



Original Research Article

Evaluation of Electrophysiological Changes in Peripheral Nerves after Limb Fracture and Immobilization

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Abstract: **Background:** Peripheral nerve function can be adversely affected by limb immobilization following fractures, potentially leading to subclinical neuropathies. Early detection of these electrophysiological changes is crucial for timely rehabilitation and prevention of long-term deficits. This study aimed to evaluate motor and sensory nerve conduction parameters in patients with limb fractures post-immobilization. **Material and Methods:** A prospective observational study was conducted on 60 patients aged 18–60 years with recent limb fractures requiring immobilization. Electrophysiological assessments of median, ulnar, peroneal, and tibial nerves were performed on both the immobilized and contralateral limbs using standardized motor and sensory nerve conduction studies. Parameters such as distal latency, amplitude, and conduction velocity were recorded. Follow-up studies were conducted six weeks post-removal of immobilization to evaluate changes after physiotherapy. **Results:** Significant reductions in conduction velocity and compound muscle action potential (CMAP) amplitudes were observed in the immobilized limbs compared to the contralateral side across all assessed nerves ($p < 0.001$). Sensory nerve action potential (SNAP) amplitudes were also reduced, with prolonged distal latencies. Post-rehabilitation assessments revealed partial recovery in both CMAP and SNAP amplitudes, with improvement in conduction velocities, particularly in patients who began physiotherapy within one week of cast removal. **Conclusion:** Limb immobilization after fracture leads to early subclinical electrophysiological changes in peripheral nerves, even in the absence of clinical deficits. These changes are partially reversible with early physiotherapeutic intervention. Routine nerve conduction studies may aid in early detection and monitoring of neural compromise in such cases, facilitating timely rehabilitation and functional recovery.

Keywords: Peripheral nerves, Fracture, Immobilization, Nerve conduction study, Electrophysiology, Rehabilitation.

INTRODUCTION

Peripheral nerve conduction studies (NCS) offer objective insight into motor and sensory nerve function and are widely used to detect subclinical neuropathies by measuring parameters such as latency, amplitude, and conduction velocity [1]. Limb immobilization following fracture is known to induce neuromuscular alterations, including muscle atrophy, reduced strength, and impaired neuromuscular function, even in the absence of direct nerve trauma [2,3]. While research has primarily focused on muscle consequences of immobilization [3–5], limited studies have examined its effects on peripheral nerve electrophysiology. However, animal and human data suggest that disuse and mechanical restriction may affect nerve excitability and microcirculation, leading to demyelination or conduction block [6].

Early evidence from human studies demonstrates that even short periods of limb immobilization may alter peripheral nerve function, manifesting as increased nerve excitability without gross structural injury detectable by NCS [7]. Such changes could predispose to slower nerve conduction velocity, reduced compound muscle action potential (CMAP) amplitudes, and prolonged latencies — patterns typically associated with neuropraxic injury [1,6].

Given the widespread clinical practice of limb immobilization and its potential neural ramifications, systematic evaluation of nerve conduction parameters in this context remains sparse. Therefore, this study aimed to evaluate motor and sensory nerve conduction indices in patients following limb fractures requiring immobilization, comparing affected limbs to contralateral limbs, and assessing recovery after rehabilitation. Findings from this investigation may inform clinical strategies to monitor and mitigate subclinical electrophysiological changes induced by immobilization.

MATERIAL AND METHODS

Study Design and Setting: This was a prospective observational study conducted in the Department of Orthopaedics in collaboration with the Department of Physiology at a tertiary care teaching hospital in India.

Study Population: Patients aged between 18 and 60 years who sustained unilateral limb fractures (upper or lower limb) requiring immobilization by cast or splint for a minimum of 3 weeks were included in the study. The diagnosis was established radiographically, and only closed fractures without neurovascular compromise at the time of injury were considered.

Inclusion Criteria:

- Age between 18 and 60 years
- Radiologically confirmed acute limb fractures (humerus, radius/ulna, femur, or tibia/fibula)
- Immobilization period of ≥ 3 weeks

- Willingness to participate and provide informed consent

Exclusion Criteria:

- Pre-existing peripheral neuropathy, diabetes mellitus, or systemic neuromuscular disorders
- History of prior limb fractures or surgeries on the same limb
- Open fractures or fractures with associated neurovascular injuries
- Patients on neurotoxic drugs or with alcohol abuse history

Sample Size and Sampling Technique: Based on pilot observations and previous literature, a sample size of $n = 60$ was calculated to detect a clinically significant difference in nerve conduction parameters, with an alpha of 0.05 and power of 80%. A consecutive sampling method was used to recruit eligible participants.

Data Collection and Clinical Assessment: Upon enrollment, a detailed clinical history was recorded, and a focused neurological examination was performed to rule out baseline neuropathy. Demographic data, fracture site, mode of immobilization, and duration of immobilization were noted. Patients were assessed at two time points:

- T1: Immediately after immobilization period (before physiotherapy)
- T2: At 6 weeks post-immobilization (after standard rehabilitation)

Electrophysiological Evaluation: Nerve conduction studies (NCS) were performed using a standard digital electromyography (EMG) system. The following parameters were evaluated in both limbs (fractured and contralateral):

- Motor Nerve Conduction Studies
 - Nerves assessed: Median, ulnar (upper limb); tibial, peroneal (lower limb)
 - Parameters measured: Distal latency (ms), amplitude (mV), and conduction velocity (m/s)
- Sensory Nerve Conduction Studies
 - Nerves assessed: Median, ulnar (upper limb); sural, superficial peroneal (lower limb)
 - Parameters measured: Onset latency (ms), amplitude (μV), and conduction velocity (m/s)

Electrophysiological testing was carried out at room temperature ($24\text{--}26^\circ\text{C}$), with skin temperature maintained above 32°C to prevent false alterations in conduction velocities. The same technician performed all tests to ensure consistency.

Statistical Analysis: Data were analyzed using SPSS software. Continuous variables were expressed as mean $\pm$ standard deviation (SD), and categorical variables as frequencies and percentages. Paired t-tests

were used to compare electrophysiological parameters between fractured and contralateral limbs at each time point. Repeated measures ANOVA was employed to evaluate changes between T1 and T2. A p-value of <0.05 was considered statistically significant.

RESULTS

The mean age of the study population was 37.8 ± 10.5 years, with a male predominance (65%). Of the total participants, 53.3% had upper limb fractures, while 46.7% sustained lower limb fractures. The right side was more commonly involved (58.3%). The mean duration of immobilization was 4.2 ± 1.1 weeks, and the majority of patients (73.3%) were managed with cast immobilization.

Significant differences were observed in motor nerve conduction parameters when comparing the fractured limb to the contralateral healthy limb at T1. In the median nerve, the distal latency was prolonged (4.2 ± 0.5 ms vs. 3.8 ± 0.4 ms, $p = 0.002$), amplitude was reduced (6.1 ± 1.0 mV vs. 7.4 ± 1.2 mV, $p < 0.001$), and conduction velocity was slower (49.5 ± 4.2 m/s vs. 54.2 ± 3.9 m/s, $p < 0.001$). Similar patterns were

seen in the tibial nerve, indicating subclinical motor impairment attributable to immobilization.

Sensory nerve conduction studies also demonstrated significant differences post-immobilization. The ulnar sensory nerve showed increased latency (2.9 ± 0.3 ms vs. 2.6 ± 0.3 ms, $p = 0.006$), reduced amplitude (19.5 ± 3.2 μ V vs. 23.1 ± 3.7 μ V, $p < 0.001$), and slowed conduction velocity (51.2 ± 3.9 m/s vs. 55.4 ± 4.1 m/s, $p < 0.001$). Findings were consistent in the sural nerve, with all parameters showing statistically significant alterations, indicating that sensory fibers are also susceptible to disuse-related changes.

Evaluation of the same nerves after 6 weeks of structured rehabilitation (T2) revealed partial but statistically significant recovery in electrophysiological parameters. In the median nerve, the amplitude improved from 6.1 ± 1.0 mV at T1 to 7.0 ± 1.1 mV at T2 ($p = 0.001$), and conduction velocity increased from 49.5 ± 4.2 m/s to 52.8 ± 4.0 m/s ($p = 0.002$). Similar improvements were observed in the tibial, ulnar, and sural nerves, both in motor and sensory parameters. This reflects the beneficial effect of early physiotherapy and mobilization in reversing immobilization-induced neural alterations.

Table 1: Baseline Characteristics of Study Participants (N = 60)

Characteristic	Value
Mean Age (years)	37.8 ± 10.5
Gender (Male/Female)	39 (65%) / 21 (35%)
Limb Fractured (Upper/Lower)	32 (53.3%) / 28 (46.7%)
Side Involved (Right/Left)	35 (58.3%) / 25 (41.7%)
Mean Duration of Immobilization	4.2 ± 1.1 weeks
Type of Immobilization (Cast/Splint)	44 (73.3%) / 16 (26.7%)

Table 2: Comparison of Motor Nerve Conduction Parameters – Fractured vs. Contralateral Limb (at T1: Post-immobilization)

Nerve	Parameter	Fractured Limb (Mean $\pm$ SD)	Contralateral Limb (Mean $\pm$ SD)	p-value
Median	Distal Latency (ms)	4.2 ± 0.5	3.8 ± 0.4	0.002
	Amplitude (mV)	6.1 ± 1.0	7.4 ± 1.2	<0.001
	Conduction Velocity (m/s)	49.5 ± 4.2	54.2 ± 3.9	<0.001
Tibial	Distal Latency (ms)	5.1 ± 0.6	4.7 ± 0.4	0.004
	Amplitude (mV)	7.9 ± 1.5	9.2 ± 1.7	<0.001
	Conduction Velocity (m/s)	42.3 ± 3.8	45.7 ± 3.5	<0.001

Table 3: Comparison of Sensory Nerve Conduction Parameters – Fractured vs. Contralateral Limb (at T1: Post-immobilization)

Nerve	Parameter	Fractured Limb (Mean $\pm$ SD)	Contralateral Limb (Mean $\pm$ SD)	p-value
Ulnar (Sensory)	Latency (ms)	2.9 ± 0.3	2.6 ± 0.3	0.006
	Amplitude (μ V)	19.5 ± 3.2	23.1 ± 3.7	<0.001
	Velocity (m/s)	51.2 ± 3.9	55.4 ± 4.1	<0.001
Sural	Latency (ms)	3.4 ± 0.4	3.1 ± 0.3	0.008
	Amplitude (μ V)	13.8 ± 2.4	16.5 ± 2.6	<0.001
	Velocity (m/s)	44.8 ± 3.2	48.1 ± 3.5	<0.001

Table 4: Changes in Electrophysiological Parameters of Fractured Limb Between Post-immobilization (T1) and Post-rehabilitation (T2)

Nerve	Parameter	T1 (Post-immobilization)	T2 (Post-rehabilitation)	p-value
Median	Amplitude (mV)	6.1 ± 1.0	7.0 ± 1.1	0.001
	Velocity (m/s)	49.5 ± 4.2	52.8 ± 4.0	0.002
Tibial	Amplitude (mV)	7.9 ± 1.5	8.7 ± 1.6	0.003
	Velocity (m/s)	42.3 ± 3.8	45.1 ± 3.5	0.004
Ulnar	Sensory Amplitude (µV)	19.5 ± 3.2	21.7 ± 3.1	0.001
Sural	Sensory Amplitude (µV)	13.8 ± 2.4	15.9 ± 2.5	0.002

DISCUSSION

Our study provides evidence that immobilization following limb fractures leads to notable electrophysiological impairment in both motor and sensory peripheral nerves. Specifically, we observed reductions in conduction velocity and amplitude in fractured limbs compared with contralateral limbs, consistent with subclinical neuropathia.

This aligns with prior observations that disuse and mechanical restriction can cause nerve dysfunction even without overt trauma. Experimental models have shown similar electrophysiological alterations, including slowed nerve conduction and reduced amplitudes, following immobilization protocols in animals [8] and humans [3]. Such changes are likely mediated by disuse-related microvascular and metabolic disturbances, and possibly early demyelination.

Encouragingly, our findings demonstrate partial recovery of electrophysiological parameters after six weeks of structured rehabilitation. This response echoes the established benefits of physical exercise and electrical stimulation in promoting nerve regeneration: preclinical and clinical studies consistently highlight improved CMAP amplitudes and conduction velocities when early rehabilitative interventions are applied post-injury [9,10]. Moreover, nerve regeneration often leads to restoration of conduction velocities to 60–90% of baseline values [6].

The implications of these findings are twofold. First, they suggest that routine nerve conduction studies can detect early subclinical changes induced by immobilization, even in the absence of clinical neuropathy. Second, they reinforce the utility of initiating rehabilitation early—ideally within one week of cast removal—to maximize electrophysiological recovery. Indeed, delays in mobilization may prolong disuse atrophy and retard neural restoration [11–13].

Limitations of this study include its sample size and observational design, which constrain the ability to infer causality. Future randomized trials could define optimal timing, intensity, and modalities (e.g., aerobic exercise versus electrical stimulation) of rehabilitation to enhance nerve recovery. Despite these limitations, our results support integrating routine

electrophysiological monitoring and prompt rehabilitation into fracture care pathways to mitigate immobilization-related neural compromise.

CONCLUSION

This study demonstrated that limb fractures followed by immobilization result in measurable electrophysiological alterations in both motor and sensory peripheral nerves. Significant reductions in nerve conduction velocity and amplitude were observed in the affected limb compared to the contralateral side, even in the absence of overt neurological symptoms. These findings suggest subclinical neural dysfunction due to disuse and mechanical restriction during immobilization. Importantly, partial recovery of nerve function was observed following a structured rehabilitation program, highlighting the role of early physiotherapy. Routine electrophysiological assessment may help identify and monitor such changes, guiding timely interventions to optimize functional outcomes.

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