

# **ELECTROENCEPHALOGRAPHIC CHANGES IN INSULIN DEPENDENT DIABETES MELLITUS IN CHILDREN**

THESIS

SUBMITTED IN PARTIAL FULFILLMENT OF  
MASTER DEGREE IN PEDIATRICS

PRESENTED BY

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UNDER THE SUPERVISION OF

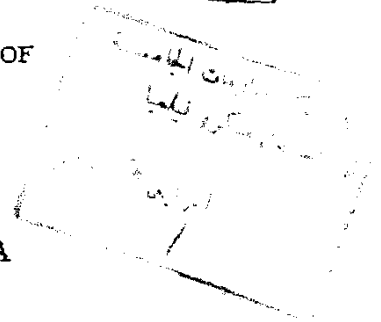
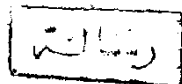
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***TO:***

***MY PARENTS,  
MY WIFE  
MY DAUGHTER***

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## LIST OF ABBREVIATIONS

DK	Diabetic Ketosis
DKA	Diabetic Ketoacidosis
DM	Diabetes Mellitus
EEG	Electroencephalogram
FCPD	Fibrocalculous pancreatic diabetes
HBA1C	Glycosylated Hemoglobin
HZ	Hertz
IDDM	Insulin dependent diabetes mellitus
JPTS	Juvenile pancreatitis tropical syndrome
MRDM	Malnutrition related diabetes mellitus
PDPD	Protein deficient pancreatic diabetes
S.D	Standerd deviation
S.E	Standerd error

***INTRODUCTION  
AND  
AIM OF THE WORK***

## INTRODUCTION

Diabetes mellitus is one of the commonest endocrinal diseases in childhood. It is classified into three forms, type I diabetes, type II diabetes and secondary diabetes. The basic cause of type I diabetes in childhood is the sharply diminished secretion of insulin.

(Genuth, 1983).

Although basal insulin concentrations in plasma may be normal in newly diagnosed patients insulin production in response to a variety of potent secretagogues is blunted and usually disappears over a period of months, to years rarely exceeding 5 years. (Sperling, 1988).

Type I diabetes is characterized by increased frequency of complications (Brittle diabetes). Among the important complications are the neurological complications. (Feingold, 1984).

EEG abnormalities constitute one of the neurological complications which are suspected to occur during the course of type I diabetes mellitus. The hypoglycemia and ketoacidosis which occur in the type I diabetes are the

major factors incriminated in the occurrence of the EEG changes. These EEG changes may vary between simple transient forms to severe and permanent EEG sequelae.

(Lerman et al., 1977).

## AIM OF THE WORK

The aim of this work is to study the electroencephalographic changes induced in insulin dependent diabetes mellitus and its correlation to the course of the disease as well as to the frequency and severity of episodes of diabetic ketoacidosis and hypoglycemic attacks.