Seminars in Arthroplasty ▮ (2016) ▮▮▮–▮▮▮

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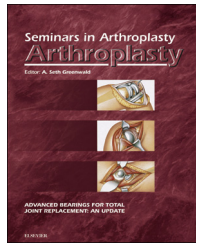

Post-surgical neuropathy after total hip arthroplasty: Causality and avoidance

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ARTICLE INFO

Keywords:
total hip arthroplasty
nerve palsy
complication
neuropathy Thx

ABSTRACT

Nerve palsy is an uncommon but distressing complication after total hip arthroplasty. Because motor function can be affected, weakness of the lower limb is a common manifestation, causing difficulty with walking and the planned rehabilitation for the hip. As such, significant disability is imparted to the affected patient. The proposed etiologies of nerve palsy include compression and tension during the course of the operation, or a collection of blood postoperatively. In the literature, hip dysplasia, lengthening of the leg, use of an uncemented femoral component, and female sex are associated with a greater risk of nerve palsy. If a compressive hematoma is found, surgical evacuation may be beneficial. As palsies are slow to recover, supportive care such as bracing, therapy, and reassurance are the mainstays of treatment.

Introduction

Nerve palsy after total hip arthroplasty (THA) is a devastating complication to both the patient and the surgeon. Most commonly, the sciatic nerve 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, both aspects can be affected, leading to dysesthesias and foot drop of the lower limb. Nerve palsies can impart significant disability to patients because motor weakness can cause difficulty walking and affect the postoperative rehabilitation. Furthermore, 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; knowledge of the risk factors can also aid in stratifying those patients at greater danger for developing a postoperative nerve palsy.

Pathophysiology and presentation

Although nerve palsy after THA is a known complication, it occurs infrequently and is unexpected; thus patients and their families may infer that a surgical error is the cause. In general, the cause is a neuropraxia, or damage to the nerve without a disruption of the axon (axonotmesis) or nerve sheath (neurotmesis). Therefore, the nerve fibers in a neuropraxia injury are still in continuity and therefore should have the potential for recovery. Neuropraxia may occur from direct

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http://dx.doi.org/10.1053/j.sart.2016.06.021
1045-4527/

compression upon the nerve, either by surgical instruments or anatomical structures. Alternatively, neuropraxia can occur from tension or ischemia to the nerve. Tension can occur during the operative procedure, or with lengthening of the leg.

The peroneal division of the sciatic nerve is thought to be particularly vulnerable to neuropraxia because of its course around the fibular head from posterior to anterior. This "tethering" of the common peroneal nerve at the level of the fibular head may make it more susceptible to tension. Furthermore the peroneal division of the sciatic nerve has less connective tissue separating the nerve fibrils as compared to the tibial division, and it 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 may have occurred is with postoperative motor weakness in the distal lower extremity. If epidural anesthesia has been given, it is necessary to discontinue the administration of anesthetic so that the lower limb motor function can be adequately assessed. If the sciatic nerve has been involved, the typical presentation is an inability to dorsiflex the foot at the ankle, 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, and 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). Because of the weakness/inability to dorsiflex the foot, the patient may have difficulty walking since the foot may catch on the ground. An ankle–foot-orthosis (AFO) is necessary to prevent contractures of the Achilles tendon and aid in 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.

Etiology

In the course of a total hip arthroplasty, the limb is manipulated and tissues are stretched, placing tension upon nerves. Specifically, the hip joint is dislocated and placed in a non-anatomic state, potentially tensioning and compressing nerves. The position of the hip and knee during surgery has been shown to affect the amount of strain and intraneural pressures in the sciatic nerve. Specifically, hip flexion and knee extension was found to increase tensile strain in the sciatic nerve by 26% [5]. Another study found that pressure transducers measured a significant increase in intraneural sciatic pressure, in the same position [6]. Thus, the position of the lower extremity during the procedure, and the length of time that it is in a particular position, may influence the development of nerve palsy.

In addition, surgical instruments are introduced in proximity to these neural structures, and as such, may inadvertently damage them. Acetabular retractors may impinge upon the femoral nerve anteriorly, or sciatic nerve posteriorly. A cadaveric study found that on average, the femoral and sciatic nerves are only 2 cm from the anterior acetabular rim and posterior acetabular rim, respectively [7]. Thus, if the retractors are placed slightly outside of the bony landmarks during acetabular preparation, nerves can be compressed. During femoral preparation, retractors are used to elevate the femoral canal for visualization. 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 internal rotation of the leg, which is commonly performed during femoral preparation. Hurd et al. [8] found a series of patients in which the gluteal sling was not released, and magnetic resonance imaging found edema in the sciatic nerve at the level of the gluteal sling. 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 15% of cadaveric specimens [9]. This has also been suggested as a mechanism by which tension is placed on the nerve by release of the tendon and subsequent retraction against the nerve.

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

In the literature, the incidence of nerve palsy after primary THA, ranges from 0.17% to 4.0% (Table).

In a large registry database, Farrell et al. examined risk factors for motor nerve palsy after primary THA, and found a preoperative diagnosis of developmental dysplasia of the hip or posttraumatic arthritis to have higher rates [1]. The theory behind this association is that the altered anatomy of the dysplastic may cause the nerve to be located in a different place than usual, and thus more susceptible to injury. In a

Table – Incidence of Nerve Palsy After THA in the Literature

	Year	# of THA	% Incidence of Palsy Overall	# Primary THA	% Incidence of Palsy in Primary THA
Schmalzried et al. [11]	1991	2355	1.8	1661	1.3
Johanson et al.	1983	5667	0.6		
Park et al. [13]	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