

Systematic review

Common Peroneal Nerve Injury and Recovery after Total Knee Arthroplasty: A Systematic Review

Christopher N. Carender, MD^{*}, Nicholas A. Bedard, MD, Qiang An, MS, Timothy S. Brown, MD

Department of Orthopedics and Rehabilitation, University of Iowa Hospitals and Clinics, Iowa City, IA, USA

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ABSTRACT

Background: Common peroneal nerve palsy (CPNP) after total knee arthroplasty (TKA) may impact extremity pain and function. Incidence and rates of recovery of CPNP after TKA vary in the current literature. The purpose of this systematic review was to evaluate the incidence of incomplete and complete CPNP after TKA and rates of incomplete and complete recovery of nerve function in the absence of further surgical treatment.

Methods: PubMed, Embase, and Cochrane Central were searched for studies published in the years 1970–2019. Studies evaluating incidence and recovery rates of CPNP in the absence of further surgical treatment were screened according to inclusion and exclusion criteria. Outcomes of interest included incidence of complete and incomplete CPNP and rates of incomplete and complete nerve recovery.

Results: Eleven studies were included for qualitative analysis. In total, there were 47,585 TKAs performed, with 203 postoperative CPNPs, for a cumulative incidence of 0.4%. One hundred twenty-nine CPNPs were classified as complete or incomplete palsies. At a mean follow-up of 3.6 years (range, 0–11 years), 24 (39%) complete CPNPs had complete recovery, 34 (56%) had incomplete recovery, and 3 were lost to follow-up. In contrast, 45 (66%) with incomplete CPNPs had complete recovery, 18 (27%) had incomplete recovery, and 5 patients were lost to follow-up.

Conclusions: Incidence of CPNP after TKA was 0.4%. Recovery of nerve function after CPNP in the setting of TKA varies by the degree of initial nerve palsy. These data may be used to inform decisions on further interventions and for the purposes of perioperative patient counseling after TKA.

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Introduction and background

Common peroneal nerve palsy (CPNP) after total knee arthroplasty (TKA) is relatively rare, with estimates of its incidence ranging from 0.01% to 4.3% [1–8]. As the volume of TKA continues to increase, more cases of CPNP after TKA are likely to be encountered [9]. CPNP may have a significant negative impact on extremity function and patient-reported outcomes (PROs) after TKA [1,10]. Injury to the common peroneal nerve may act as a neuropathic pain generator that negatively impacts performance of activities of daily living [10]. Historically, CPNP after TKA has been cited as a reason for surgeon litigation [11].

Numerous potential risk factors for CPNP after TKA have been proposed, including the use of epidural anesthesia [2,12], preoperative valgus deformity [2], preoperative flexion contracture, higher body mass index [5], and history of diabetes mellitus [8]. However, these proposed risk factors are inconsistently supported in current literature [3,4,7,12–14]. For this reason, the natural history of CPNP after TKA is incompletely understood [2,5]. Rates of complete nerve recovery without further intervention vary from 20% to 100% [1,2,5–7,14,15]; studies with a predominance of patients with incomplete nerve palsies tend to have higher rates of recovery than studies evaluating patients with complete nerve palsies or incomplete and complete palsies together [1,2,5,6,15]. Given the wide variation in published rates of nerve recovery, decision-making about the need for further intervention (ie, common peroneal nerve neurolysis) is often challenging. The relatively low incidence of CPNP makes the study difficult, requiring evaluation of a high volume of patients. Thus, the incidence of CPNP and rates of common peroneal nerve recovery may be better understood

^{*} Corresponding author. Department of Orthopedics and Rehabilitation, University of Iowa Hospitals and Clinics, 200 Hawkins Dr, 01008 JPP, Iowa City, IA 52242, USA. Tel.: +1-319-353-7550.

E-mail address: christopher-carender@uiowa.edu

through a systematic review. The purpose of this systematic review was to evaluate the incidence of incomplete and complete CPNP after TKA as well as rates of incomplete and complete recovery of CPNP in the absence of further surgical treatment.

Material and methods

This study was exempt from institutional review board approval. This study was reported in concordance with the methodology outlined by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (Fig. 1) [16].

Study eligibility

All prospective and retrospective studies evaluating CPNP after primary or revision TKA as discovered by the search algorithm outlined in the following section were included in the initial yield. Exclusion criteria were as follows: (1) unrelated subject matter (including review and technique articles); (2) no clinical follow-up or outcomes reporting after CPNP; (3) no classification of specific nerve palsies (eg, classification or examination of cohorts of any lower extremity palsy without specific data on CPNP); and (4) studies aimed at studying recovery of common peroneal nerve function after surgical neurolysis or decompression.

Search strategy and study selection

Search strategies were developed with the assistance of a health science librarian with expertise in searching for systematic reviews. Searches were developed by the primary author (C.N.C.) and librarian using an iterative process of gathering and evaluating terms. Searches were finalized in June 2019. Comprehensive strategies, including both index and keyword methods, were devised for the following databases: PubMed, Embase (Elsevier platform), and Cochrane Central (Wiley). To maximize sensitivity, no pre-established database filters were used other than the English language filter. The full PubMed search strategy included grouping terms, with the use of both Medical Subject Headings (MeSH) and text word inputs, into 3 groups. Searches were then conducted with a formula of (group 1 OR group 2) AND group 3. Search terms were as follows: (1) “Peroneal Nerve” [MeSH] OR “Peroneal Neuropathies” [MeSH] OR “Peripheral Nervous System Diseases” [MeSH] OR “Peripheral Nerve Injuries” [MeSH] OR “Paralysis” [MeSH] OR “Pressure/adverse effects” [MeSH]; (2) peroneal nerve [Text Word] OR peroneal nerves [Text Word] OR fibular nerve [Text Word] OR fibular nerves [Text Word] OR peroneal palsy [Text Word] OR peroneal palsies [Text Word] OR peroneal paralysis [Text Word] OR peroneal entrapment [Text Word] OR peroneal entrapments [Text Word] OR peroneal mononeuropathy [Text Word] OR peroneal mononeuropathies [Text Word] OR foot drop [Text Word] OR foot

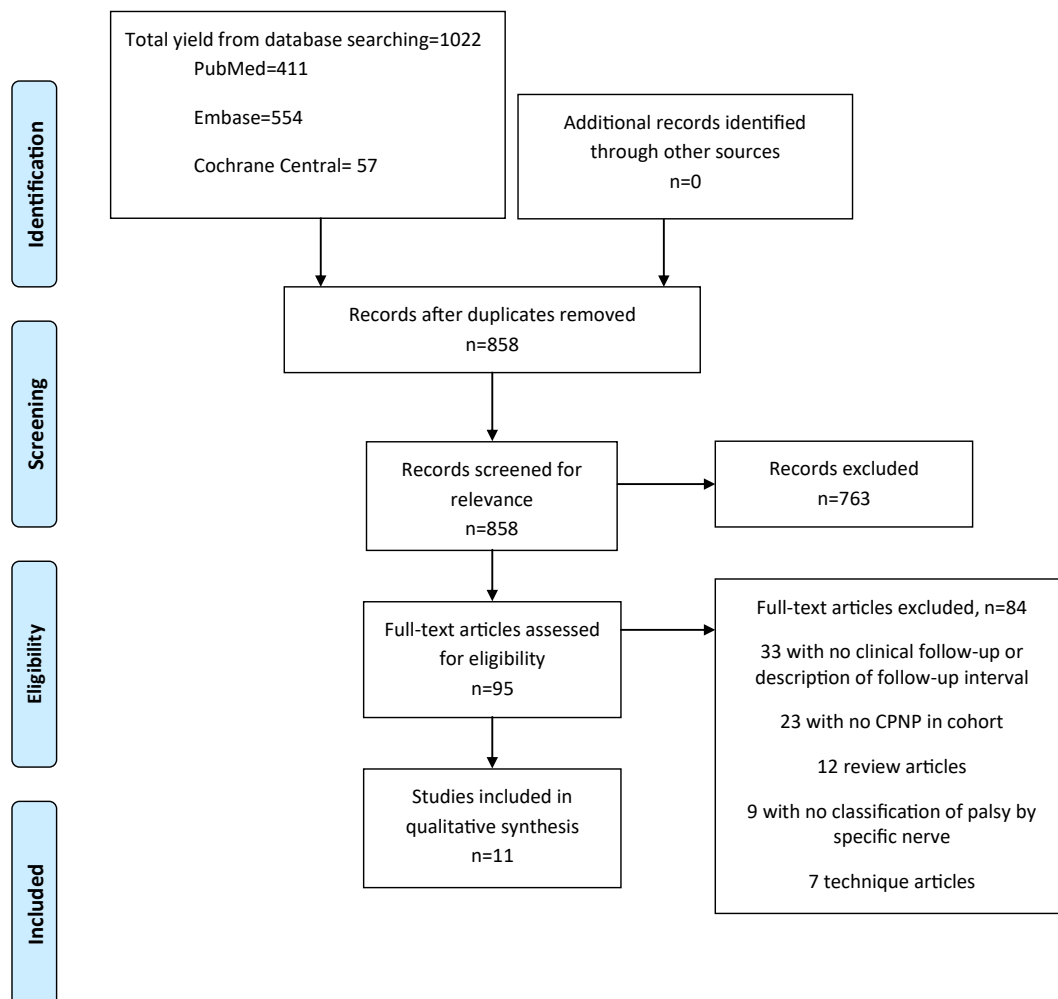


Figure 1. PRISMA flow diagram. The flow diagram of study selection per guidelines from the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) group.

drops[Text Word] OR peripheral nerve injury [Text Word] OR peripheral nerve injuries [Text Word] OR neuropathy [Text Word] OR neuropathies [Text Word]; (3) “Arthroplasty, Replacement, Knee” [MeSH] OR “Knee Joint/surgery” [Mesh] OR “Knee Prosthesis” [MeSH] OR knee replacement [Text Word] OR knee arthroplasty [Text Word] OR knee arthroplasties [Text Word] OR TKA [Text Word] OR Total Knee [Text Word]. The PubMed search strategy was adapted for use with the other electronic databases. Complete search strategies are available on request. Duplicates were removed by the librarian using an approach to ensure accuracy and prevent accidental loss of records. This process was facilitated by citation management software, supplemented by manual review of records.

The initial search, after removal of duplicate studies, yielded 858 records (Fig. 1). All records were reviewed by the primary author (C.N.C.). Screening based on the study abstract text resulted in removal of 763 studies. Ninety-five full-text articles were reviewed; 84 articles were excluded for the following reasons: 33 with no clinical follow-up available, 23 with no CPNP in the cohort, 12 review articles, 9 with no classification of palsy by specific nerve (eg, studies evaluating any lower extremity nerve palsy in one cohort), and 7 technique articles. Eleven articles remained for qualitative analysis.

Study definitions

Common data elements from studies included in the systematic review were used to create definitions for preoperative valgus deformity, varus deformity, and flexion contracture. For the purposes of this study, a preoperative valgus deformity was defined as $\geq 12^\circ$ [2,5-7]. A preoperative flexion contracture was defined as $\geq 5^\circ$ [6]. Preoperative varus deformity was defined as being $< 0^\circ$ [2,5,6]. Lumbar surgery was defined as previous laminectomy or posterior spinal fusion. Classification of complete vs incomplete palsy was made in accordance with the definition described by Park et al. [5]: complete palsy was defined as no motor function and complete loss of sensation in the neurotome; incomplete palsy was defined as greater or equal to grade 1 motor strength with full or partial sensation about the neurotome. Multiple studies included no definition of classification or description of the palsy type; such palsies were exempt from classification as complete or incomplete [6,14,17-19]. In all studies, classification of the degree of palsy into complete or incomplete was made at the time the CPNP was diagnosed. Complete recovery was defined as full resolution of sensory deficits in the neurotome and the return of full motor strength. Incomplete recovery was defined as the presence of a residual sensory and/or motor deficit. Timing of classification of the degree of nerve recovery varied between patients with complete and incomplete recovery. Patients with complete recovery were deemed to have complete recovery on the date they exhibited full motor strength and no sensory deficiencies on examination by their surgical team; patients with incomplete recovery were deemed to have incomplete recovery at their most recent follow-up visit before publication of each respective study.

Data extraction

Data were extracted from each study by the primary author (C.N.C.). Study author(s), journal of publication, year of data collection, and year of publication were noted for each study. The following data elements were extracted from each study cohort (if available): the number in cohort, number of CPNP, degree of palsy (complete vs incomplete), common peroneal nerve recovery (complete vs incomplete), preoperative diagnosis, gender, age at surgery, follow-up time (reported from the date of surgery), number lost to follow-up, preoperative valgus deformity, preoperative

varus deformity, preoperative flexion contracture, history of diabetes mellitus, history of peripheral neuropathy, history of prior tibial osteotomy, history of lumbar surgery, mean tourniquet time, range of tourniquet time, mean postoperative day (POD) of diagnosis, range of POD of diagnosis, type of anesthesia used, use of postoperative epidural anesthesia, evaluation of CPNP using electromyography, and any additional surgical treatment for CPNP.

Statistical analysis

Results from individual cohorts from each study were combined, and descriptive statistics are presented. We gathered 11 studies' data sets from our systematic review. A meta-analysis was attempted; however, there are only 3 studies with common data elements that could be analyzed. We used the I^2 statistic to assess heterogeneity of each common data element. A P value of $< .1$ was used to be the cutoff for heterogeneity. Analysis was performed using IBM SPSS Statistics for Macintosh (version 22.0; IBM Corp, Armonk, NY) and metafor (version 2.1; R package, version 3.6.1. Open source software: Wolfgang Viechtbauer).

Results

Eleven studies were reviewed (Table 1). In total, there were 47,585 TKAs performed, with 203 postoperative CPNPs, for a cumulative incidence of 0.4% (Table 1). The remainder of the results discusses only patients who developed CPNP.

The mean follow-up was 3.6 years (range, 0-11 years); 22 patients were reported as being lost to follow-up. The mean age at surgery was 63 ± 3 years. There were 35 (17%) male patients, 83 (41%) female patients, and 85 (42%) patients with unknown gender. Preoperative diagnoses included 97 (48%) cases with primary osteoarthritis, 32 (16%) with rheumatoid arthritis, 5 (2%) with post-traumatic osteoarthritis, 6 with other (3%), and 63 (31%) with unknown preoperative diagnosis. Primary TKA was performed in 143 (71%) knees and revision TKA in 15 (7%) knees; primary or revision arthroplasty status was unknown in 45 knees. Eighteen cases in the cohort had a history of diabetes mellitus, 2 had a history of peripheral neuropathy, 5 underwent a prior ipsilateral high tibial osteotomy, and 6 cases underwent a prior lumbar spine surgery.

Of the 203 postoperative CPNPs, 129 palsies were able to be classified as complete ($n = 61$) or incomplete ($n = 68$). At a mean follow-up of 3.6 years (range, 0-11 years), 24 (39%) patients with complete CPNP had complete recovery, 34 (56%) patients had incomplete recovery, and 3 (5%) patients were lost to follow-up. In contrast, 45 (66%) patients with incomplete CPNP had complete recovery, 18 (27%) patients had incomplete recovery, and 5 (7%) patients were lost to follow-up (Fig. 2). All (129 of 129) patients with classifiable nerve injury and follow-up data demonstrated at least minor recovery in nerve function over their respective follow-up periods.

One hundred and twenty-seven patients had available data regarding preoperative coronal plane alignment: 43 (34%) knees were valgus, 35 (28%) knees were varus, and 49 (38%) knees were neutral. Preoperative flexion contracture was present in 33 of 146 (23%) knees.

The mean tourniquet time for the cohort was 102 minutes (range, 13 to 255 minutes). Thirty-two (16%) patients underwent general anesthesia, 12 (6%) patients underwent spinal anesthesia, 60 (30%) patients received epidural anesthesia, and 99 (48%) patients had an unknown anesthetic type. The mean POD of diagnosis of CPNP was POD2 (range, POD 0 to 23). Sixty-five patients underwent postoperative electromyography as part of the workup of CPNP. Of 203 postoperative CPNPs included in the study, 2 were managed surgically (one palsy from the cohort of Park et al. [5]

Table 1
Study demographics and incidence and definitions of common peroneal nerve palsy.

Authors	Pub. year	Study year	No. of TKAs	No. of palsies	CPNP incidence (%)	CPNP definition
Asp and Rand [1]	1990	1972-1985	8998	26	0.3	Complete motor deficit—no contraction of peroneal innervated muscles Partial motor deficit—decreased strength but retained ankle or toe dorsiflexion Complete sensory deficit—profound sensory deficit on dorsum of the foot Partial sensory deficit—less than complete loss of sensation on dorsum of the foot
Coventry et al. [15]	1973	1971-1972	317	3	1	None
Horlocker et al. [13]	1994	1992-1993	361	8	2.2	Complete motor deficit—no contraction of peroneal innervated muscles Partial motor deficit—decreased strength but retained ankle or toe dorsiflexion Complete sensory deficit—complete loss of sensation on dorsum of the foot Partial sensory deficit—less than complete loss of sensation on dorsum of the foot
Idusuyi and Morrey [2]	1996	1979-1992	10,361	32	0.3	Per Asp and Rand [1]
Kaushal et al. [17]	1976	1972-1974	70	3	4.3	None
Nielsen et al. [18]	1985	1979-1981	247	3	1.2	None
Park et al. [5]	2013	2000-2008	8303	44	0.5	Complete injury—grade 0 muscle strength; loss of sensation in CPN neurotome Incomplete injury—disruption of motor or sensory function with grade 1 or better motor function and presence of partial or full sensation in CPN neurotome
Rose et al. [6]	1982	1974-1980	2626	23	0.9	None
Schinsky et al. [7]	2001	1970-1998	1476	19	1.3	Per Asp and Rand [1]
Speelziek et al. [14]	2019	1996-2016	14,450	37	.3	None; stratified by motor involvement or isolated sensory disturbance
Webster et al. [19]	1985	1974-1980	376	5	1.3	Loss of sensory function, motor function, or both
Total			47,585	203	0.4	

Pub., publication.

underwent surgical decompression of a postoperative hematoma around the nerve, and one palsy from the cohort of Asp and Rand [1] underwent nerve exploration and surgical decompression). Rose et al. [6] noted 2 patients with severe preoperative flexion contracture ($\geq 30^\circ$) and 3 patients with severe preoperative valgus deformity ($\geq 27^\circ$) who underwent prophylactic common peroneal nerve neurolysis at the time of TKA; CPNP occurred in all 5 patients despite prophylactic neurolysis.

I^2 statistics for heterogeneity of eligible studies containing the following common data elements: history of diabetes mellitus ($I^2 = 0\%$, $P = .45$); history of high tibial osteotomy ($I^2 = 0\%$, $P = .99$); preoperative valgus $>12^\circ$ ($I^2 = 46\%$, $P = .16$); and postoperative epidural use ($I^2 = 53\%$, $P = .12$).

Discussion

CPNP after TKA is a relatively rare complication [1,2,5,6,19], with a mean incidence of 0.4% between studies included in the present systematic review (range, 0.3%–4.3%). Prior studies evaluating greater than 5000 TKAs [1,2,5,14] all found an incidence of CPNP of $<0.5\%$. In spite of the rare incidence, an increasing volume of CPNPs may be seen in coming years as the national volume of primary and revision TKAs continues to rise [9].

Previously published rates of recovery of CPNP after TKA are highly variable, from 20% to 100% [1,2,5-7,14,15]. Studies with a greater number of incomplete palsies tend to have higher published rates of recovery relative to studies with increasing numbers of complete palsies. In the present systematic review, at a mean follow-up of 3.6 years, patients with a complete CPNP went on to full recovery in approximately 40% of cases and incomplete recovery in approximately 55% of cases. Patients with an incomplete CPNP went on to full recovery in about 65% of cases and incomplete recovery in about 25% of cases. Idusuyi and Morrey [2] evaluated a

cohort of 32 patients who developed CPNP after TKA at a mean follow-up of approximately 4 years. Seventeen patients had incomplete palsies, and 15 patients had complete palsies [2]. Incomplete palsies achieved complete nerve recovery in 76% of cases, whereas complete palsies achieved complete nerve recovery in only 20% of cases [2]. Similarly, Park et al. [5] noted incomplete palsies to have a recovery rate of 75% and complete palsies to have a recovery rate of 20%; however, the mean follow-up time for this cohort was only 7 months. In a study of 8998 TKAs and 26 CPNPs, Asp and Rand [1] noted that incomplete palsies achieved complete recovery at a rate of 86% at a mean follow-up of 3.2 years, whereas complete palsies achieved complete recovery at a rate of 35%; this difference was statistically significant ($P < .04$). Thus, if an incomplete palsy represents a less-severe nerve injury relative to a complete palsy, then the degree of initial injury seems to influence the chances of a complete nerve recovery. In a 2013 study, Park et al. [5] evaluated prognostic factors for recovery of common peroneal nerve function after palsy associated with TKA; a more severe initial insult to the common peroneal nerve was a negative prognostic factor for nerve recovery. The authors also made note of the significant time often required for nerve recovery: 62% of patients did not reach maximal recovery until after 12 months postoperatively, and 14% of patients required >2 years from surgery to achieve maximal recovery [5]. Interestingly, all (129 of 129) patients with classifiable nerve injury and follow-up data in the present study demonstrated at least minor recovery in nerve function over their respective follow-up period. However, the clinical significance of these minor improvements on an individual patient level is unknown.

Low incidence of CPNPs makes studying risk factors for development of CPNP after TKA difficult. As such, nearly all proposed risk factors for development of CPNP are incompletely supported in the literature [3,4,7,12-14]. Preoperative valgus deformity corrected at

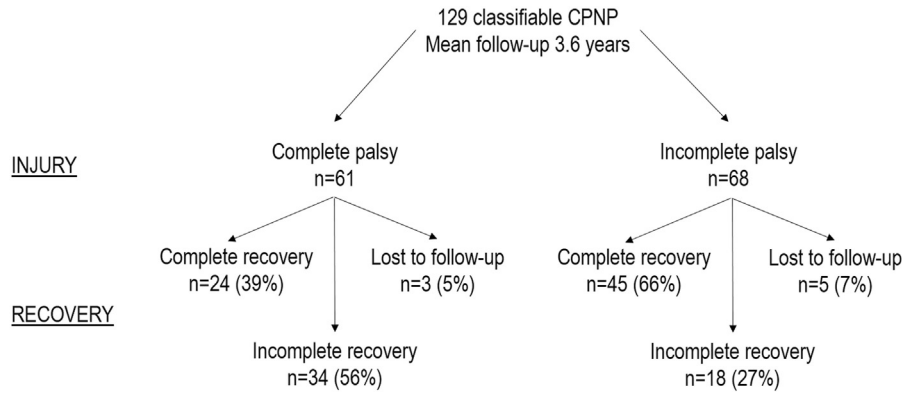


Figure 2. Rates of recovery of incomplete and complete common peroneal nerve palsy. One hundred twenty-nine CPNPs from 6 studies [1,2,5,7,13,15] were included in this analysis. Outcomes shown occurred at a mean follow-up of 3.6 y after surgery (range, 0–11 y). Data are reported in percentages of the total number of classifiable complete or incomplete palsies.

the time of surgery may create a neurapraxia on the common peroneal nerve, leading to CPNP [2,4]. Idusuyi and Morrey [2] found a twelve-fold increase in the relative risk of CPNP in patients with preoperative valgus alignment of $\geq 12^\circ$. Rose et al. [6] and Asp and Rand [1] also noted valgus deformity to be commonly associated with CPNP in their respective cohorts; however, no statistical testing evaluating the potential influence of valgus deformity on CPNP was performed in these studies. Park et al. [5] found no association between preoperative valgus deformity and development of postoperative CPNP. Idusuyi and Morrey [2] and Beller et al. [12] noted the use of epidural anesthesia to be a risk factor, hypothesizing that unrecognized pressure on the limb while epidural anesthesia is on board may contribute to nerve compression and palsy development. Horlocker et al. [13] found no increased risk of CPNP with postoperative use of epidural anesthesia but conceded that patients must be monitored carefully in the postoperative period. Diabetic patients with subclinical peripheral neuropathy may have existing nerve injury, with a potentially minor injury during TKA serving as the “second hit” that results in clinical symptoms of CPNP [2,13,20,21]. However, Park et al. [5] and Idusuyi and Morrey [2] found no significant association between a history of diabetes mellitus and development of CPNP. Patients who underwent prior decompressive spine surgery may also develop CPNP by the same mechanism; yet, history of lumbar laminectomy as a risk factor for CPNP is inconsistently supported in the current literature [2,13]. Extended tourniquet times during TKA may result in periods of ischemia for the common peroneal nerve. When tourniquet time is evaluated as a continuous variable, multiple studies have demonstrated no difference between tourniquet times in patients who develop CPNP and those who do not. Prolonged tourniquet time does not seem to influence the rate of nerve recovery; Speelziek et al. [14] noted that tourniquet time of >100 minutes does not significantly impact time to recovery in patients who develop nerve palsy after TKA. Meta-analyses were not conducted in the present study because of the limited number of common data elements between studies (4) and the limited number of studies with all common data elements (3). Furthermore, heterogeneity analyses of 2 of 4 common data elements demonstrated heterogeneity at or above the cutoff for use in a meta-analysis.

Loss of common peroneal nerve function may have an effect on extremity pain, function, and PROs in the postoperative period [1,6,10]. Asp and Rand evaluated knee function in patients with postoperative CPNP via the Hospital for Special Surgery (HSS) knee score at a mean follow-up of 4.7 years. Patients with incomplete

palsies had higher mean HSS knee scores relative to patients who had complete palsies (90 points vs 77 points, $P < .03$); importantly, preoperative HSS knee scores were not significantly different between these groups. Similarly, patients with complete recovery of their palsy had higher mean HSS knee scores relative to patients with incomplete recovery of their CPNP (87 points vs 74 points, $P < .05$). Rose et al. [6] evaluated HSS knee scores in 23 patients with peroneal nerve palsy (of any degree) after TKA; they noted a mean HSS knee score of 81.6 points in patients with a postoperative CPNP relative to 83.3 points in patients without a postoperative CPNP. No statistical testing between cohorts was performed. Additional studies evaluating PROs and knee function in patients who develop CPNP are needed.

Common peroneal nerve neurolysis has been proposed as a potential treatment for CPNP after TKA [10,22,23]. In the present study, a total of 7 patients underwent surgical decompression of a common peroneal nerve; however, only 2 underwent decompression after postoperative development of CPNP. The degree of nerve function recovered in the 2 patients who underwent surgical decompression after TKA is not known. Five patients in the cohort of Rose et al. [6] underwent prophylactic decompression of the common peroneal nerve at the time of TKA; all 5 patients developed postoperative CPNP. Recent small series demonstrate common peroneal nerve neurolysis to be relatively effective in terms of mitigating pain and improving function after CPNP [10,22,23]. Given the relatively low rates of complete recovery noted in patients with complete CPNP at >3.5 years of follow-up, additional studies specifically evaluating the efficacy of common peroneal nerve neurolysis at improving function after complete CPNP are needed.

Limitations of the present study are numerous. Above all, CPNP is a rare pathology; as such, studies on the topic are relatively rare and those that do exist are retrospective in nature with a limited number of patients. Study heterogeneity in terms of defining pathology or variables, data collection, and methodology of reporting clinical outcomes limits the inclusion of many studies into this systematic review. Similarly, the definitions of nerve recovery in the present study (eg, complete vs incomplete) may offer poor correlation to a patient’s overall post-TKA function; for example, a patient with a previously complete nerve injury that has gone onto near full recovery of common peroneal nerve function would still be classified as an incomplete recovery, although their muscle and/or sensory deficits may be very minor.

In conclusion, at a mean follow-up of 3.6 years, patients with incomplete CPNP after TKA went on to complete recovery in 66% of

cases without further surgical intervention. Patients with complete CPNP after TKA had a poorer prognosis, with 39% achieving complete recovery over the same follow-up interval. All patients with classifiable nerve injury and follow-up data demonstrated some recovery in nerve function. These data may be used to inform decisions on further intervention (ie, common peroneal nerve neurolysis) and for the purposes of perioperative patient counseling after TKA.

Conflict of interest

Timothy S. Brown, MD, Editorial Board, reports royalties from PLOS One; and a board member for the Research Committee, American Association of Hip and Knee Surgeons. All other authors declare no potential conflicts of interest.

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