ORIGINAL ARTICLE

The Role of Prophylactic Peroneal Nerve Decompression in Patients with Severe Valgus Deformity at the Time of Primary Total Knee Arthroplasty

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Abstract

Background: Common peroneal nerve (PN) palsy after total knee arthroplasty (TKA) is a serious complication. Although many authors suggest delayed or immediate PN decompression after TKA in these patients, little is known about the role of prophylactic peroneal nerve decompression (PPND) at the time of TKA. The aim is to report the results of PPND in high-risk patients at the time of TKA.

Materials and methods: A multi-institutional retrospective study review of nine patients (10 knees) who underwent PPND at the time of TKA was conducted. Patients who had severe valgus deformities (≥15° of femorotibial angle and not fully correctable by examination under anaesthesia) with or without flexion contractures were included. PPND was performed through a separate 3–4-cm incision at the time of TKA. The demographics, preoperative and postoperative anatomical and mechanical alignments, range of motion, operation time, postoperative neurological function and complications were recorded.

Results: All patients had a completely normal motor and sensory neurological function postoperatively and no complications related to PPND were reported. All patients followed the standard physical therapy protocol after TKA without modifications.

The mean preoperative femorotibial angle was 20° (range 15–33°) and the mean postoperative femorotibial angle was 6.3° (range 5–9°) ($p = 0.005$). The mean preoperative flexion contracture was 9 (range 0–20) and the mean residual contracture was 1.2° (range 2–5°) ($p = 0.006$).

Conclusion: PPND at the time of TKA is an option to minimise the risk of PN palsy in high-risk patients. This approach can be considered for patients undergoing TKA in selected high-risk patients with a severe valgus deformity.

Keywords: Foot drop, Knee arthroplasty, Knee replacement, Peroneal palsy, Valgus knee.

Strategies in Trauma and Limb Reconstruction (2022): 10.5005/jp-journals-10080-1545

Background

Common peroneal nerve (PN) palsy is a serious complication that can occur after various surgical elective procedures. It is a well-documented risk after primary total knee arthroplasty (TKA) and particularly after correction of a valgus deformity.[1,2] Some authors found that a valgus alignment of more than 12° puts the PN at risk of palsy when correcting the deformity during the TKA.[3–5] The risk of PN palsy was found to be significantly increased when the valgus deformity is associated with a flexion contracture or pre-existing spinal pathology or both at the time of TKA.[3,6] The aetiology of the nerve damage is a neurapraxia related to acute lengthening of the PN which is located in the concavity of the valgus and flexion deformity of the knee. The overall incidence of PN palsy after TKA ranges from 0.3 to 10%.[4,7,8] While many authors have suggested that the surgical decompression of the nerve be performed after a documented PN palsy following the TKA,[8–10] little is known about the role of prophylactic peroneal nerve decompression (PPND) in high-risk patients.

Surgical decompression is a well-established technique for peroneal palsy and nerve entrapment around the neck of the fibula.[8,11] In the field of limb lengthening and deformity correction, the role of PPND has been embraced for those patients at high risk at the time of acute deformity corrections around the knee.[11,12] The goal of this study is to report our experience with the use of PPND at the time of TKA in high-risk patients.

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How to cite this article: Makhdom A, Hamilton AA, Rozbruch SR. The Role of Prophylactic Peroneal Nerve Decompression in Patients with Severe Valgus Deformity at the Time of Primary Total Knee Arthroplasty. Strategies Trauma Limb Reconstr 2022;17(1):38–43.

Source of support: Nil

Conflict of interest: None

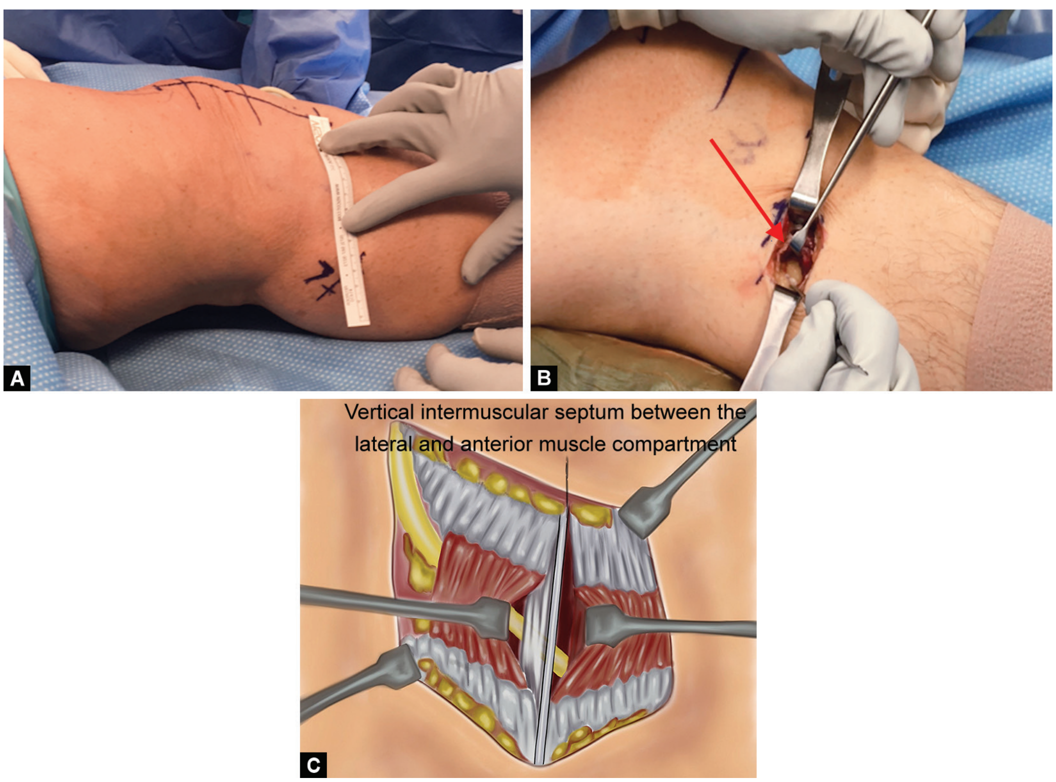
Patients and Methods

Approval from the institutional review board was obtained. A retrospective review of the medical and radiographic records was conducted on all consecutive patients who underwent PPND at the time of TKA at our institutions from January 2009 to July 2020. The demographics, pre- and postoperative anatomic alignment angles, pre- and postoperative mechanical alignment angles, pre- and postoperative neurological examination of both lower extremities, primary diagnosis, medical comorbidities, the timing of PPND, operation time, pre- and postoperative knee range of motion, complications and implants used were recorded. The indications of PPND at the time of TKA were if the preoperative anatomical femorotibial valgus was greater than 15° with or without a flexion contracture and not fully correctable by examination under anaesthesia. Patients with less than 6 months of follow-up or who had a history of pre-existing lower limb neuropathy, psychiatric illness or dementia or both were excluded from the study.

The anatomical valgus angle (also known as a femorotibial angle) was measured on the long hip to ankle radiograph.[13] This measurement represents the angle between the anatomical axis of the femur when intersecting with the anatomical axis of the tibia. The mechanical alignment was also measured on the long hip to ankle radiographs. The measurement represents the angle between the mechanical axis of the femur and the mechanical axis of the tibia.[13] A total of 11 patients were reviewed and only nine patients (10 knees) were eligible and included in the study. The other two were removed based on our exclusion criteria.

Surgical Technique

PPND was performed at the time of TKA through a separate incision ensuring a 7–8 centimetres (cm) skin bridge was maintained between the TKA standard midline incision and the PPND incision (Fig. 1A). Knees were positioned at 30–40° of flexion to ease the tension on the nerve. A 3–4 cm incision was marked just below the neck of the fibula in an oblique direction to follow the course of the common PN. Once the tourniquet was inflated, the skin and subcutaneous tissues were incised. Meticulous dissection using loupe magnification and bipolar cautery was performed to reach the fascial layer that is covering the PN. Careful dissection was performed to avoid damaging the lateral sural nerve which was often found at the posterior aspect of the incision. Prior to incising the fascia, the nerve was often easily palpable just below the neck of the fibular head. The common PN is then exposed by incising the overlaying muscular fascia (Fig. 1B) and intermuscular septa that act as compression points. The superficial and deep fascia of the peroneus longus muscle and the vertical intermuscular septum between the peroneus longus and the extensor digitorum longus muscles are sites that are decompressed (Fig. 1C). Once these septa were released, the tourniquet was deflated and haemostasis was obtained by bipolar electrocautery. This was followed by subcutaneous and skin closure. The operation of PPND ranged from 16 to 30 minutes. Thereafter, the tourniquet was re-inflated after 15 minutes and the TKA was carried out through a standard midline incision and a medial parapatellar approach. In all cases, once the tibial and femur cuts were performed, a spacer block was utilised to check the coronal balance. If the knee was tight laterally, the iliotibial band and posterolateral corner were released via a 'pie-crust' technique.[14] The lateral collateral ligament release was not required in all cases. All procedures were performed by two arthroplasty and limb lengthening and deformity correction-trained surgeons of attending level. Patients were allowed immediate weight-bearing and used a standard physical therapy protocol after TKA. Patients were seen in the office at 2- week, 6 -week and 6 -month intervals and then annually for routine follow-ups.



Figs 1A to C: (A) Intraoperative clinical photo illustrates planning the minimum distance between the two incision sites of intended midline knee incision and peroneal nerve decompression. A minimum of 7–8 cm of skin bridge is typically planned; (B) Intraoperative clinical photo illustrates a minimally invasive incision to decompress the fascial layer (red arrow) covering the common peroneal nerve around the neck of the fibula at the time of total knee arthroplasty; (C) Drawing to illustrate the vertical intermuscular septum between the peroneus longus and the extensor digitorum longus muscle that acts as a compression point

Statistical Analysis

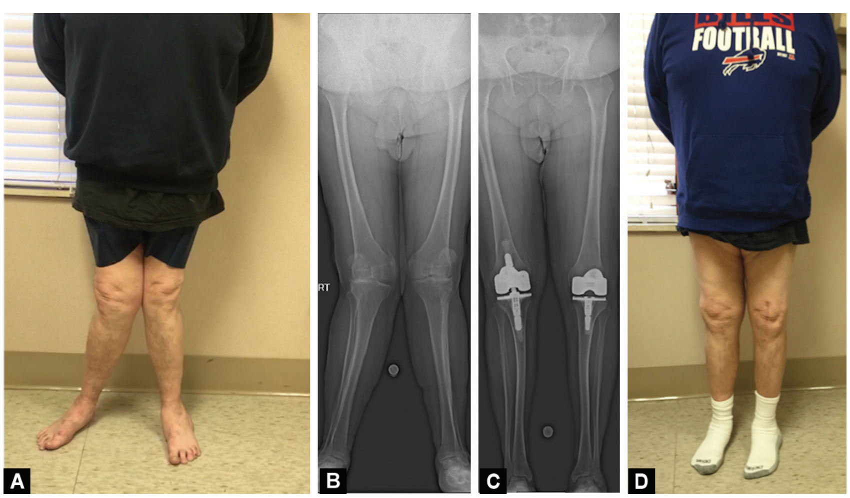
Means, ranges and standard deviations were used for descriptive statistics. The non-parametric *Wilcoxon signed-rank test* was used to compare two related means. A p value <0.05 was considered statistically significant. The statistical package for the social sciences, SSPS version 20.0 (SPSS Inc., Chicago, Illinois, USA), was utilised for the statistical analysis.

Results

Nine patients (10 knees) were eligible and included in the study. All patients underwent PPND at the time of TKA. Eight patients were diagnosed with end-stage primary osteoarthritis and one patient had secondary arthritis due to rheumatoid arthritis. There were five females and four males. There were five left and five right knees. The mean preoperative anatomical alignment was 20° of valgus (range 15–33) and the mean postoperative anatomical alignment was 6.3° of valgus (range 5–9, $p = 0.005$). The mean preoperative mechanical alignment was 15° of valgus (range 9–25) and the mean postoperative mechanical alignment was 0.6° of valgus (range 0–3). Table 1 is the summary of clinical and radiographic information from this sample. Postoperatively, all patients retained full motor and sensory function of the common PN with no changes compared to the neurological examination performed preoperatively and the contralateral limb. The detailed examination included assessment for intact sensation over the lateral aspect of the leg, dorsal aspect of the foot and the first web space of the foot bilaterally. Preserved motor function (grade 5/5) of the tibialis anterior, peroneal muscles, extensor hallucis longus and extensor digitorum longus was confirmed. The mean preoperative flexion contracture was 9° (range 0–20) and the mean postoperative residual flexion contracture was 1.2 (range 0–5, $p = 0.006$). The mean preoperative flexion was 103° (range 70–125) and the mean postoperative flexion was 111° (range 90–130, $p = 0.011$). The time for operation was 2 hours and 10 minutes (range 1 hour and 42 minutes to 2 hours and 30 minutes). All patients, but one, had no complications reported. One patient had a postoperative haematoma 5 days after the procedure. The haematoma was related to the midline TKA incision and not related to the PPND incision. This was evacuated successfully with no further consequences during the follow-up period. Three knees were reconstructed with posterior stabilising standard knee implants and seven knees were reconstructed with varus–valgus constraint (VVC) implants due to significant medial collateral ligament insufficiency (Fig. 2). Tibial and femoral stem extensions were added in those patients who had VVC implants, and one patient required a 10 mm lateral tibial metal augment. Postoperatively, all patients received chemical and mechanical deep venous thrombosis (DVT) prophylaxis. Five patients (six knees) received aspirin 81 mg twice daily and four patients received rivaroxaban 10 mg daily for 4 weeks as chemical

Table 1: Summary of mean pre- and postoperative alignment angles, range of motion and operative time in our patient population

Clinical and radiographic variables for 9 patients (10 knees)	*Mean (range)*
Preoperative anatomical alignment	20° of valgus (15–33)
Postoperative anatomical alignment	6.3° of valgus (5–9)
Preoperative mechanical alignment	15° of valgus (9–25)
Postoperative mechanical alignment	0.6° of valgus (0–3)
Preoperative flexion contracture	9° (0–20)
Postoperative residual flexion contracture	1.2 (0–5)
Preoperative flexion	103° (70–125)
Postoperative flexion	111° (90–130)
Operative time	2 hours and 10 minutes (1 hour and 42 minutes to 2 hours to 30 minutes)



Figs 2A to D: (A) Clinical photo of a 66-year-old male patient (patient 8 in Table 2) illustrating significant bilateral knee valgus deformities; (B) Preoperative hip to ankle long anteroposterior radiograph showing bilateral severe valgus deformities with evidence of primary knee osteoarthritis; (C) Postoperative hip to ankle long anteroposterior radiograph showing bilateral total knee arthroplasty with neutral alignment; (D) Postoperative clinical photo for the same patient during a follow-up visit after bilateral staged total knee arthroplasty prophylaxis for DVT. The mean follow-up period was 4 years (range 6 months to 11 years). All patients had followed the standard postoperative physical therapy protocols for immediate weight-bearing without modifications. All patients were walking without any aids at 6 weeks postoperatively. No patients had episodes of instability at the final follow-up. Detailed clinical information and demographics are in Table 2.

Discussion

A palsy of the common peroneal nerve after an elective procedure is functionally debilitating. A valgus deformity with or without a flexion contracture is well recognised as a strong risk factor[6] for this potential complication after TKA. Christ et al.[6] investigated 445 patients who developed a PN palsy after TKA and found that a preoperative valgus alignment greatly increased the risk of this complication (odds ratio 4.19). Interestingly, the risk has substantially increased when the preoperative valgus was found combined with a pre-existing spinal pathology (odds ratio 17.1). While PN palsy can occur from various factors (e.g. direct injury during the lateral release or prolonged tourniquet time) after a TKA, these findings indicate that stretching the nerve during the valgus deformity correction is most often the reason and not a direct injury.

The current management for a postoperative common PN palsy after a TKA includes flexing the knee, removing compressive dressings and initiating supportive measures (physical therapy and ankle foot arthrosis) to prevent ankle contractures. If the patient shows no recovery, electromyography (EMG) is performed at 6 or 12 weeks. Some surgeons advocate for an immediate baseline EMG study during the acute phase.[15] Several reports have suggested the role of a delayed PN surgical decompression in patients who had PN palsy after a TKA.[3,10] Mont et al.[3] retrospectively reviewed 31 patients who had a PN palsy post-TKA. Patients underwent delayed common PN decompression at a mean of 36 months post-TKA (range 12–72 months) and 97% of these patients reported functional improvement and were able to discontinue the ankle–foot orthosis. Similarly, Krackow et al.[9] reviewed five patients who underwent delayed PN decompression after a TKA. The decompression was performed at a mean of 27 months (range 5–50 months) and four patients (80%) had full neurological recovery. All five patients were able to discontinue the ankle–foot orthosis. More recently, Erikson et al.[8] have advocated for an immediate PN surgical decompression after a TKA without any waiting period for conservative management. The authors reported two patients who had valgus deformities and underwent a TKA. Both patients sustained common PN palsies postoperatively and underwent immediate surgical decompressions. Both patients improved dramatically within 2 days of post-surgical decompression with full neurological recovery prior to hospital discharge. In this study, all nine patients (10 knees) who underwent PPND at the time of TKA had no neurological consequences. The surgical decompression prior to TKA was performed through a small incision while the patient was under the spinal or epidural anaesthesia. The surgical technique is straightforward. This technique minimises the risk of PN compression or stretch or both after correcting severe valgus deformities. This approach parallels the practice patterns in the field of limb lengthening and deformity correction in which prophylactic nerve decompression procedures were adapted alongside the acute correction of sizeable deformities. Examples include planned prophylactic tarsal tunnel decompression in patients with severe distal tibial or ankle varus deformities undergoing bony correction,[16] prophylactic ulnar nerve decompression in patients with severe elbow contractures undergoing arthroscopic releases[17] and PPND in patients with severe valgus deformity or rotational deformities undergoing bony correction around the knee.[12] Patients with non-compliant soft tissues (due to scar tissue or previous trauma) are at the highest risk of nerve palsy after correction. Similarly, patients without correctable valgus on examination may pose the highest risk of PN palsy after TKA and, therefore, had a planned PPDN at the time of TKA. Erikson et al. argued that since 67% of patients with PN palsy after TKA do not recover, an immediate release is encouraged to enhance the odds of recovery. However, this is at the expense of a second procedure with anaesthetic risks and psychological distress to both patients and surgeons. Consequently, a PPND at the time of TKA in high-risk patients is an option to minimise the risk of nerve injury and obviates the potential need for a second procedure. One disadvantage would be the added operative time (16–30 minutes in our report). Some debate that the incidence of PN palsy after a TKA is probably low to justify a prophylactic decompression at the time of TKA. However, the true incidence of PN palsy after TKA in patients with severe valgus deformity remains unclear. The reported literature has focussed on the overall incidence for patients who underwent TKA rather than the incidence among high-risk groups of such patients with severe valgus deformity. To the best of our knowledge, there are no studies looking into the incidence of PN palsy in this high-risk group. Additionally, the presentation of PN dysfunction may be difficult to diagnose, and the incidence is underestimated particularly in the absence of motor deficits. Some patients may present with lateral leg pain and neuropathic symptoms, without motor deficits, that interfere with their postoperative rehabilitation and daily function.[1]

The role of PPND at the time of TKA was described many decades ago but not commonly practiced.[18–20] Patient selection and indications for PPND at the time of TKA are difficult to establish. There is a scant description of the surgical technique in the literature. Xu et al.[21] recently performed PPND at the time of TKA for 34 knees with an average valgus deformity of 31 ± 8°. All patients had no nerve palsy postoperatively with fully intact motor and sensory functions. The authors' method for the PPND was different to that described here. The nerve release was tailored and expanded based on the intraoperative ligament tension in a stepwise approach. The location of the incision was proximal to the fibular head and then the release was expanded distally if it was felt necessary while balancing the TKA. Additionally, the peroneus longus tendon was cut to allow to test the tension on the nerve by pulling it with gentle traction while balancing the TKA. Although this method is attractive, a theoretical risk of decreased ankle eversion muscle strength postoperatively is possible although the authors have reported no motor weakness for all patients. In our approach, we released all possible compression points including the fibrous arcade around the origin of the peroneus longus, the fascial compartment and intermuscular septa utilising a 3–4 cm incision. We have found the most significant point of PN compression to be at the deep fascia of the peroneus longus. After the release of the superficial fascia of the peroneus longus, the muscle is retracted medially, and the deep fascia is exposed and then released. We avoid incising the muscle fibres or tendons for two reasons: firstly to minimise the risk of bleeding and potential haematoma around the nerve and secondly to minimise the risk of muscle weakness postoperatively.

Table 2: Detailed clinical information and demograpy of our patient population

Patients	*Gender*	*Age (years)*	*Site*	*Body mass index**	*Comorbidities*	*Preoperative valgus anatomical angle (°)*	*Postoperative valgus anatomical alignment (°)*	*Preoperative range of motion (°)*	*Postoperative range of motion (°)*	*Complications*	*Follow-up period*
1	Male	73	Left	41.2	Rheumatoid arthritis	15	5	10–110	0–120	None reported	10 years
2	Female	73	Right	36	Hypertension	20	6	10–110	5–120	None reported	7 years
3	Female	66	Right	38	None reported	20	5	5–125	0–125	None reported	11 years
4	Male	43	Right	38	History of liver transplant	18	5	10–90	0–90	None reported	6 years
5	Female	67	Right	35	Hypertension	15	6	10–108	3–115	None reported	18 months
6	Female	83	Left	28	Hypertension, hyperlipidaemia	20	6	0–70	0–90	Postoperative haematoma in the knee joint. It was treated with surgical evacuation with no further consequences	2 years
7	Female	78	Right	31	Previous DVT, diabetes, hypertension	26	6	10–125	5–130	None reported	2 years
8	Male	66	Left	32	Hyperlipidaemia	33	9	20–90	0–100	None reported	6 months
9	Male	59	Right	33	Hypertension	24	9	5–100	0–110	None reported	2 years
			Left			18	6	5–110	0–115	None reported	18 months

*Body mass index formula = kg/m^2

This study is limited by the small sample size and lack of a control group. A severe knee valgus deformity >15° that does not correct fully on examination is a rare entity and multicentre recruitment is required to study a large cohort of patients. This study shows that PPND is safe to perform at the time of TKA. This encourages further studies to examine this approach in a large cohort of high-risk patients. There were no complications related to the PPND surgery in this sample. The technique is not complicated and can be performed by arthroplasty surgeons or, alternatively, by collaborating with a surgeon who has the expertise.

In conclusion, PPND at the time of TKA is an option to minimise the risk of PN palsy in high-risk patients. We believe that this approach can be considered for patients undergoing TKA for severe valgus with or without a flexion contracture deformity.

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