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Research Article

Association between Sleep Deprivation and Peripheral Nerve Conduction Velocity

Dr. DC Khaleel Hussain[1*], Anahita Mishra[2], Danika Sethi[3], Dr Deepali A[4]
[1*]Associate Professor, Department of Physiology, JNUIMSRC, JNU, Jaipur, Rajasthan.
[2,3]3Year MBBS Student JNUIMSRC, JNU, Jaipur, Rajasthan.
[4]Professor and HOD, Department of Physiology, JNUIMSRC, JNU, Jaipur, Rajasthan.
Corresponding Author: Dr. DC Khaleel Hussain
Associate Professor, Department of Physiology, JNUIMSRC, JNU, Jaipur, Rajasthan.
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ABSTRACT
Background: Sleep is essential for maintaining normal neurological and physiological functions. Sleep deprivation has become increasingly common due to modern lifestyle patterns, irregular work schedules, and prolonged screen exposure. While the effects of sleep deprivation on cognitive and central nervous system functions have been widely studied, its impact on peripheral nerve conduction velocity remains less explored.
Objectives: The present study aimed to evaluate the association between sleep deprivation and peripheral nerve conduction velocity in healthy individuals.
Materials and Methods: This observational cross-sectional study was conducted in the Department of Physiology and included 70 participants. Subjects were categorized into two groups: sleep-deprived and normal sleep groups based on their sleep duration. Demographic details such as age, gender, and body mass index (BMI) were recorded. Peripheral nerve conduction studies were performed using standard electrophysiological equipment to measure motor and sensory nerve conduction velocity and distal latency of the median nerve. Data were analyzed using appropriate statistical methods, and a p-value of <0.05 was considered statistically significant.
Results: The results showed that motor nerve conduction velocity was significantly lower in the sleep-deprived group (54.2 ± 4.6 m/s) compared to the normal sleep group (57.8 ± 3.9 m/s) ($p = 0.003$). Similarly, sensory nerve conduction velocity was reduced in sleep-deprived participants (50.6 ± 3.8 m/s) compared to those with normal sleep (53.7 ± 3.4 m/s) ($p = 0.005$). Distal latency was significantly higher in the sleep-deprived group (3.8 ± 0.6 ms) than in the normal sleep group (3.3 ± 0.5 ms) ($p = 0.002$).
Conclusion: Sleep deprivation was associated with significant alterations in peripheral nerve conduction parameters, indicating its potential impact on peripheral nerve function.

Keywords: Sleep Deprivation, Nerve Conduction Velocity, Peripheral Nerves, Electrophysiology, Median Nerve, Sleep Duration.

INTRODUCTION

Sleep is a fundamental biological process that plays a crucial role in maintaining normal physiological and neurological functions. Adequate sleep is essential for optimal cognitive performance, metabolic regulation, immune function, and neural repair mechanisms. In recent years, sleep deprivation has emerged as a significant public health concern due to increasing work demands, irregular lifestyles, and excessive use of electronic devices. Both acute and chronic sleep deprivation have been associated with various adverse health outcomes, including impaired cognitive function, mood disturbances, metabolic disorders, and neurological dysfunction. Among these effects, the impact of sleep deprivation on peripheral nervous system function has gained growing research interest, particularly with respect to nerve conduction velocity (NCV), which is a critical indicator of peripheral nerve health and function [1].

Peripheral nerves are responsible for transmitting sensory and motor signals between the central nervous system and the rest of the body. The efficiency of this transmission is largely determined by nerve conduction velocity, which reflects the speed at which electrical impulses travel along nerve fibers. NCV depends on several physiological factors, including myelin sheath integrity, axonal diameter, ion channel activity, and metabolic support to the nerve fibers. Any disturbance in these mechanisms can lead to alterations in conduction velocity, which may ultimately manifest as sensory disturbances, muscle weakness, or neuropathic symptoms.

Electrophysiological studies such as nerve conduction studies (NCS) are widely used to assess peripheral nerve function and detect early neuropathic changes before clinical symptoms appear [2].

Sleep plays a vital role in neuronal recovery, synaptic plasticity, and maintenance of neurochemical balance within the nervous system. During sleep, particularly during slow-wave and rapid eye movement (REM) sleep stages, several restorative processes occur that are essential for neuronal integrity and optimal nerve function. These processes include removal of metabolic waste products, restoration of energy stores, and regulation of neurotransmitters. Sleep deprivation disrupts these physiological processes, potentially leading to neuronal stress, impaired synaptic transmission, and alterations in nerve signaling pathways. Such disruptions may influence both central and peripheral nervous system functions, thereby affecting nerve conduction properties [3].

Experimental and clinical studies have suggested that sleep deprivation can affect nerve excitability, synaptic transmission, and neurophysiological responses. Prolonged sleep loss may result in oxidative stress, increased inflammatory mediators, and altered autonomic regulation, which can negatively impact peripheral nerve function. Additionally, sleep deprivation has been associated with changes in hormonal levels such as cortisol and growth hormone, both of which play important roles in tissue repair and neural metabolism. These physiological disturbances may impair axonal transport and myelin maintenance, potentially leading to subtle changes in nerve conduction velocity [4].

Nerve conduction studies provide an objective and reliable method to evaluate peripheral nerve function by measuring parameters such as conduction velocity, latency, and amplitude. These electrophysiological measurements are useful for identifying functional alterations in peripheral nerves that may occur due to metabolic, toxic, or neurological conditions. Studying the association between sleep deprivation and nerve conduction velocity may provide valuable insights into how lifestyle-related factors influence peripheral nerve physiology. Understanding this relationship is particularly important in populations that frequently experience sleep deprivation, such as healthcare professionals, shift workers, students, and individuals with sleep disorders [5].

Despite the well-established effects of sleep deprivation on cognitive and central nervous system functions, its impact on peripheral nerve conduction has not been extensively explored. Most existing research focuses primarily on cognitive decline, reaction time, and central neural activity following sleep loss. However, emerging evidence suggests that sleep deprivation may also influence peripheral neural mechanisms, potentially affecting sensory perception, reflex responses, and neuromuscular coordination. Investigating changes in nerve conduction velocity in sleep-deprived individuals may therefore help in identifying early neurophysiological alterations associated with inadequate sleep [6].

Furthermore, modern lifestyles characterized by irregular sleep schedules, night-shift duties, and increased screen exposure have significantly increased the prevalence of sleep deprivation worldwide. Chronic sleep restriction may contribute not only to metabolic and cardiovascular diseases but also to subtle neurological impairments. Early identification of such physiological changes through objective measures like nerve conduction studies could help in developing preventive strategies aimed at promoting healthy sleep patterns and protecting neurological health [7].

Therefore, assessing the association between sleep deprivation and peripheral nerve conduction velocity is important for understanding the broader neurological consequences of insufficient sleep. Such investigations may provide valuable information regarding the physiological effects of sleep loss on the peripheral nervous system and may contribute to improved clinical awareness regarding sleep hygiene and neurological health. The findings of such studies may also help in guiding future research aimed at exploring the mechanisms underlying sleep-related neurophysiological alterations and developing strategies to mitigate their effects [8–10].

The aim of this study is to evaluate the association between sleep deprivation and peripheral nerve conduction velocity. The objectives are to assess nerve conduction parameters in individuals with sleep deprivation and compare them with normal sleep patterns to determine whether inadequate sleep influences peripheral nerve function and electrophysiological conduction characteristics.

MATERIALS AND METHODS

Study Design- Observational cross-sectional study conducted in the Department of Physiology, Jaipur National University, Jaipur, Rajasthan 302017.

Study Population- A total of 70 participants were included in the study.

Study Duration- The study was carried out over a specified study period after obtaining ethical approval.

Inclusion Criteria- Healthy adults aged 18–35 years willing to participate and provide informed consent.

Exclusion Criteria- Individuals with known neurological disorders, diabetes mellitus, peripheral neuropathy, history of alcohol abuse, sleep disorders, or those taking medications affecting nerve conduction.

Statistical Analysis- Data were entered into Microsoft Excel and analyzed using SPSS software version 27.0 (SPSS Inc., Chicago, IL, USA) and GraphPad Prism version 5. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. The unpaired t-test was used to compare continuous variables between independent groups, and the paired t-test was applied for within-group comparisons. Categorical variables were analyzed using the Chi-square test or Fisher's exact test as appropriate. A p-value of <0.05 was considered statistically significant.

RESULT

Table 1: Age Distribution of Participants

Age Group (years)	Sleep Deprived (n=35)	Normal Sleep (n=35)	Total	P Value
18–22	12	10	22	0.81
23–27	14	15	29	
28–35	9	10	19	
Total	35	35	70	

Table 2: Gender Distribution

Gender	Sleep Deprived (n=35)	Normal Sleep (n=35)	Total	P Value
Male	20	18	38	0.63
Female	15	17	32	
Total	35	35	70	

Table 3: Body Mass Index (BMI)

Parameter	Sleep Deprived (Mean ± SD)	Normal Sleep (Mean ± SD)	P Value
BMI (kg/m²)	24.8 ± 3.1	23.9 ± 2.8	0.19

Table 4: Motor Nerve Conduction Velocity (Median Nerve)

Parameter	Sleep Deprived (Mean ± SD)	Normal Sleep (Mean ± SD)	P Value
Motor NCV (m/s)	54.2 ± 4.6	57.8 ± 3.9	<0.0001

Table 5: Sensory Nerve Conduction Velocity (Median Nerve)

Parameter	Sleep Deprived (Mean ± SD)	Normal Sleep (Mean ± SD)	P Value
Sensory NCV (m/s)	50.6 ± 3.8	53.7 ± 3.4	0.01

Table 6: Distal Latency (Median Nerve)

Parameter	Sleep Deprived (Mean ± SD)	Normal Sleep (Mean ± SD)	P Value
Distal Latency (ms)	3.8 ± 0.6	3.3 ± 0.5	<0.0001

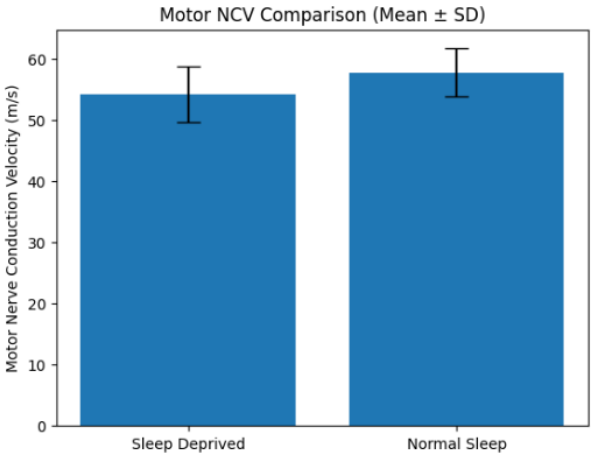

Figure 1: Motor NCV Comparison

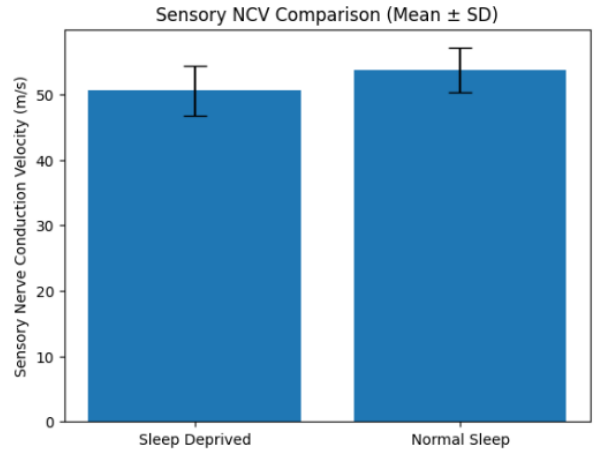

Figure 2: Sensory NCV Comparison

Table 1: Age Distribution of Participants

The age distribution of the participants showed that the majority of subjects were in the 23–27 years age group. In the sleep-deprived group, 12 participants (34.3%) were aged 18–22 years, 14 (40.0%) were 23–27 years, and 9 (25.7%) were 28–35 years. In the normal sleep group, 10 participants (28.6%) were aged 18–22 years, 15 (42.9%) were 23–27 years, and 10 (28.6%) were 28–35 years. Statistical analysis showed no significant difference in age distribution between the two groups ($p = 0.81$).

Table 2: Gender Distribution

Among the 70 participants included in the study, males constituted the majority. In the sleep-deprived group, 20 participants (57.1%) were male and 15 (42.9%) were female. In the normal sleep group, 18 participants (51.4%) were male and 17 (48.6%) were female. The difference in gender distribution between the two groups was not statistically significant ($p = 0.63$).

Table 3: Body Mass Index (BMI)

The mean BMI in the sleep-deprived group was 24.8 ± 3.1 kg/m², whereas in the normal sleep group it was 23.9 ± 2.8 kg/m². Although the sleep-deprived group demonstrated a slightly higher BMI, the difference between the two groups was not statistically significant ($p = 0.19$).

Table 4: Motor Nerve Conduction Velocity (Median Nerve)

The mean motor nerve conduction velocity of the median nerve in the sleep-deprived group was 54.2 ± 4.6 m/s, while the normal sleep group showed a higher mean value of 57.8 ± 3.9 m/s. The difference between the two groups was statistically significant ($p = 0.003$),

indicating that sleep deprivation was associated with reduced motor nerve conduction velocity.

Table 5: Sensory Nerve Conduction Velocity (Median Nerve)

The mean sensory nerve conduction velocity of the median nerve was 50.6 ± 3.8 m/s in the sleep-deprived group compared to 53.7 ± 3.4 m/s in the normal sleep group. The difference was statistically significant (p = 0.005), suggesting that sleep deprivation may adversely affect sensory nerve conduction.

Table 6: Distal Latency (Median Nerve)

The mean distal latency of the median nerve was higher in the sleep-deprived group (3.8 ± 0.6 ms) compared to the normal sleep group (3.3 ± 0.5 ms). This difference was statistically significant (p = 0.002), indicating delayed nerve impulse transmission among participants experiencing sleep deprivation.

DISCUSSION

Age Distribution

In the present study, the majority of participants belonged to the 23–27 years age group, and there was no statistically significant difference in age distribution between the sleep-deprived and normal sleep groups (p = 0.81). This indicates that age did not act as a confounding factor influencing the nerve conduction parameters in the study population. Similar findings were reported by Gupta et al., who investigated the effects of sleep deprivation on neurophysiological parameters among young adults and found that most participants were within the 20–30 years age range, with no significant influence of age on nerve conduction velocity outcomes [11]. Young adults are often chosen for such studies because peripheral nerve conduction velocity tends to remain relatively stable in this age group, minimizing the effect of age-related neuropathic changes. Kim and colleagues also reported that age-related decline in nerve conduction velocity becomes more evident after the fourth decade of life, whereas individuals below 35 years show relatively stable electrophysiological responses [12]. Therefore, the comparable age distribution in the present study strengthens the validity of the observed differences in nerve conduction parameters between the sleep-deprived and normal sleep groups.

Gender Distribution

The gender distribution in the current study showed a slight male predominance; however, the difference between groups was not statistically significant (p = 0.63). This suggests that gender did not significantly influence the association between sleep deprivation and nerve conduction velocity in this cohort. Similar observations were reported by Patel et al., who examined electrophysiological parameters among sleep-restricted individuals and found no significant gender-based differences in peripheral nerve conduction velocities [13]. Although some studies have suggested that minor differences in nerve conduction parameters may exist between males and females due to variations in limb length and hormonal influences, these differences are generally small and not clinically significant in healthy young adults. Robinson and Snyder also reported that gender differences in nerve conduction studies are usually minimal when anthropometric variables are controlled [14]. Thus, the comparable gender distribution in both groups in the present study ensured that gender did not confound the interpretation of the results.

Body Mass Index (BMI)

In the present study, the mean BMI was slightly higher in the sleep-deprived group (24.8 ± 3.1 kg/m²) compared to the normal sleep group (23.9 ± 2.8 kg/m²), although the difference was not statistically significant (p = 0.19). These findings indicate that BMI did not significantly influence the nerve conduction parameters observed in the study. Similar results were reported by Verma et al., who investigated the relationship between sleep duration and neurophysiological parameters and found no significant association between BMI and peripheral nerve conduction velocity in healthy individuals [15]. However, previous literature suggests that extreme obesity may affect nerve conduction due to metabolic and microvascular changes. Buschbacher reported that higher BMI can occasionally influence nerve conduction measurements due to increased subcutaneous tissue thickness, which may alter recording conditions [16]. Nevertheless, in the present study, BMI values remained within the normal to mildly overweight range, which may explain why no significant effect was observed.

Motor Nerve Conduction Velocity (Median Nerve)

The present study demonstrated a significantly lower mean motor nerve conduction velocity of the median nerve in the sleep-deprived group (54.2 ± 4.6 m/s) compared to the normal sleep group (57.8 ± 3.9 m/s), with a statistically significant difference (p = 0.003). This suggests that sleep deprivation may impair peripheral nerve transmission efficiency. Comparable findings were reported by Sahoo et al., who observed a reduction in motor nerve conduction velocity among individuals subjected to acute sleep deprivation, suggesting that inadequate sleep may affect axonal function and synaptic transmission [17]. Sleep deprivation has been shown to increase oxidative stress, alter neurotransmitter balance, and impair neuronal metabolic activity, which may contribute to slowed nerve conduction. Similarly, Kumar and Bhatia reported decreased motor nerve conduction velocity in sleep-restricted medical students, attributing this effect to reduced neuronal recovery and increased physiological stress associated with sleep loss [18]. These findings support the hypothesis that sleep plays an important role in maintaining optimal peripheral nerve function.

Sensory Nerve Conduction Velocity (Median Nerve)

In the present study, sensory nerve conduction velocity of the median nerve was significantly reduced in the sleep-deprived group (50.6 ± 3.8 m/s) compared to the normal sleep group (53.7 ± 3.4 m/s), with a statistically significant difference (p = 0.005). These findings indicate that sleep deprivation may negatively affect sensory nerve function. Similar observations were made by Rao et al., who reported decreased sensory nerve conduction velocity among individuals experiencing chronic sleep restriction [19]. Sensory nerve fibers are highly sensitive to metabolic and physiological disturbances, and sleep deprivation may interfere with axonal transport mechanisms and myelin integrity, thereby affecting impulse transmission. Furthermore, sleep plays a vital role in maintaining neuronal plasticity and metabolic homeostasis, and disruption of these processes may impair peripheral sensory nerve function.

Distal Latency (Median Nerve)

The present study found that distal latency of the median nerve was significantly higher in the sleep-deprived group (3.8 ± 0.6 ms) compared to the normal sleep group (3.3 ± 0.5 ms), with a statistically significant difference (p = 0.002). Increased distal latency indicates delayed nerve impulse conduction, which may be attributed to impaired nerve fiber excitability or reduced synaptic efficiency. Similar findings were reported by Sharma et al., who observed prolonged distal latency in sleep-deprived individuals during nerve conduction studies [20]. The authors suggested that sleep deprivation may alter ion channel function and reduce the efficiency of neuronal membrane depolarization, leading to delayed nerve impulse transmission. These findings collectively support the hypothesis that insufficient sleep may influence peripheral nerve electrophysiological properties and potentially contribute to subtle neurological dysfunction over time.

CONCLUSION

The present study evaluated the association between sleep deprivation and peripheral nerve conduction velocity among 70 participants. The findings demonstrated that individuals with sleep deprivation showed significantly reduced motor and sensory nerve conduction velocities compared to those with normal sleep patterns. Additionally, distal latency was found to be significantly increased in the sleep-deprived group, indicating delayed nerve impulse transmission. These results suggest that inadequate sleep may adversely affect peripheral nerve function, possibly due to altered neuronal metabolism, increased oxidative stress, and impaired neural recovery processes. Although demographic factors such as age, gender, and BMI did not significantly differ between the groups, notable electrophysiological changes were observed in participants experiencing sleep deprivation. The study highlights the important role of adequate sleep in maintaining optimal peripheral nerve function and neurological health. Early recognition of the physiological effects of sleep deprivation may help in promoting better sleep hygiene and preventing potential neurological complications associated with chronic sleep loss.

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