بسم الله الرحمن الرحيم

∞∞∞

تم عمل المسح الضوئي لهذة الرسالة بواسطة / سامية زكى يوسف

بقسم التوثيق الإلكتروني بمركز الشبكات وتكنولوجيا المعلومات دون أدنى مسئولية عن محتوى هذه الرسالة.

ملاحظات:

- بالرسالة صفحات لم ترد بالأصل
- بعض الصفحات الأصلية تالفة
- بالرسالة صفحات قد تكون مكررة
- بالرسالة صفحات قد يكون بها خطأ ترقيم

MAGNETIC MOTOR EVOKED POTENTIALS IN PARKINSONISM

THESIS

Submitted In Partial Fulfillment Of The Requirements For The Master Degree In Neurology And Psychiatry

By

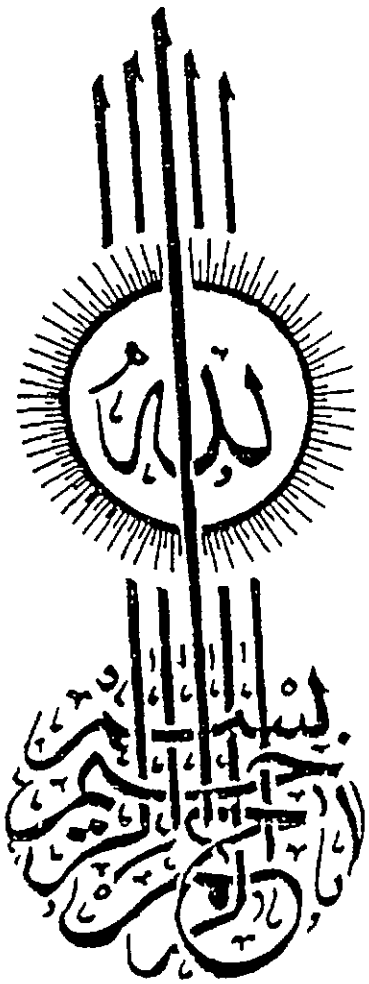
MOHAMMAD YOUSEF MOHAMMAD
(M.B. B. Ch. Assiut University

Supervised By

PROF. Dr. SAFIA ALY TOHAMY
PROFESSOR OF NEUROLOGY
FACULTY OF MEDICINE,
ASSIUT UNIVERSITY

Dr. HASSAN MOHAMMAD M. FARWEEZ
ASSISTANT PROFESSOR OF NEUROLOGY
FACULTY OF MEDICINE,
ASSIUT UNIVERSITY

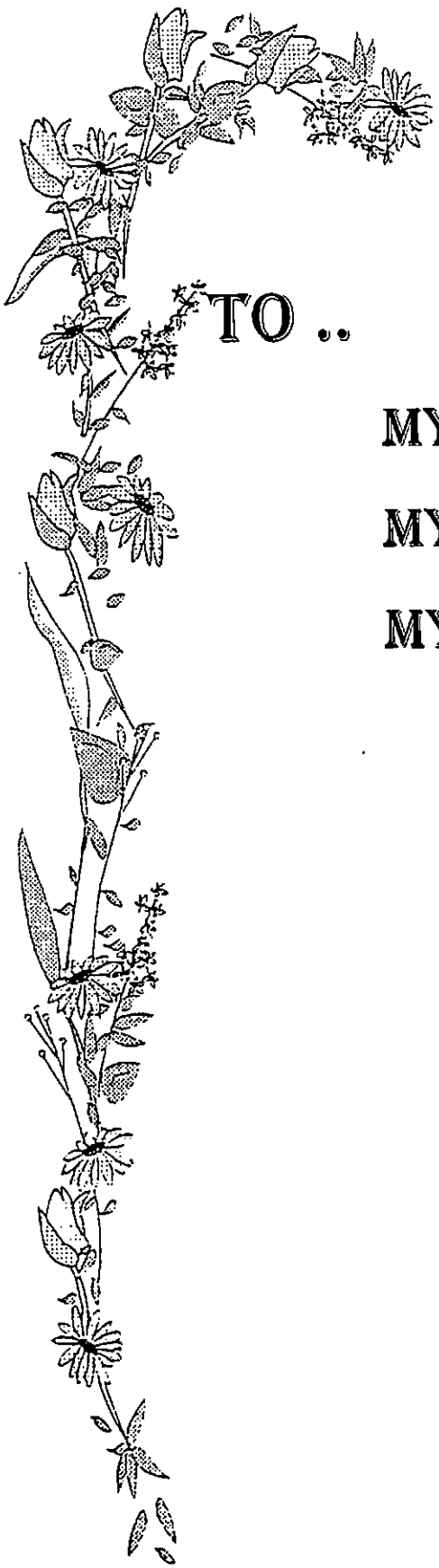
FACULTY OF MEDICINE
ASSIUT UNIVERSITY
2000



« وعلمك ما لم تكن تعلم وكان فضل الله عليك عظيما »

صدق الله العظيم

« من الآية ١١٣ سورة النساء »



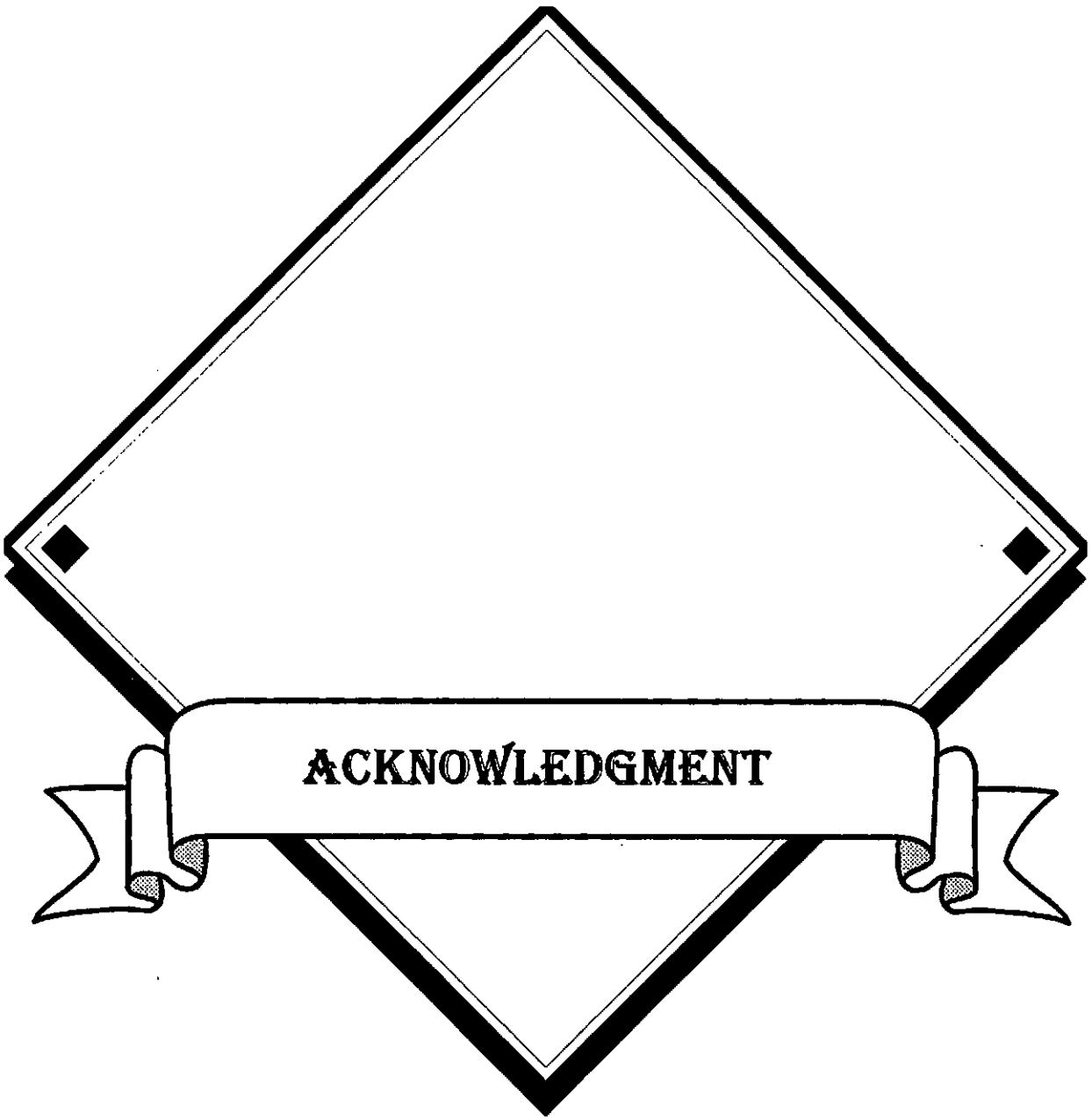
TO ..

MY PARENTS

MY BROTHERS

MY ONLY SISTER

MOHAMMAD YOUSEF


ACKNOWLEDGMENT

ACKNOWLEDGEMENTS

Praise be to **ALLAH,** the Merciful, the Compassionate for all the countless gifts have been offered. Of these gifts, those persons who were assigned to give me a precious hand so as to be able to fulfill this study. Some of them will be cordially acknoledged.

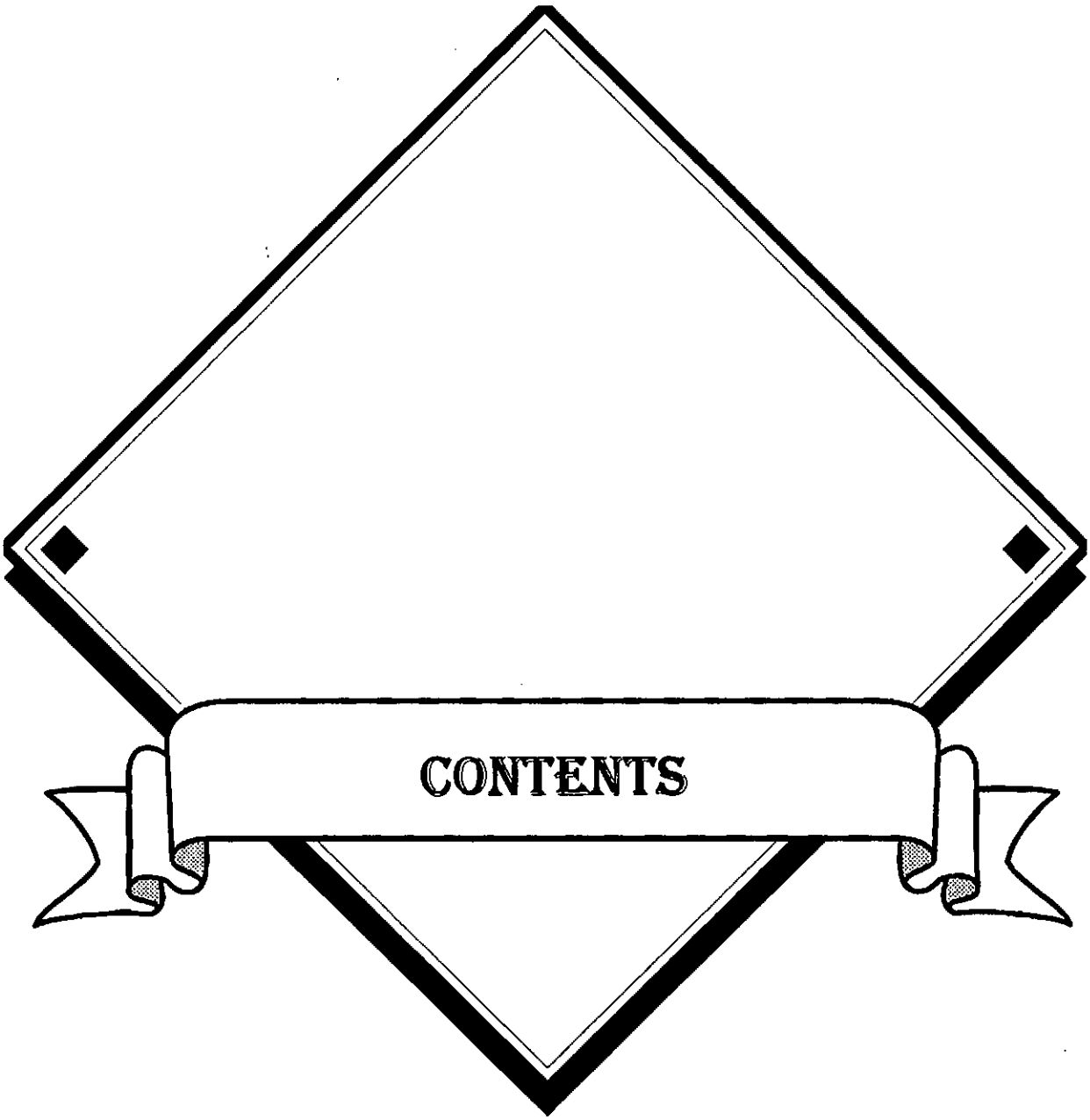
I am absolutely lucky to be supervised and directed by **Professor Dr. Safia A. Tohamy,** Professor of Neuropsychiatry Department, Faculty of Medicine, Assiut University, who kindly offered her kind guidance, sincere help. Remarkable experience and constructive remarks through revising every part of this thesis.

I would like to express my grateful appreciation and special thanks to **Dr. Hassan Mohammad Farweez,** Assistant Professor of Neurology, Faculty of Medicine, Assiut University for his unfailing support, close supervision, continuous advice, sympathetic help and kind attitude throughout this work.

With considerable appreciation, I express my great indebtedness to **Dr. Eman M. Khedr,** Assisstant Prof. of Neurology, Faculty of Medicine, Assiut University, for her fruitful discussions, significant encouragement, authentic guidance and magnificent assistance in every step of this work.

Finally, I wish to extend my appreciation and gratitude towards all my colleagues and nureses in the Neuropsychiatry Department, Faculty of Medicine, Assiut University, who gave full assistance, support and encouragement during the conduction of this study.

Mohammad Yousef

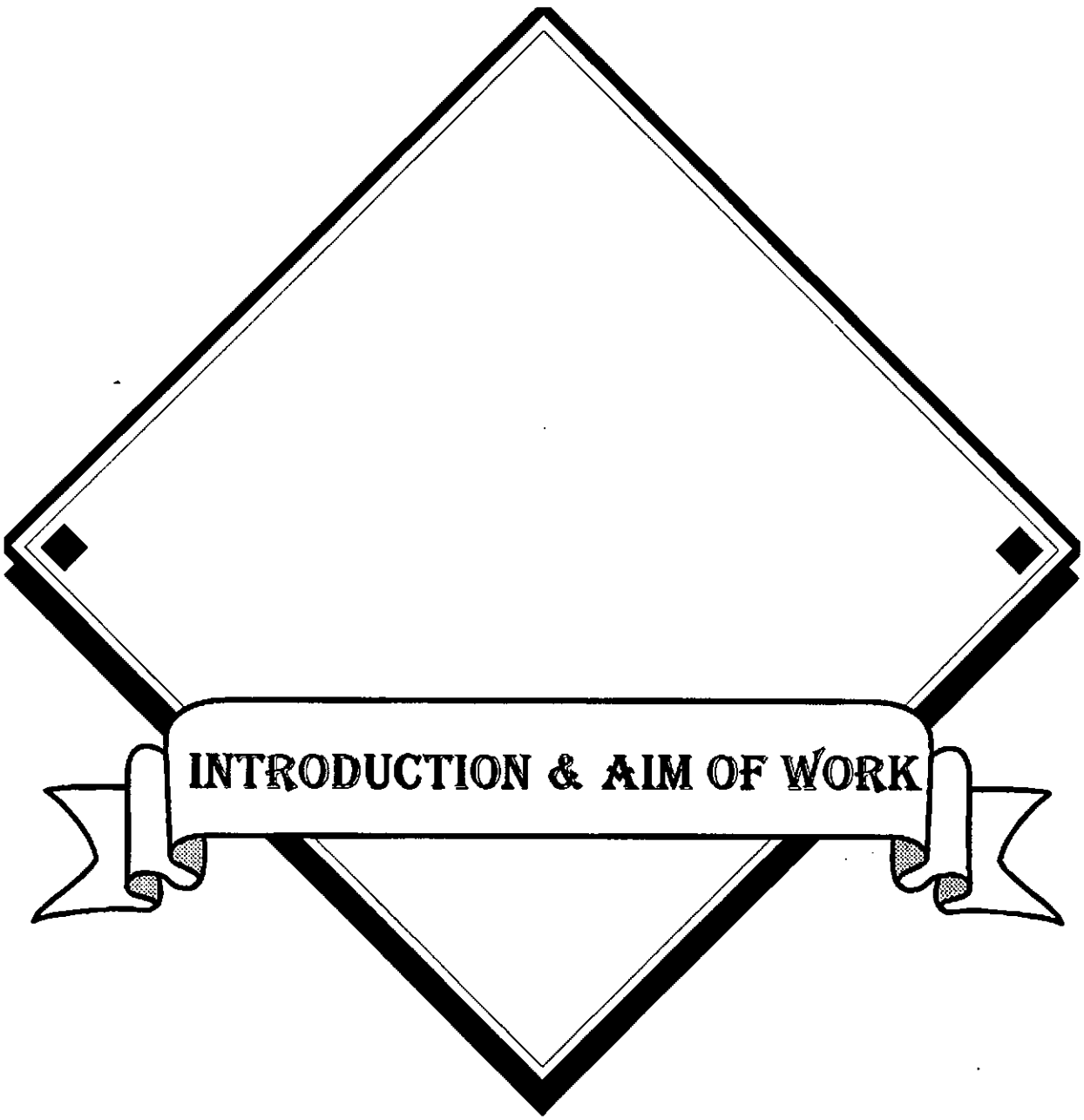

CONTENTS

CONTENTS

	Page
INTRODUCTION AND AIM OF WORK	1
REVIEW OF LITERATURE	5
* Parkinson's disease	5
* Aetiology	5
* Classification of major parkinsonion syndromes	6
* Cardinal signs	8
* Medical treatment of parkinsonism	10
* Transcranial magnetic stimulation in parkinson's disease	13
MATERIAL AND METHOD	29
RESULTS	38
DISCUSSION	98
CONCLUSIONS	107
RECOMMENDATIONS	108
SUMMARY	109
REFERENCES	112
INDEX	i-xiii
ARABIC SUMMARY	--

LIST OF ABBREVIATIONS

TMS	: Transcranial magnetic stimulation.
MEPs	: Motor evoked potentials.
PD	: Parkinson's disease.
MPTP	: 1-methyl-4-phenyl- 1,2,3,6 tetrahydropyridine.
SMA	: Supplementary motor area.
APB	: Abductor pollicis brevis.
EDB	: Extensor digitorum brevis.
CML	: Central motor latency.
CL	: Cortical latency.
RL	: Root latency.
PL	: Peripheral latency.
CML-M	: CL - RL.
CML-F	: CL - PL.


INTRODUCTION & AIM OF WORK

INTRODUCTION AND AIM OF WORK

Transcranial magnetic stimulation: a neuropsychiatric tool for the Twenty first century (George-Ms et al., 1996). The method is pain -free and very safe in the short and probably long term. Much has been learnt about the normal functioning of the motor system of man, its maturation and abnormalities that occur in a variety of diseases affecting the central and peripheral motor pathways (Eisen, 1992).

Central motor conduction times are typically calculated using the latency of motor evoked potentials (MEPs) by pulsed magnetic stimulation at the head and F wave latency by electrical stimulation of extremity. Central motor conduction time, which reflects the activity of the cerebral cortex, descending tracts, and spinal motoneurons, can fluctuate according to the condition of the central nervous system. Parkinson's disease is classified as an extrapyramidal disorders, and is primarily characterized by rigidity, bradykinesia, and tremor. However, function of the pyramidal tracts and spinal motoneurons may be altered electrophysiologically by parkinsonism because all movements are controlled by the pyramidal tracts and anterior horn cells (Ikoma et al., 1994).

Besides motor evoked potentials, transcranial brain stimulation also evokes an electromyographic silent period, which has attributed largely to intracortical inhibitory systems (Fuhr et al., 1991, Cantello et al., 1992, Kukowski and Haug, 1992, Triggs et al., 1992, Uozumi et al., 1992, Inghilleri et al., 1993, Roick et al., 1993, Wilson et al., 1993). Previous studies have revealed that patients with Parkinson's disease have a normal central conduction (Dick et al., 1984, Cantello et al., 1991, Priori et al., 1992). The central conduction time gives clues only on the output of the

neural elements of the primary motor cortex and not on the state of the intracortical neural networks. Previous reports (Cantello et al., 1991) have shown that the cortical silent period is reduced in patients with parkinson's disease and prolonged in huntington's disease (Roick et al., 1992).

Eisen et al. (1991), showed that, compared to age -matched controls, MEPs amplitude is often increased in parkinson's disease and there was a concomitant increase in F wave, in addition, cortical threshold was reduced on the involved side, the cortical silent but not the peripheral silent period were also reduced on the involved side. They also postulated that these changes result from spinal disinhibition but the possibility of hyperexcitability at the level of the cortex is also likely to play a role.

Ikoma et al. (1994), concluded that reduced central motor conduction time and high F wave amplitude reflect hyperexcitability of the anterior horn cells in patients with parkinson's disease. Also Shimamoto et al. (1996) related these changes of MEP to dysfunction of pyramidal pathways in Parkinson's disease.

And while Priori et al. (1994), concluded that dopaminergic drugs modulate the duration of the cortical silent periods and in normal subjects, through mechanisms acting mainly at basal ganglia and possibly also directly at cortical level, Nakashima et al. (1996), concluded that L- Dopa administration did not change the amplitudes of the motor responses or the auditory effects on the motor responses produced by magnetic cortical stimulation. Transcranial magnetic stimulation used to assess the relation between bradykinesia and excitability of the motor cortex where Ellawy et al. (1995), concluded that parkinsonian patients exhibiting pronounced bradykinesia have a lowered excitability of the motor cortex. Britton et al.

(1993), postulated that magnetic stimulation significantly shorten the period of parkinsonian postural tremors but did not influence the period of essential or mimicked tremors. These behavioural differences indicate differences in the pathophysiological mechanisms underlying parkinsonian postural tremor and essential tremor.

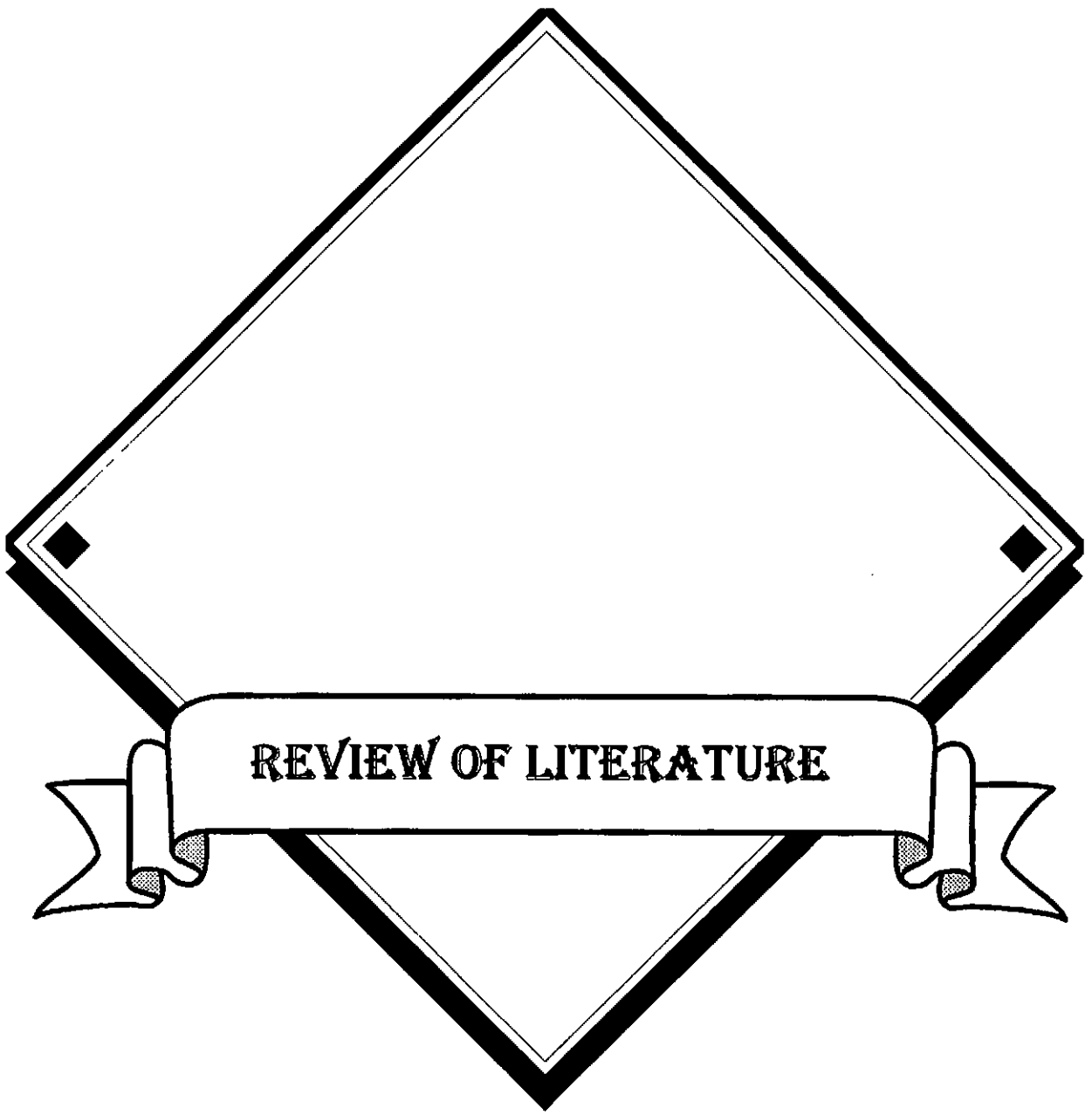
Aim of the work

1- The primary objective of this study is to demonstrate the abnormalities in magnetic MEPs in different varieties of parkinsonism, and whether these abnormalities correlate with the clinical assessment.

2- The possible use of transcranial magnetic stimulation as a diagnostic tool to differentiate between different types of parkinsonism.

3- To study more about the pathophysiological basis of tremors, rigidity and akinesia.

4- To study the effect of antiparkinsonian drugs on central and peripheral motor conduction time.


REVIEW OF LITERATURE