ELECTROPHYSIOLOGICAL STUDY OF PHRENIC NERVE IN GUILLAIN-BARRE SYNDROME

THESIS

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رسالة

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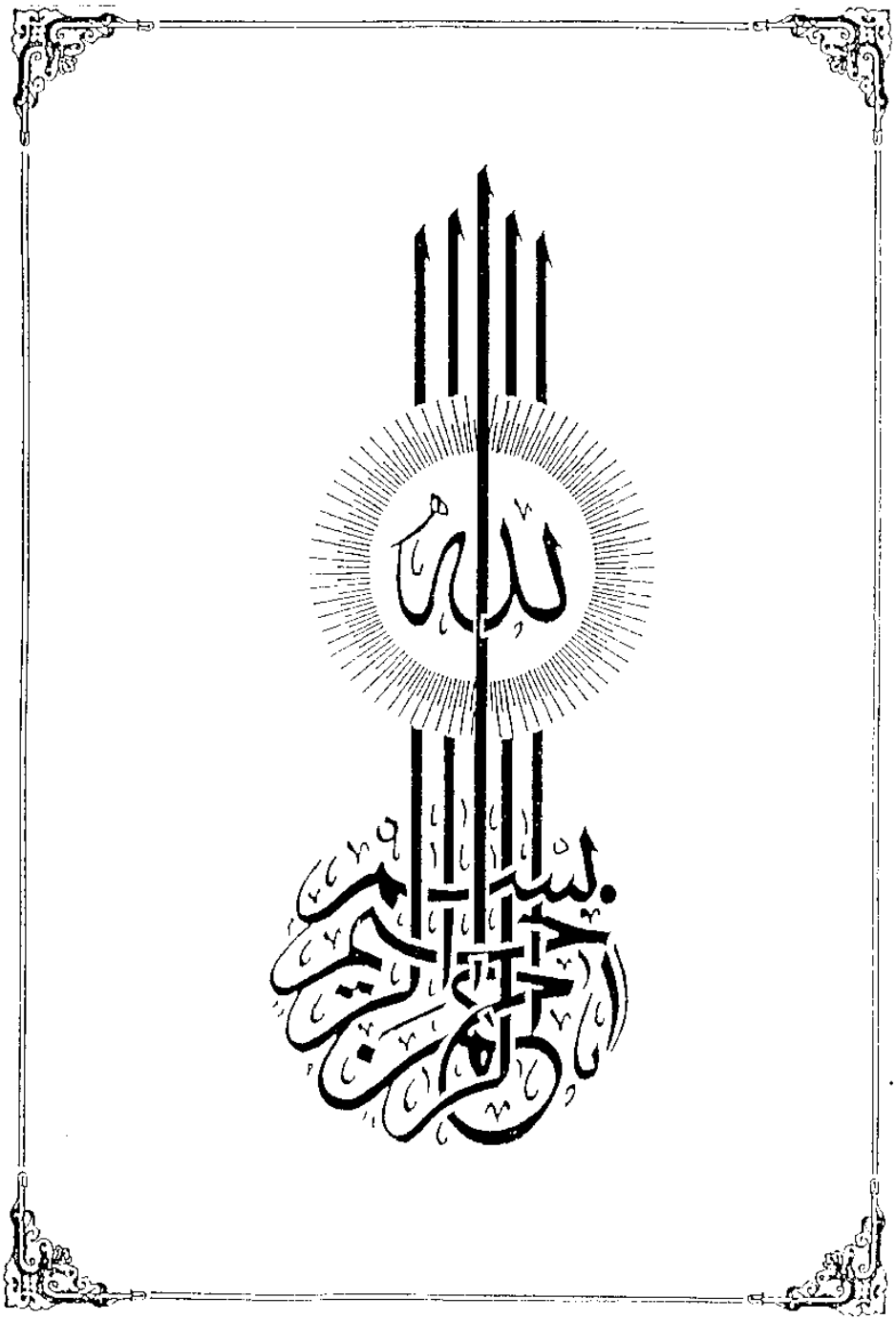
بسم الله الرحمن الرحيم

وقل اعملوا فسيرى الله عملكم
ورسوله والمؤمنون

صدق الله العظيم

التوبة / آية ١٠٥



To..

My Beloved Family

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First, thanks ***GOD*** *for blessing this work until it has reached its end.*

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LIST OF ABBREVIATIONS

AFO	Ankle foot orthosis
AIDs	Acquired immunodeficiency syndrome
CMAP	Compound motor action potential
CPM	Continuous passive motion
CSF	Cerebrospinal fluid
EMG	Electromyography
FVC	Forced vital capacity
GBS	Guillain-Barre syndrome
IL	Interleukin
KAFOs	Knee-Ankle foot orthosis
LLN	Lower limit of normal
NCV	Nerve conduction velocity
NPV	Negative predictive value
PPV	Positive predictive value
ROM	Range of motion
RR	Respiratory rate
SD	Standard deviation
ULN	Upper limit of normal
WBC	White blood cells

Introduction and Aim of the Work

INTRODUCTION

Guillain-Barre syndrome (GBS) is an acute idiopathic polyradiculoneuritis *[Rees et al., 1995]*. The exact pathogenesis of GBS is not fully understood. The major pathological change is multifocal demyelination with mononuclear inflammatory cell infiltrates in the peripheral nerves *[Vriesendorp et al., 1993]*. In rare cases there are axonal degeneration *[Feasby et al., 1986]*. Some authors suggest that an autoimmune origin is generally assumed to initiate the autodestructive process *[Vriesdorp et al., 1993]*.

GBS often follows viral infection or surgery in 70% of patients *[Enders et al., 1993]*. GBS patients presents after a non specific antecedent infection with tingling in the extremities and ascending symmetrical weakness progressing to flaccid quadriparesis *[Winner et al., 1988]*.

Respiratory failure requiring mechanical ventilation is a common complication of GBS, occurs in 14-44% of patients *[Bolton et al., 1992]*. Ventilatory failure in GBS is primarily due to diaphragmatic weakness, although weakness of intercostal, abdominal and accessory muscles of respiration, retained airway secretion, atelectasis and supine position are also contributory factors. So, early recognition of those at risk of respiratory decompensation

is important as they may benefit from intensive monitoring and early treatment *[Ropper and Kehne, 1985]*.

Phrenic nerve conduction studies may be useful in detecting respiratory involvement in patients with GBS and in identifying those at risk of respiratory failure which is the life threatening complication. So, this may give an idea about the severity of the disease *[Zifko et al., 1996]*.

On the other hand, in some cases determination of motor conduction of limb nerves may not be so beneficial in evaluating the severity of the disease *[McLeod, 1981]*.

AIM OF THE WORK

1- To detect the electrophyshiological involvement of phrenic nerve in GBS.

2- To evaluate the value of phrenic nerve electrophysiological study in early prediction of ventilatory failure.

Review of Literature

I-GUILLAIN-BARRE SYNDROME

HISTORICAL BACKGROUND

Guillain-Barre syndrome (GBS) is acute idiopathic polyneuropathy which is known in Europe as the Landry-Guillain-Barre syndrome. it is first delineated clinically by Landry in 1859, and later reevaluated by Guillain et al, in 1916. Guillain-Barre syndrome lacked a generally accepted pathologic basis for many years, even though Dumenil in 1864, had noted nerve fibre loss in the absence of spinal cord changes. Leyden in 1880 described the separation of the peripheral neuropathies from the amyotrophies of the spinal cord *[Roper et al., 1991]*. As more pathological descriptions emerged, it gradually became evident that the nerve fibre damage was associated with myelin loss, axonal preservation and endoneural inflammation *[Haymaker & Kernohan, 1949]*. It is now well established that nerve damage in this disease is not a diffuse process but occurs in the form of discrete foci of inflammation scattered, throughout the peripheral nervous system *[Prineas, 1981]*.

Incidence

GBS has an annual incidence of 1-2 per 100.000. The incidence increases gradually with advancing age for both sexes although the increase is more rapidly for men than for women *[Larsen et al., 1985]*.

Zhao et al. (1981), Schonberg et al. (1981), showed that males are affected more frequently than females.