SLEEP DISTURBANCES IN PARKINSONISM

Thesis

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The M.D. Degree in -
Neurology

By

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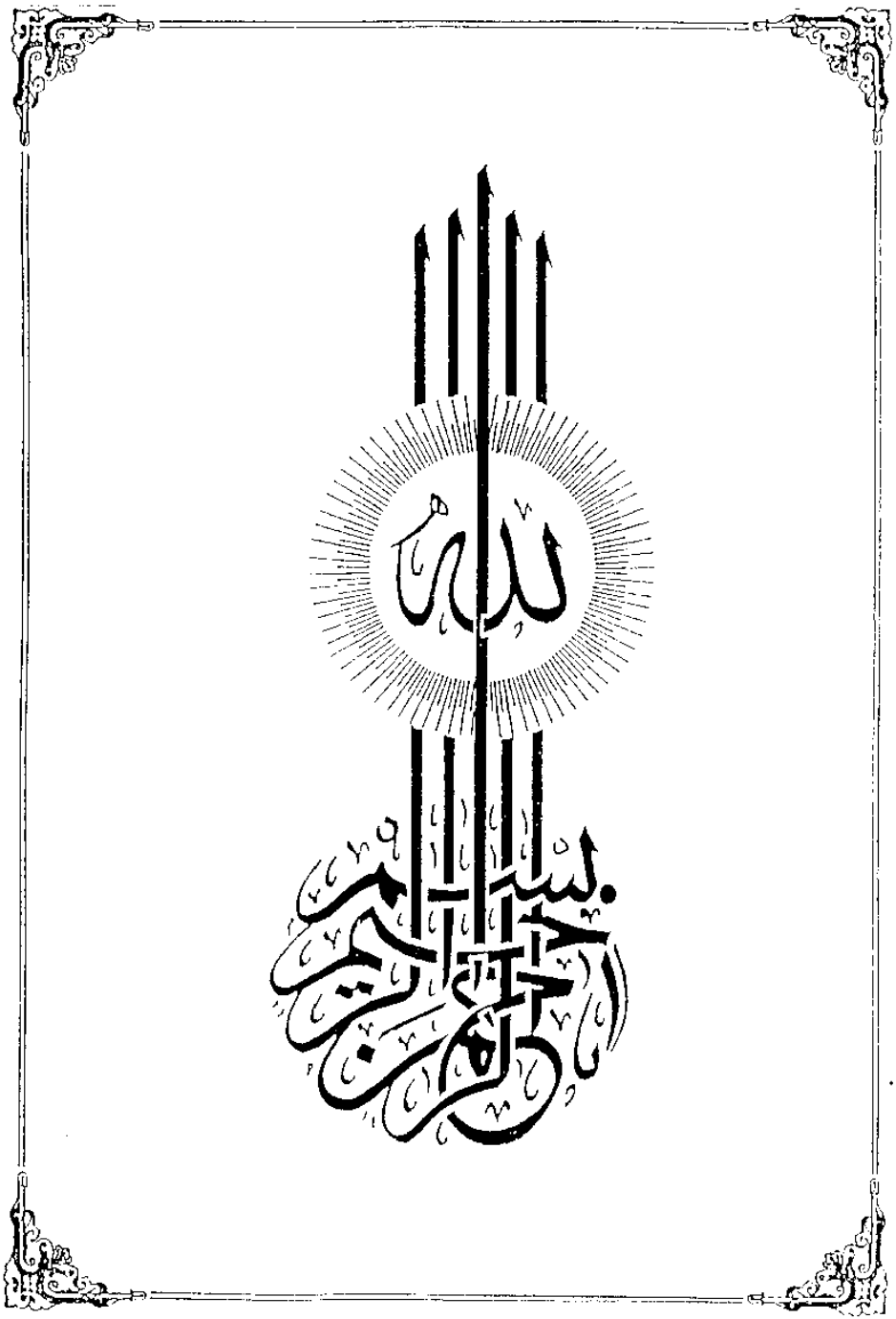
بسم الله الرحمن الرحيم
قالوا سبحانك لا علم لنا إلا ما
علمتنا إنك أنت العليم الحكيم.
صدق الله العظيم
البقرة - ٣٢ -



To ...

My Family

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LIST OF ABBREVIATIONS

PD	Parkinson's Disease
SNc	Pars compacta of the substantia nigra
LC	Locus ceruleus
DA	Dopamine
EEG	Electroencephalography
BEAM	Brain electrical activity mapping
Ach	Acetyl choline
GABA	Gamma-aminobutyric acid
Glu	Glutamic acid
C.T. scan	Computerized tomography scan
GPe	External part of globus palldus
GPi	Internal part of globus pallidus
SNr	Substania nigra pars reticulata
SN	Substantia nigra
STN	Subthalamic nucelus
ChAT	Choline acetyl transferase
PET	Positron emission tomography
5-HIAA	5-hydroxy indole acetic acid
VTA	Ventral tegmental area
MMSE	Mini-Mental state examination
SPECT	Single phaton emission computerized tomography
SWS	Slow wave sleep
REM	Rapid eye movement
NREM	Non rapid eye movement
HPA	Hypothalamic-pituitary adrenal
SCN	Suprachiasmatic nucleus
RF	Reticular formation
5-HT	Serotonin
MSH	Melanocyte stimulating hormone
EOG	Electrooculogram
EMG	Electromyogram
CSF	Cerebrospinal fluid
HVA	Homovanillic acid
CRF	Corticotropin-releasing factor
WMST	Wechsler memory scale test
PSG	Polysomnography

Introduction and Aim of the work

INTRODUCTION

Although the day time phenomena of Parkinson's Disease (PD) have been well recognized for almost 200 years, the frequent nocturnal symptoms, which occur in up to 75 percent of patients, and the associated sleep abnormalities were not systematically studied until the 1960s *(Aldrich, 1994).*

Sleep related complaints are reported in patients with PD, with various presentations including difficulty in initiating sleep, poor interrupted sleep by night with excessive day somnolence, unrefreshing sleep and early morning awakening *(Nausieda, 1987).*

The hypotheses of the pathogenesis of sleep disturbance in parkinsonian patients are different, i.e. many factors interplay making a certain single pathogenic mechanism almost impossible. First, bradykinesia and rigidity may reduce the number of normal body shifts during sleep, leading to discomfort and increased frequency of awakenings *(Mouret, 1975).* Second, increased muscle tone of upper airway muscles can produce disordered breathing *(Hardie et al., 1986).* Third, periodic leg movements, tremors or medication - induced myoclonic movements can produce arousals *(Nausieda, 1987).*

Fourth systems responsible for sleep - wake regulation may be affected by the neurochemical changes of PD, resulting in sleep disruption *(Aldrich, 1994).* Fifth, the association between depression and PD was found to be approximately 25-40% with mild to moderate intensity, while that between dementia and PD was 8-10% *(Okasha, 1988).* All of these effects on sleep have implications for planning the treatment of PD.

AIM OF THE WORK:

To study the prevalence of different types of sleep disorders and sleep profile in parkinsonian patients.

We will also compare the profile of sleep pattern in PD and that of depression. In addition to evaluation of cognitive functions in PD, we will discuss the possible etiological factors of sleep disturbance in parkinsonian patients.

All the above for suitable strategy of management and better quality of life.

Review of Literature

Pathogenesis of PD

PD can be produced by several different pathologic processes involving the basal ganglia *(Harding, 1993)*. In the past 20 years much progress has been made in identifying the anatomical connections and characterizing the regional neurochemistry of the basal ganglia (Fig. 1, 2, and 3).

The pathognomonic pathologic finding is loss of the large, pigmented neurons of the pars compacta of the substantia nigra (SNc). Loss of neurons has also been seen in other pigmented nuclei, including the locus ceruleus (LC) and dorsal vagus nucleus, and in the basal nucleus of Meynert. PD occurs when the number of dopamine (DA) neurons is reduced by more than 75% to 80% *(McGeer et al., 1977; McDowell and Cedarbaum, 1992)*.

Patients with PD may also have diffuse cortical atrophy. Mohamed (1993) found that frontal and temporal cortex are affected as evidenced by neuropsychological tests, electroencephalography (EEG) and Brain Electrical Activity Mapping (BEAM).