primary hip arthroplasties. Risk factors found to be associated with the incidence of nerve palsy at our institution included the presence of spinal stenosis or lumbar disc disease, age younger than 50, and smoking. If a nerve palsy is diagnosed, imaging is mandatory and surgical evacuation or compressive haematomas may be beneficial. As palsies are slow to recover, supportive care such as bracing, therapy, and reassurance are the mainstays of treatment.**

**Cite this article: *Bone Joint J* 2017;99-B(1 Supple A):46–9.**

Nerve palsy after total hip arthroplasty (THA) is a devastating complication to both the patient and the surgeon because it is unexpected and debilitating. The most common nerve to be affected is the sciatic nerve, which is involved in over 90% of cases, followed by the femoral nerve.[1,2] As both of these peripheral nerves are mixed nerves with both sensory and motor components, injury to either nerve can lead to sensory changes and weakness of the lower limb. Nerve palsies can cause significant disability to patients because motor involvement can cause a foot drop, which will affect walking. Recovery from a nerve palsy can be very slow and incomplete, leading to anxiety and frustration. As such, nerve palsies are a frequent cause of malpractice lawsuits after THA. A survey of the American Association of Hip and Knee Surgeons found that nerve injury was the number one reason for litigation after hip arthroplasty surgery.[3] Surgeons should be aware of the incidence of nerve palsy following THA so that they may be better able to counsel their patients pre-operatively. Furthermore, knowledge of the risk factors can also aid in stratifying those patients at greater danger for developing a post-operative nerve palsy.

## Pathophysiology and presentation

Although nerve palsy is a known complication of THA, it occurs infrequently and is unanticipated. As a result patients and their families may ascribe the development of a nerve palsy to surgical error. In general, post-operative nerve palsies are neuropraxias, where damage is done to the nerve without a disruption of the axon (axonotmesis) or nerve sheath (neurotmesis). Following a neuropraxia, the nerve fibres remain in continuity and therefore have the potential to recover function. Neuropraxia may occur from direct compression, either by surgical instruments or anatomical structures, or from tension or ischemia to the nerve, which can occur as a result of intra-operative manipulation of the limb or as a result of lengthening of the limb.

The peroneal division of the sciatic nerve is thought to be particularly at risk for neuropraxia because of its "tethering" at the level of the fibular head, making it more susceptible to tension. Furthermore, the peroneal division has less connective tissue separating the nerve fibrils compared with the tibial division, making it more vulnerable to compression. Finally, the peroneal nerve is in a more lateral location anatomically, placing it closer to the surgical field of the posterior approach.[4]

The first indication that a neuropraxia has occurred is with post-operative motor weakness in the dorsiflexion of the foot. If epidural anesthesia has been used, it is necessary to

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doi:10.1302/0301-620X.99B1.BJJ-2016-0430.R1 $2.00

*Bone Joint J* 2017;99-B(1 Supple A):46–9.

**Table I.** Incidence of nerve palsy after total hip arthroplasty (THA) in the literature

| | Yr | THAs (n) | % incidence of palsy overall | Primary THA (n) | % incidence of palsy in primary THA |
|---|---|---|---|---|---|
| Schmalzried et al[2] | 1991 | 2355 | *1.8* | 1661 | *1.3* |
| Johanson et al[10] | 1983 | 5667 | *0.6* | | |
| Park et al[11] | 2013 | 9570 | *0.32* | | |
| Farrell et al[1] | 2005 | 27 004 | *0.17* | 27 004 | *0.17* |
| Navarro et al[12] | 1995 | 1000 | *0.8* | 630 | *0.5* |
| Wilson and Scales[13] | 1973 | 108 | *3.7* | 108 | *3.7* |

discontinue the administration of anesthetic so that the lower limb motor function can be properly assessed. If the sciatic nerve has been involved, the typical presentation is a foot drop, with weakness of the tibialis anterior, extensor hallucis longus, and extensor digitorum longus. Eversion with the peroneal muscles is also commonly affected. Often, the tibial division of the sciatic nerve is unaffected; thus, the tibialis posterior, gastrocnemius and soleus muscles continue to function. There are usually sensory deficits in the skin areas supplied by the peroneal division (dorsum of the foot and first web space). Because of the weakness or inability to dorsiflex the foot, patients may be unable to clear their toes from hitting the ground, causing them to trip. An ankle-foot-orthosis (AFO) that maintains the ankle in a neutral or dorsiflexed position is necessary to prevent contractures of the Achilles tendon and to aid walking.

If the femoral nerve is involved, patients may have weakness in the quadriceps and iliopsoas, with sensory loss over the anterior thigh. Patients may also have difficulty with walking and activities that require quadriceps function, particularly stair climbing.

## Aetiology

In the course of a THA, the lower limb is manipulated and tissues are stretched for exposure, placing tension upon nerves. Specifically, the act of dislocating the hip may cause tension or compression of nerves. The position of the hip and knee during surgery have been shown to affect the amount of strain and intraneural pressures in the sciatic nerve: flexion of the hip and extension of the knee has been found to increase tensile strain in the sciatic nerve by 26%, and leads to a significant increase in intraneural pressure.[5,6]

A second cause is the placement of surgical instruments in proximity to neural structures. Acetabular retractors may impinge upon the femoral nerve anteriorly, or on the sciatic nerve posteriorly. A cadaveric study found that on average, the femoral and sciatic nerves are only 2 cm from the anterior and posterior acetabular rim, respectively.[7] Placement of retractors on the periphery of the acetabular rim during preparation of the acetabulum may therefore compress the nearby neural structures. During femoral preparation, a femoral elevator retractor may be used to aid exposure of the femoral canal. This compressive force placed on the posterior soft tissues may be a cause of sciatic nerve palsy.

Some surgeons also believe that the gluteus maximus femoral insertion (gluteal sling) can compress the sciatic nerve upon 90° of internal rotation of the leg, a position which is necessary during femoral preparation. Hurd et al[8] found that magnetic resonance imaging revealed oedema within the sciatic nerve at the level of the gluteal sling, in a series of patients who had post-operative nerve palsy. Furthermore, there was a significant difference in the incidence of sciatic nerve palsy in patients who had the gluteal sling released *versus* those who did not.

Anatomical variants in which the sciatic nerve penetrates the piriformis muscle exist in up to 20% of cadaveric specimens.[9] This is also a possible location of tension to the sciatic nerve, as in a posterior approach, the tendon is released and may pull on the nerve as it retracts. It is also another point at which the nerve may be tethered.

Finally, bleeding at the surgical site can collect in an enclosed space after surgery, causing compression of nearby nerves, leading to neuropraxia.

## Incidence and risk factors

The incidence of nerve palsy after primary THA ranges from 0.17% to 4.0% (Table I).[1,2,10-13] In a large registry database, Farrell et al[1] examined risk factors for motor nerve palsy after primary THA, and found that preoperative diagnoses of developmental dysplasia of the hip or post-traumatic arthritis had higher rates. The theory behind this association is that the altered anatomy of dysplastic patients may result in an aberrant course of the sciatic nerve, making it more susceptible to injury. In the post-traumatic situation, scar tissue or adhesions may tether the sciatic nerve. Other associated risk factors found in this study were the use of a posterior approach, lengthening of the extremity, and the use of an uncemented femoral implant.

Excessive lengthening of the extremity places tension upon the nerve; in this study, the average lengthening of the limb in the patients who sustained a nerve palsy was 1.7 cm, significantly greater than a matched cohort who did not have a nerve palsy. The use of an uncemented femoral implant may result in more axial impaction into the femoral canal, which could repeatedly stretch nerves.

Other investigators have reported on a higher incidence of nerve palsy in female patients.[10,14-16] Schmalzried, Noordin and Amstutz[15] found that in a meta-analysis of over 34 000 THAs, females had a 1.5% risk of nerve palsy, whereas male patients had a 0.77% risk. Johanson et al[10] and Edwards, Tullos and Noble[14] reported that 79% and 74%, respectively, of their cohorts of patients with nerve palsies were female. These analyses, however, did not adjust for confounding factors such as the prevalence of dysplasia. Other theories of why females may be more subject to nerve palsy include different skeletal structure (gynecoid pelvis and reduced hip offset) and lower muscle mass to protect against nerve compression.[14,16]

A higher body mass index (BMI) is also commonly thought to be associated with a greater risk of nerve palsy because of a need to apply more forceful retraction during surgery, but was not found to be a risk factor by either Farrell et al[1] or Park et al.[11]

## Our experience

We reviewed all patients with nerve palsy after primary THA at the Hospital for Special Surgery over an approximately 15-year period (between 1 January 1998 and 31 October 2013) in order to calculate our overall incidence and examine associated risk factors. Over that period, 93 patients had experienced nerve palsies (0.24%) of a total of 39 056 primary THA performed through a posterior approach.[10] Of these 93 patients, 63% were female, with an mean age of 62.6 years (21.3 to 86.4). The mean BMI for nerve palsy patients was 28.2 kg/m$^2$ (11.1 to 60).

We performed a univariable analysis of demographic parameters and risk factors for nerve palsy previously described in the literature, such as age, sex, BMI, diagnosis and smoking history. Peri-operative parameters such as duration of surgery, implant fixation, amount of limb lengthening, type of anesthesia, procedure start time, and the level of training of the surgeon were also investigated. Additionally, radiographic variables such as lateral centre-edge angle, lateral offset, neck-shaft angle, acetabular component alignment and stem alignment were analysed for a correlation with nerve palsy. The parameters that reached a p-value of 0.20 or less in the univariable analysis were included in a multivariable regression analysis.

From our multivariable analysis, the risk factors found to be associated with nerve palsy were a history of lumbar spine disease or spine surgery, spinal stenosis, smoking, and age younger than 50. The strongest risk factor for the development of nerve palsy at our institution was the presence or history of spine disease, leading to the possibility of a "double crush" phenomenon causing injury. No significant association was found with medical comorbidities such as diabetes or obesity. Contrary to other reports in the literature, we did not find an association with the type of femoral fixation, amount of limb lengthening, or gender. There was significant variability amongst different surgeons, leading to the belief that surgical technique and patient selection play a large role in the development of post-operative nerve palsy.

## Management

The management of nerve palsy certainly warrants a careful discussion between the surgeon and patient regarding the etiology and prognosis. At our institution, the recognition of a post-operative nerve palsy initiates a special protocol. The wound site and dressings are inspected to look for any sites of constriction. The limb is positioned to decrease stretch on the affected nerve: with sciatic involvement, the hip is extended and the knee is flexed while for a femoral nerve palsy, the hip is flexed. A neurologist is consulted and a detailed neurological examination is performed. Though orthopaedic surgeons are familiar with the basic neurological examination, the neurologist can perform other tests such as two-point discrimination, and testing of vibration and proprioception, which may be important in gauging future improvement. In general, acute electromyography (EMG) is not performed during the inpatient stay, as it is too early to yield information of benefit. EMG is typically obtained at three weeks following surgery, with a follow-up study at one year to assess for recovery.

Imaging of the affected area surgical site is also performed using MRI, to assess for compressive haematoma, impingement by the implants, or direct nerve injury. If a neuraxial anesthetic was used for the procedure, an MRI of the spine is also performed to rule out epidural or spinal haematoma.

If a compressive haematoma or implant impingement is identified, then the patient is returned to the operating theatre for exploration and evacuation of the compression. If the limb appears to have been lengthened excessively, then a shortening of the limb could also be considered at this juncture.

In the absence of any pathology that would warrant a return to the operating theatre, management of a nerve palsy consists of supportive measures: physical therapy to maintain range of movement in the affected lower limb, bracing during ambulation and night time splinting, and reassurance that the majority of these problems will resolve slowly over time.

## Prognosis

The prognosis of a nerve palsy is unpredictable. Although classic teaching is that the palsy will resolve completely, many researchers have found persistent deficits upon careful examination at follow-up. Edwards et al[14] found that at one year after surgery complicated by sciatic nerve palsy, only 16% of patients had complete recovery of their motor function, and that 58% had a persistent mild deficit. Schmalzried et al[15] reported that 81% had a persistent neurological deficit at two years, in their cohort of both sciatic and femoral nerve palsies. Park et al,[11] in their series of sciatic nerve palsies, found that only 57% of palsy patients recovered completely at two years. Several

authors have sought to define prognostic factors for recovery such as the degree of the palsy. Schmalzried et al[15] reported that the lesser the degree of motor involvement or the quicker recovery of motor function (within two weeks), consequently the better the prognosis. Dysesthesias seemed to be associated with a poorer outcome.[15] By contrast, Park et al[11] did not find that the degree or completeness of palsy was related to prognosis; the only prognostic factor for a full recovery was a lower BMI. The nerve affected appears to be important with femoral nerve palsy appearing to have a better outcome than a sciatic nerve palsy.[15] This is thought to be due to the closer proximity of the lesion to the spinal cord compared with the sciatic nerve, perhaps requiring less regeneration of nerve fibres for recovery.

Nerve palsy following THA is a serious complication that leads to anxiety, concern, and potential litigation. Due to the proximity of nerves to the surgical site, manipulation of the leg during the procedure, insertion of surgical retractors and instruments, and post-operative swelling, neuropraxia of the femoral or sciatic nerves can occur. This will result in sensory disturbances and/or motor weakness, affecting the patient's recovery and rehabilitation. Upon recognition of a nerve palsy, it is advisable to image the surgical area to assess for compressive lesions that could be alleviated by a repeat procedure. However, most cases will not reveal any pathology that can be corrected surgically. At our institution, a neurologist is an integral part of the team evaluating the patient with a nerve palsy, to aid the assessment the likely recovery of nerve function. The prognosis after nerve palsy is generally good, with at least partial recovery in the majority of cases. More research is needed to investigate the risk factors for this unfortunate complication.

**Take home message:**

Nerve palsy after THA is a rare but distressing complication that negatively affects patient outcomes. The arthroplasty surgeon should be familiar with the incidence, risk factors, and potentially modifiable elements in order to better counsel patients and avoid its occurrence.

**Author contributions:**
E. P. Su: Literature review, Writing the paper.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

This article was primary edited by A. D. Liddle.

This paper is based on a study which was presented at the 32nd annual Winter 2015 Current Concepts in Joint Replacement meeting held in Orlando, Florida, 9th to 12th December.

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