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Review Article

Peroneal Nerve Palsy After Total Knee Arthroplasty

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Peroneal nerve palsy is a potentially devastating complication of total knee arthroplasty (TKA). Fortunately, review of the literature reveals that this is not a common problem. Retrospective studies examining a large number of consecutive TKAs (> 1000) performed at various institutes show an incidence ranging from 0.3% to 1.3% (Table 1).

In smaller studies, the incidence can vary more widely. The reported incidences have been as low as 0% and as high as 10%. This variability may be caused by the specific patient population enrolled in the studies [5-7] or the specific prostheses used in the studies [8,9]. In addition, the number of surgeons involved and their experience may have a greater impact on the incidence in studies with small sample sizes.

The reported clinical incidences may not represent the true incidence of peroneal nerve palsy. The actual incidence of nerve compromise may be underestimated because of the presence of subclinical palsy, which may only be diagnosed by means of electrodiagnostic tests such as electromyography (EMG) or nerve conduction studies. Moreover, the different methods of gathering data (chart review vs follow-up vs complication reports) may also result in discrepancies between reported and true incidences. We found no prospective studies specifically addressing these issues.

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Predisposing Factors

Several preoperative, perioperative, and postoperative factors have been reported to be significant for the development of peroneal nerve palsy after TKA. However, no single entity has been consistently shown in all studies to be significant, and some patients without any known risk factor still develop peroneal nerve palsy. Because of the lack of establishment of a cause-effect relationship and yet unidentified risk factors, it is best to be aware of all of the following risk factors and avoid this troublesome complication. The etiology of peroneal nerve palsy is multiple, and the relative contributions of the different risk factors, if present within a single patient, are so far unknown.

Valgus Deformity and/or Flexion Contracture

Early experiences with TKA have resulted in isolated cases of peroneal nerve palsy that were assumed to be caused by the correction of severe valgus deformity and/or flexion contracture [10,11]. Subsequent studies that sought to identify specific risk factors for peroneal nerve palsy have found that these preoperative deformities are indeed significant risk factors [1-3,12]. The studies that support the role of preoperative valgus deformity defined the term *valgus* differently; it ranged from 10° or higher to 15° or higher valgus angulations. In these studies, the average preoperative valgus angulations in the patients who developed peroneal nerve palsy ranged from 18° to 23.3°. In studies that supported the role of preoperative flexion contracture, the average contractures ranged from 15.5° to 22°.

The mechanism by which the nerve injury occurs is thought to be via direct stretching of the nerve or

Table 1. Retrospective Studies Involving More Than 1000 TKAs

Authors	Period of study	No. of TKAs	Incidence (%)
Rose et al [1]	1974-1980	2626	0.9
Asp and Rand [2]	1972-1985	8998	0.3
Idusuyi and Morrey [3]	1979-1992	10361	0.3
Schinsky et al [4]	1970-1998	1476	1.3

via stretching of the soft tissue surrounding the nerve with consequent compression of the nerve or its vascular supply, which is possible to occur during the correction of a severely deformed knee [13-15]. Information that support this hypothesis exist. Severe histological damage has been shown to occur in the axons after 4% to 11% elongation of a human nerve [16,17], and impaired microcirculation of a nerve can be seen after 8% elongation in the tibial nerve of rabbits [15]. It is thought that the stretching narrows both the extraneural and intraneural microvasculatures and results in impaired blood flow [15]. It has also been hypothesized that the more deformed knees require more extensive soft tissue dissections, which increase traction on the nerve or compromise its vascular supply [1].

The importance of vascular supply to the nerve from the surrounding tissues is debatable. The sciatic nerve of the rabbit undergoes extensive degeneration after ligation of the gluteal artery [18], whereas it has been shown in cats that ligation of all vessels to the sciatic nerve did not impair nerve conduction [19]. Hence, 2 schools of thought exist regarding peripheral nerve suturing—that which advocates wide exposure and mobilization of nerve during the operation [20,21] and that which advocates minimized mobilization [22,23].

However, not all studies found the preoperative valgus deformities and/or flexion contractures to be significant risk factors for the development of peroneal nerve palsy [4,7,9]. Interestingly, the study of Idusuyi and Morrey [3] showed that although valgus deformity was significantly associated with peroneal nerve palsy, flexion contracture was not. In Miyasaka et al's study [7] of 60 TKAs performed solely on valgus knees (the average valgus angulation was 16.8°), there were no peroneal nerve palsies. This study, however, was not performed to identify the risk factors of the peroneal nerve palsy or to contest the role of preoperative deformities; the authors attributed the lack of palsy to prophylactic flexion of all knees in the recovery room.

Although there are a few studies that do not support the role of preoperative deformities in the development of peroneal nerve palsy, most of the data in the orthopedic literature suggest otherwise. To our knowledge, there are no prospective studies looking into the role of preoperative deformity in postoperative peroneal palsy.

Postoperative Epidural Analgesia

The first case of peroneal nerve palsy after administration of postoperative epidural analgesia was reported in a patient who had undergone exploratory laparatomy [24]. The authors attributed the palsy to the resting of the fibular head against the bed rail with subsequent pressure on the peroneal nerve, which the patient was unaware of because of the effects of epidural analgesia. The sensory block (including proprioception) achieved by the epidural analgesia may cause the patient to unwittingly rest the limb in a position that directly compresses the peroneal nerve at the fibular head level. It may also tolerate constrictive dressing and excessive extensions on continuous passive motion machines, which may predispose to injury and prevent the early diagnosis of peroneal nerve palsy [3,12].

Since that first case report, Idusuyi and Morrey [3] found that epidural analgesia was significantly associated with peroneal nerve palsy after TKA. Moreover, most of the diagnoses were made later (only 1 of 32 palsies discovered on the day of operation) than in the previous study of Rose et al [1] (8 of 23 palsies discovered on the day of operation). This study [1] did not provide any anesthesia data. Although the study of Horlocker et al [12] did not find the relationship between epidural analgesia and peroneal palsy to be significant, all cases of severe peroneal nerve palsy that did not recover completely were in the epidural analgesia group and were diagnosed only after discontinuation of the analgesia. Although the association was not significant, the authors theorized that it might indeed be a relative risk factor. The delay in diagnosis of peroneal nerve palsy was seen in a previous study by Asp and Rand [2] in which most of the palsies were diagnosed on postoperative day 2. The delay in diagnosis mentioned in these studies supports the role of epidural analgesia in the development of peroneal nerve palsy by the aforementioned postoperative mechanism, but the delay may simply be caused by the analgesia's masking of the injury incurred by other means, which is made apparent only after the analgesia is discontinued [3].

This relationship between postoperative epidural analgesia and peroneal nerve palsy was not supported in all studies; Schinsky et al [4] found that none of the 19 of 1467 TKR patients who developed peroneal palsy was exposed to postoperative epidural analgesia and that, moreover, 79% of the palsies were diagnosed early while the patients were in the recovery room.

Previous Neuropathy

Previous neuropathy, whether occurring centrally (ie, spinal stenosis, lumbar radiculopathy) or peripherally (owing to nerve injury away from the spinal origin), has been significantly associated with the development of peroneal nerve palsy. These neuropathies were diagnosed preoperatively either clinically or via EMG studies [3,12]. It is plausible that a nerve that is already compromised may be more susceptible to a second insult. Upton and McComas [25] set forth the concept of a "double-crush" phenomenon and hypothesized that the decreased axoplasmic flow occurring as a result of a proximal lesion makes the nerve more vulnerable to a distal injury. It is interesting that although diabetes is a prevalent chronic illness that is associated with a progressive peripheral neuropathy, it has not been shown to be a risk factor associated with peroneal nerve palsy after TKA [2-4,12].

Rheumatoid Arthritis

Some studies have shown that rheumatoid arthritis is significantly associated with the development of peroneal nerve palsy after TKA [4,5]. Unlike the low incidence of 0.3% to 1.3% seen in large retrospective studies, Knutson et al's study [5] of 42 TKAs performed solely on rheumatoid knees showed clinical and subclinical peroneal nerve palsy (as diagnosed via EMG) in 7 knees, which is an incidence of 16.7%.

Knutson et al [5] implied that the effect of rheumatoid arthritis on the development of peroneal nerve palsy was via indirect risk factors already discussed—the preoperative valgus deformity and flexion contracture. The palsy group patients required significantly more correction of deformities than did patients from the nonpalsy group (mean valgus correction of 7° in the palsy group vs 0° in the nonpalsy group; flexion contracture correction of 20° in the palsy group vs 5° in the nonpalsy group). However, the mean correction of valgus deformity of rheumatoid knees in Knutson et al's study [5] (7°) was no more than that in Rose et al's data [1] (14°), which did not show rheumatoid arthritis to be a significant risk factor. Schinsky et al [4] found rheumatoid arthritis and not preoperative valgus deformity and flexion contracture to be the significant risk factor for peroneal nerve palsy. Thus, it is possible that the predisposition of rheumatoid knees to developing peroneal nerve palsy may be via a mechanism unrelated to the deformity of the knee.

Rheumatoid arthritis is an illness known to be associated with neuropathies, and these preexisting neuropathies may predispose to nerve injury during TKA. Although often asymptomatic, the peroneal nerve in rheumatoid arthritis patients has been known to show slowed motor nerve conduction velocity and abnormal sensory conduction in the superficial peroneal nerve branch [26-28].

Tourniquet

The early literature of extremity surgery recognized nerve palsy as a complication of tourniquet use, but the incidence was believed to be low [29-31]. According to Rudge [31], the advent of the pneumatic tourniquet made the incidence even more uncommon. However, the pneumatic tourniquet used in lower-extremity surgery has been associated with not uncommon occurrences of EMG-evident nerve injury, especially in knee meniscectomy surgeries [32,33]. Some experiments have shown deleterious effects of tourniquet application on the function and structure of the peripheral nerve; both the compressed segment directly underneath the cuff and the uncompressed area distal to the cuff are affected. The mechanism appears to be caused by both ischemia and mechanical deformation with more prominent functional and histological changes seen in the area directly underneath the cuff [19,34-36].

In studies of patients who underwent knee meniscectomy, EMG changes were seen in 71% to 75% of patients whose surgery was performed with the pneumatic tourniquet. The tourniquet pressures ranged from 300 to 450 mm Hg, and the average tourniquet times ranged from 44 to 59 minutes. One study examining EMG changes 3 weeks postoperatively noted that most changes were in the distribution of the femoral nerve, with tibial nerve involvement being the next most common [32]. Another study, which examined EMG changes 6 weeks postoperatively, noted that most changes were in the distribution of the tibial nerve [33]. The EMG changes in both studies

resolved to normal 6 months following the surgical procedure. An important question is whether the EMG changes in these studies were clinically significant. None of the 2 studies correlated EMG changes with physical examination immediately postoperatively. Moreover, no sensory examinations were performed. Dobner and Nitz [33] reported a functional capacity (as measured by vertical leap) of 39% in normal legs at 6 weeks as opposed to that of 79% in control legs that were not exposed to the pneumatic tourniquet. Because a vertical leap is a physically demanding test, a patient showing a 39% functional capacity may be clinically indistinguishable from a patient showing a 79% functional capacity if tested by a routine physical examination. Thus, it is possible that the 2 groups may not have shown any obvious physical examination differences during the routine physical examinations performed immediately in the postoperative period.

Most studies that sought to identify risk factors for the development of peroneal nerve palsy after TKA showed that pneumatic tourniquet pressure and duration of application were not significant risk factors [1,3-5]. The tourniquet pressures of patients who developed peroneal nerve palsy in these studies ranged from 300 to 450 mm Hg and the duration ranged from 82 to 129 minutes, which is more than the duration experienced by meniscectomy patients. One study in which 8 of 361 TKA patients developed peroneal palsies found that in addition to a preoperative valgus deformity of 10° or higher, preexisting neuropathy, and postoperative bleeding, total tourniquet time longer than 120 minutes was a significant risk factor for development of peroneal palsies ($P < .05$) [12]. The tourniquet pressure in this study was not reported.

The tourniquet is an important part of extremity surgery. Its use is critical in achieving adequate hemostasis in many surgical procedures that require clear visualization of structures. Its use may also be required in procedures to minimize blood loss and operative time, but its use is not without complications. The complications include postoperative swelling, nerve injury, wound hematoma, vascular injury, tissue necrosis, and even cardiovascular complications [37]. The concept of safe duration of tourniquet time is controversial. Values ranging from 1 hour to even as high as 3 hours are quoted in the literature [38-41]. In practice, tourniquet use limited to 2 hours or less is considered safe based on studies assessing neural and muscular injury [19,29,42-44] and continues to be used until a randomized trial can define the optimal time.

Although most of the studies performed to identify the risk factors for peroneal nerve palsy after TKA did not find the use of tourniquet to be a significant risk factor if the duration was 2 hours or less, one study [12] with a higher average tourniquet time (141 minutes) showed otherwise. This fact combined with the known complications of tourniquet use advocates constant caution whenever one uses a tourniquet during surgery.

Constrictive Dressing

The superficial anatomical location of the peroneal nerve as it winds around the fibular head makes it especially vulnerable to compression injury. Although difficult to prove, it is plausible that application of constrictive dressing after the operation may play a role in the development of peroneal nerve palsy after TKA [8,9].

Hematoma

In several studies, authors attributed formation of hematoma postoperatively at the wound site along with the resultant compression on the peroneal nerve as the cause of the nerve palsy. This, however, represented only a small percentage of peroneal nerve palsy in the studies [2,3,5]. One study even found postoperative bleeding complication as a significant risk factor in the development of peroneal nerve palsy after TKA [12].

Prognosis

The potential for complete recovery once peroneal nerve palsy is diagnosed after TKA differs among various studies (Table 2).

Except for Rose et al's study [1], other studies show that at least 50% of patients with peroneal nerve palsy made complete recoveries. Moreover, the less severe the initial peroneal nerve palsy, the

Table 2. Percentage of Patients Making Complete Recovery

Authors	Percentage of complete recovery (%)	Length of follow-up
Rose et al [1]	9.1	6 mo-7 y
Webster and Murray [9]	60	6 mo
Asp and Rand [2]	50	5.1 y
Idusuyi and Morrey [3]	50	3.9 y
Schinsky et al [4]	68	18 mo

higher the likelihood of complete recovery and higher the knee scores [2,3]. Although Rose et al's study [1] showed a very low percentage of complete recovery, the Hospital for Special Surgery knee scores in the peroneal nerve palsy group averaged 81.6 compared with 83.3 of the entire study population. A similar Hospital for Special Surgery knee score in the peroneal nerve palsy group (81) is seen in Asp and Rand's study [2], which showed 50% of peroneal nerve patients showing complete recovery. This implies that regardless of the percentage of complete recovery, patients demonstrated good functional capacity after TKA [1].

Treatment

Once peroneal nerve palsy is diagnosed after TKA, immediate removal of any constrictive dressing and flexion of knees are recommended [1,2]. Theoretically, this maneuver releases the compression caused by constrictive dressing and relieves the tension on the peroneal nerve. However, at best, a partial improvement is seen and most patients do not show any improvement after these maneuvers are performed.

A more invasive intervention of exploration and decompression of peroneal nerve has been advocated by Krackow et al [45]. The authors recommended operative management for any patient whose palsy, especially if severe, does not improve after 3 months as evidenced by clinical assessment or EMG. They performed operative exploration and decompression of the peroneal nerve on 5 patients who failed nonoperative management 5 to 45 months after their index TKA. All 5 patients demonstrated improved nerve function and 4 of them had a full peroneal nerve recovery. However, the beneficial outcome of this procedure has not been substantiated by the experience of other authors and its routine use remains controversial.

Summary

Peroneal nerve palsy after TKA, although uncommon, can be a troublesome complication for patients and surgeons. The predisposing factors must be recognized and meticulous surgical techniques and vigilant postoperative care must be undertaken to minimize their effects. Although effective intervention still remains to be determined, most patients do proceed to complete recovery from the nerve palsy but demonstrate good functional capacity.

To our knowledge, there are no prospective studies that clearly elucidate the effect of the previously mentioned risk factors on the incidence of peroneal nerve palsy after TKA. More research is needed to tease out these risk factors and explore treatment strategies. A prospective randomized controlled study may help determine the optimal treatment strategy for this important clinical problem.

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