EVALUATION OF CONSERVATIVE VERSUS SURGICAL THERAPY IN CARPAL TUNNEL SYNDROME ACCORDING TO TYPE OF NERVE LESION

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بسم الله الرحمن الرحيم

اقرأ باسم ربك الذي خلق

خلق الإنسان من علق

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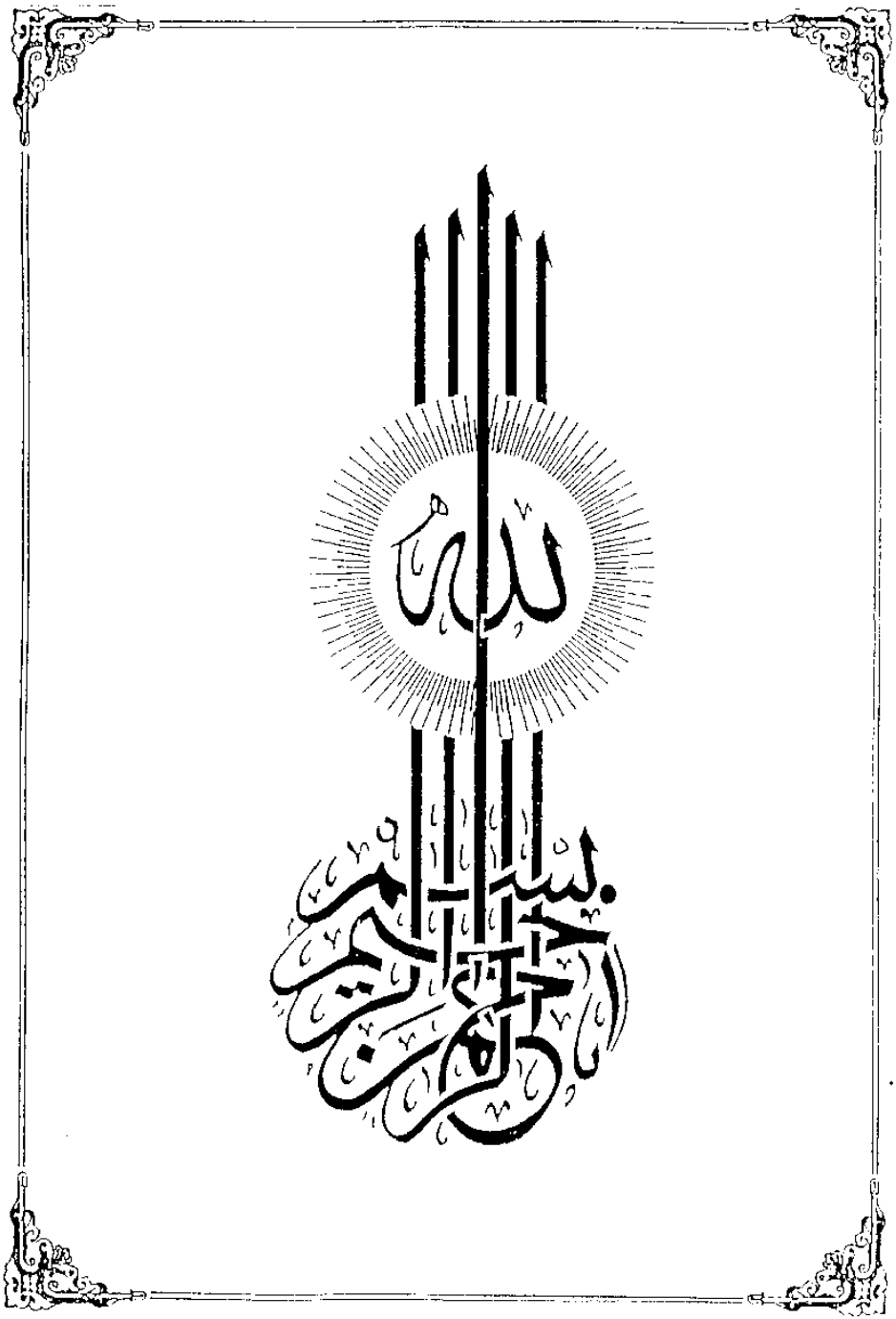
العلق ١-٥



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CONTENTS

LIST OF ABBREVIATIONS

CMAP:	**Compound muscle action potential.**
CNS:	**Central nervous system.**
CTS:	**Carpal tunnel syndrome.**
DIF:	**Difference.**
DML:	**Distal motor latency.**
DSL:	**Distal sensory latency.**
EA:	**Elbow amplitude.**
EMG:	**Electromyography.**
MUAPs:	**Motor unit action potentials.**
NCSs:	**Nerve conduction studies.**
PMA:	**Palmar motor amplitude.**
PSA:	**Palmar sensory amplitude.**
PSL:	**Palmar sensory latency.**
SNAP:	**Sensory nerve action potential.**
SOD:	**Superoxide dismutase.**
TCL:	**Transverse carpal ligament.**
WA:	**Wrist amplitude.**

INTRODUCTION

INTRODUCTION

Carpal tunnel syndrome (CTS) is one of the most commonly encountered peripheral entrapment neuropathy and is a common cause of paresthesia in the hands. It is seen in many fields of medicine- general practice, Physical Medicine and Rheumatology, Orthopaedic Surgery, Plastic Surgery, Internal Medicine... etc.

The basic lesion of the syndrome is compression of the median nerve within the narrow fibro-osseous carpal tunnel at the wrist, and any condition which reduces the capacity of the carpal tunnel may compress the median nerve with progressive distal sensorimotor impairment and pain (Phalen, 1966).

The signs and symptoms resulting from the median nerve compression at the wrist have previously been termed median neuritis, median neuropathy, and tardy median palsy, but since 1947 the condition has become known as CTS (Szabo, 1989).

Several methods are used in the treatment of CTS, and are divided into two main lines: the first one includes the non surgical or conservative line of treatment. This form of treatment typically consists of; avoiding the activity precipitating the condition, splinting the wrist in neutral position, a short course

of either steroids or non steroidal anti-inflammatory drugs, and a trial of diuretics, especially when the symptoms are perimenstrual (Phalen, 1972). Recently there has been a great interest in treating CTS with pyridoxin (vitamin-B6). Ellis (1982) has claimed that CTS is a primary deficiency of vitamin-B6.

The next tool of conservative treatment includes local steroid injection into the carpal tunnel due to its anti-inflammatory effect and fibrinolytic action, its effects on collagen synthesis, and its effect on the synovial vascular bed (Harvey et al., 1974 and De Ceulaer et al., 1979).

Lehamann et al. (1958) reported that pain threshold can be elevated by application of ultrasonic energy to the peripheral nerve on the area of the nerve endings.

Today, the low power infrared laser therapy is a new, non invasive and promising therapeutic tool in many branches of medicine (Kovacs, 1980 and Goldman et al., 1980). Zatelli et al. (1986) proved that low power laser therapy has a pain relieving effect. Also, it has an anti-inflammatory action through its anti-prostaglandin mechanism (Essaman, 1988), and it has been reported to be valuable in nerve regeneration as it increases collateral nerve sprouting and the regeneration of damaged nerve (Cheng and Kibbins, 1988).

The second line of treatment of CTS is the surgical method which entails surgical decompression of the median nerve. It is considered one of the most successful operations that can be performed on the hand. Even though the operation is a simple, not difficult minor surgical procedure there are important technical considerations that must be understood (Conolly, 1984).

The choice of selection of the different lines of treatment will depend upon the patient's condition and the result of the electrodiagnostic study.

CTS can be diagnosed electrically using proximal and distal nerve conduction latency and velocity of the median nerve, and in the evaluation of CTS by nerve conduction studies it is desirable to produce diagnostic as well as prognostic information.

In CTS the type of nerve lesion can not be clearly differentiated using routine nerve conduction techniques (Kemble, 1968). The finding of neurapraxia is regarded as an evidence that conservative treatment can result in prompt recovery of nerve function (Albers et al., 1985).

Johnson (1988) and Pease et al. (1988) reported that neurapraxia can be identified in CTS by comparing the

amplitudes of the compound muscle action potential (CMAP) recorded over the thenar muscles after stimulation of the median nerve at the wrist and in the palm. Comparison of the amplitudes of the CMAP provides the most reproducible information regarding neurapraxia. This evaluation adds substantial information to the study of the patients with CTS at a minimal cost of time, effort and discomfort.

AIM OF THE WORK

The aim of this work is:

1- To establish a new technique for more accurate diagnosis and determination of the type of nerve lesion in cases of CTS.

2- To evaluate the efficacy of conservative versus surgical therapy in treatment of CTS according to the type of nerve lesion.

Four years after Hunt's original article, Marie and Foxi (1913) performed an autopsy on a patient with marked atrophy of the thenar muscles but with no history of injury. Neuromas were noted in both median nerves just proximal to the TCL. They were the first physicians to recommend decompression of the median nerve by sectioning the TCL to prevent paralysis of the thenar muscles.

Watson (1929) described median neuropathy following carpal dislocations, and he published a large series of patients with chronic median nerve compression following fractures of the distal forearm. In the same year Learmonth decompressed the TCL in a patient with CTS, secondary to osteoarthritis of the wrist.

There was relatively slow recognition, however, of the occurrence of compression of the median nerve in the carpal tunnel without previous trauma. The first description of spontaneous compression was not made until 1938. In that year Moresch, a neurologist at the Mayo Clinic, described a syndrome of spontaneous median nerve compression, which he thought typically appeared in the later decades of life, included thenar atrophy and in some instances paresthesia and even sensory changes. Although he believed, as had Hunt thirty years earlier, that motor changes were due to compression of the thenar

motor branch beneath the TCL, he suggested that sensory changes were caused by a coexistent lesion in the carpal tunnel. Also, Woltman, another neurologist at the Mayo Clinic, in 1941 proposed that in acromegaly the production of hand pain, median sensory loss and thenar atrophy was due to pressure on the median nerve.

In 1945, Zachary rejected Hunt's diagnosis of isolated thenar motor branch compression, and explained that both acromegaly and post-traumatic arthritis of the wrist produced narrowing of the carpal tunnel causing compression of the median nerve.

In the following year, Cannon and Love (1946) reported on the first carpal tunnel release for spontaneous median nerve compression in the carpal tunnel.

In 1947, Brain, Wright and Wilkinson, published the first paper describing in details the clinical signs, diagnosis and pathophysiology of spontaneous median nerve compression in the carpal tunnel. The syndrome occurred in the absence of trauma, they explained, and could produce both sensory and motor findings. They believed that spontaneous recovery did not occur, and recommended early operative release of the TCL.