Deep Peroneal Nerve: From an Anatomical Basis to Clinical Implementation

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ABSTRACT

The deep peroneal nerve (DPN) is considered one of the clinically significant nerves of the lower extremity since several clinical abnormalities can commonly be caused by its defects, either in its sensory or motor functions. Its derivatives, classified as muscular, cutaneous, and articular, mainly supply the muscles in the anterior fascial compartment of the leg and the dorsum of the foot, the 1st dorsal web space of the foot, the ankle joint, and certain joints of the foot. To improve the effectiveness of clinical practices involving the DPN, it is important to first understand its anatomical nature, including its typical characteristics and the variants (orientation, branching, and analogous structure), prior to applying such practices in clinical implementation. This review, therefore, aims to review the previously studied information of DPN on its fundamental anatomy and link it to the provided examples of current commonly used procedures, both non-invasive and invasive, e.g., nerve imaging, nerve block, neuro-electrophysiological study, and free autologous tissue transfer, thereby giving an integrated view in the translational medicine of DPN. Conclusively, the ultimate goal of this review is to help maximize the therapeutic effectiveness and to minimize the unanticipated complications of any clinical practices involving the DPN by inferring from its anatomical knowledge.

Keywords: Deep peroneal nerve; accessory deep peroneal nerve; nerve imaging; nerve block; electrophysiological study; surgical reconstruction (Siriraj Med J 2022; 74: 448-462)

Abbreviations: DPN; deep peroneal nerve, CPN; common peroneal nerve, PL; peroneus longus, EDL; extensor digitorum longus, ATA; anterior tibial artery, TA; tibialis anterior, EHL; extensor hallucis longus, SER; superior extensor retinaculum, IER; inferior extensor retinaculum, DPA; dorsalis pedis artery, MTB; medial terminal branch, LTB; lateral terminal branch, SPN; superficial peroneal nerve, LTA; lateral tarsal artery, EDB; extensor digitorum brevis, EHB; extensor hallucis brevis, ATT; anterior tarsal tunnel, ADPN; accessory deep peroneal nerve, PB; peroneus brevis, US; ultrasonography, MRI; magnetic resonance imaging, CMAP; compound muscle action potential, NCV; nerve conduction velocity, AUC; area under the curve, SNAP; sensory nerve action potential, MNAP; mixed nerve action potential, EMG; electromyography

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INTRODUCTION

DPN is recognized as one of the clinically significant nerves since several clinical manifestations can commonly be observed attributed to its defective components in either sensory or motor function. These abnormalities are caused by either intraneural (e.g., peripheral nerve sheath tumor or intraneural ganglion cyst from metatarsophalangeal joint distension) or extraneural (e.g., tumor, extensor retinaculum, bony spur, ganglion cyst or inadequate foot device) compressive lesions. In addition, they can also be due to the traumatic etiologies. For example, common injuries near the proximal part of the fibula, especially the iatrogenic ones after certain interventions around the lateral side of the knee and upper part of the leg, e.g., the proximal fibular osteotomy, percutaneous wire placement in the proximal tibia or total knee arthroplasty, can result in DPN dysfunction. Not only the proximal part, the clinical procedures near the distal part of DPN, e.g., ankle arthroscopy, may also cause the disturbance to this nerve and lead to the sensory impairment of the cutaneous area supplied by this nerve. Besides its clinical significance in terms of nerve injury, several clinical manipulations with this nerve, both non-invasive and invasive, are commonly employed in the medical practice nowadays, e.g., imaging, anesthetic procedure, functional study, or even surgical intervention. In this study, we, therefore, aimed to review its anatomical basis and variations reported in the previous literature and to link these to its current applications that are commonly used in clinical service, e.g., clinical imaging, nerve block, electrophysiological study, and employing this nerve in surgical procedures, to establish definitive translational knowledge for its clinical implementation.

The review process was carried out by using groups of keywords for searching on the standard online publication database, i.e., PubMed (https://pubmed.ncbi.nlm.nih.gov/). Certain keywords used for searching comprised "deep peroneal nerve", "deep fibular nerve", "anatomical variations and deep peroneal nerve", "dorsalis pedis artery", "accessory deep peroneal nerve", "accessory deep fibular nerve", "deep peroneal nerve imaging", "deep peroneal nerve block", "electrophysiological study and deep peroneal nerve", "surgical reconstruction and deep peroneal nerve". All searched records available on the PubMed database were screened for their relatedness to the aim of this review and only the full articles of the related ones were retrieved. The publication resource used in this review, starting from the latest ones in 2021, dates back to 1896 as the earliest one. All publication resource, after being categorized according to the disciplines (anatomy, radiology, rehabilitative medicine and plastic surgery), was then distributed to the authors with specialty corresponding to each discipline, i.e., anatomist, radiologist, rehabilitation physician and plastic surgeon. After reviewing all related resource, authors altogether conceptualized and drafted the contents of this review to propose the systematic knowledge of its anatomical nature linking to the application useful in current clinical practice.

Anatomical background of the DPN

General information and origination of the DPN

DPN, also known as the deep fibular nerve (L. nervus peroneus profundus, nervus fibularis profundus), is one of the nerves in the lower extremity that functions as combined sensory and motor fibers, mainly supplying the leg and foot area.[1] This nerve is a bifurcating branch of the common peroneal (fibular) nerve (CPN), which is one of the two divisions of the sciatic nerve (ventral rami of the 4th and 5th lumbar together with the 1st, 2nd, and 3rd sacral spinal nerves).

Continuing from the distal part of the CPN that passes posterior to the head of fibula, the bifurcation is located near the neck of the fibula and terminates as two branches: the superficial and deep peroneal nerves (Fig 1A). Traumatic/non-traumatic abnormalities of the proximal part of the fibula[2] or iatrogenic injuries around this zone[3], e.g., after fibular biopsy, high tibial osteotomy, or total knee arthroplasty, can result in dysfunction of the DPN. The superficial branch courses downward laterally along the length of the leg into the lateral fascial compartment while the deep one deviates medially, pierces through the anterior intermuscular septum, and then passes downward anteriorly into the anterior fascial compartment.

Proximal part of the DPN

The proximal part of the DPN runs deep to the peroneus (fibularis) longus (PL) and then the extensor digitorum longus (EDL) muscles to lie anterior to the interosseous membrane. It becomes laterally parallel to the anterior tibial artery (ATA, in the proximal third of leg) as the neurovascular supply for the surrounding extensor muscular tissue, i.e., the tibialis anterior (TA), EDL, extensor hallucis longus (EHL), and peroneus (fibularis) tertius muscles. Therefore, lower motor neuron disturbance or neuropathy of this nerve, e.g., complications of diabetes, intraneural ganglion cyst, compartment syndrome, or inflammatory problems, commonly causes the clinical sign of foot drop, which is the inability to achieve ankle dorsiflexion and toe extension.[4]

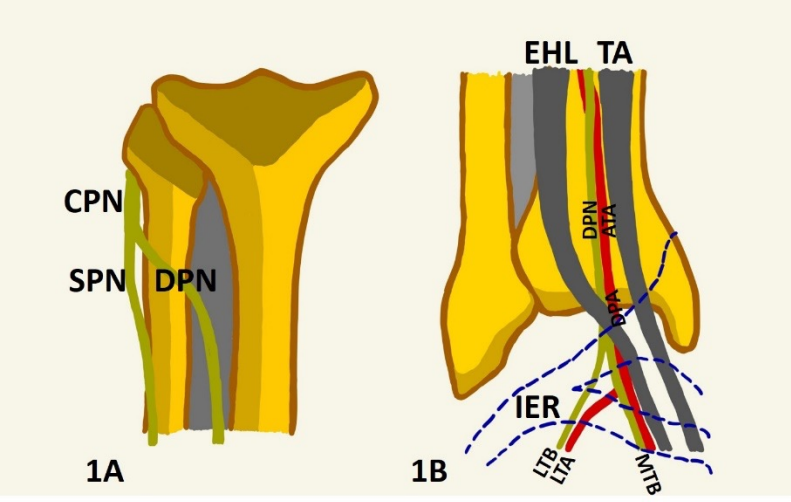

Fig 1. **1A:** Schematic demonstrating the right DPN originating from the bifurcation of CPN near the neck of fibula. **1B:** Schematic demonstrating the orientation and branching of the distal part of the right DPN at the lower leg and ankle areas. CPN = common peroneal nerve; DPN = deep peroneal nerve; SPN = superficial peroneal nerve; EHL = extensor hallucis longus (tendon); TA = tibialis anterior (tendon); ATA = anterior tibial artery; DPA = dorsalis pedis artery; MTB = medial terminal branch; LTB = lateral terminal branch; LTA = lateral tarsal artery; IER = inferior extensor retinaculum (borders represented by the blue curved dashed lines).

Distal part of the DPN

During its passage downwards through the distal part of the leg, these neurovascular structures course more superficially than the proximal part and lie posterior to the superior and inferior extensor retinacula (SER and IER, respectively) and in between the TA (medial) and EHL (lateral) muscles (Fig 1B). When the course of DPN passes anterior to the ankle joint, the tendon of the EHL crosses superficially over the descending neurovascular structures (DPN and dorsalis pedis artery, DPA) to lie medially to them (Fig 1B). The DPN is typically lateral to the DPA. At this level, the DPN shares an articular branch for the ankle joint and then divides into two terminal branches, i.e., the medial and lateral terminal branches (MTB and LTB, respectively), although division proximal or distal to this level can also be found as positional variations (Fig 1B).

Terminating branches of the DPN

The MTB runs dorsally along with the DPA and provides the articular branch for the 1st metatarsophalangeal joint and the muscular branch for the 1st dorsal interosseous muscle. Finally, the MTB communicates with the medial branch of the superficial peroneal nerve (SPN) in the 1st interosseous space and reaches the proximal zone of the 1st interdigital cleft to terminate as two digital cutaneous nerves supplying the 1st dorsal web space of the foot. This explains the sensory impairment of this cutaneous area in the case of DPN disturbance, even when it's due to the iatrogenic etiology, e.g., ankle arthroscopy.[5] Likewise, the LTB runs dorsolaterally along with the branch of the DPA, the lateral tarsal artery (LTA), and passes beneath the extensor digitorum brevis (EDB) and the extensor hallucis brevis (EHB) muscles (as a pseudoganglion enlargement) to supply them (Fig 1B). Moreover, the LTB also provides the articular branches for the tarsal and the middle three metatarsophalangeal joints.

Key takeaway

DPN is one of the lower extremity nerves supplying structures in the anterior fascial compartment, ankle region and dorsum of foot in both sensory and motor functions. Two superficial parts of DPN course, i.e., near the neck of the fibula and anterior to the ankle region, should be clinically focused as the common sites for iatrogenic and non-iatrogenic nerve damage. In addition, anatomical relations to its adjacent structures are the fundamental concept for implementing in various clinical applications.

Anatomical variations of the DPN

Proximal part of the DPN

Owing to the superficial lying of the proximal part of the DPN adjacent to the fibular head, the anatomical variation of this part is vulnerable to pathological disturbance by the surrounding structures.[6] Thus, understanding the positional variation of the proximal DPN plays an important role in clinical practice in order for physicians to find a safe area or angle for procedures that can avoid injury to the DPN. Iatrogenic injury caused by clinical procedures around the lateral side of the knee and upper part of the leg, e.g., proximal fibular osteotomy during high tibial osteotomy or percutaneous wire placement in the proximal tibia, significantly contributes to cases of DPN damage, and have prompted a number of studies into the variation of DPN in this region to find a safe area that can allow avoiding the risk of damage to the DPN.[7,8]

1. Variations of the DPN originating position and orientation of the proximal part

A study in Thai cadavers was performed to find the ethnic-specific distance from the tip of the fibular head to the DPN originating point (28.4 ± 4.8 mm).[9] Compared with the Thai study, a study in the Japanese

cadavers showed an approximate average of this distance of 26.0 ± 0.32 mm.[10] However, studies in cadavers of non-Thai ethnicity indicated a safe distance for fibular head procedures of only about 20 mm distal to the tip of the head of the fibula.[7,8] In terms of the angle of proximal DPN relative to the vertical axis of the fibula, it was reported to be about 28.1° ± 7.2° in the study of Thai cadavers.[9] Compared with the Thai study, the results from the Japanese cadavers showed a slightly narrower angle (23.5° ± 3.5°).[10]

2. CPN entrapment by the peroneal (fibular) tunnel

In addition to iatrogenic injury, the peroneal tunnel is one common cause of idiopathic entrapment of the CPN, which also affects the function of the DPN. A study in cadavers was carried out in Ireland and found that the compression site of DPN was about 3.2 ± 1.0 cm (at the entering point of the CPN into the tunnel) and 7.0 ± 1.5 cm (at the exit point of the DPN from the tunnel) from the apex of the fibula.[11] These results can be implemented in planning a procedure to release a nerve entrapment, but they need to confirm in Thai subjects to avoid the risk of existing ethnic-specific variations.

Distal part of the DPN

In contrast to the proximal part of the DPN, the distal part demonstrates a larger number of both positional and branching variations due to the larger possibility of diverse orientations relative to neighboring structures along the distal coursing and also the branching circumstances. As the DPN divides into the MTB and LTB at the approximate zone of the ankle joint without a particular location, the branching patterns and levels have been investigated in a number of studies with an aim to identify their distribution.

1. Variations of the DPN branching level and pattern

Based on the previous study using the ankle mortise as the landmark, only 23% of the studied specimens were found to be bifurcated either above or at this level (Fig 1B).[12] Likewise, using an imaginary line connecting the medial to the lateral malleoli as the landmark instead of the ankle mortise showed the prevalence of bifurcation at and distal to this level as about 21% and 27.1%, respectively (Fig 1B)[13] Further compared with the IER (the roof of the anterior tarsal tunnel, ATT), most of the bifurcation (86.1%) was observed in the ATT, while only a small number of the studied specimens (5.6%) showed bifurcation below the ATT, and no bifurcation above the ATT was seen (Fig 1B).[14] Nonetheless, the absence of bifurcation[14-16] and multiple branching[13] were found at low prevalence of 6.7-8.3% and 4.16%, respectively.

2. Positional variations of the DPN and its branches in relation to the DPA

Not only have branching variations of the distal part of DPN been significantly observed but the positional variations of the DPN and its branches in relation to the DPA have also been explored to reveal their orientation. Inside the ATT, Rab *et al.* reported that the DPN was found lying anterolaterally to the DPA in almost all of the studied specimens (92.9%) (Fig 2).[17] When combined with observations of the DPN branching level, the prevalence of the DPA orientation medial to the DPN in the ATT and medial to the MTB distal to the ATT was about 36.1%–36.7% (Fig 2A), whereas the prevalence of the DPA orientation lateral to the MTB was reported to be 25%–30% (Fig 2B).[15,16] Crossover of the DPN and DPA at multiple levels was also reported in wide-ranging frequency of about 8.4%–30.6%, which may depend on the ethnicity of the cadavers in each study (Fig 2C).[13,15,16]

Accessory deep peroneal nerve (ADPN)

1. General information and course of the ADPN

The accessory deep peroneal nerve (ADPN; L. nervus peroneus profundus accessorius), first reported in 1896[18], is a nerve variant emerging from the SPN, carrying both sensory and motor fibers, and descending along the anterior surface of the posterior intermuscular septum and the posterior border of the peroneus brevis (PB) muscle in the lateral fascial compartment of the leg until reaching the lateral malleolus, while some studies demonstrated its possible existence in the anterior fascial compartment.[19–21] The course of the ADPN can bifurcate to reunite at the proximal part of the PB muscle and can also be partially covered by the slip of PB muscle.[21] During passing inferior to the distal part of the fibula with the tendons of two peronei muscles, the ADPN runs through the superior peroneal tunnel and then the deep-anterior compartment of the inferior peroneal tunnel.[21] Finally, it courses anteriorly near the sural nerve and deep to the tendon of the PB muscle to the dorsum of the foot.

2. Structures innervated by the ADPN

As commonly reported for innervation of the EDB muscle, the ADPN was revealed to provide muscular branches for not only the PL and PB muscles (75%, 33.3%, and 100% of the studied cases for the EDB, PL and PB, respectively) but also for other small peronei muscles (peroneus quartus and peroneus digiti quinti).[21]

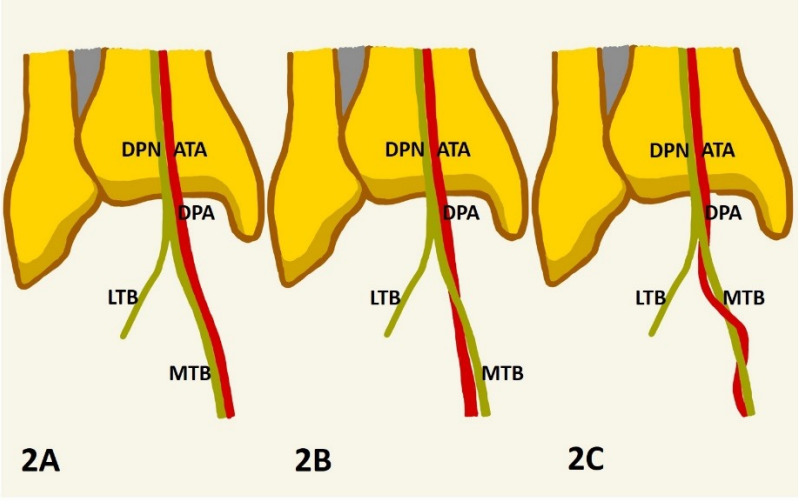

Fig 2. Schematic demonstrating the orientation of the right DPN and its branches at the ankle area and the dorsum of foot in comparison with the right ATA and DPA. **2A:** The vessel is medial to both DPN and MTB. **2B:** The vessel is medial to DPN but lateral to MTB. **2C:** Crossover of the DPN and the vessel at multiple levels. DPN = deep peroneal nerve; ATA = anterior tibial artery; DPA = dorsalis pedis artery; MTB = medial terminal branch; LTB = lateral terminal branch.

It commonly innervates the EDB muscle for the extensor part of the 4th toe rather than the 3rd and the 5th toes.[22] For the sensory component, the ADPN supplies the fibular periosteum, lateral region of the ankle (e.g., the ankle joint, ligaments, tarsal periosteum, and synovial sheath of the peroneal muscles), and the metatarsal region (37.5%, 100%, and 20.8% of the studied cases for each, respectively).[19,21,23]

3. Study methods for the occurrence of the ADPN

Study into the occurrence of the ADPN can be performed by two approaches, i.e., by direct dissection of cadaveric specimens and by electrophysiological study in living subjects. Nonetheless, the latter approach usually gives a lower prevalence than the former (13.6% vs. 39.3%)[24], as a positive verification of the ADPN by the latter requires the presence of a muscular branch to the EDB muscle. Identification of ADPN existence is crucial for clinical practice in cases with peroneal lesions that may involve a confused diagnosis. Furthermore, clinical procedures at the lateral side of the ankle, e.g., ankle arthroscopy, orthopedic procedures at the lateral malleolus, and sural nerve biopsy should raise some concern for iatrogenic complications that may occur to the ADPN. This risk can be reduced by performing a prior electrophysiological study to detect the existence of ADPN. Besides, the existence of ADPN may account for certain unexplained clinical conditions, such as EDB muscle atrophy and chronic ankle pain.

4. Prevalence of the ADPN and its branches

A number of ADPN studies, both cadaveric and electrophysiological, among different ethnicities have been conducted to find the prevalence of ADPN, prevalence differences between the sexes and sides, bilateral similarity, and the prevalence of a muscular branch to the EDB muscle. A meta-analysis was done to collect eligible published data from 19 ADPN studies.[24] The pooled ADPN prevalence was 18.8% (95% CI: 14.2–24.0%). Variability in the pooled prevalence among different ethnicities was also found (Asian = 50.3% (95% CI: 21.1–79.5%), North American = 17.6% (95% CI: 13.3–22.3%), and European = 12.1% (95% CI: 8.3–16.5%)).

The pooled prevalence on each side (from 4 out of 19 studies) was 6.4% (95% CI: 4.8–8.2%) for the right side and 8.5% (95% CI: 7.3–9.8%) for the left side. The pooled unilateral prevalence was two times higher (67%, 95% CI: 53.8–79.1%) than the pooled bilateral one (33%, 95% CI: 20.9-46.2%) in cases with ADPN present (from 11 out of 19 studies). The pooled prevalence of a muscular branch to the EDB muscle was 79.5% (95% CI: 53.5–97.4%) of all specimens with ADPN (from 4 out of 19 studies). There was no statistical significance in prevalence difference between the sexes.[25,26] Nevertheless, the occurrence of ADPN seems to run among family members as a hereditary trait with the proposed autosomal dominant mode of inheritance.[27]

Key takeaway

DPN is one of certain nerves that its anatomical variants can be found in all aspects, e.g., position, orientation, branching level and pattern or even additional accessory. For the proximal part, they are mostly about the positional and orientational variants, whereas the significant variabilities observed in the distal part are involved with aspects of branching and relations to its adjacent structures. These anatomical observations are the key translational knowledge to effectively practicalize the clinical approaches dealing with DPN.

Current clinical applications of the DPN

Application in radiology

1. General imaging characteristics of DPN

Diseases of the DPN are composed of many factors, including traumatic[28] and non-traumatic diseases, such as intraneural and extraneural compressive lesions.[29] Nowadays, imaging techniques play a major role in the diagnosis of peripheral nerve diseases, such as high-frequency ultrasonography (US) and magnetic resonance imaging (MRI).[30,31]

1.1 Ultrasonographic imaging

In US, the normal appearance of the nerve appears as a honeycomb pattern in the short axis, in which the hypoechoic nerve fiber is enveloped by the hyperechoic epineurium (Fig 3). In the long axis, it appears as a coarse hypoechoic longitudinal structure without anisotropy as in the tendon.[32] The landmark for the depiction of the DPN in US corresponds with its anatomy. First, in the proximal part, it continues on from the CPN after bifurcation at the peroneal tunnel, where the anterior one is the DPN and the other posterior one is the SPN. Second, in the leg region, it lies in the anterior fascial compartment of the leg, which is deep to the TA muscle and anterior to the interosseous membrane, and laterally accompanies the ATA (Fig 3). Last, in the ankle and foot regions, it lies between the EHL and EDL muscles and distally gives two terminal branches: the MTB, which accompanies the DPA until the 1st web space (Fig 4), and the LTB, which supplies the EDB muscle, tarsal joints, and 2nd to 4th metatarsophalangeal joints.[31,33]

1.2 Magnetic resonance imaging

In MRI, a normal appearance of the nerve is revealed with an iso-signal intensity (SI) in T1W and T2W as the SI of the muscles (or a slightly higher SI than that of the muscle in T2W), and it is surrounded by high SI fat (Figs 5&6).[34-36]

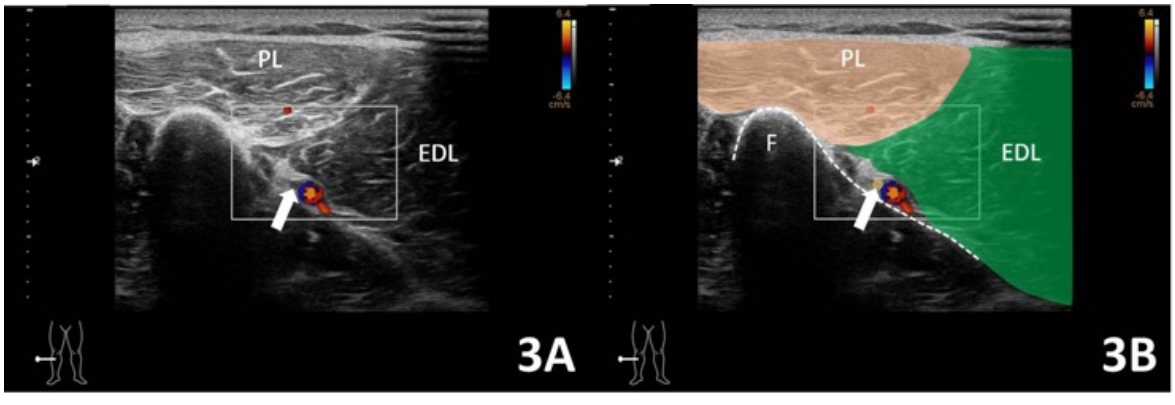

Fig 3. **3A:** Axial scan of US using a 12 MHz linear array transducer at the proximal aspect of the right leg (28-year-old man, below the fibular head) demonstrating the DPN coursing laterally to the ATA in the anterior fascial compartment with a honeycomb appearance (white arrow). **3B:** Area-representing schematic for US image demonstrating the DPN (yellow area pointed by the white arrow) in anterior fascial compartment of leg laterally accompanying the anterior tibial vessels (the structures with blue and red colors representing the blood flow directions in color-Doppler US). PL = peroneus longus muscle; EDL = extensor digitorum longus muscle; F = fibula with the curved dashed line as its boundary.

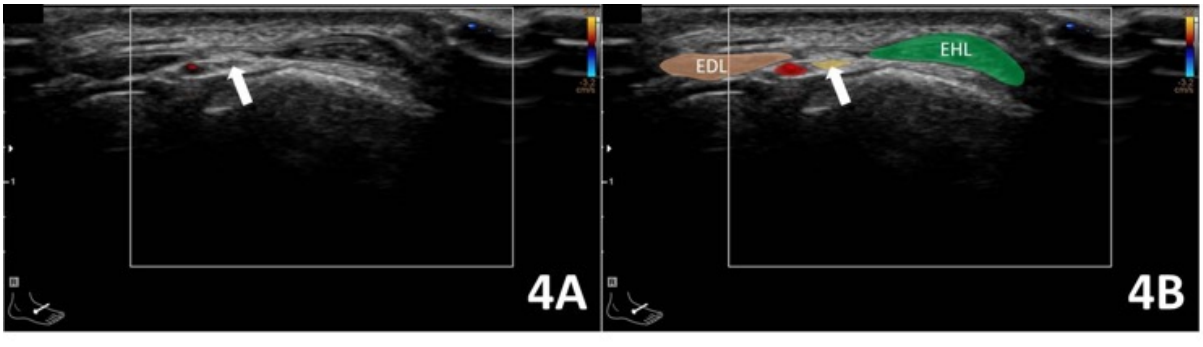

Fig 4. **4A:** Axial scan of US using a 12 MHz linear array transducer at the dorsum of the right foot (28-year-old man) demonstrating the MTB of the DPN, laterally accompanying the DPA, with a honeycomb appearance (white arrow). **4B**: Area-representing schematic for US image demonstrating the DPN (yellow area pointed by the white arrow) in the dorsum of foot located medial to the DPA (red area). EHL = extensor hallucis longus (tendon); EDL = extensor digitorum longus (tendon).

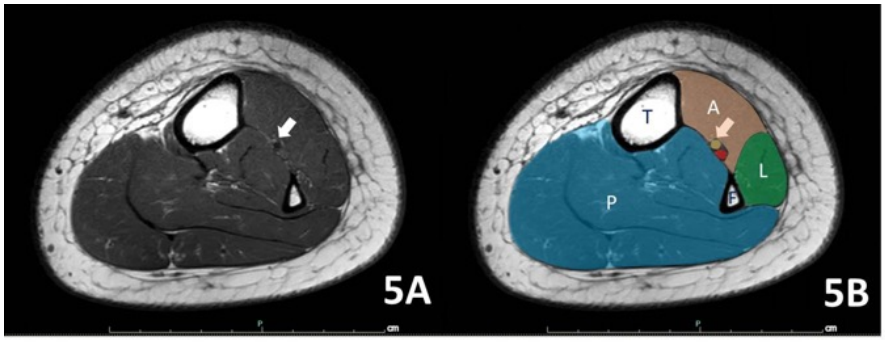

Fig 5. 5A: Axial T1-weighted MR image of the proximal aspect of the left leg (12-year-old girl, below the fibular head) demonstrating the DPN accompanying the ATA in the anterior fascial compartment (white arrow). **5B:** Area-representing schematic for MR image demonstrating the DPN (yellow area pointed by the white arrow) at the proximal aspect of the left leg. Red area = ATA; A = anterior fascial compartment; L = lateral fascial compartment; P = posterior fascial compartment; T = tibia; F = fibula.

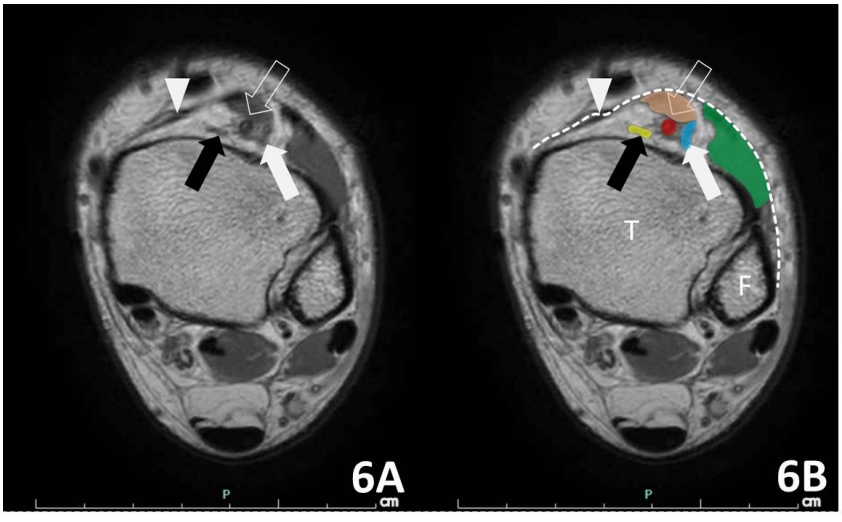

Fig 6. 6A: Axial proton density-weighted MR image of the left ankle (41-year-old man) demonstrating the MTB (black solid arrow) and the LTB (white solid arrow) of the DPN. The DPA (white open arrow) courses laterally to the MTB of the DPN. Extensor retinaculum is marked by the white arrow head. **6B:** Area-representing schematic for MR image demonstrating the MTB (yellow area pointed by the black solid arrow) and LTB (blue area pointed by the white solid arrow) of the DPN at the left ankle with the DPA (red area pointed by the white open arrow) located lateral to the MTB. Brown area = EHL (muscle and tendon); green area = EDL (muscle and tendon); curved dashed line pointed by the white arrow head = extensor retinaculum; T = tibia; F = fibula.

2. Direct imaging characteristics of DPN pathology

MRI and US can be used to evaluate the DPN pathology of both traumatic and non-traumatic causes as described below.

2.1 Traumatic pathology of the DPN

In traumatic causes, including direct contusion, traction injury, penetrating injury, and also post-surgical iatrogenic events, the DPN appears swollen and edematous, with hypoechoic thickening in US[33] with an increased T2W SI and decreased T1W SI in MRI. If partial or complete disruption of the nerve occurs, post-traumatic or stump neuroma may be seen in US as a hypoechoic mass-like lesion[28,33] and in MRI with an iso- to low T1W SI and iso- to high T2W SI with enhancement.[37,38] Other traumatic causes include compartment syndrome in the anterior or lateral fascial compartments[39,40] and compression from a fractured bone fragment or late sequelae of heterotopic ossification.[41]

2.2 Non-traumatic pathology of the DPN

Non-traumatic causes include intraneural and extraneural causes. Intraneural causes include a peripheral nerve sheath tumor either benign or malignant[42], and the less common intraneural ganglion cyst from metatarsophalangeal joint distension.[29,43] Extraneural causes include tumor (e.g., exostosis) and compression by adjacent structures, such as an extensor retinaculum, bony spur, ganglion cyst, or inadequate foot device.[43] The imaging findings depend on each pathologic nature. For example, a ganglion cyst may be found as a well-defined round-shaped high T2W SI lesion and iso- to hypo-T1W SI depending on its content[44] with either non- or rim enhancement in the post-gadolinium sequence of MRI.[45] Nerve thickening can be seen with an increased T2W SI in MRI.[43]

3. Indirect imaging characteristics of DPN pathology

Beside direct nerve abnormalities in peripheral neuropathy, indirect signs of nerve injury can also be observed in clinical imaging. One of the useful indirect signs is the corresponding denervation of the muscle. Varied imaging findings of muscle denervation depend on the timing, site, and degree of neural involvement[46,47], but they can still be used as a beneficial clue for the detection of neuropathy. Muscle denervation is edematous in the acute stage and then atrophic with fatty infiltration in the chronic stage. In the acute phase, US of denervated

muscle shows a, sometimes subtle, hypoechoic edematous appearance, while better evaluation can be achieved by utilizing fat suppression techniques with T2W MRI[43,48], seen as an increased SI in 24 to 48 hours after the event.[46,47,49] In the chronic phase, the denervated muscle shows increased echogenicity with a smaller size[33], whereas an increased fat SI of atrophic muscle can be observed in MRI.[46,47,49] Like other peripheral nerves, DPN neuropathy can be involved with muscles in the anterior fascial compartment (Fig 7) and, less frequently, the EHB muscle according to the degree and site of neuropathy.[46,49]

4. Key takeaway

Normal anatomy, including position, orientation, branching pattern and variation, is the key for understanding normal findings in radiology. Likewise, normal imaging appearance is also the key for abnormality detection. US and MRI have the capacity *per se* to evaluate the DPN pathology in different aspects. In certain aspects, efficacy of one modality can also be enhanced by the other. Not only the direct sign of nerve abnormality but also the indirect sign of muscle innervation is beneficial in detection of the DPN abnormality by clinical imaging.

Application in anesthesiology

1. Regional nerve block of the DPN

Regional nerve block of the DPN is an anesthetic approach that is preferably used in practice due to its low systemic risk.[50,51] Application in the ankle region is used in many situations, including ankle surgery and pain control.[52,53] However, certain conditions may contraindicate this method, such as a local infection at the injected site or in patients at risk of compartment syndrome.[53] Accurate knowledge of the DPN anatomy makes this nerve block perfect.

The landmark for DPN block in the ankle region is located lateral to the ATA[52], just lateral to the tendon of EHL[54], which can be easily found by active dorsiflexion of the big toe. US guidance for peripheral nerve block seems to be advantageous[50], such as using a directly observable nerve with adjacent vascular structures, for avoiding direct vessel injection and reducing the amount of drug used.[55–57] However, John *et al.* found that the ultrasound guidance technique showed no improvement in overall quality in DPN block at the ankle level.[58] Therefore, an anatomical landmark for DPN block is still important.

2. Key takeaway

Normal DPN anatomy is essential for successful regional DPN block in the ankle region, including adequacy of local anesthesia and complication avoidance, whether the ultrasound guidance technique is applied or not.

Application in rehabilitation medicine

1. Electrodiagnosis for DPN

Electrodiagnosis is a physiologic study that can help in localizing the DPN pathology, and for assessing the severity and signs of recovery. Moreover, it can be used in differential diagnosis and for ruling out other causes mimicking DPN mononeuropathy; for example, sciatic neuropathy, lumbosacral plexopathy, L5 radiculopathy, peripheral polyneuropathy, and upper motor neuron diseases. There are two types of electrodiagnostic study.

1.1 Nerve conduction study (NCS)

1.1.1 Motor NCS

Motor NCS evaluates the functioning motor axons by sending electrical impulses at the proximal site and recording the response over the muscle belly. The action potential recorded is a summation from the motor nerve,

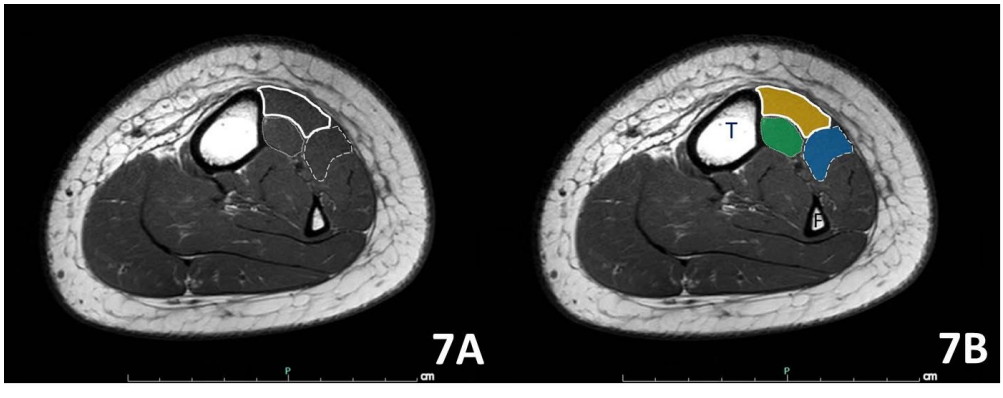

Fig 7. 7A: Axial T1-weighted MR image of the proximal aspect of the left leg (12-year-old girl, below the fibular head) demonstrating the muscles of the anterior fascial compartment, including the TA (white solid line), EDL (white dashed line) and EHL (white encircling dots) muscles. **7B:** Area-representing schematic for MR image demonstrating the muscles in the anterior fascial compartment of the proximal aspect of left leg, including the TA (yellow area), EDL (blue area) and EHL (green area) muscles. T = tibia; F = fibula.

neuromuscular junction, and all the muscle fiber action potentials (compound muscle action potential, CMAP) (Fig 8). The stimulation sites for the DPN are at the anterior ankle, fibular head, and lateral area of the popliteal fossa. Recording electrodes are usually placed on the EDB (Fig 9A) and TA muscles. The onset latency, amplitude, and nerve conduction velocity (NCV) are the common parameters recorded (Table 1).

Demographic factors can also affect some other parameters. A previous study found that a greater height of the subject was associated with increased onset latency and decreased NCV. In addition, it was also found that an older subject was correlated with a lower amplitude, area under the curve (AUC) and NCV. However, there was no statistically significant difference in NCS between the sides.[59,60] Moreover, the NCV across the fibular head when recorded at the TA was faster than that recorded from the EDB, with the mean difference in the NCV being 5 m/s.[60]

To use these parameters in testing, Buschbacher *et al.* reported a cutoff value as a 50% amplitude drop when compared to the contralateral side and recorded at the TA muscle for diagnosing an abnormality of the motor amplitude, if the affected limb showed normal values.[60] When recording at the EDB muscle, the upper limit of normal amplitude difference from side-to-side was 61%, which might not be sensitive enough to make a diagnosis of mononeuropathy.[59] To find a focal demyelinating lesion of the nerve, comparing the amplitude between stimulating at the proximal (popliteal fossa or fibular head) and distal (ankle) points, and recording at the EDB muscle is usually done. The upper limit of normal amplitude decrease was found as a 32% drop across the lower leg and 25% drop across the knee in one study.[59] While recording at the TA muscle, the upper limit of normal drop in amplitude from distal to proximal stimulation was 36%. This normal drop was high for a short nerve distance. However, it was possible in this condition because the phase cancellation from a volume-conducted tibial waveform could not be ruled out.[60] Additionally, when recorded at the EDB and comparing the NCV between below-knee and across-knee segments, the upper limit of normal drop was 10 m/s.[59]

1.1.2 Anatomical variation

The ADPN primarily emerges as both the motor nerve from the SPN to the EDB muscle and the sensory branches to the ankle joint. The presence of ADPN should be suspected if the EDB motor amplitude is smaller when stimulated at the ankle than at the fibular head. This can be confirmed by obtaining the CMAP from stimulating behind the lateral malleolus.[20,59] Another method is by comparing the CMAP area ratio between stimulating at the distal and proximal. The area ratio is higher in subjects without ADPN than those with ADPN (1.15 and 0.96, respectively). From a previous study, the distal CMAP amplitude of ADPN was around 1.27 mV with an average distal latency at 4.84 ms.[20]

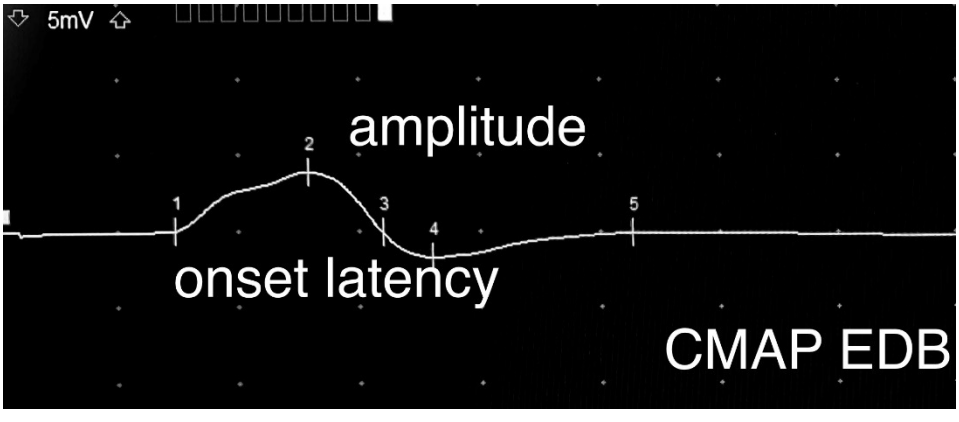

Fig 8. DPN motor waveform recorded at the EDB muscle. (marker 1 = onset latency; vertical distance from marker 1 to marker 2 = amplitude; area under peak from marker 1 to marker 3 = area under the curve)

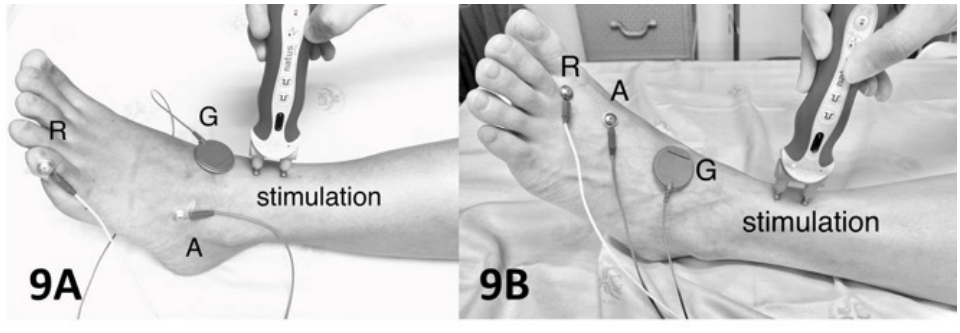

Fig 9. DPN stimulation for motor NCS recorded at the EDB muscle **(9A)** and for antidromic sensory NCS **(9B)**. (A = active electrode; R = reference electrode; G = ground electrode)

TABLE 1. Reference parameters for DPN in electrodiagnosis.

Electro-diagnosis study	Muscle/nerve, electrode placement	Stimulation	Age (year)	Sex	Onset latency (mean, ms)	Amplitude (mean, mV)	Velocity (mean, m/s)	Reference
CMAP	EDB - Active: midpoint of the muscle belly - Reference: slightly distal to the 5th metatarsophalangeal joint	(1) 8 cm proximal to the active electrode lateral to the TA tendon (2) posterior and inferior to the fibular head (3) 10 cm from (2) medial to the tendon of the biceps femoris	All 19–39 40–79		4.8 (6.5)^{U} 4.8 (6.5)^{U} 4.8 (6.5)^{U}	5.9 (1.3)^{L} 6.8 (2.6)^{L} 5.1 (1.1)^{L}	47 (38)^{L} 47 (38)^{L} 47 (38)^{L}	(59)
CMAP	TA - Active: one-third of the distance from tibial tubercle to lateral malleolus - Reference: on the bony surface of the tibia	(1) posterior and inferior to the fibular head (2) 10 cm from (1) medial to the tendon of the biceps femoris	All 19–29 30–79		3.6 (4.9)^{U} 3.6 (4.9)^{U} 3.6 (4.9)^{U}	3.8 (1.7)^{L} 4.3 (2.1)^{L} 3.6 (1.5)^{L}	62(43)^{L} 62(43)^{L} 62(43)^{L}	(60)
CMAP	EDB, ADPN	Posterior to lateral malleolus	All male female		4.84 4.95 4.69	1.27 0.97 1.67	55.7 56.85 58.11	(20)
SNAP	DPN - Active: the interspace between the 1st and 2nd metatarsal heads - Reference: 3 cm distal to the active electrode on the 2nd digit	12 cm proximal to the active electrode and just lateral to the EHL tendon	35 35–50 >50			9.73 μV 7.95 μV 6.65 μV	41.01 41.23 40.28	(61)

^{U} Upper limit of normality. ^{L} Lower limit of normality.

1.1.3 Sensory NCS

Sensory NCS assesses the density of the functioning nerve fibers. The action potential recorded is a summation of all the sensory fibers (sensory nerve action potential, SNAP). The study of the DPN is usually done in an antidromic manner and recorded at the first dorsal web space of the foot. The stimulation site is at the anterior ankle (Fig 9B). The estimated normal values are shown in Table 1. Lo *et al.* reported that the SNAP amplitude could be diminished in a normal population and even more in the elderly.[61] This should be considered in interpreting the results. Furthermore, anatomical variations could also affect the results. From a former study in healthy volunteers, the first dorsal web space of the toes was supplied by an isolated DPN for 34% of all studied limbs, SPN for 10%, both nerves for 44%, and

no response for 12%. The gross diameter of the nerve is very small and it is more difficult to stimulate in swollen limbs.[62]

One study reported that the NCV from the orthodromic method was more sensitive than that from the antidromic method in detecting lesions. The orthodromic method in the DPN involves stimulating at the ankle and recording the response at above and below the fibular head. The action potential recorded is a mix and not only from the DPN; hence it is called the mixed nerve action potential (MNAP). In healthy subjects, the mean sensory orthodromic NCV of the DPN was found to be 55.27 m/s. The mean MNAP amplitudes of the DPN under and above the fibular head were 1.13 μV and 0.52 μV, respectively. This method is easier to apply even in an elderly population. Therefore, an absent response may indicate a true abnormality.[63]

1.2 Needle electromyography (needle EMG)

This study can provide more information, such as whether the pathology is acute or chronic, and the signs of denervation and re-innervation from axonal loss, than NCS. In this method, a needle electrode is inserted into the muscle and electrical signals from muscle contractions are recorded. Spontaneous activity, and the number and morphology of the motor units are observed. However, a study by Dumitru *et al*. in an asymptomatic healthy population reported a prevalence of abnormal spontaneous activity of EDB in 60% of all subjects.[64] History taking and physical examination should be performed and the findings correlated since foot trauma can result in spontaneous activity in this muscle. Spontaneous activity in diffuse, decreased recruitment and an abnormal morphology may indicate the true pathology.[65]

2. Evaluation of DPN abnormality

Most pathologies of the DPN near the fibular head are axonal loss[66,67] because the fascicles at this level are located anteriorly, and thus are more sensitive to pressure or stretching. ATT is another common suspected area for DPN pathology, but is a rare condition. The mechanism is nerve compression by IER. The common presentations are a dull ache, numbness, or paresthesia radiating to the 1st interdigital space of the dorsum of the foot. An extremely serious condition is a weakness of the EDB muscle. The electrophysiologic study mostly finds chronic neuropathic patterns.[65] Two common electrodiagnostic findings are axonal loss and demyelination. For axonal loss, needle EMG can be used to show signs of denervation, whereas NCS can show an absent, low, or more than 50% decreased amplitude compared to the unaffected side. For focal demyelination, NCS may show a conduction block pattern (CMAP amplitude drop at the distal compared to in proximal stimulation) and a decreased NCV difference between segments of more than 10 m/s as described above. Significant demyelination is usually found co-existing with mild axonal loss.

3. Evaluation of DPN recovery

Derr *et al*. studied peroneal nerve recovery in both traumatic and non-traumatic cases by electrodiagnosis at 2 weeks to 24 months after onset. The stimulating site was at the fibular head. The recording sites were at the TA and EDB muscles. A good outcome was a TA motor power by manual muscle testing from grade 4 and higher. Presentation of a CMAP response in both EDB and TA muscles was a predictor for good outcome recovery (94% in all cases, 80% in trauma, and 100% in non-trauma). A good outcome was found in 81% and 94% of subjects who had CMAP presentation only in the TA and EDB, respectively. However, the absence of EDB CMAP and TA CMAP did not mean a poor outcome (46% had good outcome recovery). Most traumatic cases showed an absence of EDB CMAP. A response of EDB CMAP in the first three months can predict a good outcome (100% of all cases had good outcome recovery). From one needle EMG study, discrete or absence of motor unit recruitment in a trauma group predicted a worse outcome recovery, but without statistical significance. However, this was a small retrospective study and might not have had a large enough sample size.[68] Another study was performed in non-traumatic subjects and reported that if only a conduction block, which means demyelination, was found at 2–12 weeks after onset, a good recovery at 6 months could be anticipated.[69]

4. Evidence for the use of electrodiagnostic studies

Due to the nature of study, the supporting evidence was limited to class III for NCS and class IV for needle EMG for peroneal neuropathy diagnosis, according to an American Association of Neuromuscular and Electrodiagnostic Medicine review.[70]

5. Key takeaway

Being a nerve with both sensory and motor components, DPN can be electrophysiologically evaluated via the NCS for its sensory and motor function together with the needle EMG at its muscle targets. These studies can be summed up for identifying the abnormality and following the recovery of the DPN. Electrodiagnostic finding of the DPN should also be correlated with the

history and the physical examination of the patient to effectively evaluate the abnormality and to set the plan for treatment. However, evaluation techniques and anatomical variations can affect this finding. Therefore, anatomical knowledge is the important foundation to conduct the electrodiagnosis accurately and efficiently.

Application in surgery

1. Surgical application as a sensory nerve: Burned hand

The first web space contracture in a burned hand is one of the most common problems resulting in significantly decreased hand function. To achieve the goals following specific treatment principles[71,72], there are many options possible for the surgical reconstruction, including skin grafting[73,74], local flaps[75,76], regional flaps[77], and free flaps.[78,79,80] According to the principles, healthy tissue replacement or rearrangement is not sufficient. It requires sensory restoration together with glabrous skin restoration to achieve the ultimate goals, nearly-normal hand function.

With the aforementioned goals in mind, transferring tissue from another glabrous area with its sensory innervation is the choice. In the first web space, the connection between the MTB of the DPN and SPN provides sensory supply as the dorsal digital nerves to the 1st dorsal web space of the foot.[15] Either the DPN or the dorsal digital nerve can be transferred for harvesting an innervated free flap based on the shape and extension of the defect in the hand area. Understanding its anatomical variations, including level of bifurcation and absence of bifurcation will help surgeons prepare for additional steps in advance, such as intraneural dissection or nerve graft harvest. The former may require additional instrument for more meticulous dissection to minimize donor site morbidity such as motor nerve injury. On the other hand, the latter requires preparation of another donor site for nerve graft harvest. Either of the options, it is to perform tension-free microsurgical anastomosis of the donor nerve to a sensory nerve at the recipient site for ensuring the best condition for sensory recovery. In clinical scenarios, this innervated tissue transfer has been used with successful outcomes, such as a near-normal range of motion and sensation (2-point discrimination) of the reconstructed 1st web space.[80]

2. Surgical application as a motor nerve: Facial reanimation

The ultimate goal of reconstruction in patients with facial paralysis is to restore the lost function to normal, i.e., facial reanimation. Nevertheless, in cases of long-standing paralysis, the facial muscle is assumed to be atrophic after 24 months and muscle transfer is the option.[81] The transferred muscle can be connected with a source nerve for neural regeneration, resulting in a functional muscle. Principally, the donor muscle should be expendable and have a reliable neurovascular pedicle, sufficient length, and excursion muscle inset. In facial reanimation, especially smile reconstruction, gracilis muscle transfer is the gold standard.

Although not the gold standard option, another possible option is the DPN-innervated EDB muscle. The possibility of this flap in facial reanimation is not limited to smile reconstruction, but also to eyelid function.[82] This flap is mainly supplied by the LTA, and additional branches from the DPA and the peroneal artery[82], thereby considered as a Mathes–Nahai classification type 2.[83] The mean length and external diameter of the LTA were 2.3 cm and 2.4 mm, respectively, which were considered suitable as a choice for microsurgical anastomosis.[82] In addition to the vascular supply, the native function of EDB muscle is to assist the EDL muscle, harvesting this EDB muscle therefore results in a minimal deficit of the donor site.[84,85] Regarding this, understanding an anatomical course of the LTB of the DPN is important for safe dissection and flap harvest. It runs dorsolaterally along with the LTA and passes beneath the EDB muscle. Proximally, there is anatomical variation about its level of bifurcation. This may limit the length of the donor nerve possibly requiring nerve graft harvest. These issues share a similar principle of avoiding donor site morbidity (sensory nerve preservation in this application) and tension-free anastomosis of the nerve. In clinical scenarios, its application as a free flap in facial reanimation has been reported with successful results in variable cases.[86,87]

3. Key takeaway

Understanding the anatomy of the DPN, its variations, and its relation to adjacent structures is crucial for harvesting a flap for reconstruction such as the 1st web space reconstruction and free-functioning muscle transfer for facial reanimation. This is not only important for operative success, but also helps minimize both the donor site morbidities and the operative time due to the appropriate surgical planning.

CONCLUSION

As one of the nerves that is commonly manipulated in current clinical practice, anatomical knowledge and information on anatomical variations of the DPN are mandatorily essential for clinicians to understand the demographic nature of the DPN prior to its proper

application in patients. Nowadays, the clinical procedures implicated with this nerve can widely range from non-invasive ones, e.g., nerve imaging, to sophisticated surgical approaches, such as flap transfer for the reconstruction of a lower extremity, which have been regularly conducted in specialized clinical institutes.[88] This review therefore linked the fundamental anatomy of this nerve to its practical importance visualized in the examples of current clinical applications reviewed in this study by the main focus on its practicality and utilization. This review therefore presented the usefulness of translational research in this area for clinical implementation. Conclusively, the ultimate goal of this review is to help maximize the therapeutic effectiveness and to minimize the unanticipated complications of any clinical practices involving the DPN by inferring from its anatomical knowledge.

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