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Postural Threat and Balance Control in Diabetic Neuropathy: Evidence from Anticipatory Postural Adjustments

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Abstract

Background: Postural threat can influence both emotional and motor responses, particularly in individuals with diabetic peripheral neuropathy (DPN), who exhibit impaired balance and increased fall risk. This study examined the effects of postural threat on anticipatory postural adjustments (APAs) and anxiety in people with DPN compared with healthy controls.

Methods: This quasi-experimental study included twenty-five participants with DPN and twenty-five healthy controls. All participants completed the rise-on-toes task under two conditions: threat and no-threat. Center of pressure (COP) and electromyographic (EMG) signals were collected using a force platform and wireless EMG system. Anxiety was measured before and during each condition. Nonparametric Mann–Whitney and Wilcoxon tests with false discovery rate (FDR) correction were used, and effect sizes ® were calculated for significant comparisons.

Results: Individuals with DPN showed greater anxiety $P<0.001$, r=0.75) and delayed APA onset ($P<0.001$, r=0.62) compared with healthy controls. Both groups exhibited higher anxiety and larger, faster COP and EMG responses under threat ($P<0.001$, r=0.50–0.70), but the magnitude of these adjustments was smaller in the DPN group.

Conclusion: Postural threat increased emotional arousal and APA magnitude in both groups, though individuals with DPN demonstrated attenuated adaptive modulation. These findings suggest that incorporating psychological and sensorimotor balance training may enhance stability and reduce fall risk in this population.

Keywords: Anticipatory Postural Adjustment, Postural Threat, Diabetic Peripheral Neuropathy, Anxiety, Balance Control

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Introduction

Diabetic peripheral neuropathy (DPN) is one of the most prevalent complications of diabetes mellitus and significantly contributes to impaired balance and increased fall risk in affected individuals (1, 2). Alterations in sensory and motor pathways caused by neuropathy lead to deficits in postural control, gait instability, and delayed muscular responses (3-6). These balance impairments are not only due to peripheral nerve dysfunction but also to higher-level changes in the central nervous system that affect sensorimotor integration and emotional regulation (7, 8).

Maintaining postural stability requires efficient anticipatory postural adjustments (APAs)—automatic preparatory muscle activations that stabilize the body before voluntary

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↑What is "already known" in this topic:
Individuals with diabetic peripheral neuropathy (DPN) show impaired balance and delayed anticipatory postural adjustments due to sensory and motor deficits. Emotional factors such as anxiety and postural threat can further influence postural control efficiency.

→What this article adds:
This study provides novel evidence that postural threat increases anxiety and modifies anticipatory postural adjustments in DPN. It highlights that emotional and sensorimotor training targeting threat-related balance responses may improve stability and reduce fall risk in this population.

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movement (9). However, individuals with DPN show delayed and reduced APA responses due to altered proprioceptive feedback and muscle weakness(10, 11). Previous studies in DPN primarily examined static or reactive balance, whereas APA have remained largely unexplored (12, 13).

Postural threat refers to a condition in which individuals perceive an increased risk of instability or falling. Such threat-related states are known to influence balance control and APAs, particularly by altering postural muscle activation and center of pressure (COP) dynamics (14). In neurologically impaired populations, including those with Parkinson's disease and stroke, postural threat elicits exaggerated anxiety and less efficient postural strategies (15, 16). However, the specific impact of postural threat on APA responses in DPN remains poorly understood (17, 18).

One of the most common experimental paradigms to evaluate anticipatory postural adjustments under challenging balance conditions is the "rise on toes" task, in which participants are asked to stand barefoot and lift their heels off the ground to rise onto their toes. In individuals with DPN, this task provides valuable insight into how somatosensory loss and fear of instability interact to alter postural preparation mechanisms (19, 20).

Emotional and cognitive theories such as the Attentional Control Theory (ACT) (21) provide a useful framework to explain how anxiety influences postural control. According to this theory, heightened anxiety reallocates attentional resources toward threat-related stimuli at the expense of motor efficiency, leading to less automatic and more constrained movement control (21). This concept has been expanded by recent models showing that anxiety can impair postural automaticity through maladaptive attentional shifts and altered cortical processing (22). Despite being conceptually relevant, this theoretical perspective has rarely been integrated into DPN research.

Given these sensory-emotional interactions, the present study specifically aimed to examine how postural threat alters both anxiety and APA in individuals with DPN compared to healthy adults. We hypothesized that exposure to postural threat would increase anxiety and alter APA responses in both groups, with individuals with DPN exhibiting greater anxiety and more pronounced changes in postural control. By examining these interactions, this study provides novel insights into how emotional and sensorimotor processes interact under threatening balance conditions, with potential implications for targeted rehabilitation and fall prevention in diabetic neuropathy. These findings may inform the design of anxiety-regulated balance training programs (23, 24).

Methods

Study design

This research was a quasi-experimental study incorporating both between-subject factors (DPN vs. healthy) and within-subject conditions (threat vs. no-threat). This quasi-experimental design was chosen due to practical and safety considerations, which precluded randomization of participants or threat order. No randomization or clinical intervention was performed; instead, postural threat was experimentally manipulated (25). Participants were tested in a fixed order (No-Threat → Threat), consistent with established postural threat protocols, to ensure comparability with previous studies and to minimize habituation effects (26, 27).

Participants

Twenty-five adults with type 2 diabetes and clinically confirmed diabetic peripheral neuropathy (DPN) (18 females) and twenty-five age- and sex-matched healthy adults (18 females) participated in this study. The sample size was a convenience sample.

The sample size was estimated a priori using G*Power 3.1 software based on prior studies demonstrating threat-induced changes in anticipatory postural control. The calculation was based on a single primary outcome, namely COP onset latency during the anticipatory phase, as this parameter has been consistently reported as a sensitive indicator of postural threat–related modulation of anticipatory postural adjustments (26, 28). A conservative effect size of $r = 0.40$ was selected based on this literature and conventional benchmarks for effect size magnitude in behavioral research (29), with $\alpha = 0.05$ and statistical power set at 0.80, resulting in a required sample size of 25 participants per group. Participants under 65 years were included to minimize aging effects on balance and motor control (30).

The inclusion criteria for the DPN group were:

(1) Neuropathy Disability Score (NDS) ≥ 3;

(2) reduced sural or peroneal nerve conduction velocity;

(3) abnormal results on the 10-g Semmes-Weinstein monofilament test.

Only individuals with mild to moderate neuropathy (NDS 3–6) were recruited (31-33). Healthy controls were required to have normal fasting blood glucose (<100 mg/dL) and HbA1c <5.7%, verified by laboratory tests (30). All participants scored below 40 on the State–Trait Anxiety Inventory (STAI) and above 67 on the Activities-Specific Balance Confidence (ABC) Scale (24). Exclusion criteria included cognitive impairment, major depression, poor visual acuity, vestibular dysfunction, or other neuromuscular disorders affecting balance (32, 33). All participants provided written informed consent prior to participation, and the study protocol was approved by the Institutional Ethics Committee (Approval Code: PHT-0104).

Experimental Protocol

Balance performance was evaluated using a Computerized Dynamic Posturography system (NeuroCom EquiTest CRS, USA, 2015). The protocol consisted of six main trials: three under non-threat and three under postural threat conditions. Participants stood on a dual-force platform wearing a safety harness to prevent falls, while a visual target was positioned one meter ahead (34). Participants completed trials in a fixed order, beginning with the No-Threat condition followed by the Threat condition, in accordance with established postural threat paradigms (26, 28). In the threat condition, the platform delivered unpre-

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dictable forward–backward translations at a velocity of 0.6 m/s, peak acceleration of 0.25 m/s², and peak displacement velocity of 1.4 m/s². These perturbations occurred randomly at 15, 30, or 45 seconds, always before the "Go" cue. Unpredictability was maintained by randomizing timing across trials and participants (35). Participants were verbally informed that perturbations might occur during the threat condition but were unaware of the exact timing, ensuring psychological threat induction without physical harm (15). Immediately after each trial, participants rated their state anxiety using two 9-point Likert items adapted from the STAI, assessing cognitive and somatic anxiety components. Though concise, this measure has been shown to sensitively detect situational anxiety fluctuations during postural threat tasks (24).

Data Collection and Analysis

Center of Pressure (COP) Measures

Ground reaction forces and torques were sampled at 1000 Hz. Raw COP data were low-pass filtered using a fourth-order Butterworth filter with a 10 Hz cutoff (34). Four APA-related parameters were extracted during the preparatory phase: COP onset latency – time after the "Go" cue when COP deviated by ≥2 SD from baseline for ≥50 ms, Peak posterior COP displacement – maximum posterior shift from baseline, Time to peak displacement – interval between COP onset and maximum displacement, and Peak posterior COP velocity – calculated as displacement/time to peak.

During the execution phase, three parameters were analyzed: Peak anterior COP displacement – maximum forward shift from baseline, time to peak anterior displacement – duration from onset to maximum anterior movement, and Peak anterior COP velocity – calculated as anterior displacement divided by time to peak. These parameters quantify latency, amplitude, and velocity components of postural control and have been validated as sensitive markers of APA efficiency (23, 34).

Electromyography (EMG) Measures

Surface EMG signals were recorded using a wireless Biometric Lite Data system (USA) from the tibialis anterior (TA) and soleus (SOL) muscles. Skin was prepared by shaving and cleaning with alcohol to ensure low impedance. Electrode placement followed SENIAM guidelines (36, 37). EMG signals were band-pass filtered (10–500 Hz), digitized at 1000 Hz, and full-wave rectified. The root mean square (RMS) amplitude was computed over a 250-ms post-activation window. EMG onset latency was defined as activity exceeding 2 SD above baseline (200 ms pre-"Go") sustained for ≥50 ms. Latency was expressed relative to COP onset. EMG amplitude reflected activation magnitude within the first 250 ms of movement (24, 34).

Statistical Analysis

Descriptive statistics (medians, interquartile ranges [IQR]) summarized demographic and outcome data. Normality was tested using the Shapiro–Wilk test (38). Since most COP, EMG, and anxiety variables violated normality (P<0.01), nonparametric tests were used. Although some descriptive variables (e.g., Beck and STAI scores) showed approximately normal distributions, all variables were reported as median (IQR) for consistency and due to the small sample size. Given the repeated-measures structure, mixed-effects models were considered; however, due to the small sample size and non-normal distributions, nonparametric paired tests were used. To control for Type I error inflation due to multiple comparisons, False Discovery Rate (FDR) correction was applied using the Benjamini–Hochberg method (39). The Benjamini–Hochberg False Discovery Rate correction was applied separately within each outcome domain (psychological measures, COP measures, and EMG measures), rather than across all statistical tests combined. No additional sensitivity analysis (e.g., parametric robustness checks) was performed given the quasi-experimental design.

Between-group comparisons employed the Mann–Whitney U test, while within-group (threat vs. non-threat) comparisons used the Wilcoxon Signed-Rank test(40). Effect sizes ® were calculated for all significant results using the formula $r = Z / \sqrt{N}$, where Z is the standardized test statistic and N the total number of observations (41). All statistical analyses were conducted using IBM SPSS Statistics version 26 (IBM Corp., Armonk, NY, USA), with the significance level set at P<0. The results of normality tests (Shapiro–Wilk) confirmed that most COP, EMG, and anxiety variables were not normally distributed ($p < 0.01$). Therefore, non-parametric analyses were used, and effect sizes ® and FDR-adjusted p-values were also reported to account for multiple comparisons and practical significance (42). Effect sizes are reported without confidence intervals due to the non-parametric nature of the analyses and small sample size.

Results

As shown in Table 1, significant differences were observed in the STAI, ABC, and Beck Depression scores between the DPN and healthy groups, while the two groups were matched for BMI, sex, and age.

Psychological Measures

Median (IQR) was reported as Figure 1. The Wilcoxon signed-rank test showed a significant within-group increase in anxiety from the No-Threat to the Threat condition in both groups — DPN (P<0.001, r=0.59, FDR-adjusted P<0.001) and healthy controls (P<0.001, r=0.60, FDR-adjusted P<0.001) (Table 2).

Between groups, the Mann–Whitney U test showed that anxiety was significantly higher in the DPN group than in the control group under both conditions: Threat (P<0.001, r=0.67, FDR P<0.001) and No-Threat (P<0.001, r=0.69, FDR P<0.001). These findings show an association of postural threat with emotional response in both groups, with higher baseline and reactive anxiety among DPN participants (Table 2).

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Table 1. Descriptive characteristics of participants (Median [IQR])

Variable	DPN (n = 25)	Healthy (n = 25)	*P*-value	Effect size (r)
Age (years)	54 [49–60]	51 [46–58]	0.640	0.09
Gender (M/F)	7 / 18	7 / 18	1.000	—
BMI (kg/m²)	22.1 [20.0–24.3]	21.0 [19.5–23.9]	0.370	0.15
MMSE score	28 [27–29]	30 [29–30]	<0.001	0.72
Beck depression score	8 [7–9]	6 [5–7]	0.020	0.45
STAI score	32 [31–34]	24 [21–27]	<0.001	0.81
ABC score	71 [68–73]	89 [85–92]	<0.001	0.78
DM duration (years)	11 [7–15]	N/A	—	—
NDS score	4 [3–5]	N/A	—	—

Notes: Continuous variables are presented as median [interquartile range (IQR)] and analyzed using the Mann–Whitney U test. Categorical variables (gender) were analyzed using the Chi-square test. Effect size (r) was computed as r = Z / √N for non-parametric comparisons. All tests were two-tailed, with statistical significance set at *P*<0.05. Although some descriptive variables showed approximately normal distributions, all variables were reported as median [IQR] for consistency and due to small sample size. DPN: diabetic peripheral neuropathy; BMI: body mass index; MMSE: Mini-Mental State Examination; STAI: State–Trait Anxiety Inventory; ABC: Activities.

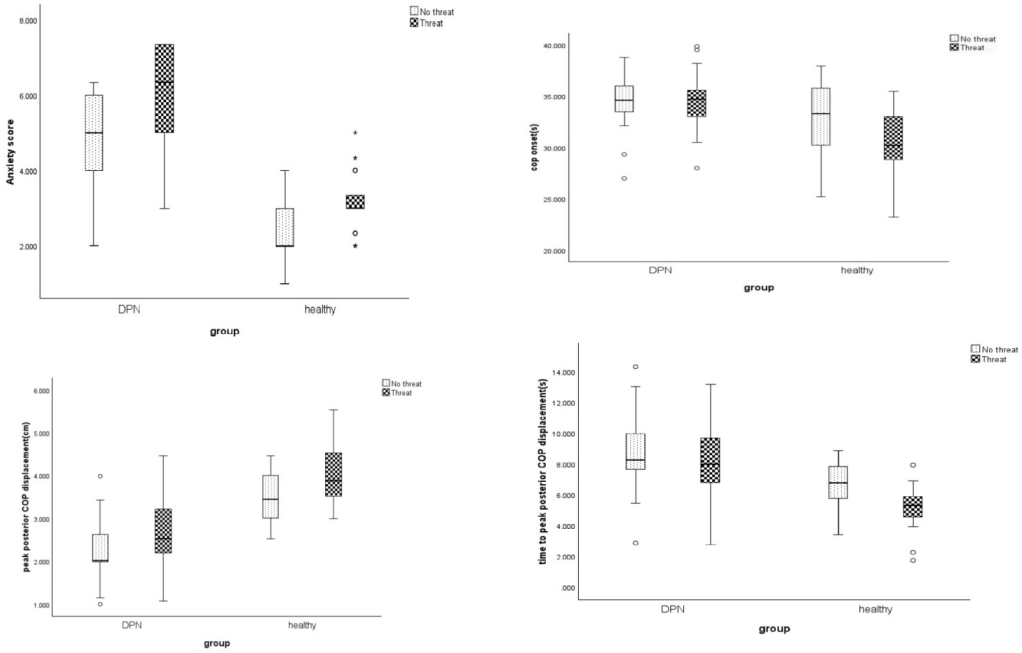

Figure 1. Boxplots of anxiety scores and COP parameters under threat and no-threat conditions
The boxplots illustrate medians and interquartile ranges (IQRs) for anxiety scores and COP displacement/timing measures across groups (DPN vs. healthy). Higher anxiety and reduced COP modulation are observable in the DPN group, particularly under threat.

Center of Pressure (COP) Measures

Median (IQR) was reported as Figure 1. During the anticipatory phase, the Wilcoxon test revealed an earlier COP onset latency in the Threat condition for the healthy group (*P*<0.001, r=0.61, FDR *P*<0.001), while no significant difference was observed in the DPN group (*P*=0.540). Between groups, COP onset was significantly delayed in DPN compared with controls for both Threat (*P*<0.001, r=0.65, FDR *P*<0.001) and No-Threat (*P*=0.070). Both groups showed larger posterior COP displacement in the Threat condition (DPN: *P*<0.001, r=0.60; healthy: *P*<0.001, r=0.61), yet DPN participants had smaller magnitudes under both conditions (Threat: *P*<0.001, r=0.67; No-Threat: *P*<0.001, r=0.69) (Table 3).

The time to peak posterior displacement was shorter in the Threat condition for both groups (DPN: *P*=0.009; healthy: *P*=0.003), but DPN participants exhibited longer latencies overall (Threat: *P*<0.001, r=0.64; No-Threat: *P*<0.001, r=0.59). Peak posterior COP velocity increased significantly under threat for both groups (DPN: *P*<0.001, r=0.61; healthy: *P*<0.001, r=0.60), but remained lower in DPN (Threat: *P*<0.001, r=0.69; No-Threat: *P*<0.001, r = 0.68) (Table 3).

During the execution phase, both groups showed greater anterior COP displacement under threat (DPN: *P*<0.001; healthy: *P*<0.001). However, DPN participants had smaller displacements in both conditions (Threat: *P*<0.001; No-Threat: *P*<0.001). The time to peak anterior displacement was shorter in the Threat condition (DPN: *P*<0.001;

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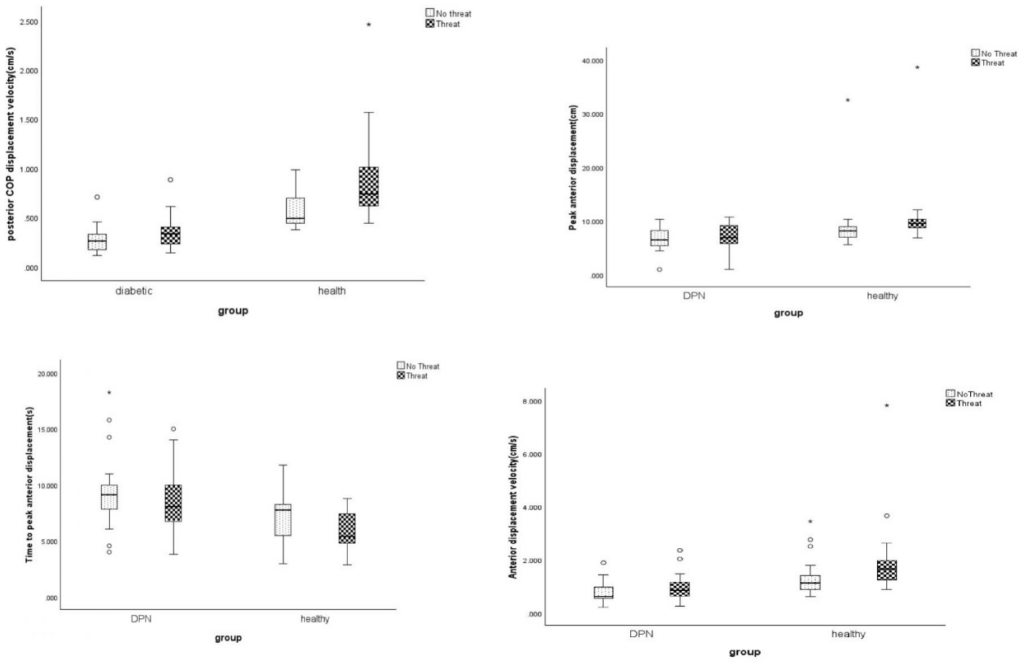

Figure 1 (continued). Additional COP parameters
Boxplots display IQRs and medians for COP onset latency, posterior displacement, and peak velocity under threat and no-threat conditions. DPN participants show delayed onsets and smaller displacement amplitudes compared with healthy controls.

Table 2. Summary of between- and within-groups comparison for anxiety under Threat and No-Threat conditions in DPN and healthy participants

Variable	Comparison type	Z / U	*P* (uncorrected)	r (effect size)	*P* (FDR-corrected)
Anxiety	Within-group (Healthy: No-Threat vs Threat)	Z = -4.23	<0.001	0.85	<0.001
	Within-group (DPN: No-Threat vs Threat)	Z = -4.17	<0.001	0.83	<0.001
	Between-group (DPN vs Healthy: No-Threat)	U = 49 (Z = -5.62)	<0.001	0.79	<0.001
	Between-group (DPN vs Healthy: Threat)	U = 25.50 (Z = -5.28)	<0.001	0.75	<0.001

Notes: DPN = Diabetic Peripheral Neuropathy; Z = Standardized statistic for Wilcoxon signed-rank test; U = Test statistic for Mann–Whitney U test; r = Effect size calculated as $|Z|/\sqrt{N}$ (N = 25 for within-group and 50 for between-group analyses); p = Uncorrected significance; p (FDR) = Adjusted p-value using Benjamini–Hochberg False Discovery Rate correction. Between-group comparisons were conducted using the Mann-Whitney test, and within-group comparisons using the Wilcoxon Signed-Rank test. Effect sizes are reported. All results were considered statistically significant at $p < 0.05$ (two-tailed).

healthy: *P*<0.001) but remained longer for DPN across conditions (Threat: *P*<0.001; No-Threat: *P*<0.001). Peak anterior velocity also increased under threat (DPN: *P*<0.001; healthy: *P*<0.001), though DPN participants demonstrated lower values (Threat: *P*<0.001; No-Threat: *P*<0.001) (Table 3).

Electromyography (EMG) Measures

Median (IQR) was reported in Figure 2. Both TA and SOL EMG onset latencies were shorter under Threat compared with No-Threat for DPN (TA: *P*=0.004, r=0.41; SOL: *P*<0.001, r=0.52) and healthy groups (TA: *P*<0.001, r=0.56; SOL: *P*<0.001, r=0.44). Between groups, DPN participants showed delayed onsets in both muscles and conditions (TA Threat:, *P*<0.001, r=0.50; TA No-Threat: *P*=0.004, r=0.41; SOL Threat: *P*<0.001, r=0.70; SOL No-Threat: *P*<0.001, r=0.58). Both TA and SOL amplitudes increased significantly under threat (TA DPN: *P*=0.001, r=0.55; TA healthy: *P*<0.001, r=0.61; SOL DPN: *P*=0.001, r=0.50; SOL healthy: *P*<0.001, r=0.61), though amplitudes were smaller in DPN for both muscles (TA Threat: *P*<0.001; SOL Threat: *P*<0.001) (Table 4).

Discussion

This study was the first to examine how postural threat influences both emotional responses and anticipatory postural adjustments (APAs) in individuals with diabetic peripheral neuropathy (DPN). Consistent with our hypotheses, postural threat increased anxiety and modified APA characteristics in both DPN and healthy groups, though the magnitude and temporal patterns differed significantly (6, 17).

The current findings demonstrated that individuals with DPN exhibited higher anxiety levels and smaller, slower APA responses under both threat and no-threat conditions compared with healthy participants. These results align with prior research showing that neuropathic populations have diminished postural adaptability due to sensory and motor impairments (18, 23). The elevated anxiety observed under postural threat was associated with

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Table 3. Summary of between-group- and Within-group comparisons for COP measures in DPN and healthy participants under Threat and No-Threat conditions

Variable	Group comparison	Z / U	*P* (uncorrected)	r (effect size)	*P* (FDR-corrected)
COP onset latency (s)	Within-group (Healthy: No-threat vs. Threat)	Z = –4.37	<0.001	0.87	<0.001
	Within-group (DPN: No-threat vs. Threat)	Z = –0.60	0.54	0.12	0.560
	Between-group (DPN vs. Healthy; No-threat)	U = 222 (Z = –1.75)	0.07	0.25	0.090
	Between-group (DPN vs. Healthy; Threat)	U = 87 (Z = –4.38)	<0.001	0.62	<0.001
Posterior COP displacement (cm)	Within-group (Healthy)	Z = –4.37	<0.001	0.87	<0.001
	Within-group (DPN)	Z = –4.37	<0.001	0.87	<0.001
	Between-group (No-threat)	U = 51.50 (Z = –5.06)	<0.001	0.72	<0.001
	Between-group (Threat)	U = 62.50 (Z = –4.85)	<0.001	0.69	<0.001
Time to peak posterior displacement (s)	Within-group (Healthy)	Z = –3.00	0.003	0.60	0.005
	Within-group (DPN)	Z = –2.62	0.009	0.52	0.015
	Between-group (No-threat)	U = 122.50 (Z = –3.68)	<0.001	0.52	<0.001
	Between-group (Threat)	U = 70 (Z = –4.70)	<0.001	0.67	<0.001
Peak posterior COP velocity (cm/s)	Within-group (Healthy)	Z = –4.31	<0.001	0.86	<0.001
	Within-group (DPN)	Z = –4.37	<0.001	0.87	<0.001
	Between-group (No-threat)	U = 37 (Z = –5.34)	<0.001	0.76	<0.001
	Between-group (Threat)	U = 31 (Z = –5.46)	<0.001	0.77	<0.001
Anterior COP displacement (cm)	Within-group (Healthy)	Z = –4.34	<0.001	0.87	<0.001
	Within-group (DPN)	Z = –3.53	<0.001	0.71	<0.001
	Between-group (No-threat)	U = 163 (Z = –2.90)	0.004	0.41	0.007
	Between-group (Threat)	U = 168 (Z = –2.80)	0.005	0.40	0.009
Time to peak anterior displacement (s)	Within-group (Healthy)	Z = –4.29	<0.001	0.86	<0.001
	Within-group (DPN)	Z = –4.37	<0.001	0.87	<0.001
	Between-group (No-threat)	U = 163 (Z = –2.90)	0.004	0.41	0.007
	Between-group (Threat)	U = 168 (Z = –2.80)	0.005	0.40	0.009
Peak anterior COP velocity (cm/s)	Within-group (Healthy)	Z = –4.37	<0.001	0.87	<0.001
	Within-group (DPN)	Z = –4.37	<0.001	0.87	<0.001
	Between-group (No-threat)	U = 118 (Z = –3.77)	<0.001	0.53	<0.001
	Between-group (Threat)	U = 78 (Z = –4.55)	<0.001	0.64	<0.001

Notes : Summary of Center of Pressure (COP) measures within and between groups under threat and no-threat conditions. DPN = Diabetic Peripheral Neuropathy; COP = Center of Pressure; Z = standardized statistic for Wilcoxon signed-rank test; U = test statistic for Mann–Whitney U test; r = effect size calculated as |Z| / √N (N = 25 for within-group, N = 50 for between-group analyses); p (FDR) = adjusted p-value using Benjamini–Hochberg False Discovery Rate correction. . Between-group comparisons were conducted using the Mann-Whitney test, and within-group comparisons using the Wilcoxon Signed-Rank test. Effect sizes are reported. All results were statistically significant at $p < 0.05$ (two-tailed).

motor control changes rather than implying causation directly, further supporting the idea that emotional arousal modulates motor control efficiency (15).

Based on ACT, anxiety may shift attentional focus in ways that could influence motor preparation; however, this interpretation remains speculative (21, 43). Applying ACT to our findings, it is possible that heightened anxiety in DPN participants influenced attentional allocation, potentially contributing to delayed APA responses. This interpretation is consistent with evidence that anxiety-induced attentional shifts can reduce postural automaticity (16, 22). Additionally, diminished APA responses in DPN participants may also reflect neuropathy-related sensory deficits (23), which can impair the integration of sensory information for anticipatory balance control. These combined emotional and sensory constraints may result in smaller postural adjustments and slower muscle activation and lower balance confidence (as reflected by ABC scores) may be associated with a cycle of anxiety and avoidance, rather than proving causation directly (44).

Our findings also revealed that despite impaired APA magnitude and timing, individuals with DPN maintained a qualitatively similar pattern of adaptation to postural threat compared with controls. This suggests that while the amplitude and velocity of APA responses were reduced, the direction of adjustment (i.e., faster and larger APA under threat) remained consistent. This indicates some degree of preserved adaptability in individuals with DPN, though this interpretation should be viewed cautiously (8).

The reciprocal relationship between anxiety and postural control, as highlighted by the reviewer, deserves further consideration. On one hand, anxiety may reduce APA magnitude by disrupting attentional resources; on the other, impaired balance performance can itself increase anxiety and fear of falling. This bidirectional mechanism, sup-

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ported by recent neurobehavioral models (45) emphasizes the need for integrative frameworks that combine emotional and motor control perspectives in balance research (15, 46).

During the execution phase, both DPN and healthy participants exhibited increased anterior center of pressure (COP) displacement and velocity under threat conditions. These findings are consistent with previous studies show-

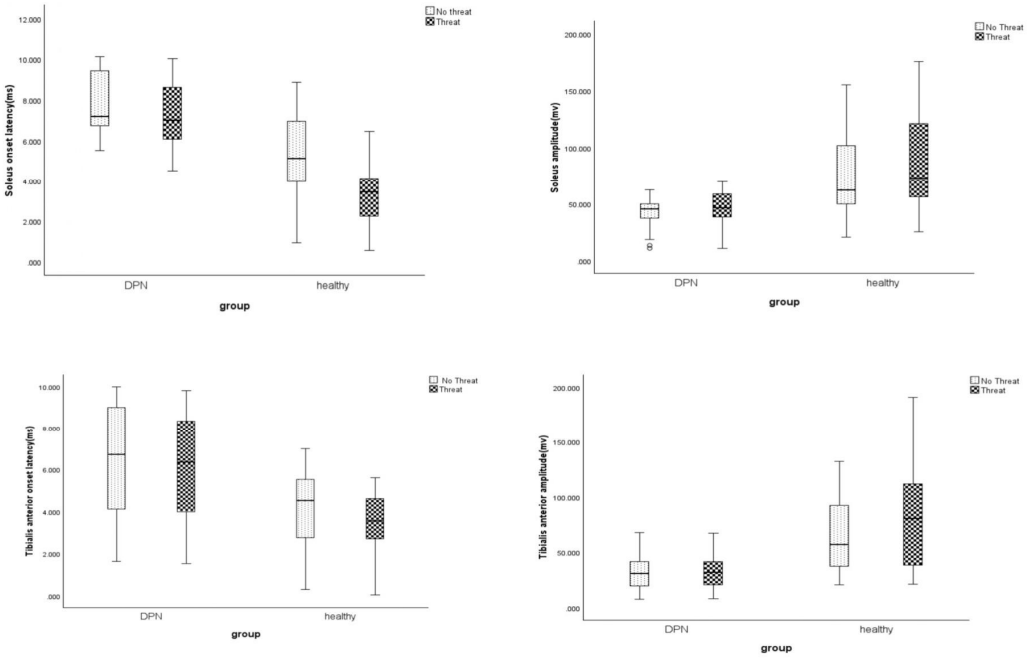

Figure 2. Electromyographic (EMG) activity of tibialis anterior (TA) and soleus (SOL)
Boxplots represent group medians and IQRs of EMG onset latency and amplitude under threat and no-threat conditions. Threat generally reduced EMG amplitudes in both groups, with DPN participants demonstrating delayed activation and lower amplitudes.

Table 4. Summary of between-group- and Within-group comparisons for EMG measures in DPN and healthy participants under Threat and No-Threat conditions

Variable	Group comparison	Z / U	*P* (uncorrected)	r (effect size)	*P* (FDR-corrected)
TA onset latency (ms)	Within-group (Healthy: No-threat vs. Threat)	Z = –3.91	<0.001	0.78	<0.001
	Within-group (DPN: No-threat vs. Threat)	Z = –2.86	0.004	0.57	0.008
	Between-group (DPN vs. Healthy; No-threat)	U = 165 (Z = –3.86)	0.004	0.55	0.007
	Between-group (DPN vs. Healthy; Threat)	U = 130 (Z = –3.54)	<0.001	0.51	<0.001
SOL onset latency (ms)	Within-group (Healthy)	Z = –3.02	<0.001	0.60	<0.001
	Within-group (DPN)	Z = –3.61	<0.001	0.72	<0.001
	Between-group (No-threat)	U = 100 (Z = –4.12)	<0.001	0.58	<0.001
	Between-group (Threat)	U = 16 (Z = –5.75)	<0.001	0.81	<0.001
TA amplitude (mV)	Within-group (Healthy)	Z = –4.37	<0.001	0.87	<0.001
	Within-group (DPN)	Z = –3.96	<0.001	0.79	<0.001
	Between-group (No-threat)	U = 134 (Z = –3.46)	<0.001	0.49	<0.001
	Between-group (Threat)	U = 123 (Z = –3.67)	<0.001	0.52	<0.001
SOL amplitude (mV)	Within-group (Healthy)	Z = –4.37	<0.001	0.87	<0.001
	Within-group (DPN)	Z = –3.42	<0.001	0.68	<0.001
	Between-group (No-threat)	U = 137 (Z = –3.40)	<0.001	0.48	<0.001
	Between-group (Threat)	U = 98 (Z = –4.16)	<0.001	0.59	<0.001

Notes: Summary of EMG comparisons between and within groups under threat and no-threat conditions. DPN = Diabetic Peripheral Neuropathy; TA = Tibialis Anterior; SOL = Soleus; Z = standardized statistic for Wilcoxon signed-rank test; U = test statistic for Mann–Whitney U test; r = effect size calculated as |Z| / √N (N = 25 for within-group, N = 50 for between-group analyses); p (FDR) = adjusted p-value using Benjamini–Hochberg False Discovery Rate correction. . Between-group comparisons were conducted using the Mann-Whitney test, and within-group comparisons using the Wilcoxon Signed-Rank test. Effect sizes are reported. All results were statistically significant at $P<0.05$ (two-tailed).

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ing that individuals respond to instability by enhancing COP velocity to minimize time spent in unstable postures (34). Such changes indicate a compensatory motor adaptation to threat, possibly mediated by increased cortical engagement and heightened arousal (24). While our results provide important insights, several methodological factors should be acknowledged. The small sample size, predominantly female participants, and reliance on nonparametric tests limit statistical power and generalizability; these are noted as study limitations rather than implying causation. However, the inclusion of effect sizes and FDR correction in the current analyses enhances interpretability and reduces Type I error risk, addressing reviewer concerns about statistical rigor.

Another limitation is the use of a brief anxiety scale derived from the State-Trait Anxiety Inventory (STAI). Although this measure captured essential changes in self-reported anxiety, future studies should incorporate validated multi-item scales and physiological indices such as heart rate or galvanic skin response to better capture emotional reactivity (47). Furthermore, incorporating direct measures of attentional focus (e.g., dual-task paradigms or eye-tracking) would allow stronger inference regarding the cognitive mechanisms proposed by the Attentional Control Theory. Despite these limitations, the present findings support the view that people with DPN are capable of modulating anticipatory postural strategies under postural threat, albeit less effectively than healthy controls, acknowledging that this association is observed, not causally proven. From a clinical perspective, this suggests that balance rehabilitation programs incorporating mild postural challenges (such as perturbation-based or height-induced training) could promote adaptive anxiety regulation and improve motor control in DPN (20, 23, 24, 48) .

Stuudy Limitations

While this study provides novel insights into the interaction between emotional and motor factors during anticipatory postural adjustments (APAs) in individuals with diabetic peripheral neuropathy (DPN), several limitations should be acknowledged.

1. Sample size and gender imbalance: the relatively small sample size (n = 50), particularly with a predominance of female participants, may limit the statistical power and generalizability. Future studies should include larger and more gender-balanced cohorts to enhance representativeness and strengthen the external validity of these results (49).

2. Anxiety measurement-2-item scale: Anxiety measure used in this study—two Likert-type items derived from the State-Trait Anxiety Inventory (STAI)—provided a quick assessment of situational anxiety but lacked comprehensive psychometric validation in this experimental context. Including more comprehensive, validated anxiety scales (e.g., full STAI or Beck Anxiety Inventory) and physiological indicators such as heart rate or galvanic skin response(47) would improve accuracy in capturing emotional interactions. Given that a brief two-item scale may not fully reflect the complexity of emotional responses under postural threat, future studies should use multi-item validated tools.

3. Limited clinical detail/neuropathy severity and residual confounding: The current study did not control for potential confounding variables such as physical activity level, duration of diabetes, medication use, or comorbidities that might influence both anxiety and postural control. Moreover, detailed clinical grading of neuropathy severity was not collected, limiting interpretation of how different stages of DPN may differentially affect APA responses. Given the quasi-experiment and non-randomized design, residual cofounding due to unmeasured variables (e.g., lifestyle, medication) cannot be ruled out. and sensitivity analyses to test the robustness of the results were not performed. Future research should incorporate standardized neuropathy assessments and include sensitivity analyses to better isolate the effects of threat on motor preparation.

4. Lack of direct attentional or neural measures: No direct measure of attentional focus or neural activation was included. As suggested by the Attentional Control Theory, anxiety-related attentional shifts may alter motor efficiency, which was not directly assessed in this study. Therefore, further studies could incorporate neurocognitive or eye-tracking techniques to directly assess attentional mechanisms during postural threat tasks (35, 43).

5. Trial-order/ habituation effects: Although the study included repeated trials, potential effects of habituation or fixed trial order were not explicitly analyzed. Future protocols should evaluate learning or adaptation effects across multiple exposures to threat stimuli to clarify temporal dynamics in APA modulation (15).

Conclusion

In summary, this study showed that postural threat is linked to increased anxiety and altered anticipatory postural adjustments in individuals with diabetic peripheral neuropathy. Compared with healthy controls, participants with DPN exhibited smaller and slower APA responses under both threat and no-threat conditions, consistent with known sensory and motor impairments. These findings should be interpreted as hypothesis-generating, given the non-interventional and quasi-experimental nature of the study. While no causal or clinical recommendations can be drawn, the observed associations may inform future interventional research exploring the relevance of graded postural challenges for understanding emotional–sensorimotor interactions and balance rehabilitation in individuals with diabetic neuropathy.

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Conflict of Interests

The authors declare that they have no competing interests.

Authors' Contributions

Shirin Rahimzadeh: Writing – review & editing, Writing – original draft, Methodology, Investigation, Concep-

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tualization. Amin Behdarvandan: Writing – review & editing, Writing – original draft, Validation, Supervision, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. Mehrnoosh Zakerkish: Writing – review & editing, Writing – original draft, Validation, Supervision, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. Neda Orakifar: Writing – review & editing, Writing Original draft, Validation, Methodology, Conceptualization. Razieh Mofateh: Writing – review & editing, Writing – original draft, Methodology, Conceptualization. Saeed Hesam: Writing – review & editing, Writing – original draft, Formal analysis, Conceptualization. Ramin Saki: Writing – review & editing, Writing – original draft, Software, Data curation, Conceptualization.

Ethical Considerations

This study was approved by the Ethics Committee of Ahvaz Jundishapur University of Medical Sciences (Ethics Code: IR.AJUMS.REC.1401.038). Written informed consent was obtained from all participants prior to participation.

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Data Availability

Data of this study is available from the corresponding author upon reasonable request.

AI Use Statement

AI was used solely to improve language and readability. The author takes full responsibility for the final content.

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