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Research Article

The Effects of Ankle Joint Position on Deep Peroneal Nerve Latencies

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ABSTRACT

Introduction: Joint positioning can impact nerve function. Few studies have explored the effects of ankle positions on deep peroneal nerve conduction. This cross-sectional study investigated the influence of different ankle joint positions on the deep peroneal nerve's distal motor and sensory onset latencies.

Materials and Methods: A total of 31 healthy adults (23.4±3.9 years old) underwent a deep peroneal nerve conduction study. Distal motor and sensory onset latencies were measured at neutral (0°), dorsiflexion (20°) and plantar flexion (40°) ankle positions.

Results: Changing ankle position significantly affected distal motor (P=0.001) and sensory onset latencies (P=0.001). Latencies were shortest in dorsiflexion (motor: 3.8±0.46; sensory: 2.4±0.2 ms), followed by neutral (motor: 4.2±0.5; sensory: 2.6±0.3 ms) and most prolonged in plantar flexion (motor: 5±0.6; sensory: 3.3±0.2 ms).

Conclusion: Ankle position impacts deep peroneal nerve conduction. Dorsiflexion and neutral positions reduced distal motor and sensory latencies compared to plantar flexion. These findings provide preliminary evidence that may help optimize ankle positioning in electrodiagnostic testing. Further blinded research with larger, more diverse samples is warranted.

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Introduction

Peripheral nerves possess viscoelastic properties, enabling adaptation to repetitive force and positional changes imposed by limb movements [1]. As joints move through the range, associated nerves must stretch and slide to accommodate changes in length [2]. The deep peroneal nerve innervates muscles controlling ankle position and movement [3]. Dorsiflexion is primarily mediated by deep peroneal-innervated tibialis anterior, while plantar flexion relies more on triceps surae muscles supplied by the tibial nerve [4]. Given its role at the ankle, the function and conduction of the deep peroneal nerve may be impacted by ankle joint positioning [5].

Several studies have revealed that joint positions affect the conduction parameters of associated nerves. Sustained elbow flexion prolongs ulnar motor distal latency [6]. Similarly, median sensory latency increases with wrist hyperextension [7]. At the lower limb, common peroneal latency varies with knee and hip position [8]. However, few studies have specifically investigated the impact of ankle angles on deep peroneal nerve function. This gap exists in the current literature.

With ankle motions, the deep peroneal nerve must slide longitudinally and transverse within its interface to avoid excessive strain [9]. However, adverse neural effects may occur if positioned in slack or excessive tension for prolonged periods. Animal studies reveal that 6-15% tensile strain on nerves reduces action potential amplitude and axonal transport [10]. In humans, prolonged nerve bed elongation increases interfascicular pressure and slows conduction velocities [11].

The deep peroneal nerve is under the greatest tension in plantar flexion at the ankle as muscle origins and insertions are pulled apart [12]. In contrast, dorsiflexion may slacken the nerve as muscle length decreases [13]. If plantar flexion is sustained, the heightened strain could perturb deep peroneal conduction [14]. This concept is supported by trials in carpal tunnel syndrome, showing that wrist flexion stresses the median nerve, delaying distal latencies [15]. However, few electrodiagnostic studies have specifically assessed deep peroneal conduction in different ankle positions.

Quantifying the impacts of ankle angles is important, given that certain occupations require prolonged postures. For example, high heel shoes worn by many women maintain the ankle in plantarflexion [16]. Prolonged driving can also sustain dorsiflexion [17]. If ankle positions affect deep peroneal conduction acutely, long-term effects may manifest in those with occupational ankle postures.

Clinically, optimizing ankle positioning during electrodiagnostic testing could maximize nerve conduction. This action may enhance diagnostic sensitivity in conditions like deep peroneal neuropathy. Furthermore, recognizing detrimental positions could better inform conservative care. Patients with deep peroneal entrapment often receive stretching and footwear advice [18]. Guiding exercise and ergonomics based on ankle angles that minimize nerve strain may improve rehabilitation.

This study aimed to address the gap in the literature by investigating the effects of different ankle positions on deep peroneal nerve distal motor and sensory latencies. We hypothesized that plantar flexion would prolong latencies compared to neutral and dorsiflexion angles due to heightened nerve strain. The findings may have implications for electrodiagnostic testing, conservative management, and ergonomic guidance in deep peroneal neuropathy.

Materials and Methods

Study design

This observational cross-sectional study involved one group of participants measured at three different ankle positions. The independent variable was ankle position at three levels: Neutral, 20° dorsiflexion and 40° plantar flexion. The dependent variables were the distal motor latency and sensory onset latency of the deep peroneal nerve measured bilaterally at each ankle position.

Study setting

The study took place in the physical therapy laboratory at Ahram Canadian University between December 5, 2022 and January 3, 2023. All data collection and procedures were conducted in a controlled laboratory environment. Participants were positioned supine on a plinth with their lower legs exposed for electrode placement and stimulation.

Study participants

A total of 31 participants aged 20-40 years with a body mass index of 18.5-24.9 kg/m^2 and no history of obesity, diabetes, hypertension, peripheral nerve injury or dysfunction, or previous lower extremity fracture or surgery

Figure 1. Neuropack S1 MEB-9004 Nihon Kohden, Japan

were recruited by convenience sampling from the local university population.

A priori power analysis using G*Power software, version 3.1 determined that a sample size of 28 was required to detect a medium effect size of 0.25 at an alpha of 0.05 and power of 0.80 for the primary outcome measure of deep peroneal nerve distal motor latency. Accounting for 10% dropouts, the final sample size was 31 participants. This sample size was sufficiently powered to detect clinically meaningful differences between ankle positions for the primary outcome measure.

Standardization procedures

The principal investigator performed all experimental preparation, instructions, electrode placement, ankle goniometry, and data collection to reduce measurement variability. Electrode placement was determined using precise anatomical landmarks according to surface electromyography for a non-invasive assessment of muscle guidelines to improve inter-rater reliability [19]. The participants were given standardized instructions for positioning and relaxation. Trials were discarded and repeated if submaximal effort was observed. Room temperature was closely monitored and controlled throughout data collection. Room temperature was confirmed within the 22±2 ˚C range.

Outcome measures

Distal motor latency

Distal motor latency of the deep peroneal nerve was the primary outcome measure, quantifying the time from stimulation to the onset of muscle response in the extensor digitorum brevis [20].

We employed Neuropack S1 MEB-9004 (Nihon Kohden, Japan) to objectively evaluate both motor distal and sensory onset latencies. It comprises a main unit with high-performance 2-channel amplifiers and a junc-

Figure 2. Ground electrode

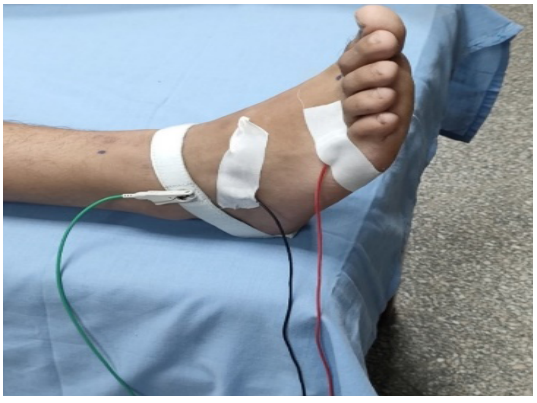

Figure 3. Recording electrodes

Figure 4. Stimulating electrode

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tion box with an articulated arm. The recording, stimulating, earth electrodes are attached to the junctional box (Figure 1).

The electrodes attached to the junctional box are divided into ground electrodes used to prevent or minimize noise (Figure 2) and two recording electrodes (one is negative and black while the other is positive and red) (Figure 3) used to pick up the signals. The last electrode is the stimulating one used to stimulate the nerve at a certain predetermined site (Figure 4).

The active recording electrode was positioned over the muscle belly of the extensor digitorum brevis, identified through palpation and muscle contraction during toe extension [21]. Correct placement was confirmed by observing the largest motor response on the electromyography (EMG) monitor during low-intensity stimulation. The reference electrode was placed electrically neutral at the fifth metatarsophalangeal joint. The ground electrode was secured around the ankle joint to reduce interference. Stimulation of the deep peroneal nerve was performed using a surface stimulator. The cathode was positioned over the deep peroneal nerve at the level of the fibular head, slightly anterior to the biceps femoris tendon. The anode was placed 2 cm distal to the cathode. A square-wave pulse with a duration of 0.2 ms was used for stimulation. To minimize the risk of movement of the stimulating electrodes, they were secured in place with adhesive tape (Figure 5).

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Figure 6. Placement of recording electrodes for sensory branch of deep peroneal nerve

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Figure 5. Placement of recording electrodes for motor branch of deep peroneal nerve

Latency was measured from the onset of the stimulus artifact to the first major negative deflection of the compound muscle action potential, indicating muscle depolarization [22]. Sensitivity was set at 1 mV per division as recommended for motor nerve conduction studies to accurately detect the compound muscle action potential response without exceeding the amplifier limits [23]. Signals were amplified with a gain of up to 10000 to sufficiently resolve the waveform for onset latency and amplitude measurements [23]. Latency values were measured in ms with 100 µs precision. Latency was measured from the origin of the stimulus artifact to the first positive deflection of the sensory nerve action potential. Prolonged latencies indicate slowed nerve conduction velocity. Normal distal motor latency value ranges from 3.5 to 6.0 ms [24].

Sensory onset latency

The active recording electrode was positioned in the first web space between the metatarsal heads of digits 1 and 2 [25]. This position maximizes the sensory re-

Table 1. Repeated measures analysis of variance for measured variables

Ankle Position / Deep Peroneal Nerve	Mean±SD			F Value	P	Effect Size Cohen's f
	Neutral Position	Planter Flexion	Dorsiflexion			
Distal motor latency (ms)	4.2±0.5	5±0.6	3.8±0.46	30.39	0.001*	0.727
Sensory onset latency (ms)	2.6±0.3	3.3±0.2	2.4±0.2	25.9	0.001	0.700

* Significant.

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sponse from digital nerve fibers of the deep peroneal nerve under the extensor hallucis brevis. The reference electrode was placed 3 cm distal to detect potential travel toward the recording electrode. The ground electrode reduced interference (Figure 6).

A minimum of 50 traces were averaged for each sensory nerve action potential recording to obtain a robust response for accurate latency and amplitude measurements, as recommended for low amplitude potentials [23, 26]. The initially acquired signals at a gain of 20 µV/division were further amplified by a factor of 3x during analysis, resulting in a final amplification of 60 µV/division used for measuring the averaged waveform parameters [23]. Latency was measured from stimulation onset to the first major positive deflection of the sensory nerve action potential [27]. Latency values were recorded in ms with 100-microsecond precision. Normal upper limits are <4.5 ms [24].

Instrumentation

We used a Neuropack S1 MEB-9004 EMG system (Nihon, Kohden, Japan) to record distal motor latency and sensory onset latency of the deep peroneal nerve. For distal motor latency recordings, filters were set at 10 Hz to 10 kHz and the sweep speed was 5 ms/division to capture the compound muscle action potential accurately. Signals were sampled at 5 kHz to satisfy the Nyquist rate. For distal sensory latency recordings, filters were set at 20 Hz to 2 kHz and the sweep speed was 1 ms/division to maximize the resolution of the lower amplitude sensory nerve action potential. Signals were sampled at 5 kHz to satisfy the Nyquist rate. Latency values were measured in ms. A handheld universal goniometer was used to measure the maximal ankle joint range of motion.

Experimental protocol

The participants first underwent a familiarization session where electrode placement was determined. They were seated comfortably with their legs exposed. Skin preparation involved shaving and cleaning with alcohol pads at electrode sites. The participants must also acclimate in the room for 10 minutes before testing. Before data collection, the skin temperature was measured over the anterior ankle region, 5 cm proximal to the stimulation site for the deep peroneal nerve, using an infrared thermometer. The temperature was confirmed to be within the range of 33-35 °C.

After electrode placement, the participants were positioned supine on the plinth with hips and knees in neutral rotation and 0° flexion. The ankle was positioned in neutral (0°) plantar flexion or dorsiflexion with the foot relaxed.

Table 2. Post Hoc test between different positions

Variables		Distal Motor Latency	Sensory Onset Latency
Neutral vs planter flexion	Difference	-0.75	-0.7
	P	0.001*	0.001*
Neutral vs dorsiflexion	Difference	0.4	0.2
	P	0.001*	0.001*
Planter flexion vs dorsiflexion	Difference	1.2	0.7
	P	0.001*	0.001*

*Significant.

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EMG recordings were anonymized and analyzed by an assessor blinded to ankle positioning to reduce potential bias during latency measurement. The principal investigator set up the ankle positioning delivered electrical stimulations, and collected the EMG recordings. To facilitate blinding, the order of ankle positioning was randomized across participants. The secondary assessor was not present in the room during data collection. This assessor received the de-identified EMG recordings and measured onset latencies. The assessor was blinded to the ankle position associated with each recording until all latency measurements were completed.

The ankle was positioned in neutral, full dorsiflexion, or full plantarflexion, and held continuously for 5 minutes. After 5 minutes of sustained positioning, electrophysiological stimulation and recording were performed for each position. Distal motor latency was recorded first, followed by sensory onset latency measurements at the neutral ankle position. The ankle was then moved into maximal dorsiflexion latencies and maximal plantarflexion with repeat measurements at each position. A 30-s rest was provided between ankle repositioning to avoid fatigue. After final measurements, maximal ankle dorsiflexion and plantarflexion range of motion were recorded.

Data processing and statistical analysis

Latency values were averaged across 3 trials at each ankle position. Normality was confirmed with the Shapiro-Wilk test. Repeated measures analysis of variance (ANOVA) was used to compare mean distal motor and sensory onset latencies between the three ankle positions. Pairwise comparisons were made with Bonferroni correction. The Pearson correlation coefficients were calculated between latency values and maximal ankle range of motion. Statistical significance was set at $P<0.05$. All analyses were performed using SPSS software, version 25.

Results

As shown in Table 1, the Mean±SD distal motor latencies of the deep peroneal nerve at neutral, plantar flexion, and dorsiflexion positions were 4.2±0.5, 5±0.6, and 3.8±0.46 ms, respectively. The univariate tests of repeated measure ANOVA revealed a statistically significant difference in distal motor latency of the deep peroneal nerve among the three measurements ($F=30.39$, $P<0.001$; Cohen's $f=0.727$). As observed in Table 2, pairwise comparison (post hoc test) revealed significant differences between distal motor latency at the neutral position and plantar flexion position, neutral position and dorsiflexion, between plantar flexion and dorsiflexion ($P=0.001$). This significant reduction is in favor of the ankle dorsiflexion position and ankle neutral position compared to the ankle plantar flexion position.

As shown in Table 1, the Mean±SD sensory onset latencies of the deep peroneal nerve at neutral, plantar flexion, and dorsiflexion positions were 2.6±0.3, 3.3±0.2 and 2.4±0.2 ms, respectively. The univariate tests of repeated measures ANOVA revealed a statistically significant difference in sensory onset latency of the deep peroneal nerve among the three measurements ($F=25.9$, $P=0.001$, Cohen's $f=0.700$). As observed in Table 2, pairwise comparison (post hoc test) revealed significant differences between sensory onset latency at the neutral position and plantar flexion position, neutral position and dorsiflexion, and plantar flexion and dorsiflexion ($P=0.001$). This significant reduction favors ankle dorsiflexion and ankle neutral positions compared to ankle plantarflexion positions.

Discussion

The ability of peripheral nerves to extend and slide is essential for maintaining proper neural function [1]. As a key nerve controlling ankle dorsiflexion and foot inversion, the deep peroneal nerve must adapt its position within surrounding tissues in response to biomechanical loads from routine joint motions like walking or more extreme ankle positions [28].

Our study reveals that ankle joint position significantly influences deep peroneal motor and sensory nerve conduction. We found that 20° dorsiflexion and neutral positions reduced distal motor and sensory onset latencies compared to 40° plantar flexion. The significant reduction in latencies at 20° dorsiflexion and neutral positions compared to 40° plantar flexion was consistent across measurements, suggesting that the position itself, rather than its duration, was the primary factor influencing nerve conduction. Our findings suggest that the mechanical and physiological changes associated with different ankle positions, such as stretching or compression of the nerve, can acutely affect nerve conduction properties. This finding is important for clinical nerve conduction studies, where the limb's position being tested could potentially influence the results.

Our findings suggest that the ankle position can significantly affect the distal motor and sensory latencies of the deep peroneal nerve. Therefore, it is possible that the standard practice of performing neurographic stud-

ies with the ankle in a neutral position may not provide a complete or accurate assessment of nerve function. One potential implication of our study is that clinicians and technicians who perform neurographic studies of the deep peroneal nerve may want to consider assessing nerve function in multiple ankle positions to obtain a more comprehensive understanding of nerve function. However, we acknowledge that changing the standard practice of neurographic assessment is not a decision to be taken lightly.

The physiological basis for the effects of ankle position on deep peroneal nerve conduction likely involves the biomechanical impacts on the nerve itself. In plantar flexion, the nerve comes under increased tensile stretch as the posterior calf muscles, like gastrocnemius, elongate [29]. This position places a traction force on the deep peroneal nerve since it runs adjacent to and innervates muscles in the anterior compartment [30]. The tensile load alters nerve function through mechanical effects on axonal microtubules and neurofilaments that transmit the nerve impulse [31, 32]. Over 8-15% elongation, vascular perfusion within the nerve also becomes impaired, compounding the functional effects [33].

In contrast, a neutral position avoids excessive stretch, while dorsiflexion may allow slight nerve relaxation [34]. The nerve can glide more optimally with less mechanical deformation of axonal cytoskeletal elements [35]. This position helps preserve conduction velocity and activation timing [36].

The prolonged latency with plantar flexion can be explained by the increased stretch force imposed on the nerve in this position [37]. This tensile load likely alters nerve function and conduction by increasing the distance signals travel from the stimulation to the recording site [38]. Over time, sustained stretch may decrease nerve conduction velocity by causing intraneural changes like reduced blood flow [39]. Cadaveric research shows that peripheral nerves can elongate around 6% before adverse impacts occur, including decreased action potential amplitude, venule flow reduction at 8% strain, and intramural vascular occlusion at 15% strain [1]. Prolonged elongation increases interfascicular pressure and slows conduction time [40].

Numerous studies support that nerve positioning in a lengthened state negatively affects conduction parameters [6]. They found prolonged ulnar nerve stretching from elbow flexion during phone use reduces motor conduction velocity and increases latency, especially in those with ulnar neuropathy [7]. Also, studies show that wrist hyperextension positions the median nerve under stretch, worsening motor and sensory conduction while preparing for radial catheterization. Prolonged hyperextension could progress to a full conduction block.

However, one study [41] found no impact of elbow flexion up to 120° on ulnar latency, amplitude, or action potential duration. This disagreement may stem from their narrow 18 to 25 year old sample. Lack of temperature control during testing may also explain their discrepancy.

This preliminary study has limitations. First, the study was cross-sectional in design. Therefore, we cannot determine whether the differences in DML and DSL values between the different ankle positions are due to a causal relationship or other factors. Second, the study exclusively enrolled young, healthy adults, and different effects may be observed in older populations. Third, we did not control for confounding factors such as physical activity level and medical history. Practical issues also exist. Those with tight gastrocnemius may not tolerate 20° dorsiflexion, suggesting a neutral position may be optimal. Having subjects actively hold dorsiflexion is complex, and passive positioning by a brace or examiner may be required.

Our preliminary findings reveal that ankle position significantly impacts deep peroneal nerve conduction. Prolonged plantar flexion appears to adversely affect parameters, while neutral and dorsiflexion are optimal. We also emphasize the importance of consistent positioning when collecting normative nerve conduction data. Further research could examine the effects on older adults, obese populations, and those with pre-existing neuropathies.

Conclusion

In conclusion, this preliminary study shows prolonged ankle plantar flexion worsens deep peroneal nerve conduction compared to neutral or dorsiflexed positions, likely due to increased nerve stretch.

Ethical Considerations

Compliance with ethical guidelines

This study was approved by the Ethics Committee of Cairo University (Code: P.T.REC/012/004076). All participants read and signed a written informed consent before testing. The study participants were informed about the purpose of the research and assured of the confidentiality of their information. Moreover, they were allowed

to discontinue participation in the study as desired. Before participant recruitment, the trial was prospectively registered on Clinical Trials.gov (ID: NCT05635721).

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Authors' contributions

Conceptualization: Mohamed Hussein El-Gendy, Mahmoud Salah Abd El-Fattah and Yasser Ramzy Lasheen; Methodology: Mohamed Magdy El Meligie and Efrem Kentiba; Investigation, formal analysis and writing-original draft: Mahmoud Salah Abd El-Fattah and Efrem Kentiba; Supervision: Mohamed Hussein El-Gendy and Yasser Ramzy Lasheen; Review and editing: Mohamed Hussein El-Gendy, Mahmoud Salah Abd El- Fattah and Yasser Ramzy Lasheen; Reading and final approval: All authors.

Conflict of interest

The authors declared no conflict of interest.

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