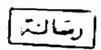
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ELECTROPHYSIOLOGICAL STUDY OF PHRENIC NERVE IN **GUILLAIN-BARRE SYNDROME**

THESIS

Submitted in Partial Fulfillment of the Requirements for Master Degree (M.Sc) in Physical Medicine and Rehabilitation

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To.. My Beloved Family

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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 of the peripheral neuropathies from the separation 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.