Femoral neuropathy following primary or revision total hip arthroplasty (THA) is a rare but acknowledged complication. Treatment of femoral neuropathy has long been debated and there is a paucity of accepted principles on which to base management. Currently, no definitive management protocol exists in the literature. A literature search was performed by a review of PubMed, Google Scholar and OVID articles published from 1972-2011. The literature reports an incidence rate of femoral neuropathy following THA ranging from 0.1 to 2.4 percent. Determining the precise aetiology, establishing a diagnosis and subsequent treatment of femoral nerve injury remains a difficult task, with conservative management remaining the treatment benchmark. In this review, we aim to summarise the aetiologies and risk factors associated with femoral neuropathy following THA and provide management guidelines.

Keywords: femoral neuropathy; total hip arthroplasty; nerve palsy; management; review.

INTRODUCTION

Total hip arthroplasty (THA) is a widely recognized procedure for the treatment of degenerative hip disease. THA has evolved considerably in recent years and is highly effective in relieving pain, improving function and restoring motion (11,14-16,23).

Femoral neuropathy following primary or revision THA is a rare but acknowledged complication and represents a challenge for both the patient and orthopaedic surgeon. Treatment of femoral neuropathy has long been debated and there is a paucity of accepted principles on which to base management of this rare complication (42). Currently, no definitive management protocol exists in the literature.

A literature search was performed from PubMed, Google Scholar and OVID articles published from 1972-2011. The purpose of this study was to provide
a concise and detailed description of the incidence, presentation, aetiology, risk factors, prevention, treatment and prognosis associated with femoral neuropathy following THA.

Anatomy

The femoral nerve is the largest branch of the lumbar plexus, arising from the dorsal divisions of the ventral rami of the second, third and fourth lumbar nerves. The nerve originates in the abdomen within the psoas major and descends postero-laterally through the pelvis to approximately the midpoint of the inguinal ligament. It then passes deep to this ligament and enters the femoral triangle, lateral to the femoral vessels. After entering the triangle, the femoral nerve divides into several branches to the hip and knee joints and provides several cutaneous branches to the anteromedial side of the thigh (Fig. 1A). The terminal cutaneous branch of the femoral nerve, the saphenous nerve descends through the femoral triangle, lateral to the femoral sheath containing the femoral vessels. The femoral nerve supplies motor innervation to the iliacus, pectineus, sartorius and quadriceps femoris muscles (Fig. 1B).

Incidence

Determining the incidence of femoral nerve injury is difficult, partially because of under diagnosis. A review of the literature reports an incidence rate ranging from 0.1% to 2.4% following primary THA (mean 0.8%) and 0.3% to 2.3% following revision THA (mean 1.1%) (Table I) (3,5,6,9,10,12,13,17,24,26-28,30,31,34-36,39-41).

Presentation

Symptoms and signs of femoral neuropathy may vary depending on both severity and location of the injury, but are characterized typically by groin or thigh pain, weakness of the iliopsoas, paralysis of the quadriceps femoris, loss of the knee jerk and sensory loss over the anteromedial aspect of the lower extremity and swelling or haematoma in the wound or in the inguinal region (18). Patients are often able to stand and walk on flat surfaces with the usual postoperative assistive devices; however the majority of patients will experience difficulty with climbing stairs. The residual functional disability, instability and pain associated with nerve injuries may be incomplete or prolonged. Any persistent postoperative motor and sensory abnormalities may impair the rehabilitation process and adversely affect patient satisfaction.

Aetiology

It is often difficult to ascertain the precise aetiology of femoral nerve injury following THA, partially because of a lack of reporting in the literature (19). Farell et al reported that only 55% of patients had a presumably identifiable cause of the nerve injury (13), whilst Barrack reported that the aetiology was unknown in 47% of patients (1). Schmalzried et al reviewed 126 consecutive total hip replacements in which the cause of the nerve palsy remained unknown in 57% of cases (34). Potential mechanisms of femoral neuropathy associated with THA have been variously reported (Table II).
Risk Factors

Owing to its infrequent occurrence, it is difficult to determine the precise risk factors that predispose certain patients to femoral neuropathy following THA.

Patients taking anticoagulants, or those with intrinsic coagulopathies such as haemophilia, may develop spontaneous bleeding in the soft tissues adjacent to the nerve. The formation of a haematoma may be the result of the perforation of the medial wall of the acetabulum during the arthroplasty and can produce delayed onset nerve palsy (4,25,35,43).

In a study of 2012 THA’s, Weber et al reported that the female gender was the only factor that correlated with development of neuropathy (41). Johanson et al reported 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 the female gender (19,31,36). The higher incidence of femoral neurologic injuries in females versus males has been postulated to result from females’ reduced muscle mass, different local vascular anatomy and shorter limbs (36,41). Experimental models have shown that neural injury will occur if the nerve is elongated more than 6 percent of its length. Given this limit, smaller individuals with shorter limbs, and thus shorter nerves, have less of a tolerance for absolute neural retraction than do much larger individuals (19,36). This factor may help to partially explain the differences in prevalence observed between genders (25).

Several studies have evaluated the prevalence of femoral nerve palsy based on the origin of hip disease (including hip dysplasia and dislocation). Altered anatomy in the region of the hip has been considered a factor that increases the potential for

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Table I. — Incidence of reported femoral nerve palsies in primary and revision total hip arthroplasty. An asterisk (*) indicates data not available. No, number

<table>
<thead>
<tr>
<th>Study</th>
<th>No. of patients</th>
<th>Total no. of femoral palsies (%)</th>
<th>No. of primary cases</th>
<th>Total no. of femoral palsies in primary cases (%)</th>
<th>No. of revision cases</th>
<th>Total no. of femoral palsies in revision cases (%)</th>
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<tr>
<td>Chapchal et al, 1973</td>
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<td>340</td>
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<td>1684</td>
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<td>1,684</td>
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<td>328</td>
<td>*</td>
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<tr>
<td>Eggli et al, 1999</td>
<td>370</td>
<td>2 (0.5)</td>
<td>508</td>
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<td>Eskelinen et al, 2006</td>
<td>63</td>
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<td>*</td>
<td>48</td>
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<td>Farrell et al, 2005</td>
<td>27004</td>
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<td>27,004</td>
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<td>557</td>
<td>10 (1.8)</td>
<td>251</td>
<td>5 (2.0)</td>
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<tr>
<td>Navarro et al, 1995</td>
<td>1000</td>
<td>8 (0.8)</td>
<td>630</td>
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<td>15 (0.6)</td>
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<td>*</td>
<td>327</td>
<td>*</td>
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<td>Schmalzried et al, 1991</td>
<td>2355</td>
<td>43 (1.8)</td>
<td>1661</td>
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<td>Simmons et al, 1991</td>
<td>440</td>
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<td>7 (1.9)</td>
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<td>*</td>
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<td>*</td>
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<td>1 (0.2)</td>
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<td>*</td>
<td>*</td>
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<td>Weale et al, 1996</td>
<td>42</td>
<td>1 (2.4)</td>
<td>42</td>
<td>1 (2.4)</td>
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<td>0</td>
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<tr>
<td>Weber et al, 1976</td>
<td>2012</td>
<td>14 (0.7)</td>
<td>2012</td>
<td>14 (0.7)</td>
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</table>
neural injuries (28). Schmalzried et al reported that patients with developmental dysplasia of the hip (DDH) and patients undergoing revision surgery were at significantly increased risk for neurologic injury (34). In a series of 27,004 patients, Farrell et al reported that DDH was a significant risk factor in those undergoing primary THA and 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 (13). Acute limb-lengthening of more than two to four centimetres during arthroplasty has been associated with an increased risk of neural injury (19).

There has been some disagreement in the literature regarding the evaluation of the role of surgical approach in the development of femoral neuropathy following THA. Navarro et al prospectively compared 1000 consecutive primary and revision THA cases for neurologic injury when performed through the transtrochanteric or posterior approach and concluded that anatomical variation and the complexity of the reconstruction, but not the surgical approach was associated with femoral neuropathy (28). Johansen et al and Weale et al also concurred that incidence of neurologic injury was unrelated to surgical approach (19,40).

However, several authors have disputed that surgical approach is unrelated to incidence of femoral neuropathy following THA. Simmons et al reported 10 femoral nerve palsies from a review of 440 (360 primary, 80 revision) THA’s. All were associated with anterolateral approach. Their anatomic studies show the proximity of the femoral nerve to the anterior aspect of the hip joint and thus to anterior acetabular retractors (35).

Revision of a failed implant has been associated with an increased risk of injury to the femoral nerve after THA (28,34). This increased risk may be due to the more extensive and difficult dissection required with revision procedures and perhaps, in some patients, to uncertainty regarding the location of major neurological and vascular structures when a previous operative exposure has been performed (25,27). In addition, tethering by scar tissue may predispose the nerve to stretch with retraction, dislocation of the hip, or limb-lengthening. During isolated revision of the acetabular component through a posterior approach, retraction of the intact femoral component anteriorly may increase the risk of compression of the femoral nerve (25).

### Prevention

The best treatment of any complication is prevention. It is advised that the treating surgeon preoperatively identify the patients who are most at risk and take the measures necessary to avoid nerve injury. Once a patient is identified as being at an increased risk, preoperative planning is mandatory. Identification and protection of the nerve, particularly when there may be alterations in the local anatomy, is indicated. Careful surgical technique and avoidance of direct injury of the nerve during exposure, gentle retraction and judicious placement of the retractors are important. Operative techniques that emphasize the use of instruments and sharp dissection away from rather than toward肩

<table>
<thead>
<tr>
<th>Haemorrhage/Haematoma</th>
<th>Fixation</th>
<th>Anatomical</th>
<th>Surgical</th>
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<td>Anticoagulant therapy</td>
<td>Extravasation/Impingement</td>
<td>Anatomy</td>
<td>Difficulty of surgery</td>
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<td>Cement Pressuring-Device</td>
<td>Displasia of hip</td>
<td>Intraoperative positioning</td>
</tr>
<tr>
<td>Haematoma</td>
<td>Direct encasement of nerve</td>
<td>Female gender</td>
<td>Leg lengthening or stretch</td>
</tr>
<tr>
<td></td>
<td>Heat of polymerization</td>
<td></td>
<td>Post-operative anterior dislocation</td>
</tr>
</tbody>
</table>

Table II. — Possible aetiologies of femoral neuropathy following total hip arthroplasty, as reported in the literature
important neurological or vascular structures are suggested. Such techniques make it possible to avoid neural injury when those instruments slip. There is no strong evidence favouring any one approach for preventing nerve injury (7,25).

Maintenance of the knee in flexion during retraction for exposure about the acetabulum through a posterior approach can reduce tension on the nerve. Avoidance of acetabular screw-placement in the anterior-superior and anterior-inferior quadrants is recommended (25). Minimizing the amount of leg lengthening during preoperative planning and using leg-length measurement techniques may decrease the risk of the nerve. Less than four centimetres or six percent of the calculated length of the nerve (which ever is less) should be used as an approximate guideline.

Intraoperative neural monitoring has been explored as a potential means to reduce the risk of nerve injury (2,20,29,33). It is possible to document intraoperative irritation of the nerve and, if necessary, to alter the operative technique or the position of the limb. Such monitoring has been recommended as a surveillance method during revision total hip arthroplasty or when major limb-lengthening efforts are planned (25,33). Simultaneous changes in amplitude and latency appear to be predictive of an alteration in the function of the nerve postoperatively. However, the actual reduction in the risk of nerve palsy due to intraoperative neural monitoring has not been well established and thus the ability of this method to prevent neural injury remains in debate (2,20,29,33).

**TREATMENT**

**Recommended Pre-Operative Considerations and Management**

Patients should be advised of the possibility of developing femoral neuropathy, particularly high-risk patients. With challenging surgery or uncommon anatomic variations, an experienced orthopaedic surgeon should be involved. There is no proven benefit of preoperative neurophysiological testing to predict or prevent femoral nerve damage.

**Recommended Intra-Operative Considerations and Management**

It is recommended that the following precautions be taken:

- A thorough awareness of the regional anatomy, its relevant functions and associated patterns of pain is essential;
- Care with the use of surgical instruments, especially retractors; avoid excessive traction, especially during dislocation of the hip;
- The limb should be supported throughout the operation;
- Consideration for intraoperative monitoring of the femoral nerve in high risk cases, and precise documentation of intraoperative (and postoperative) findings.

**Recommended Post-Operative Considerations and Management**

It is often difficult to establish a diagnosis of nerve injury following THA. It is recommended that the following be considered:

- The neurological status should be examined and recorded on the first post-operative day and repeated during the first post-operative week. Some authors recommend the routine use of electrodiagnostic testing however this has not been substantiated (8,13,37,38).
- Post-surgical care should include attention to the clinical recognition, accurate diagnosis and assessment of severity of nerve damage that may occur as a complication of the surgery.
- If femoral nerve damage is indicated and requires surgery (such as those caused by haematomas), early surgical decompression may be recommended to prevent further nerve compression and irreversible damage (13). However, this decision should be determined by the treating surgeon. The surgeon should also provide realistic recovery outcomes to the patient.
- If femoral nerve damage is indicated but surgery is not required, conservative management is the cornerstone treatment of the neuropathy (36,43). An electromyogram at six weeks and at three
months should be performed. If by three months, there is no improvement, magnetic resonance imaging may be of assistance.

**PROGNOSIS**

The potential for recovery (whether partial or complete) of femoral neuropathy after THA is generally proportional to the severity and location of the injury, although clinical factors such as unrecognized severity of the injury and delays in diagnosis, have precluded establishing an exact relation. This principle underlies the policy of watchful waiting in certain surgical aetiologies where the nerve is known or even suspected to be intact.

Establishing the degree of damage early (within 2-4 months) may minimize further nerve damage (21). Optimal management of femoral nerve injuries can be obtained by close interdisciplinary co-operation among the treating orthopaedic surgeon, neurologists and physical therapists.

Pess et al recommend decompression and/or neurolysis to improve motor function and relieve the pain associated with femoral nerve injury following cement encasement (32). However, direct envelopment of the nerve by cement carries a worse prognosis than injury by retractors or haemorrhage (35).

We recommend that the surgeon discuss with the patient the realistic outcome of recovery based on the location and severity of the femoral nerve injury. Improvements may continue for at least one year after the injury but the status is unlikely to change after two years (4,21,22,25,32,43). Some patients may never fully recover.

**REFERENCES**


