REVIEW ARTICLE

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Nerve palsy after hip replacement: medico-legal implications

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Introduction

Nerve palsy following hip replacement can be a devastating complication that is likely to face all surgeons at some stage who perform the operation regularly. Revision surgery has a greater risk than primary surgery, but the greatest risk is for replacement in developmental dysplasia of the hip. It is not just the sciatic nerve and its divisions that are at risk during hip replacement surgery; the femoral and obturator nerves may also be damaged.

Incidence of nerve palsy following total hip replacement

The literature of nerve injury following hip replacement surgery is scant. There is a paucity of accepted principles on which to base the management of these difficult problems [24].

The first large series of nerve palsies to be collected was that by Ratcliffe in 1984 [15]. This was a postal survey of orthopaedic surgeons in the United Kingdom, with 50 cases of nerve palsy presented. His conclusions were: (a) that there was no relation between nerve injury and surgical approach to the hip; and (b) traction was the most common cause of the palsy. A more recent series is that presented by Birch [2], and this work is to be presented more fully in due course. He reviewed 37 cases of injury to the sciatic and femoral nerves. His conclusions were: (a) there was no relation between nerve injury and the surgical approach to the hip. (b) In over half the series, the precise cause of the palsy was unknown, even after detailed investigation and in some cases surgery. (c) Of the 37 palsies 12 were due to direct trauma to the

nerve. (d) Of the 37 palsies 4 were due to extra-neural or intra-neural bleeding. (e) In 1 case, the injury was due to a cement burn. (f) In 2 cases, the injuries were due to ischaemia secondary to injury to nearby large vessels. Birch thus concluded that a combination of traction and compression was probably responsible for the majority of these injuries.

Several authors have documented the nerve palsy rate following hip replacement and the figures vary widely. Much of the variability in results reflects the differing patient populations and the indications for surgery:

1. Early series found a high incidence of sciatic nerve palsy following hip replacement. Wilson and Scales [23] found a 3.7% incidence of nerve palsy in 108 replacements. These figures should be regarded with caution as the review was of replacements using early arthroplasty techniques.
2. Eftekhari and Stinchfield [8] had no cases of nerve palsy in 700 hip replacements.
3. Lazansky [12] found a 1.4% incidence of nerve palsy following primary hip replacement.
4. Weber, Daube and Coventry [22] documented a 0.7% rate of clinical sciatic nerve palsy following 2012 hip replacements. However, by neurophysiological assessment, they demonstrated that over two thirds of the patients had a subclinical nerve palsy rate following arthroplasty. This series emphasises the high subclinical insult to peripheral nerves around the hip during arthroplasty.
5. Edwards, Tullos and Noble [7] recorded a palsy rate of 1.1% after 614 replacements (11 complete and 12 partial palsies). They noted a higher frequency in revision surgery and developmental dysplasia of the hip. They concluded that risk factors for nerve palsy were leg lengthening, traction at surgery and revision surgery.
6. Schmalzried, Amstutz and Dorey [17] reviewed the nerve palsy rate after 3000 consecutive arthroplasties. The rate was 1.3% after primary replacement, 3.2% after revision surgery and 5.2% in hips with develop-

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mental dysplasia. Eftekhar [9] has stated that he believes the nerve palsy rate after standard primary hip replacement to be of the order of 1%.

Aetiology of nerve palsy following total hip replacement

No direct cause can be found for most nerve palsies after hip replacement, and this fact is useful for the surgeon in medico-legal cases. In two large series [10, 17] 50% of the palsies had no clear cause. Birch [2] has also noted that a significant proportion of his series had no diagnosis. There are usually more partial than complete palsies [2, 15]. Postulated causes of nerve palsy after hip replacement include the following:

Traction or compression upon the nerve

This is probably the most common cause of postoperative nerve palsy in practice, and may be responsible for many of the "undiagnosed" cases. Acute stretching of a nerve by as little as 6% can cause permanent injury [12]. The traction may be: (a) transient, e.g. at dislocation of the hip perhaps from the use of a retractor (a Hohman retractor placed over the anterior lip of the acetabulum is a potent cause of danger to the femoral nerve), or (b) permanent, e.g. with lengthening of the limb (Edwards et al. [7] have concluded that lengthening of more than 4 cm risks nerve palsy). Where a nerve is embedded in dense scar tissue (as in revision procedures), it has even less potential to stretch without damage.

Extraneural and intraneural haematoma

This cause has probably been under-reported and, with the increasing use of prophylactic anticoagulation to prevent thrombo-embolism, should be positively excluded when investigating postoperative nerve palsies. The syndrome of haematoma-induced palsy is one of intense buttock and leg pain with tension and swelling around the wound. Neurological deficit is not always present in the early stages. Nerve conduction studies are not helpful in the acute management of these problems – the syndrome is a clinical diagnosis. Haematoma-induced palsy requires prompt decompression if the viability of the nerve is to be saved – decompression should be performed as an emergency. Delay in decompression may result in a poor outcome [21].

Intra-operative laceration of the nerve

Examples include injury by scalpel, penetration of the femoral cortex by reamers or entrapment by wire or suture. This cause is uncommon in most series [7, 15, 22]. When damage to a nerve causes continued irritation or

compression, then exploration, decompression or grafting may result in minimal improvement in neurological function, there is often significant pain relief. The most severe pain may result from continued irritation or strangulation of a nerve trunk.

Thermal burns from extraneous cement

This is a diagnosis that should be considered in all cases of postoperative palsy where methylmethacrylate cement has been used. Several cases of cement burns of the sciatic, femoral and obturator nerve have been described [4, 18, 22]. Birch [1] has shown that thermal damage extends as little as 1 cm from the site of damage; segmental excision and grafting of the nerve can be very helpful in the relief of pain. The postoperative radiograph should be carefully evaluated to exclude extraneous cement as a cause of nerve palsy.

Common peroneal nerve damage at the knee due to preoperative compression

This is a relatively uncommon cause of palsy after hip replacement. Apparently isolated common peroneal palsies are usually caused by a lesion at the level of the hip; this reflects the relative vulnerability of the peroneal division as compared to the tibial division. It has been said that females are more prone to nerve palsy after hip replacement. This is probably not the case, and as Eftekhar [9] noted most series are biased with the increased rate of arthroplasty in females and developmental dysplasia of the hip.

Anaesthetic practice can also affect the management of nerve palsies after hip replacement surgery. Some of the palsies may be induced by anaesthetic procedures, e.g. epidural anaesthetics. Epidural and local nerve blocks may also delay the diagnosis of a postoperative nerve palsy. Cooperation between surgeon and anaesthetist in this regard is essential.

Treatment and prognosis of nerve palsy after total hip replacement

The literature on recommended treatment and prognosis of nerve palsy following hip replacement is sparse and poorly controlled. The first major series on all sciatic nerve injuries (including traumatic as well as iatrogenic) by Clawson and Seddon [5, 6] concluded that there is a very wide variability in recovery between patients with essentially similar lesions, and they noted that the discrepancy between function and neurological deficit was wider for the sciatic nerve than for almost any other. In their series, there were 229 injuries of the tibial division and 345 injuries of the common peroneal division of the nerve. Their papers documented the results of 119 nerve sutures and 13 nerve grafts. They concluded that where

the tibial division was damaged and repaired, 80% regained plantarflexion of the foot, whereas only one third of common peroneal nerve repairs regained ankle dorsiflexion. The intrinsic foot musculature rarely recovered. One third of cases had no sensory recovery, with 14% of the total number of patients having trophic ulcers. The main functional complications were pain (50%), fixed ankle plantarflexion and toe clawing.

Ratcliffe [15] notes that in cases where there was postoperative compression by suture, wire or other agent, urgent exploration resulted in a better prognosis. In the series of Schmalzried and Amstutz [17] 20% were left with severe disability. If significant recovery was to occur, it would do so within 6 months. Those patients with severe pain fared badly. Birch [2] has reported that in cases where there is pain in addition to the palsy, prompt re-exploration resulted in better results than where no or delayed action had been taken.

In general, the results of exploration and neurolysis in nerve palsy due to traction are disappointing [6]. The prognosis may be determined at the time of injury [20].

The consequences of nerve injury in children are particularly disabling. This article does not comment on pelvic osteotomy, which carries a risk of nerve palsy. However, joint replacement in juvenile chronic arthritis is indicated as early as 9 years of age in some series [16]. Nerve damage in the growing skeleton carries with it the risk of limb shortening and foot deformity.

Sciatic nerve palsy

Sciatic nerve palsy is clinically the most common affliction of the three major nerves after hip replacement. The clinical effects of injury to the nerve are variable and not accurately predictable. The variability in pain following postoperative sciatic nerve palsy is remarkable. Clawson and Seddon [5, 6] commented that "the discrepancy between function and neurological deficit was wider for the sciatic nerve than almost any other"; this also applies to pain.

Several series have shown that sciatic nerve palsy after hip replacement is not correlated to surgical approach; the commonly held view that there is a higher incidence with the posterior approach is not supported by the literature [2, 15]. This reflects the fact that traction is probably the most common cause of sciatic nerve palsy, which is possible with all approaches. The common peroneal division is more susceptible to division than the tibial division, on account of its oblique course, the distal tethering both of its terminal branches and through the fibular arcade at the knee, and finally due to its intrinsic connective tissue structure [17].

Femoral nerve palsy

Femoral nerve palsy after hip replacement is probably under-reported, with the loss of quadriceps function of-

ten overlooked. Of the 50 palsies documented by Ratcliff [15] 16 were of the femoral nerve. The retractor placed around the anterior lip of the acetabulum is the principal cause of palsy during hip arthroplasty. Simmons [19] found a 2.3% femoral neuropathy rate in a series of 440 consecutive hip arthroplasties – all were associated with the anterolateral approach, and placement and management of acetabular retractors was the most common fault. Ratcliffe [15] demonstrated a residual deficit in nearly all cases.

Obturator nerve palsy

The obturator nerve is at risk in any violation of the floor of the acetabulum and may be damaged more frequently than previously realised [9]. Obturator nerve damage may account for obscure patterns of pain following hip arthroplasty. Again, extraneous cement should be excluded in such cases [18].

Recommended preoperative management

A patient undergoing hip replacement surgery should be warned of the risk of nerve palsy as a complication of the procedure. Risk factors for peripheral nerve damage during hip surgery include diabetes mellitus, thyroid disease, hypertension, hereditary sensory and motor neuropathy, anaemia and malignancy. There is no proven benefit of preoperative neurophysiological testing as a predictor of nerve palsy.

Recommended operative management

Neurophysiological monitoring may have a place in the management of high risk cases. Necessian et al. [13] used somatosensory evoked potentials (SSEP) during revision arthroplasty and noted neurological compromise in 32%, most of which was due to excessive traction during exposure of the acetabulum or extremes of positioning of the extremity. Byrne et al. [11] monitored the sciatic nerve during revision hip arthroplasty using SSEPs, and found them useful when lengthening the limb. Reckling and Porter [3], also using SSEPs, found femoral reaming and joint reduction to be related to abnormal function in the surrounding peripheral nerves.

However, intraoperative SSEPs may not be effective – Rasmussen et al. [14], in a series of 290 patients monitored by SSEPs while undergoing hip replacement surgery, found that the method was not effective in predicting or preventing sciatic nerve palsy. In reality, intraoperative neurophysiological monitoring should be reserved for high risk cases, e.g. for lengthening of the limb in developmental hip dysplasias.

To avoid iatrogenic injury to peripheral nerves during hip replacement surgery, we recommend:

- (a) A thorough awareness of the surgical anatomy of the region
- (b) Care with the use of retractors, especially around the anterior lip of the acetabulum
- (c) That the sciatic nerve should be identified during revision hip surgery
- (d) That the limb should be supported throughout the operation with great care
- (e) Excessive traction should be avoided, especially during dislocation of the hip
- (f) Neurophysiological monitoring of the sciatic nerve in high risk cases

Recommended postoperative management

The distal neurovascular status of the operated limb should always be checked in the recovery room following surgery. Importantly, this needs to be repeated during the first postoperative week (e.g. haematoma may compress the nerve over a delayed period). There is often a delay in diagnosis, and this is especially true of femoral nerve palsy.

Recommended action to take in cases of nerve palsy

Medico-legal actions more often involve a failure to diagnose the palsy than the fact that it has been induced. The first step in management is the accurate diagnosis of the palsy, especially with regard to the level of the lesion; thus, the following actions should be taken:

- (a) Preoperative risk factors should be evaluated.
- (b) Knowledge of the operative details from the operating surgeon is essential, e.g. difficulties with dislocation, femoral cortical penetration and acetabular penetration.
- (c) A radiograph of the region is essential to exclude cement extrusion, acetabular penetration and to assess prosthesis positioning.
- (d) Neurophysiological tests have no role to play in acute management – the tests will frequently be normal for up to 3 weeks following injury, and even if abnormal, will offer little guidance on diagnosis and management. Delay while such tests are performed should not hinder prompt exploration if indicated.

The most difficult management decision when faced with an acute palsy is that of whether to explore the nerve lesion, and if so, when. The wound should always be explored if there is any suggestion of intraneural or extraneural haematoma; prompt decompression as a surgical emergency may significantly improve outcome. In addition, if there is the possibility of cement extrusion, damage by knife, suture or diathermy, prompt exploration is important. For all the above, the single most accurate diagnostic feature is the presence of pain in the distribution of the affected nerve. Pain suggests a continu-

ing insult to the nerve. We therefore recommend that all nerve palsies associated with pain are explored urgently.

The lesion with little or no pain is more difficult to manage. The benefits of exploration must be balanced against the risks of reoperation in an often elderly patient, combined with the risks of the introduction of sepsis at a second procedure. Skin protection and physiotherapy are important in the acute management of these cases. A foot-drop splint should be applied if possible, although distal oedema should not cause insensate skin to abrade against the splint.

Conclusions

1. There is a high subclinical nerve palsy rate in total hip replacement.
2. Preoperative warning of the risk of palsy should be given.
3. Medico-legally, what is often considered negligent is not that a palsy has occurred, but the faulty management of the palsy postoperatively.
4. It is the painful palsy that should be explored as an emergency. Non-painful palsies are more difficult to manage; the lesser chance of neurological improvement following exploration should be balanced against the risks of additional surgery and sepsis in an often elderly patient.
5. The incidence of nerve palsy appears higher since the introduction of routine thrombo-embolic prophylaxis. Haematoma as a cause of the palsy should be excluded in all cases.
6. Neurophysiological testing in the acute palsy is not indicated and delays prompt treatment.
7. Neurological function should be monitored in the recovery room and in the early postoperative course (up to 1 week), and this fact documented in the medical records.

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