International Research Journal of Natural and Applied Sciences
Vol. 3, Issue 11, November 2016 Impact Factor- 5.46
ISSN: (2349-4077)

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PERIPHERAL COMPRESSIVE NEUROPATHY
ROLE OF NCS AND EMG IN DIAGNOSIS AND FOLLOW UP

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ABSTRACT

Background: *Compression / entrapment lesions are the most common disorders of the peripheral nervous system as they comprise between 60-70% of all peripheral nerve problems.*

Electrodiagnostic examination can be helpful in the assessment of these lesions, it is the premier diagnostic laboratory procedure in this regard.

Aim of the study: *The aim of this study is to determine the role of NCS and EMG in the diagnosis and follow up.*

Patients and methods: *It is a prospective study on (42) patients with (51) peripheral nerve lesions who were divided into two groups, traumatic and non-traumatic. All of them were subjected to clinical and electrophysiological assessment followed by surgical decompression and external neurolysis.*

Results: *It has been found that NCS and EMG are very helpful in confirming the diagnosis of nerve compression and predicting the surgical outcome.*

Early decompression (which is our policy in this study) resulted in clinical and electrophysiological recovery earlier than late decompression.

Conclusions: *NCS and EMG can localize the site of nerve compression, determine the type of axon injured, depth and severity of compressive injury.*

Introduction

Nerve entrapment / compression lesions are one of the major causes of peripheral nerve dysfunction. These lesions are acute or chronic, traumatic or non-traumatic [(1)].

In addition to the clinical findings, electrophysiological studies are used for the assessment of these lesions by means of Nerve Conduction Study (NCS) and Electromyography (EMG). The introduction of electrodiagnostic techniques in the evaluation of these disorders began with the pioneering work of Professor Fritz Bucthal and colleagues in 1950 and the clinical studies of the motor units especially with the work of Professor Erik Stalberg[(2-7)].

Electromyography (EMG) is the study of the electrical properties of skeletal muscles as it reflects the integrity of the connection between the nerve and its innervated muscle and the muscle itself[(1)]. Electrical activity of muscles can be recorded from the skin overlying the muscle with surface electrode or with needle electrodes inserted into the bulk of the muscle originally introduced by Adrian and Bronk 1929 [(15)].It is possible to measure three recordings, these are[(8-16)]:

1. Insertion potential activity
2. Spontaneous activity (fibrillation, sharp wave and fasciculation)
3. Motor units potential

When voluntary contraction occurs in the muscle, motor units will be seen. The normal amplitude is 100Mv and duration is 10ms.

Nerve Conduction Study (NCS) is used to measure the action potentials resulting from peripheral nerve stimulation recorded over the nerve or innervated muscle. It evaluates both motor and sensory components. The measurements:

1. Latency: is the time elapsed between onset of the stimulus and onset of the motor or sensory nerve action potential.
2. Amplitude: is the height measured from baseline to peak.

3. Conduction Velocity (CV): by stimulating and recording from two separate sites (proximal and distal) over the nerve, it can be measured by the following equation[17-19]:

$$CV = \frac{Distance\ (between\ proximal\ and\ distal\ stimuli)}{Time\ (proximal\ latency - distal\ latency)}$$

Other diagnostic approaches is the plain x-ray, which determines the state of soft tissue, any retained radio-opaque foreign body, the presence of fracture with degree of displacement and the presence of bone spike which may be the cause of nerve injury.

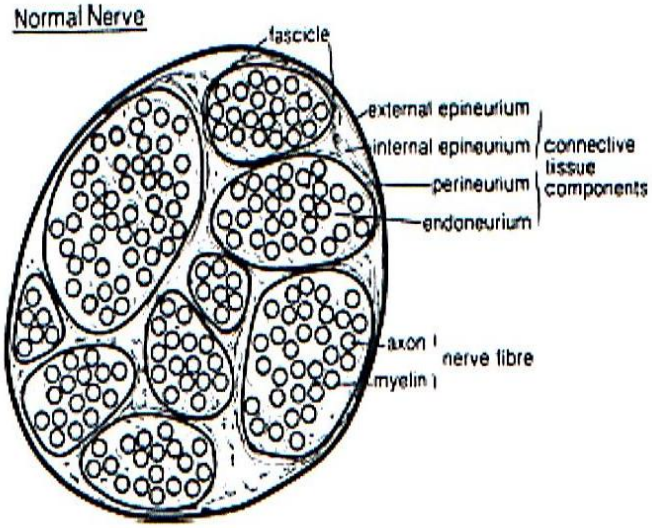


Figure (1): Peripheral nerve microanatomy

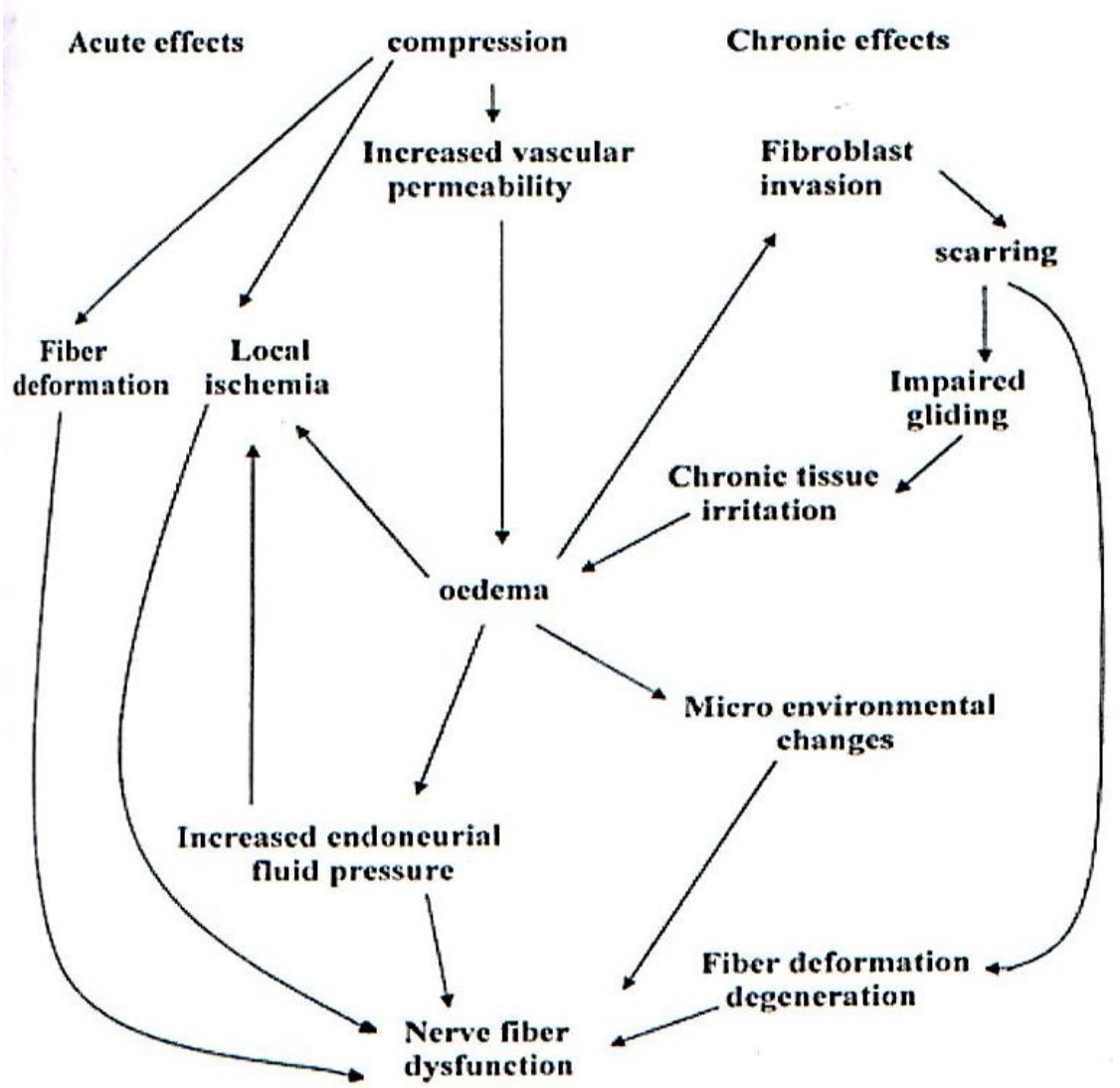


Figure (2): Pathophysiology of peripheral nerve compression

Patients

This is a prospective study on (51) peripheral nerves of (42) patients (26 females "62%" and 16 males "38%") admitted to the departments of Plastic and Orthopaedic surgery in Al-Wasity Teaching Hospital between October 2003 and February 2005.Female to Male ratio was "1.6:1". The mean age of the patients at time of admission was (41) years ranging between (12-70) years as shown in the table.

Table (1): Distribution of patients according to age group and sex

Age group	Patients number	Male	Female
10-19	4	3	1
20-29	9	4	5
30-39	10	4	6
40-49	10	4	6
50-59	6	1	5
60-69	2	-	2
70-79	1	1	-

The patients were divided into two groups: traumatic group (A) and non-traumatic group (B). Group (A) includes (22) patients, (13) males and (9) females. Group (B) includes (20) patients, (17) females and (3) males. In group (A) the upper limb was affected in (16) patients and the lower limb in (6) patients while all patients in group (B) have upper limb involvement.

The entrapment was unilateral in all patients of group (A) while in group (B) it was unilateral in (11) patients and bilateral in (9) patients. At presentation, the duration of symptoms ranged between 1 month-12 years and it was generally shorter in group (A) than group (B). The median nerve was the most commonly involved.

Table (2): Frequency and percentage of specific nerve involvement including bilateral cases

Nerve	Number	Percentage %
Median	37	72.5%
Radial	5	9.8%
Ulnar	3	5.8%
Common Peroneal	2	4%
Sciatic	4	7.9%
Total	51	100%

The patients sustained different causes of peripheral nerve compression as shown in the table.

Table (3): Distribution of patients according to the cause

Cause	Group A	Percentage %	Group B	Percentage %
Bullet	9	21.4%	-	-
Fracture distal radius	7	16.6%	-	-
Stab	4	9.5%	-	-
F.B.	1	2.4%	-	-
Iatrogenic (ligation)	1	2.4%	-	-
Idiopathic	-	-	8	19%
Pregnancy	-	-	6	14.3%
Mass (TB or Bursitis)	-	-	2	4.8%
Diabetes	-	-	2	4.8%
Drugs (Contraceptive pills)	-	-	1	2.4%
Repetitive trauma (hand stick)	-	-	1	2.4%
Total	22	52.3%	20	47.7%

Methods

All patients were evaluated by detailed history according to a special questionnaire prepared for this purpose.Thorough physical examination concentratingon peripheral nerve examination both clinically and electrophysiologically. The examination includes:

1. **Sensory examination:**Touch, pain and temperature. In our study we examined these modalities by using cotton wool for touch, a pin for pain and a hot or cold object contact for warmth and cold.
2. **Innervation density test: Static two-point discrimination:** In our study, we depended on the static test because the moving test can be applied only in the distal phalangeal pud[(19)].This test more accurately reflects the quantity of innervated sensory receptors. The normal

threshold for the volar surface of the hand (divided into 7 zones) is 7-12 mm, below elbow and knee 40-50 mm, above elbow and knee 65-75 mm[12].

3. **Threshold test: Tourniquet test:**By applying a pneumatic blood pressure cuff proximal to the elbow and inflation higher than the patient's systolic pressure. The test is positive when tingling develops in 60 seconds [13].
4. **Tinel's sign:Formication sign:** A positive test indicates that regenerating axons are progressive along the endoneurial tube. The speed of regeneration is 1 mm/day or 25 mm/month in adults and two or three times of that in children [14].
5. **Phalen test**
6. **Reverse Phalen test**
7. **Percussion test**
8. **Autonomic function tests:** Sudomotor, Pilomotor and Vasomotor.
9. **Motor examination:** according to the following classification:

Table (4): Classification of severity of motor deficits caused by peripheral nerve disorders [20]

Grade	Description of muscle function	Percentage of motor deficit
5	active movement against gravity with full resistance	0
4	active movement against gravity with some resistance	1-25
3	active movement against gravity only without resistance	26-50
2	active movement with gravity eliminated	51-75
1	slight contraction with no movement	76-99
0	no contraction	100

In our study we adopted the classification of nerve injuries by Seddon 1972 and Sunderland 1978 [21], although grade 1 and 2 are relevant only as shown in the following figure:

		Sunderland				
		1st degree	2nd degree	3rd degree	4th degree	5th degree
Seddon	Neuropraxia					
	Axonotmesis					
	Neurotmesis					

Figure (3): Correlation of Seddon's and Sunderland's classification of nerve injuries. Shaded areas indicate equivalent terms [22]

The diagnosis and follow up of patients with peripheral nerve compression / entrapment were based on clinical examination and electrophysiological study. The clinical findings at the time of diagnosis are shown in the following table.

Table (5): Frequency and percentage of clinical findings

Clinical findings	Group A	Group B	Total	Percentage
Pain	3	16	19	45.5%
Paresthesia	13	16	29	69%
Tinel's sign	11	14	25	59.5%
Tourniquet test	7	12	19	45.2%
Motor deficit	15	8	23	54.7%
Sensory loss	10	1	11	26.1%
Two-point discrimination	15	14	29	69%
Phalen test	7	16	23	54.7%

In the traumatic group (A), (7) patients with distal radius fracture had positive tourniquet test and Phalen test, paresthesia with or without positive Tinel's sign or abnormal static two-point discrimination so the clinical diagnosis was median nerve compression within the carpal tunnel.

(15) Patients with motor deficit like claw hands, wrist drop and foot drop with sensory loss or paresthesia were diagnosed as severe nerve injury.

In the non-traumatic group (B), the clinical diagnosis was carpal tunnel syndrome in all patients except one who presented with a lump in the wrist with neither pain nor paresthesia with negative Tinel's sign, Phalen test and Tourniquet test. All patients specifically in group (A) were sent for radiological examination (plain x-ray).

EMG and NCS were done for all patients before surgery and two weeks after surgery when pain had subsided then every 2-4 weeks depending on patient's cooperation.Regarding the electrophysiological diagnosis, assessment of the results of NCS depends on the comparison with the normal values as shown in the table.

Table (6): Normal values of NCS

Nerve		Latency (m/sec)	Amplitude (Mv)	Conduction Velocity(m/sec)
Ulnar	Motor	3	5	51
	Sensory	3.2	5	50
Median	Motor	3.8	5	50
	Sensory	3.5	10	51
Radial	Motor	3.6	7	52
	Sensory	3.3	15	50
Sciatic	Motor	45	-	-
Common Peroneal	Motor	5	4	44
	Sensory	3.5	5	40

NCS was done for all patients in our study and the following results were obtained.

Table (7): Pre-operative results of patients NCS

Patients	Sensory			Motor		
	Latency (m/sec)	Amplitude (Mv)	Velocity(m/sec)	Latency (m/sec)	Amplitude (Mv)	Velocity(m/sec)
Group A						
6	Normal	Decreased	Normal	Increased	Decreased	Decreased
3	Increased	Decreased	Decreased	Normal	Decreased	Normal
4	Absent	Absent	Absent	Normal	Decreased	Normal
7	Absent	Absent	Absent	Absent	Absent	Absent
2	Normal	Normal	Normal	Normal	Normal	Normal
Group B						
13	Increased	Normal	Decreased	Increased	Normal	Normal
7	Absent	Absent	Absent	Increased	Decreased	Decreased

This table shows that (9) cases in group (A) were diagnosed by NCS as moderate nerve injury because of abnormal sensory and motor response. (11) Cases were diagnosed as severe injury due to absent or abnormal sensory response and absent motor response[(23)]. (2) Cases had normal recordings.

In group (B), (13) cases were diagnosed as moderate carpal tunnel syndrome (CTS) because of abnormal sensory and motor responses. (7)Cases with absent sensory response and abnormal motor response were diagnosed as severe CTS[(24)].

The EMG was done simultaneously with NCS to support diagnosis.

Table (8): Distribution of patients according to EMG findings

Patients	Insertional activity		Spontaneous activity		Exertional activity (mup)	
	Normal	Increased	Fibrillations	Absent	Normal	Reduced
Group A	15	7	15	7	4	18
Group B	-	-	-	-	16	4

Fibrillation potentials were recorded in (15) patients in group (A) and absent in (7) patients. Reduction of motor unit potential (mup) recruitment was recorded in (18) patients in group (A) while it was normal in (4) patients. In group (B) normal (mup) was recorded in (16) patients while it was reduced in (4) patients.

Results

Surgical treatment was performed in all cases when the diagnosis was confirmed clinically and electrophysiologically. (3)Patients were excluded from our study.

The first one suffered from bullet injury to the back of the thigh, exploration revealed sciatic nerveneuroma in continuity. The second one had the same injury but the sciatic nerve was completely severed. The third one suffered from stab injury to the lower forearm, the ulnar nerve was completely severed. All of these injured nerves were treated with nerve graft. The surgical findings are summarized in the following table.

Table (9): Operative findings

Patients	Cause	Number	Procedure	Findings
Group A	Bullet	4	External neurolysis	Intact nerve + fibrous adhesions + stenosis and prestenotic dilatation
	Fracture	4		
	Ligation	1		
	Stab	3		Intact oedematous nerve + fibrous adhesions
	F.B.	1		
	Fracture	3		
	Bullet	3		
Group B	Non traumatic median nerve compression	9	Flexor retinaculum release + epineurial neurolysis	Fibrous adhesions + stenosis and prestenotic dilatation
		6		Fibrous adhesions + flat congested nerve
		5	Flexor retinaculum release	Oedematous nerve

After surgery, the patients were followed up clinically and electrophysiologically for variable periods ranging from (2) weeks to (7) months depending on patient's cooperation. (8) Patients in group (A) and (9) patients in group (B) were not followed up due to loss of contact.

The first signs of clinical recovery were decreased or absence of paresthesia and improvement of muscle power grading.The first signs of electrophysiological recovery were reduced latency, increased amplitude and increased nerve conduction velocity. The following table shows the results of follow up and the correlation between clinical and electrophysiological recovery.

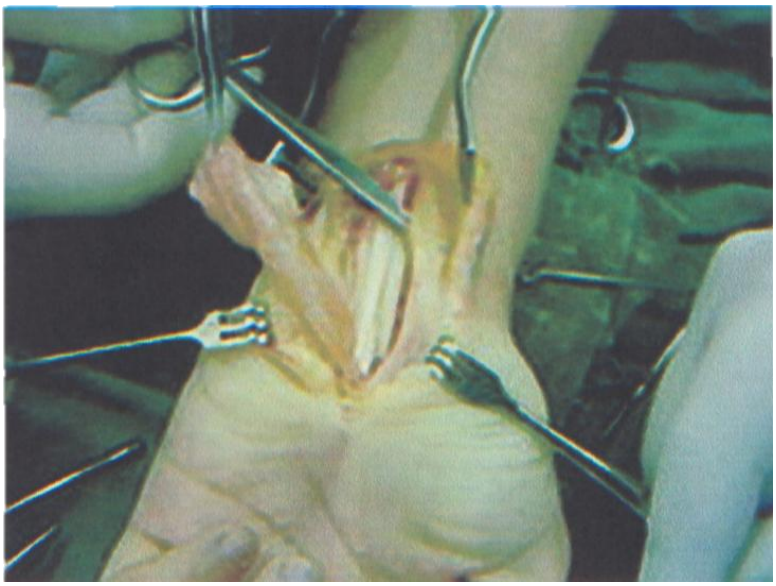
Table (10): The results of patients follow up

Patients	No.	Follow up	2w	4w	6w	8w	12w	4m	5m	6m	7m
Group A	8	Clinical	+	+	+	F					
		EMG	+	+	+	+	+	+	+	+	+
	3	Clinical	+	+	+	+	+	+	+	+	+
		EMG	-	-	-	-	-	-	-	-	-
Group B	2	Clinical	F								
		EMG	+	F							
	6	Clinical	+	F							
		EMG	+	+	+	+	+	+	+	+	+
	2	Clinical	+	+	+	+	F				
		EMG	+	+	+	+	+	+	+	+	
	1	Clinical	+	+	+	F					
		EMG	-	-	-	-					

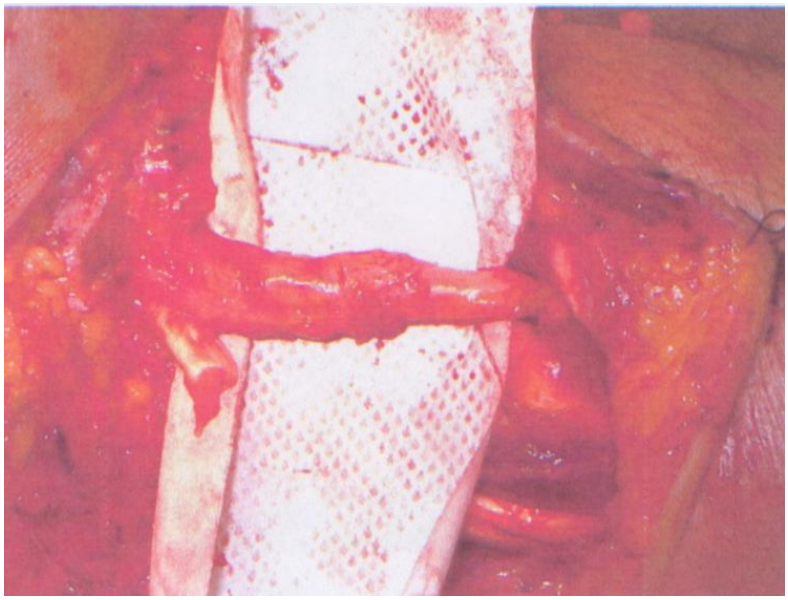
(No.) number,(w) week,(m) month, (+) progressive recovery, (-) static or no recovery,(F) full recovery depending on muscle power grading

The table shows that (8) patients have full clinical recovery after 8weeks while EMG recovery was progressive at 7months. (3) Patients did not reach full clinical recovery even after 7 monthson EMG with no signs of recovery.

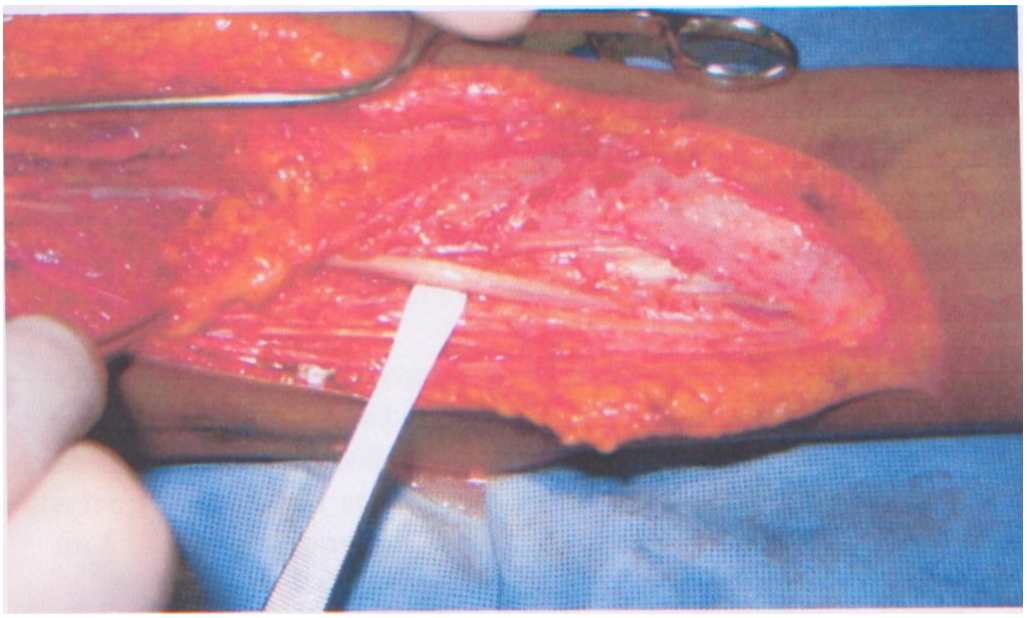
(2) Patients were followed up 4weeks in which clinical recovery was full after 2weeks and EMG recovery after 4weeks. (6) Patients were followed up 7months, full clinical recovery after 4weeks and EMG recovery was progressive after 7months. (2) Patients were followed up 6months, full clinical recovery after 12weeks and EMG recovery was progressive after 6months. (1) Patient was followed up 8weeks, full clinical recovery after 8weeks and EMG results were static without any sign of recovery during this period.



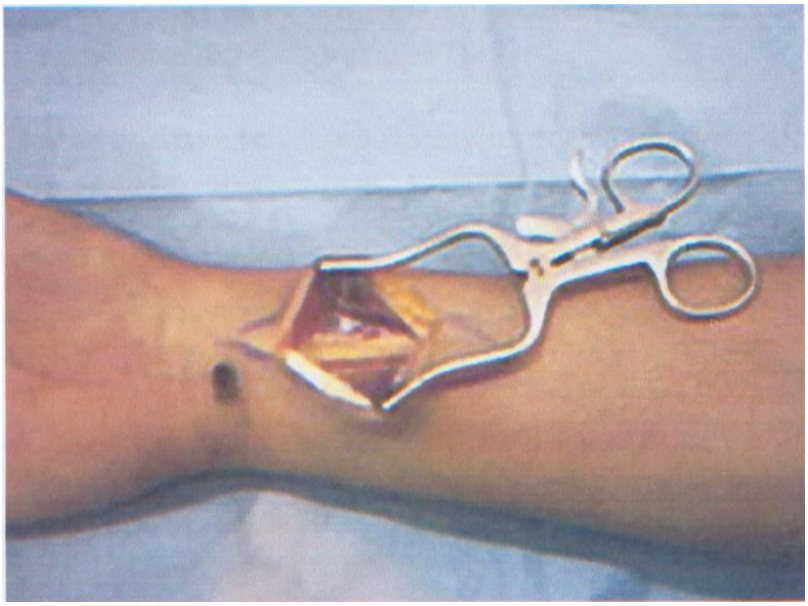
Picture 1: Big mass (bursitis) compressing the median nerve within the carpal tunnel in 35years old female patient



Picture 2: 27years old male patient suffered from stab wound injury in the lower forearm, external neurolysis revealed intact median nerve compressed with fibrous tissue



Picture 3: 15 yearsold male patient presented with foot drop and sensory loss in the dorsum of the foot 3 weeks after stab injury in the popliteal fossa, exploration revealed intact compressed common peroneal nerve with fibrous tissue



Picture 4: 28years old male patient with motor weakness and paresthesia along the median nerve distribution 2 weeks after shell injury in the lower forearm exploration revealed intact median nerve with fibrous tissue

Discussion

The timing of electrophysiological diagnosis is critical regarding when nerve injury had occurred. In our patients, NCS and EMG were performed at least 1 month after injury in which all abnormalities could be detected at that time.The explanation is that although the clinical motor and sensory deficits are maximal at onset, most electrophysiological abnormalities are not, they first appear after 2-3 weeks and all abnormalities are fully developed 3-5 weeks after injury[23].

In group (A), (7) patients presented with history of fracture distal radius had the classical signs and symptoms of median nerve compression within the carpal tunnel. (15) Patients subjected to trauma presented clinically with sensory or motor loss, NCS and EMG were required to confirm the presence of nerve compression or nerve Injury secondary to trauma.This finding is in agreement with the study of Chavin (2002) and Seivak (1993) who stated that traumatic peripheral nerve dysfunction results in pathophysiological changes along nerve fibers and electrophysiological study can detect such changes along the motor and sensory axons[25,26].

In group (B), all patients were diagnosed clinically as CTS except (1) patient who did not present with the classical signs and symptoms, therefore NCS and EMG were required to confirm the diagnosis. This finding is supported by the study of spinner (1989) who mentioned that diagnosis is easy when all signs and symptoms and confirmatory tests are present, however when isolated symptom is present without any findings, positive electrical studies are helpful in confirming diagnosis [27].

NCS was positive in (20) patients in group (A) in which abnormal or absent sensory and motor responses were found while it was negative (normal study) in (2) patients (7%) with fracture distal radius and diagnosed clinically as CTS. This finding is supported by the study of Louis et al (1987) who found that false negative tests exist but the rates have decreased with improving techniques [28] and the study of Spinner (1989) and Stevens (1988) who found that (5%) of clinically certain CTS patients have normal studies[27-29].The explanation of false negative results is that the symptom of paresthesia is related to excessive neuronal firing of sensory neurons (Both of our patients have paresthesia). This symptom has no electrophysiological correlation and may occur without sufficient compression[30].

All patients in group (B) have positive NCS findings in which abnormal or absent motor and sensory responses were found.A study by Oh SJ (1993) supports this finding in which NCS was positive in 91-98% of patients with clinical CTS, where as 100% patients were positive in our study[31].

NCS can determine the depth of nerve lesion whether it is in the myelin sheath (neuropraxia or demyelination) or in the axon (axonotmesis). Demyelination is presented electrophysiologically with increased motor and/or sensory latency and when it is more severe to cause conduction block or axonal loss even NAP amplitude is reduced.

In group (A), (20) patients had reduced or absent motor and/or sensory amplitude (axon loss), this finding is supported by the study of Wilbourn (2002) who proved that axonotmesis is the most common type of injury in traumatic compressive neuropathy[32].

In group (B), (13) patients had prolonged latency with normal amplitude (neuropraxia) and (7) patients with prolonged latency and reduced amplitude (conduction block). This finding is supported by the same study which determines that the primary pathology in CTS is demyelination rather than axon loss.

NCS can determine the type of axonal injury whether motor or sensory through the measurement of (NAP) amplitude.When the motor amplitude is reduced it means motor axon loss and when sensory amplitude is reduced, it is sensory axon loss.NCS can also determine the severity of the compressive injury, according to Wilbourn classification [23] of axonal injury in traumatic peripheral compressive neuropathy which is based on NCS changes. Axon loss is mild when both motor and sensory amplitudes are normal, moderate when both are reduced, severe when the sensory amplitude is absent and motor amplitude is severely reduced, and total or complete axon loss when both amplitudes are absent.In our study, (9) patients have moderate or partial axon loss, (4) patients have severe axon loss and (7) patients have total axon loss, all of them were explored and found intact compressed except (2) patients with complete lesion (total axon loss) had completely severed nerve and (1) patient with neuroma in continuity. This means that although NCS and EMG report reveals a complete injury, it does not necessarily mean complete severance of the nerve but complete axonal loss with intact compressed nerve.This is of paramount clinical implication,such cases must be explored surgically without delay. The

purpose is to repair completely divided nerve or to decompress an intact nerve, the latter gives dramatic post-operative recovery.

Regarding the non-traumatic compressive neuropathy, CTS can be classified in order of severity according to Padua classification [(24)] which is based on NCS changes into mild CTS when median sensory latency is abnormal with normal motor latency, moderate when both are abnormal, severe when sensory latency is absent with abnormal motor latency and extreme when both are absent. (13) Patients had moderate CTS and (7) patients had severe CTS.

NCS and EMG can differentiate whether the cause of muscle weakness is neurogenic or myogenic (e.g.: myopathy). When the motor nap amplitude is reduced or absent with reduced or absent map recruitment then the cause is nerve injury and when both are normal then the cause is myogenic.

NCS and EMG can localize the site of compression which is situated along the injured nerve at a site where stimulation can be applied immediately proximal and distal to it, for example: a suspected common peroneal nerve compression at fibular head can be localized by stimulation at two sites between the popliteal fosse and below the fibular head.When this stimulation is not possible, the injury is located at a site below the origin of the motor branch that innervates the most distal muscle which is clinically normal and that supplies the most proximal muscle which is abnormal clinically (weak). This is helpful in multiple level injuries to determine whether one or more sites are responsible for nerve damage.

All patients were explored 34 months after injury except (2) patients explored 1 month after injury and (1) patient explored after 14 months. The delay was due to delay in referral and late arrival of the patients. Our policy is to explore surgically as soon as practical.The reason is that the extent of nerve fiber damage increases with time as the mechanical compressive force continues to influence the lesion[(36)].Although Kline and Hackett (1975) who recommended that during the first 3-4 months after injury, one may identify the potential for spontaneous recovery and allow the nerve to regenerate[(37)]. We believe that early decompression produces dramatic full recovery as long as patients who explored early show progressive electrophysiological recovery while in those with late decompression, the NCS and EMG remains static during the follow up period.

External neurolysis was performed in all cases even the severe ones in which the nerve was severely compressed with stenosis and prestenotic dilatation or flat and congested. The explanation is that internal neurolysis can result in excessive interfascicular fibrosis [38].

These surgical findings were found in (9) patients in group (A) and (9) patients in group (B) all of them were diagnosed as severe cases with NCS and EMG.This suggests that there is a correlation between NCS/EMG and the surgical findings as mentioned in the study of Sunderland (1978), that in severe cases of compression neurolysis reveals intact nerve with narrow area of indentation [22].

Regarding the role of NCS and EMG in follow up and prognosis;as previously shown in table (10), (8) patients in group (A) and (10) patients in group (B) who were diagnosed as partial or moderate lesions with mainly neuropraxia (demyelination) with some axon loss, these patients recovered clinically and electromyographically earlier than the other (4) patients who were diagnosed as severe or total (complete) axon loss.This finding is supported by the study of Robert and Morton Spinner (1998) which determines that owing to segmental demyelination, recovery following neurolysis of neuropraxic lesion takes 1-2 months and owing to Wallarian degeneration distal to the level of compression, recovery following neurolysis of axonotmetic lesion takes more than 6 months[39].

With successful regeneration, NCS shows decreased latency, increased amplitude and velocity and EMG shows increasedinsertional activity, decreased in number of fibrillation potentials and examples of nerve action potentials.

The post-operative electrodiagnostic study can help to predict the surgical outcome of nerve decompression. That is because partial improvement of abnormal pre-operative electrodiagnostic study provides reassurance that adequate decompression was performed[40].Table (10) also shows that there is suboptimal correlation between clinical and electrical recovery as the patients recovered clinically, while NCS and EMG were progressive or the clinical recovery was progressive while NCS and EMG were static. This is supported by the study of Goodwill (1965) and Melvin (1968) who proved that complete clinical improvement may be expected despite incomplete improvement of NCS to normal range[41,42].

Conclusions

1. Nerve compression should be suspected whenever there is a closed or open trauma to a limb.
2. Early referral of cases to specialized centers is essential for proper investigations and early decompression.
3. NCS and EMG can support the diagnosis and predict the surgical outcome and can determine the following:
   A. Localization of the site of nerve compression in multiple level injuries.
   B. Type of axon injured whether it is motor or sensory.
   C. Depth of the nerve lesion (myelin or axon).
   D. Severity of compressive injury.
4. NCS and EMG are helpful 1 month after injury when all the electrophysiological abnormalities are well developed.

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