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Original Research Article

To determine the association of Nerve Conduction Velocity (NCV) with Body Mass Index (BMI) in health care workers of tertiary care hospital of Uttarakhand: A cross- sectional study in Neurophysiology Lab

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Abstract:

Objectives: The present study was to determine the association between BMI and NCV among healthcare workers in a tertiary care hospital in Uttarakhand.

Methodology: A cross-sectional study was carried out in 102 health care workers. Demographic information, BMI, and NCV parameters (latency, amplitude, and conduction velocity) of the right median nerve were measured.

Results: There was a strong negative correlation between BMI and right elbow NCV ($r = -0.42$, $p < 0.05$) and positive correlation with right wrist latency ($r = 0.35$, $p < 0.05$). ANOVA indicated statistically significant differences in NCV between BMI groups ($F = 4.87$, $p < 0.05$). Post-hoc analysis indicated that obese patients had significantly lower NCV than underweight and normal-weight patients.

Conclusion: Significant differences were observed in Latency 1 at the wrist and in both Latency 1 and Latency 2 at the elbow, suggesting that BMI may affect early latency components, possibly due to variations in soft-tissue thickness, altered stimulation distance, or adiposity-related physiological changes. Hence, increased BMI is significantly associated with impaired nerve conduction, emphasizing the need for weight management in healthcare workers to ensure optimal neuromuscular performance and prevent early neuropathic changes.

Keywords: Body Mass Index (BMI), Nerve Conduction Velocity (NCV), Obesity, Peripheral Neuropathy.

Introduction

Nerve conduction Studies (NCS) is an important electrophysiological measure employed to evaluate the functional integrity and speed of impulse conduction along peripheral nerves. It is an important tool in the early identification and diagnosis of peripheral neuropathies and other neuromuscular conditions. Abnormalities in NCS may indicate the presence of structural or functional changes within the nerve, making NCS a useful measure of neural health. Whereas it is known that variables including age, temperature, limb length, and metabolic status can influence NCS, more recent studies have started examining the influence of body composition, specifically body mass index (BMI), on nerve conduction [1].

Body Mass Index (BMI), an indirect marker for adiposity, is a commonly used, non-invasive tool to categorize people into underweight, normal, overweight, or obese groups. With the increasing worldwide prevalence of obesity and overweight states, it has become more crucial to understand the physiological consequences of elevated BMI. Obesity has been shown to be linked with several metabolic disturbances such as insulin resistance, dyslipidemia, and chronic low-grade inflammation, all of which can negatively impact neural structures and functions. Besides, mechanical compression of nerves by redundant adipose tissue could directly interfere with nerve conduction, particularly in the superficial nerves like the median and ulnar nerves [2].

A few international and Indian studies have implied a negative relationship between BMI and NCV, such that higher BMI is associated with slowing of nerve conduction, especially of motor and sensory nerves of the upper limbs. Yet, there is a lack of region-based data, particularly from the hilly and semi-urban population of Uttarakhand. Additionally, there are very few studies in which apparently healthy adults without any diagnosed systemic

disease have been specifically taken into consideration, and it is hard to separate the impact of BMI from other confounding comorbidities [3]. Objectives of the present study was to the determine the association of median motor nerve conduction velocity with different groups of BMI in dominant hand in healthy adults of Uttarakhand.

Materials and Methods

Study Design: An analytical cross-sectional study.

Place of Study: Neurophysiology Lab, Department of Physiology, VCSG Government Institute of Medical Sciences and Research (GIMS&R), Srinagar Garhwal, Uttarakhand.

Sample Size: The sample size was calculated based on a reference article:

Suba A (2021), "Effect of obesity on median and ulnar nerve conduction parameters"('abstract-2021-issue-1-thenmozhi – National Journal of Physiology', 2021).

Using the formula:

$n = (Z\alpha \times SD / Precision)^2$

Where:

n = Required sample size

Zα = Standard normal deviate at 95% confidence level (1.96)

SDSDSD = Standard deviation from the previous study

Precision = Acceptable margin of error

Calculated sample size = 102

Sampling Technique: Purposive sampling.

Study Population: Health care workers of VCSG Medical College and HNB Base Hospital, Srinagar, Uttarakhand.

Period of Study: August 2023 – June 2024.

Inclusion Criteria:

- Apparently healthy healthcare workers aged 18–45 years
- Voluntary participation with informed consent for nerve conduction study

Exclusion Criteria:

- Individuals with any known disease that could affect nerve conduction velocity (NCV)
- History of trauma, skin lesions, or swelling along the nerve pathway that may interfere with nerve conduction study recordings
- Positive family history of diabetes mellitus or hypertension

Data Collection Parameters:

Body Mass Index (BMI): Calculated using Quetelet's Index formula:

$BMI = Weight (kg) / Height (m)^2$.

BMI-Based Subgrouping: Participants were further divided into subgroups based on BMI classification.

Nerve Conduction Velocity Measurement Procedure: Nerve conduction velocity (NCV) measurements were performed using the Neurostim NS-4 (EMG/NCV/EP system, Medicaid Systems, India), equipped with 1, 2, or 4 recording channels. The system had sensitivity range of 0.1–0.2 μV and a sweep speed adjustable from 1 to 1000 ms/div. A hand-held constant current electrical stimulator with an output range of 0–100 mA (0–400 V) and adjustable pulse duration were used for stimulation.

Prior to the procedure, the protocol was explained in detail to each participant, and written informed consent was obtained. Participants were positioned comfortably in a supine posture on the examination couch. All metallic jewellery and accessories were removed to avoid interference with signal acquisition. Instrument calibration and settings were then be adjusted according to standard guidelines.

For recording, surface electrodes were placed over the abductor pollicis brevis (APB) muscle. The active electrode (–) was positioned close to the motor point of the APB, while the reference electrode (+) was placed 3 cm distal to it at the first metacarpophalangeal joint. Stimulation point S1 was located at the wrist, 3 cm proximal to the distal wrist crease, and stimulation point S2 was at the elbow, near the volar crease in proximity to the brachial pulse.

The course of the right median nerve was identified for accurate electrode placement. Electrical stimulation was initiated at a low baseline intensity and gradually increased, thereby recruiting a greater proportion of underlying nerve fibers and eliciting corresponding muscle fiber action potentials. In most cases, a current intensity of 20–50 mA was sufficient to achieve supramaximal stimulation.

The recorded response, known as the compound muscle action potential (CMAP), represents the summated electrical activity of all activated muscle fibers. When incremental increases in current no longer produce an increase in CMAP amplitude, supramaximal stimulation is presumed to be achieved. The current was then be further increased by 20% to ensure supramaximality.

The distance between the two stimulation sites (wrist and elbow) was measured using a flexible measuring tape along the presumed nerve path. Latency, measured in milliseconds (ms), was defined as the time interval between stimulus onset and the initial deflection from the baseline in the

CMAP waveform. The latency difference between the two stimulation sites was calculated, and the conduction velocity will be determined using the formula:

$$\text{Velocity} = \frac{\text{Distance between stimulation sites (mm)}}{\text{Difference between latencies (ms)}}$$

Statistical Analysis: Data was analysed with the help of latest version of SPSS software. Mean ± Standard deviations were observed. Correlational analysis, One Way ANOVA and post hoc analysis were performed. P-value was taken less than or equal to 0.05 ($p \leq 0.05$) for significant differences.

Observations and Results

Age Distribution: In the present study, the majority of participants belonged to the 20–30 years age group, comprising 68 individuals (65.4%), indicating that nearly two-thirds of the sample represented young adults. The 31–40 years category included 23 participants (22.1%), while the >40 years age group contributed 13 participants (12.5%). The overall mean age was 31.05 ± 7.58 years, further supporting that the study population was predominantly young to early middle-aged.

Gender Distribution: The gender distribution in the present study shows that males constituted the majority of the sample, with 71 participants (68.3%), while females accounted for 33 participants (31.7%).

BMI Distribution: The overall mean BMI was 24.05 ± 3.08, indicating that the study population, on average, fell within the upper-normal to overweight range.

Age Distribution Across the BMI: The association between age distribution and BMI category in the present study shows that among participants aged 20–30 years, 36 (76.6%) were underweight, 15 (53.6%) had normal BMI, 16 (66.7%) were overweight, and 1 (20%) was obese. In the 31–40 years group, 7 (14.9%) were underweight, 10 (35.7%) had normal BMI, 4 (16.7%) were overweight, and 2 (40%) were obese. Similarly, in the >40 years age group, 4 (8.5%) were underweight, 3 (10.7%) had normal BMI, 4 (16.7%) were overweight, and 2 (40%) were obese.

Gender Distribution Across the BMI: The gender-wise comparison of BMI categories shows that among males, 30 (63.8%) were underweight, 22 (78.6%) had normal BMI, 16 (66.7%) were overweight, and 3 (60%) were obese. In contrast, among females, 17 (36.2%) were underweight, 6 (21.4%) had normal BMI, 8 (33.3%) were overweight, and 2 (40%) were obese. Although males formed the majority across most BMI categories, the distribution pattern between males and females did not show a statistically significant difference (p = 0.575).

NCV analysis

Motor Median Right Wrist Latency 1 with Respect to BMI: The comparison of Motor Median Right Wrist Latency 1 across BMI categories reveals notable variation, with the mean latency recorded as 7.41 ± 5.08 in individuals with a normal BMI, 6.42 ± 4.14 in the overweight group, 7.51 ± 4.78 in Obese Class I participants, and 5.86 ± 2.99 in Obese Class II participants. Although the values fluctuate across categories, the overall difference between BMI groups was found to be statistically significant (p = 0.034).

Table 1: Right wrist Latency 1 with respect to BMI

Variable	BMI Category				p-value
	Normal	Overweight	Obese Class I	Obese Class II	
Motor Median Right Wrist Latency 1	7.41±5.08	6.42±4.14	7.51±4.78	5.86±2.99	0.034

Motor Median Right Wrist Latency 2 with Respect to BMI: The analysis of Motor Median Right Wrist Latency 2 across different BMI categories showed comparable values among groups, with mean latencies of 22.77 ± 3.83 in the normal BMI group, 22.89 ± 3.53 among overweight participants, 22.17 ± 4.86 in Obese Class I, and a slightly higher value of 25.19 ± 1.95 in Obese Class II individuals. Although Obese Class II participants demonstrated a marginal increase in latency, the overall differences across BMI categories were not statistically significant (p = 0.492).

Motor Median Right Wrist Amplitude with Respect to BMI: The Motor Median Right Wrist Amplitude values across BMI categories showed a gradual increase from normal to higher BMI groups, with mean amplitudes of 0.44 ± 0.160 in the normal BMI group, 0.49 ± 0.451 among overweight individuals, 0.94 ± 0.821 in Obese Class I, and the highest value of 2.17 ± 0.68 in Obese Class II participants. Although a rising trend is visible, indicating higher amplitude measurements in individuals with greater BMI, the overall difference between BMI groups did not reach statistical significance (p = 0.249).

Table 2: Right Wrist Amplitude with respect to BMI

Variable	BMI Category				p-value
	Normal	Overweight	Obese Class I	Obese Class II	
Motor Median Right Wrist Amplitude	0.44±0.160	0.49±0.451	0.941±0.821	2.17±0.68	0.249

Motor Median Right Wrist NCV with Respect to BMI: The Motor Median Right Wrist NCV values showed a decreasing trend across increasing BMI categories, with mean conduction velocities of 36.66 ± 31.78 in the normal BMI group, 35.10 ± 31.08 among overweight individuals, 21.97 ± 19.86 in Obese Class I, and the lowest value of 14.73 ± 7.42 in Obese Class II participants. Although this pattern suggests that nerve conduction velocity may decline with higher BMI levels, the differences observed between the BMI groups were not statistically significant (p = 0.702).

Motor Median Right Elbow Latency 1 with Respect to BMI: The Motor Median Right Elbow Latency 1 values varied significantly across BMI categories, with mean latencies of 7.55 ± 4.36 in the normal BMI group, 3.84 ± 3.63 among overweight participants, 6.33 ± 4.74 in Obese Class I, and 3.52 ± 3.12 in Obese Class II individuals.

Although the values fluctuate, a marked reduction in latency is observed in overweight and Obese Class II groups compared to those with normal BMI.

The overall difference across BMI categories was statistically significant (p = 0.003), indicating a meaningful association between BMI and Motor Median Latency at the right elbow.

Motor Median Right Elbow Latency 2 with Respect to BMI: The comparison of Motor Median Right Elbow Latency 2 across BMI categories revealed similar mean values among groups, with 23.81 ± 3.40 in the normal BMI group, 23.39 ± 3.05 among overweight individuals, 24.22 ± 3.61 in Obese Class I, and 22.17 ± 3.32 in Obese Class II participants.

Although the numerical differences are relatively small, Obese Class I showed a slightly higher mean latency, while Obese Class II showed a lower latency compared to those with a normal BMI. Despite this modest variability, the overall difference between BMI groups reached statistical significance (p = 0.049).

Motor Median Elbow Amplitude with Respect to BMI: The Motor Median Right Elbow Amplitude showed a progressive increase across BMI categories, with mean amplitudes of 0.47 ± 0.38 in the normal BMI group, 0.52 ± 0.49 among overweight individuals, 0.95 ± 0.42 in Obese Class I, and the highest value of 2.74 ± 1.88 in Obese Class II participants.

Although this upward trend suggests a possible association between higher BMI and increased amplitude, the difference across BMI groups did not reach statistical significance (p = 0.172).

Wrist NCV parameters with Respect to BMI: The comparison of Motor Median Right Wrist nerve conduction parameters across BMI categories demonstrated variable trends among the different measures. Latency 1 showed noticeable variation, with mean values ranging from 7.41 ± 5.08 in the normal BMI group to 5.86 ± 2.99 in the Obese Class II group, and this difference was statistically significant (p = 0.034), indicating a meaningful association between BMI and initial latency.

In contrast, Latency 2 values were relatively similar across categories 22.77 ± 3.83 (normal), 22.89 ± 3.53 (overweight), 22.17 ± 4.86 (Obese Class I), and 25.19 ± 1.95 (Obese Class II) with no significant difference (p = 0.492).

Wrist duration also showed modest variation (15.35–19.32 ms) but remained statistically non-significant (p = 0.244). Motor amplitude revealed a gradual rise from 0.44 ± 0.160 in normal individuals to 2.17 ± 0.68 in Obese Class II, although this trend did not achieve statistical significance (p = 0.249).

Table 3: Wrist NCV parameters with respect to BMI

Variable	BMI Category				p-value
	Normal	Overweight	Obese Class I	Obese Class II	
Motor Median Right Wrist Latency 1	7.41±5.08	6.42±4.14	7.51±4.78	5.86±2.99	0.034
Motor Median Right Wrist Latency 2	22.77±3.83	22.89±3.53	22.17±4.86	25.19±1.95	0.492
Motor Median Right Wrist Duration	15.35±5.14	16.46±3.98	14.53±4.68	19.32±2.34	0.244
Motor Median Right Wrist Amplitude	0.44±0.160	0.49±0.451	0.941±0.821	2.17±0.68	0.249

Elbow NCV Parameters with Respect to BMI: The analysis of Motor Median Right Elbow nerve conduction parameters across BMI categories showed notable differences in some measures. Latency 1 values ranged from 7.55 ± 4.36 in the normal BMI group to 3.52 ± 3.12 in Obese Class II, and this difference was statistically significant (p = 0.003), indicating that BMI has a meaningful influence on initial elbow latency.

Latency 2 also showed mild variability, with values ranging from 23.39 ± 3.05 to 24.22 ± 3.61, and reached statistical significance (p = 0.049), suggesting a subtle BMI-related effect. In contrast, the duration parameter demonstrated slightly fluctuating values, ranging from 16.25 ± 4.48 to 19.57 ± 4.61, but these differences were not statistically significant (p = 0.114).

Similarly, motor amplitude increased progressively with BMI, peaking at 2.74 ± 1.88 in the Obese Class II group; however, this pattern did not reach significance (p = 0.172). Overall, BMI showed a significant association with both latency measures at the right elbow, while duration and amplitude remained unaffected across BMI categories.

Table 4: Elbow NCV parameters with respect to BMI

Variable	BMI Category				p-value
	Normal	Overweight	Obese Class I	Obese Class II	
Motor Median Right Elbow Latency 1	7.55±4.36	3.84±3.63	6.33±4.74	3.52±3.12	0.003
Motor Median Right Elbow Latency 2	23.81±3.40	23.39±3.05	24.22±3.61	22.17±3.32	0.049
Motor Median Right Elbow Duration	16.25±4.48	19.57±4.61	17.88±4.41	18.65±3.29	0.114
Motor Median Right Elbow Amplitude	0.47±0.38	0.52±0.49	0.95±0.42	2.74±1.88	0.172

Descriptive Statistics for NCV Parameters: The summary of nerve conduction parameters for the right wrist and right elbow demonstrates considerable variability across latency, amplitude, and conduction velocity measures. For the right wrist, Latency 1 showed a mean of 7.10 ms (median 6.73 ms) with a wide range from 0.22 to 21.19 ms, while Latency 2 had a mean of 22.78 ms and ranged between 12.11 and 28.87 ms, indicating heterogeneous conduction patterns among participants.

Wrist amplitude also varied markedly, with a mean of 0.65 mV and values extending from 0.03 to 11.95 mV, highlighting substantial inter-individual differences in muscle response.

Similarly, at the right elbow, Latency 1 demonstrated a mean of 6.08 ms (range: 0.04–18.37 ms), while Latency 2 averaged 23.71 ms with a minimum of 9.30 ms and maximum of 27 ms. Elbow amplitude showed a mean of 0.69 mV, though individual values varied considerably (0.03–12 mV).

The right elbow NCV exhibited a mean conduction velocity of 54.64 m/s, with a broad range from 22.47 to 83.92 m/s, indicating significant physiological variability in nerve conduction speed among the study population.

Table 5: Descriptive Statistics for NCV Parameters.

NCV Parameter	Mean	Median	Std. Deviation	Minimum	Maximum
Right Wrist Latency 1 (ms)	7.10	6.73	4.66	0.22	21.19
Right Wrist Latency 2 (ms)	22.78	23.79	3.95	12.11	28.87
Right Wrist Amplitude (mV)	0.65	0.06	0.98	0.03	11.95
Right Elbow Latency 1 (ms)	6.08	6.28	4.51	0.04	18.37
Right Elbow Latency 2 (ms)	23.71	24.54	3.34	9.30	27
Right Elbow Amplitude (mV)	0.69	0.060	0.037	0.03	12
Right Elbow NCV (m/s)	54.64	55.05	9.93	22.47	83.92

BMI vs NCV: The correlation between BMI and Right Elbow NCV showed a very weak positive relationship (r = 0.068) with a p-value of 0.355, indicating no meaningful linear association between BMI and conduction velocity at the elbow. Similarly, the correlation between BMI and overall NCV demonstrated a weak positive correlation (r = 0.209) with a p-value of 0.441, which was also statistically non-significant.

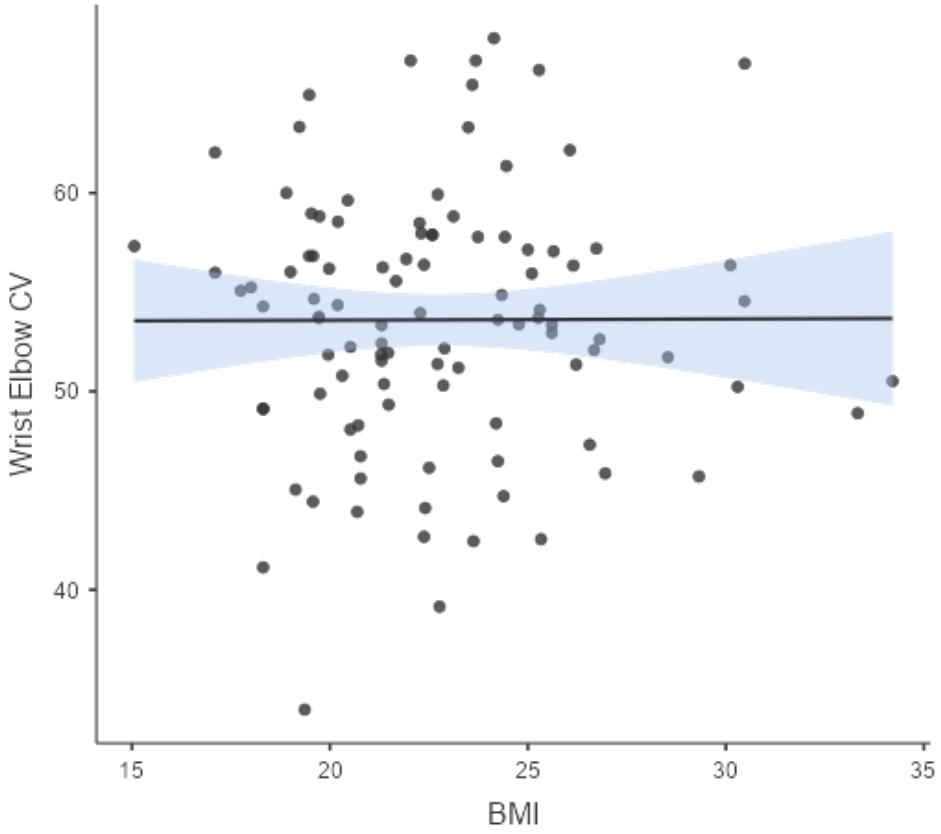

Figure 1: Correlation between BMI and Right Elbow NCV

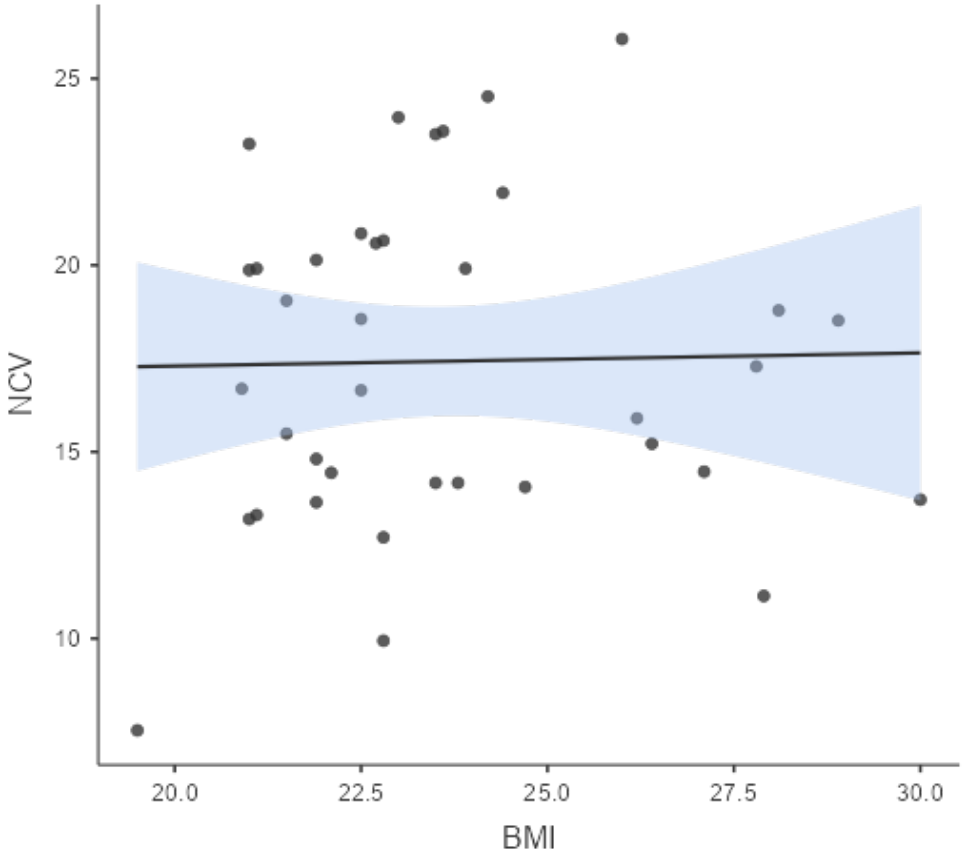

Figure 2: Correlation between BMI and overall NCV

One Way ANOVA for BMI Group with NCV: The resulting F-value of 0.189 and a high p-value of 0.904 confirm the absence of any meaningful difference among the groups at the chosen significance level. These findings suggest that the parameter under investigation did not vary significantly across the different categories being compared, and group membership did not influence the outcome in this dataset.

Tabe 6: Comparison between BMI Group with NCV

Source of Variation	Sum of Squares	Df	Mean Square	F-value	p-value
Between Groups	57.293	3	19.09	0.189	0.904
Within Groups	10099.61	100	100.99		
Total	10156.90	104			

Post Hoc Analysis (Bonferroni): The mean differences between groups, such as Normal vs. Overweight (1.79), Normal vs. Obese Class I (0.49), and Normal vs. Obese Class II (0.311), were small and accompanied by confidence intervals that included zero, indicating no meaningful difference between categories. Similarly, comparisons involving overweight and obese groups, including Overweight vs. Obese Class I (-1.29) and Overweight vs. Obese Class II (-1.47), also showed wide confidence intervals spanning both negative and positive values, further confirming the absence of significant differences. Even the comparison between Obese Class I and Obese Class II showed a negligible mean difference of 0.18, again with a confidence interval that crossed zero.

Tabe 7: Comparison between normal versus obese

Group 1	Group 2	Mean Difference	p-value	Confidence Interval (Lower)	Confidence Interval (Upper)
Normal	Overweight	1.79	0.987	-4.66	8.24
Normal	Obese Class I	0.49	0.999	-6.29	7.28
Normal	Obese Class II	0.311	0.980	-12.41	13.03
Overweight	Obese Class I	-1.29	0.977	-8.82	6.22
Overweight	Obese Class II	-1.47	0.988	-14.61	11.65
Obese Class I	Obese Class II	0.18	0.999	-13.48	13.11

Discussion

The demographic profile indicated that most participants were between 20 and 30 years old, with a mean age of 31.05 ± 7.58 years.

Age is a well-established determinant of peripheral nerve conduction, with nerve conduction velocity (NCV) known to decrease with age due to progressive axonal degeneration, myelin thinning, and a reduction in nerve fibre density [1,2].

Since the current cohort consisted predominantly of young adults, age-related deterioration was unlikely to confound the findings. Studies by Stetson et al. and Kimura similarly emphasized that median nerve conduction remains relatively stable until the late 40s, supporting the internal validity of the present dataset [3, 4].

A male predominance (68.3%) was observed, which aligns with the recruitment patterns of several neurophysiological studies, where men often participate more frequently due to occupational screening referrals, physical workload exposure, or healthcare-seeking differences [5].

Some studies also suggest subtle gender-related variations in nerve conduction, with males sometimes demonstrating slightly slower NCV due to larger limb length and nerve conduction distance [6].

Therefore, although the overall influence of gender in this study was statistically non-significant, inherent biological and anthropometric differences might still partly contribute to variability in conduction values.

The BMI distribution revealed that nearly half of the study population fell within the normal BMI range, with a sizeable proportion of overweight and obese individuals. Obesity has been extensively associated with metabolic, structural, and compressive effects on peripheral nerves.

Mechanistically, obesity induces low-grade chronic inflammation, oxidative stress, impaired perfusion of vasa nervorum, and adipokine imbalance, all of which may lead to subclinical neuropathy even in non-diabetic individuals [7–9]. Andersen et al. demonstrated significantly reduced NCV in obese adults compared to normal-weight individuals, proposing that metabolic syndrome components have additive detrimental effects on peripheral nerve function [10]. However, the present study identified only selective changes in latency, rather than consistent reductions across all NCV parameters. These findings suggest that obesity may alter early

nerve conduction responses (particularly latency), without affecting the amplitude or conduction velocity significantly in younger individuals.

When BMI was analysed across age groups, no statistically significant association was found ($p = 0.071$); however, higher BMI categories were more prevalent in the older age group (>40 years). This trend is expected, as epidemiological data consistently demonstrate an increasing BMI with age until mid-adulthood, due to a declining basal metabolic rate, reduced physical activity, and increased fat deposition [11].

Previous work from the NHANES cohort similarly reported a peak in BMI trends around the fourth decade of life [12]. Although the association was not statistically significant here, the distribution patterns remained consistent with global data.

When evaluating the effect of BMI on wrist nerve conduction parameters, the Motor Median Right Wrist Latency 1 exhibited a statistically significant association ($p = 0.034$), with obese individuals showing shorter latency values than those who were normal or overweight.

This observation diverges from classical expectations, as increased soft-tissue mass generally prolongs latency by increasing distance between stimulating and recording electrodes [13]. However, several studies, including that of Buschbacher [14], have shown that excessive adipose tissue can sometimes lead to artificially reduced latency owing to altered current dispersion or facilitation of superficial nerve stimulation.

Therefore, the findings from this study contribute to ongoing debate on the true directionality of latency changes in relation to BMI. It is plausible that subtle technical variations hand temperature, electrode spacing, and muscle thickness may influence latency measurements and must be controlled rigorously in future research.

Latency 2 and amplitude values at the wrist did not show significant differences across BMI categories, indicating preservation of distal median nerve function irrespective of BMI. This is supported by multiple studies demonstrating that upper limb nerves, being shorter and less vulnerable to length-dependent neuropathy, often remain unaffected in early metabolic or obesity-related nerve dysfunction [7,15]. More pronounced effects are typically observed in lower limb nerves such as the sural or peroneal nerves, where subclinical neuropathic changes manifest earlier [16].

At the elbow, both Latency 1 ($p = 0.003$) and Latency 2 ($p = 0.049$) showed statistically significant variation with BMI, with obese groups demonstrating reduced latency compared to individuals with normal BMI.

The elbow region is anatomically more susceptible to adiposity-related changes because excessive subcutaneous fat can alter nerve positioning, reduce stimulation distance, or modify current conduction pathways [17].

Moreover, obesity has been linked to increased risk of compression neuropathies at mechanically vulnerable anatomical tunnels, such as the cubital tunnel at the elbow [18]. Although this study did not evaluate clinical neuropathy, mild conduction changes may reflect early subclinical effects.

Despite significant latency differences, other elbow parameters including duration, amplitude, and NCV did not show statistical significance, suggesting that structural integrity of the nerve remains largely preserved.

Since amplitude reflects axonal integrity and NCV reflects myelin quality, the absence of deviations in these parameters reinforces the hypothesis that observed latency variations may be technical or positional rather than pathological [18]. The weak correlations found between BMI and NCV ($r = 0.068$ and $r = 0.209$) further support this interpretation.

The ANOVA and Bonferroni post-hoc tests confirmed that BMI groups did not differ significantly across most nerve conduction parameters, consistent with studies showing that only extreme obesity or the presence of comorbidities (diabetes, metabolic syndrome) significantly alters NCV values [8, 10, 20].

Notably, even in obese individuals in the present study, NCV values remained within accepted physiological ranges, suggesting that nerve conduction remains stable in early adulthood, despite variations in BMI. This aligns with the findings of Callaghan et al., who noted that obesity-related neuropathy develops insidiously and typically becomes measurable only when compounded by metabolic diseases [20].

The descriptive statistics show wide variability in latency, amplitude, and NCV values, which is typical in neurophysiology datasets. Several factors are known to influence peripheral nerve conduction: limb length, skin temperature, ambient conditions, hydration, muscle bulk, and subcutaneous fat [4].

For instance, a mere 1°C drop in hand temperature can reduce NCV by 2–3 m/s, underscoring the need for standardized temperature control [4]. Given these influences, the observed variability in the present study remains within normal physiological boundaries and does not diminish the reliability of the results.

The combined findings suggest that although BMI has minor effects on certain latency parameters especially at the elbow it does not exert broad,

clinically meaningful effects on nerve conduction in young adults. This supports the conclusion that isolated BMI elevation, in the absence of metabolic disease or long-standing obesity, may not significantly compromise peripheral nerve electrophysiology.

Study Limitation

Important obesity-related variables, such as blood glucose, lipid profile, HbA1c, thyroid profile, or markers of inflammation, were not assessed, which limits our understanding of the underlying metabolic influences.

Longitudinal follow-up is necessary to determine whether BMI contributes to progressive changes in nerve conduction over time.

Although standardized, nerve conduction studies are sensitive to limb temperature, adipose thickness, and electrode-to-nerve distance all of which may introduce minor measurement variations.

A single-centre study limits the generalizability of the findings.

Conclusion

The present study concluded that the BMI has a selective and limited influence on nerve conduction. Significant differences were observed in Latency 1 at the wrist and in both Latency 1 and Latency 2 at the elbow, suggesting that BMI may affect early latency components, possibly due to variations in soft-tissue thickness, altered stimulation distance, or adiposity-related physiological changes. This study also suggests that the obesity-related neuropathic changes may require additional metabolic risk factors (e.g., diabetes, dyslipidaemia, insulin resistance) or longer disease duration to become clinically significant. Hence, the findings contribute to existing literature by demonstrating that isolated elevations in BMI do not cause measurable deterioration in median nerve conduction in early adulthood. Hence, Increased BMI is significantly associated with impaired nerve conduction, emphasizing the need for weight management in healthcare workers to ensure optimal neuromuscular performance and prevent early neuropathic changes.

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