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Electrodiagnostic Findings in Upper Limb Traumatic Nerve Injuries: A Pattern-Based Analysis at Ghazi Al-Hariri Hospital

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Abstract:

Peripheral nerve injury is a serious and significantly disabling disorder that affects the daily life of patients as well as their quality of life. Reaching an accurate injury classification helps to find the optimal treatment, which in turn leads to a reduction of disabilities and, in many cases, regaining functionality. This study aims to characterize the electrodiagnostic findings in patients with traumatic peripheral nerve injuries of the upper limbs presenting to Ghazi Al-Hariri Hospital in Baghdad. A total of 50 adult patients, with (40) men and (10) women; mean age (39.8± 11 years), with a total of (55) peripheral nerve injuries (duration of 3 months to 20 years). NCS and EMG were done for all patients. Axonotmesis or partial nerve injury was diagnosed in 45 (81.8%) nerves, neurotmesis or complete nerve injury in 2 (3.6%), and intact nerves were found in 8 (14.5%). Electrodiagnostic studies showed important informative details regarding traumatic peripheral nerve injuries. A pattern-based analysis of CMAPs, SNAPs and EMG findings can reliably differentiate between injury groups and thus lead to the optimum choice of management. To our knowledge, this is the first study to introduce a pattern-based EDX approach to traumatic upper limb nerve injuries in an Iraqi population, highlighting its importance for both clinical decision and regional epidemiological data.

Keywords*: Peripheral nerve injury, upper limb trauma, Electrodiagnostic study, military.*

1. Introduction:

Peripheral nervous system (PNS) trauma, though relatively uncommon, represents an important clinical issue because it leads to severe sensorimotor dysfunction and chronic neurogenic pain. These injuries can significantly affect a patient's quality of life and functional capacity. They account for 2–3% of all cases presenting to trauma centers worldwide [1, 2]. Traumatic peripheral nerve injury (TPNI) that results from blunt trauma is more frequently observed of injury in the civilian population, whereas penetrating trauma is more commonly observed during wartime settings. Different mechanisms are responsible for PNI, including the stretch mechanism, which is the most common, followed by laceration and compression mechanisms, respectively [3,4]. Peripheral nerve fibers, being highly delicate, are more prone to injury. Upper extremities are particularly more susceptible to such traumatic damage than lower limbs. Despite the global literature on TPNI, information from Iraq is limited. Factors such as war-related trauma place a significant burden on the population. EDX assessments are often delayed or performed incompletely due to severe injuries and the immediate focus on life-saving decisions. This shortage of data in Iraq underscores the need for systematic electrodiagnostic assessment of upper limb nerve injuries. Leveraging existing healthcare resources—trained clinicians, diagnostic tools, and rehabilitation services—can significantly improve diagnosis, guide timely interventions, and enhance patient recovery outcomes [5, 6].

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1.1 Classifications

Historically, the first formal classification of nerve injuries was introduced in 1942 by H.J. Seddon, a British orthopaedic surgeon. His system categorized nerve injuries into three distinct types based on anatomical disruption and patterns of recovery. Neuropraxia represents the first and least severe degree of injury, characterized by preserved nerve continuity despite functional impairment. Axonotmesis, the second degree of peripheral nerve injury, is defined by the disruption of the axon and its myelin sheath, while the surrounding perineurium and epineurium remain preserved. The third degree of nerve injury, known as neurotmesis, comprises a complete transection of the nerve, disrupting not only the axons but also all surrounding connective tissue layers. In 1951, S. Sunderland, an Australian neuroanatomist and clinician, expanded upon Seddon's classification by introducing two additional intermediate grades of nerve injury.

These provided a more detailed description of connective tissue involvement. While grade II injuries involve axonal damage with preservation of connective tissues, higher grades (III to V) reflect increasing levels of disruption to the endoneurium, perineurium, and epineurium, with more severe injuries typically necessitating surgical repair [7, 8]. The clinical assessment of a patient with PNI consists of a detailed history, focused neurological examination, and electrophysiological studies. These studies represent the gold standard method in diagnosing, localizing and grading PNI as well as determining the pathophysiology of injury, whether demyelination or axonal loss and the type of involved nerve, whether motor or sensory [9-12].

1.2 Electrodiagnosis:

A. Nerve conduction study*:* A sensory nerve action potential (SNAP) is recorded from the afferent fibers of a sensory nerve or the sensory branch of a mixed nerve, with key parameters including amplitude, latency, duration, and waveform configuration. Similarly, a compound muscle action potential (CMAP) reflects the summated electrical response of nearly synchronous action potentials from multiple muscle fibers, typically elicited by stimulating the motor nerve supplying the muscle. The important parameters to analyze include amplitude, latency, and duration [13, 14].

***B. Electromyography*:** Normally, a single motor unit innervates specific muscle fibers, and when activated, will produce a motor unit action potential (MUAP). In cases when the muscle is denervated, muscle fibers contract autonomously without any control. The detection of these spontaneous activities is one of the most significant findings in EMG. In neuropraxia, stimulation of the distal nerve segment typically elicits normal CMAP and SNAP responses. However, when stimulation is applied proximal to the lesion with distal recording, there is often a reduction in CMAP amplitude, partial or complete conduction block, and slowed conduction velocity across the injured segment. SNAPs may also show decreased amplitude with proximal stimulation. A conduction block is suggested if the amplitude drops by 50–70%. These abnormalities usually resolve as remyelination progresses. On needle EMG, spontaneous activity like fibrillation potentials is generally absent. During voluntary contraction, recruitment is reduced, and MUAPs are fewer with normal morphology [14].

In contrast, axonotmesis and neurotmesis share similar electrodiagnostic features, largely dependent on the time since injury. In the initial phase (up to 7 days for CMAP and 11 days for SNAP), distal stimulation may still yield normal or near-normal responses due to preserved axonal excitability. Thus, early differentiation from neuropraxia is difficult. After 10–12 days, Wallerian degeneration leads to a complete loss of distal responses, distinguishing these injuries from neuropraxia, where distal responses remain intact. A conduction block with preserved distal responses is strongly indicative of neuropraxia. Therefore, NCS are optimally performed 10–14 days post-injury. EMG changes in axonotmesis and neurotmesis develop over weeks. Electrophysiological evidence of denervation, such as fibrillation potentials and positive sharp waves, typically emerges within 2–3 weeks following injury. With reinnervation, MUAPs shift from small and unstable to larger and more complex [14].

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This study aims to characterize the electrodiagnostic patterns associated with traumatic peripheral nerve injuries of the upper limb in Iraqi patients. By analyzing nerve conduction and electromyography findings across affected median, ulnar, and radial nerves, the study seeks to identify specific findings that differentiate between partial and complete nerve injuries, thereby enhancing diagnostic accuracy and informing clinical decision-making.

2. Methods

Outpatients' attendants with a history of traumatic insult to the upper limb, representing all eligible individuals, were collected consecutively from the Neurophysiology clinic at Ghazi Al-Hariri hospital between July 2024 to March 2025. This case series is with an analytic comparison study. No power calculation was used. Inclusion criteria comprised individuals with a history of trauma and clinical symptoms indicative of peripheral nerve injury, such as weakness, numbness, or pain. Patients with non-traumatic neuropathy, such as diabetes mellitus, pre-existing peripheral neuropathy, or those who declined participation were excluded. All participants underwent a detailed trauma history and focused neurological examination, including power, sensory assessments and reflex testing. Informed consent was obtained from each participant, and the study protocol was approved by the Ethical Committee of the College of Medicine / University of Baghdad.

2.1 Electrodiagnostic Study

EDX studies, including NCs and EMG, were performed using the Natus system (2019, Ireland). NCS assessment typically involves evaluation of the median, ulnar, and radial nerves (sensory and motor). The patient was examined while lying comfortably in a supine position, and before starting the alcohol was applied to sterilize the skin. Adequate hand warming was ensured to avoid slow conduction because of a cold limb, and then electrodes were positioned at their specific recording sites. Regarding EMG, the same preparations were used. All the steps were explained in advance to the patients thoroughly. EMG was performed for the group of muscles innervated by the affected nerve in two phases, first during resting and relaxed muscle to assess spontaneous activity and the second one during minimal contraction to assess MUAP morphology.

2.2 Statistical analysis

The analysis of data was by SPSS software version 26.0 (SPSS Inc., Chicago, IL, USA). Continuous data were summarized as the mean with standard deviation. Categorical variables were represented as frequencies and percentages. Differences in nerve conduction study parameters and EMG across varying degrees of severity were assessed using Welch's t-test and one-way Anova. A p-value of < 0.05 was considered statistically significant.

3. Results and Discussion

Despite the high incidence of TPNI in Iraq because of war-related trauma, there is a lack of published local data, although proper data documentation is essential in estimating the burden on our healthcare resources and importantly, providing insight to optimal utilization of EDX studies in the diagnosis of TPNI. However, EDX may not be performed at the appropriate timing due to the severity of injuries and prioritization of life-saving interventions, leading to delayed diagnosis and poor prognosis. In this case series study, we analyzed the electrodiagnostic findings in patients with traumatic peripheral nerve injuries in the upper limbs. Our demographic data revealed 40 cases (80%) and females 10 cases (20%). This male predominance can be attributed to the type of patients attending the clinic, as most of them are military personnel, either currently serving or previously having worked in the military field. This aligns with the findings of Magnéli and Axenhus [15], who reported higher incidences of peripheral nerve injuries in men in comparison to women, across all age groups.

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As the majority of patients who participated in this study were military personnel, this demographic detail is of particular relevance, because military individuals in Iraq are at higher risk of traumatic upper limb injuries as they operate in a hazardous environment such as armed conflicts and explosive operations. Therefore, the findings of this study may hold practical value for military healthcare systems in Iraq, offering insights into the utility of EDX assessments for early detection, classification, and management of PNI in this high-risk population. Such targeted applications could contribute to optimized rehabilitation protocols and improved functional outcomes among injured service members.

Missile injuries were the most common cause of PNI, representing 40% of cases, with motor vehicle accidents accounting for 18%. By contrast, Aman et al. [16] reported motor vehicle accidents as the leading mechanism of traumatic injuries observed in 7% of 110,667 patients evaluated at their trauma center. The mean age was 39.8±11 years. Some patients had multiple nerve injuries. Twenty-six (47.3%) had injury on the right side and 29 (52.7%) were on the left side. Forty-three (78.2%) patients with laceration injury, 12 (21.8%) with traction injury, as shown in Table 1

Table 1: Demographic and clinical data amongst involved patients.

Gender	Male	40	80%
	Female	10	20%
Side	Right	26	47.3%
	Left	29	52.7%
Mechanism of injury	Laceration	43	78.2%
	Traction	12	21.8%
Duration of injury	<1 year	15	27.3%
	1-5 years	21	38.2%
	>5 years	19	34.5%

3.1 Median nerve

CMAP amplitude for the median nerve averaged 8.2 ± 2.2 mV, with 76.4% of nerves demonstrating normal motor amplitudes. A smaller proportion showed reduced (16.4%) or absent (7.3%) motor responses, indicating varying degrees of axonal loss. Distal motor latency (DML) averaged 3.5 ± 0.7 ms, and conduction velocity was within normal limits at 52.0 ± 5.8 m/s. SNAP amplitudes averaged 26.9 ± 13.4 μV, with half of the nerves exhibiting normal sensory amplitudes, while 40% showed reductions and 10% were absent. Distal sensory latency (DSL) averaged 3.1 ± 0.9 ms. As shown in Table 2 and Fig. 2.

3.2 Ulnar nerve

The ulnar nerve showed slightly lower CMAP amplitudes, with a mean of 7.4 ± 2.0 mV. Normal CMAP amplitudes were found in 72% of cases, while 20% exhibited reductions and 7.3% had absent responses. The DML was 2.8 ± 0.7 ms, and conduction velocity averaged 53.6 ± 8.2 m/s, consistent with preserved nerve function in most cases. SNAP amplitudes averaged 19.1 ± 9.4 μV, with half the nerves showing normal responses. However, 34.5% had absent sensory signals, and 14.5% were reduced. The DSL was 2.6 ± 0.9 ms. In our study, the ulnar nerve was the most frequently affected, involved in 20 cases (40%), consistent with findings reported by Elfayoumy et al. [17] and Kouyoumdjian et al. [18], who also reported the ulnar nerve as the most frequently injured nerve, either alone or in combination with other nerves. In our study, six cases were found to have combined nerve injuries, four of which affected both the ulnar and median nerves in the same trauma. In contrast, Jung

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et al. (2024) found that radial nerve injury, related to humeral fractures, constituted the largest subgroup in a cohort of Korean soldiers, as shown in Table 2 and Fig. 1.

3.3 Radial nerve

The radial nerve demonstrated the highest proportion of normal CMAP amplitudes, with 90% of nerves showing normal values averaging 4.6 ± 1.0 mV. Reduced and absent CMAP amplitudes were less common (7.3% and 1.8%, respectively). DML was 2.5 ± 0.4 ms, and conduction velocity was 54.3 ± 7.2 m/s. SNAP amplitudes averaged 21.9 ± 9.0 µV, with 74% normal, 20% reduced, and only 5.5% absent. DSL was 3.4 ± 4.1 ms, reflecting relatively preserved sensory function, as shown in Table 2 and Fig. 1.

Table 2: Electrodiagnostic Findings in median, ulnar and radial nerves. (n=50; total nerves=55)

Parameter	Median nerve	Ulnar nerve	Radial nerve
Motor NCS (MNCS):			
CMAP amplitude (mV)	8.2 ± 2.2	7.4 ± 2.0	4.6 ± 1.0
Normal	42 (76.4%)	40 (72.0%)	50 (90.0%)
Reduced	9 (16.4%)	11 (20.0%)	4 (7.3%)
Absent	4 (7.3%)	4 (7.3%)	1 (1.8%)
Distal motor latency (ms)	3.5 ± 0.7	2.8 ± 0.7	2.5 ± 0.4
Conduction velocity (m/s)	52.0 ± 5.8	53.6 ± 8.2	54.3 ± 7.2
Sensory NCS (SNCS):			
SNAP amplitude (µV)	26.9 ± 13.4	19.1 ± 9.4	21.9 ± 9.0
Normal	25 (50.0%)	28 (50.0%)	41 (74.0%)
Reduced	20 (40.0%)	8 (14.5%)	11 (20.0%)
Absent	5 (10.0%)	19 (34.5%)	3 (5.5%)
Distal sensory latency (ms)	3.1 ± 0.9	2.6 ± 0.9	3.4 ± 4.1

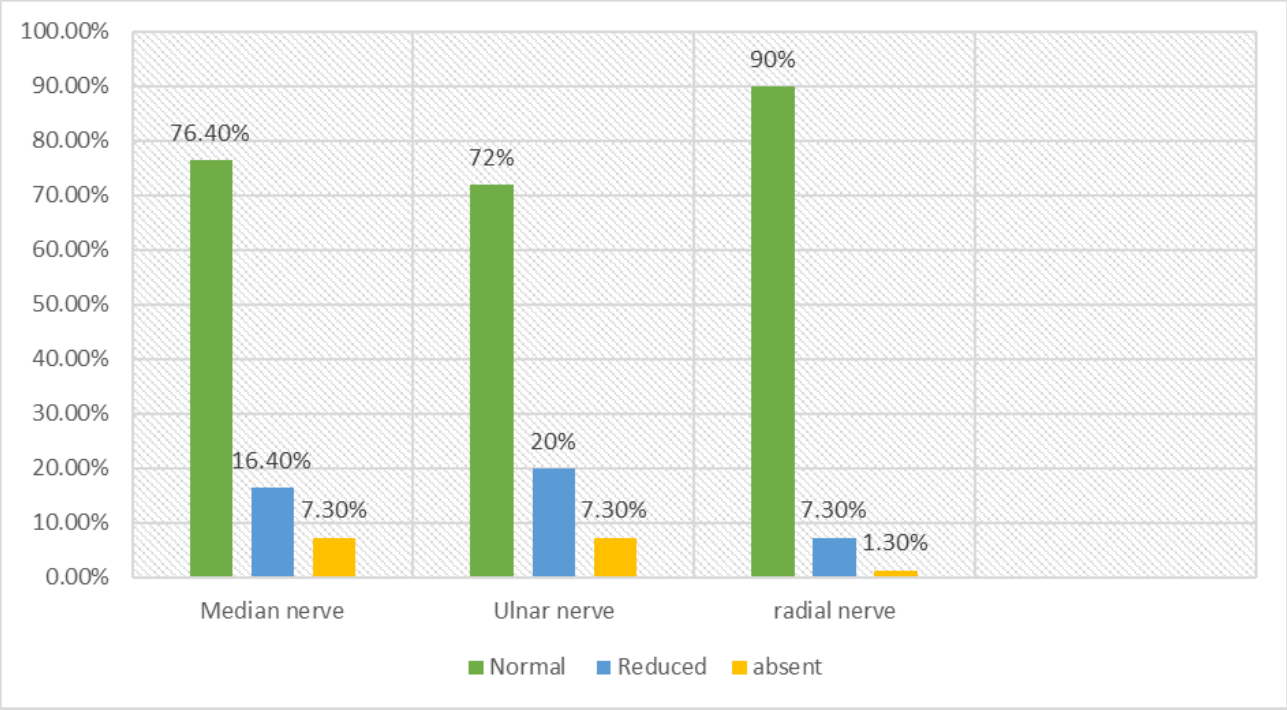

Fig. 1: Motor and sensory amplitudes between median, ulnar and radial nerves.

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Regarding EMG examination, we found spontaneous activity in 11 cases (20%). Additionally, 10 (18.2%) showed abnormal reinnervated MUAPs (large duration and high amplitude), which can be explained by collateral sprouting after partial axonal loss [17], and fourteen (25.4%) had absent MUAPs, indicating a complete nerve cut as shown in Figs 2 and 3.

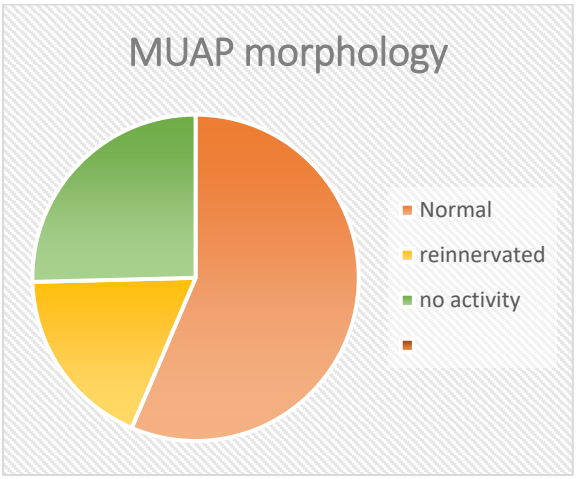

Fig. 2 MUAP morphology

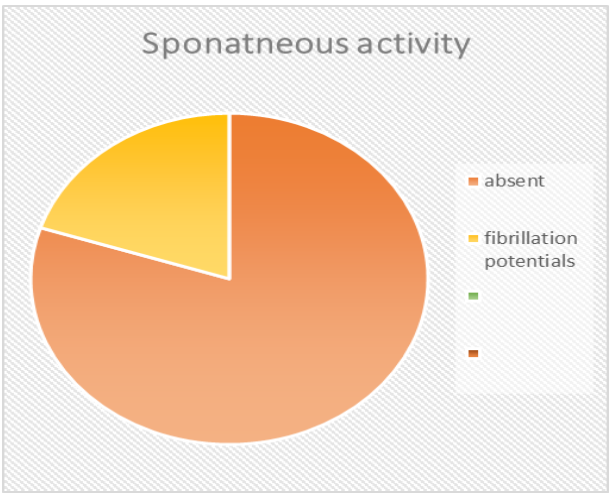

Fig. 3 Spontaneous activity

EDX findings revealed partial axonal loss (axonotmesis), complete axonal loss (neurotmesis), and normal nerve conduction. This classification follows the approach of Elfayoumy et al. [17] and Padua et al. [20], who categorized injuries based on the severity of axonal loss. The statistical analysis revealed highly significant differences across all measured EDX parameters among normal, partial, and complete nerve injury groups. One-way ANOVA demonstrated p-values < 0.0001 for CMAP amplitude, latency, and conduction velocity, as well as SNAP amplitude and latency, underscoring their diagnostic utility. CMAP amplitude was significantly reduced in complete injuries, moderately reduced in partial injuries, and highest in normal nerves. This gradation supports the utility of CMAP amplitude as a reliable marker of axonal integrity. Regarding sensory fibers, SNAP amplitudes and latencies followed similar patterns. The absence of SNAP amplitude in complete injuries indicates significant axonal loss. These findings validate the use of nerve conduction studies for grading injury severity. Furthermore, the EMG findings—particularly spontaneous activity seen exclusively in complete injuries—provide further electrophysiological confirmation of neurotmesis. As shown in tables 3,4,5.

Table 3: Comparison of median nerve CMAP parameters between normal and partially injured nerves. (n=55)

Characteristic	Normal MNCS= 42[1], SNCS= 25[1]	Partial nerve injury MNCS=9[1] SNCS= 20[1]	Complete injury MNCS=4[1] SNCS=5[1]	p-value[2]
CMAP amplitude	8.8 ± 2.4	1.99 ± 0.54	0	$p < 0.0001$
CMAP latency	3.2 ± 0.3	5.45 ± 1.37	0	$p < 0.0001$
Conduction velocity	55.7 ± 3.8	37.06 ± 8.77	0	$p < 0.0001$
SNAP amplitude	34.2 ± 13.2	12.3 ± 5.2	0	$p < 0.0001$
SNAP Latency	3.02 ± 0.44	4.54 ±1.14	0	$p < 0.0001$

[1]Mean ± SD; n (%)

[2]Welch Two Sample t-test; Fisher's exact test

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Table 4: Comparison of ulnar nerve CMAP parameters between normal and partially injured nerves. (n=55)

Characteristic	Normal, MNCS =40[1] SNCS=28[1]	Partial nerve injury MNCS = 11[1,] SNAP= 8[1]	Complete nerve injury MNCS= 4[1], SNAP=19[1]	p-value[2]
CMAP amplitude	7.7 ± 1.7	4.2 ± 1	0.1 ± 0.2	p < 0.0001
CMAP latency	2.9 ± 0.8	4.6 ± 0.96	0	p < 0.0001
conduction velocity	54.7 ± 7.0	37.7 ± 5	0	p < 0.0001
SNAP amplitude	24.4 ± 8.4	11.8 ± 3.4	0	p < 0.0001
SNAP Latency	2.6 ± 0.5	4.22 ± 1.3	0	p < 0.0001
[1]Mean ± SD; n (%) [2]One-way ANOVA; Fisher's exact test				

Table 5: Comparison of radial nerve CMAP parameters between normal and partially injured nerves. (n=55)

Characteristic	Normal MNCS = 50[1] SNCS=41[1]	Partial nerve injury MNCS= 4[1] SNCS = 11[1]	Complete nerve injury MNCS= 1[1] SNCS=3[1]	p-value[2]
CMAP amplitude	4.6 ± 0.8	1.7 ± 0.1	0.6±0	p < 0.0001
CMAP latency	2.5 ± 0.2	4 ± 1.16	6	p < 0.0001
Conduction velocity	55.1 ± 5.7	36.2 ± 6	16.6	p < 0.0001
SNAP amplitude	26.1 ± 9.1	10.5 ± 3.6	0.23 ± 0.4	p < 0.0001
SNAP Latency	2.14 ± 0.3	4.25 + 1.4	0	p < 0.0001
[1]Mean ± SD; n (%) [2]One-way ANOVA; Fisher's exact test				

4. Conclusion

Electrodiagnostic studies provide vital diagnostic and prognostic information in traumatic peripheral nerve injuries. A pattern-based interpretation of NCS and EMG findings highlights the importance of early and accurate EDX evaluation, it can reliably differentiate between injury severities and guide treatment planning.

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