STUDY OF THE IMPLICATION OF DIABETIC CONTROL ON GASTRIC EMPTYING

THESIS
SUBMITTED FOR PARTIAL FULFILMENT OF
M. Sc. Degree in
INTERNAL MEDICINE
By

Ahmed Ahmed Eltokhy
M.B.B.Ch.

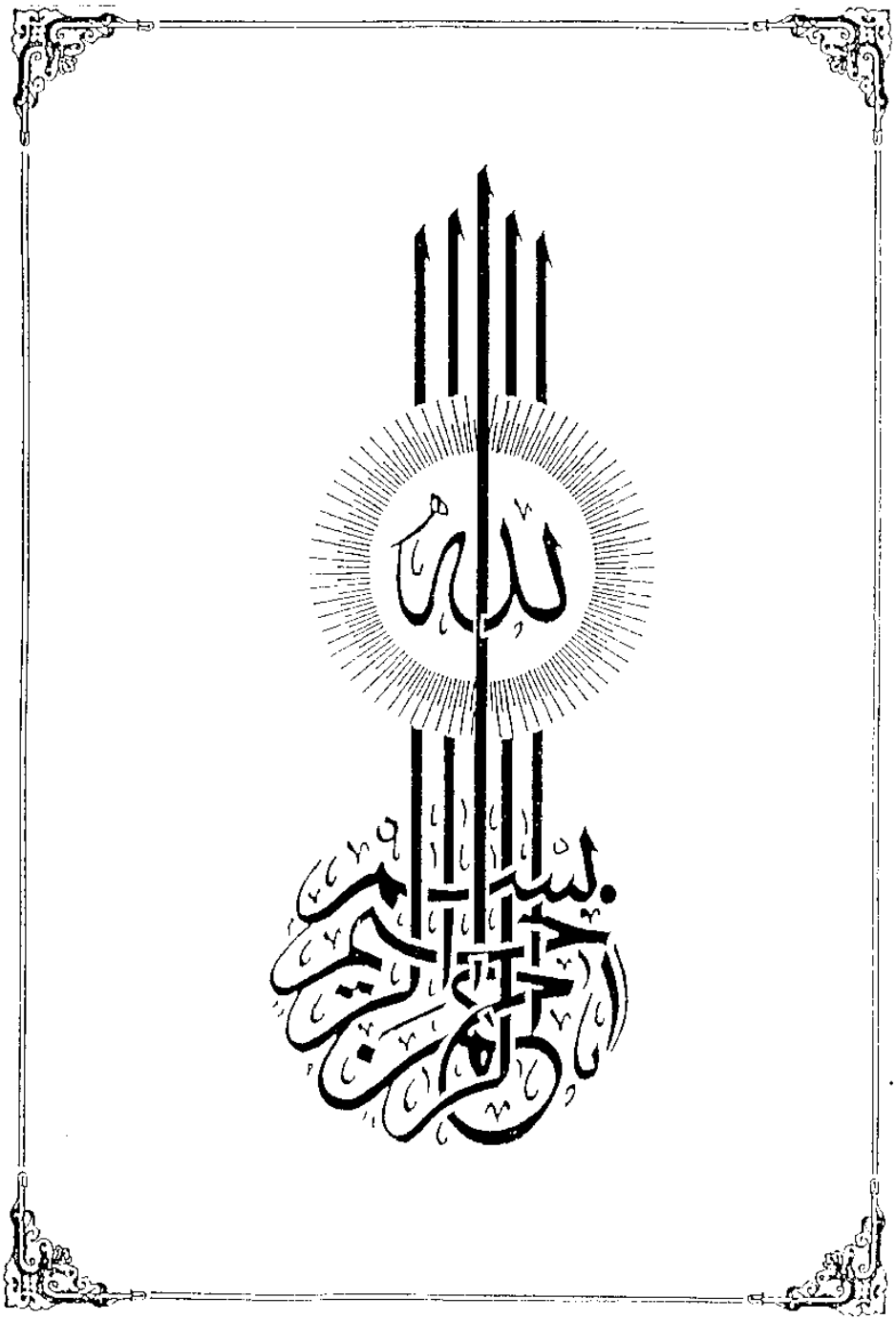
SUPERVISORS

Prof. Dr. Sami Abd Allah Abdel Fattah
Prof. of Internal Medicine
Ain Shams University

Prof. Dr. Prof. Fadila Gadallah
Prof. of Internal Medicine
Ain Shams University

Ass. Prof. Hala Abou Senna
Ass. Prof. of Radiology
Ain Shams University

Faculty of Medicine
Ain Shams University
1996



ACKNOWLEDGMENT

I have great pleasure to express my utmost gratitude to **Professor Dr. Sami Abd Allah Abdel Fattah, Professor of Internal Medicine, Faculty of Medicine, Ain Shams University** for the supervision of this thesis and for his continuous support that I have received throughout this work.

I would like to forward my sincere thanks to **Professor Dr. Fadila Gadallah,** Professor **of Internal Medicine, Faculty of Medicine, Ain Shams University** for her moral support and close follow-up of this work.

I am also grateful to **Ass. Prof. Dr. Hala Abou-Senna, Ass. Prof. of Radiology, Faculty of Medicine, Ain Shams University**, whose indispensable help in doing the ultrasonography.

I would like to offer many thanks to **Dr. Afaf Abdel-Wahab Haggag, MD of Endocrinology and Metabolism, Ain Shams University Hospitals** for her advice in writing this thesis.

I appreciate the help and co-operation of every person who gave me a hand so that this work could come to existence.

LIST OF ABBREVIATIONS

AMP:	Adenosine monophosphate
ATP:	Adenosine triphosphate
BLI:	Bombesin-like immunoreactivity
CCK:	Cholecystokinin
CHO:	Carbohydrate
CIIP:	Chronic idiopathic intestinal pseudoobstruction
ECA:	Electrical control activity
GER:	Gastrooesophageal reflex
GIP:	Gastric inhibitory polypeptide
GLI:	Glucagon like immunoreactivity
GRP:	Gastric releasing peptides
IRG:	Immunoreactive glucagon
SLE:	Systemic lupus erythematosus
VIP:	Vasoactive inhibitory peptides

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INTRODUCTION
AND
AIM OF THE WORK

Introduction and Aim of the Work:

Gastroparesis is one of the sequelae of diabetes. Symptoms may include post-prandial nausea, epigastric pain, bloating, vomiting, early satiety *(Drenth, Engels, 1992)*. Delayed gastric emptying in diabetic patients occurs with progress of autonomic neuropathy as one late complication correlated with poor glycemic control *(Yamada, Hongo, 1992)*. Delayed gastric emptying causes unstable glycemic control because of unpredictable emptying of food from the stomach *(Chaudhuri, Fink, 1992)*. The improvement of delayed gastric emptying in patients with autonomic neuropathy should be achieved not only for relief of GIT symptoms but also for stable glycemic control *(Okumo, Ango, 1993)*.

In diabetic patients, gastric emptying have been shown to be impaired during hyperglycemia. The hyperglycemia may affect GIT function through vagal-cholinergic inhibition by alteration in serum osmolarity or perhaps by alteration in GIT hormone secretion *(Schvarcz, Palmar, 1993)*.

The aim of this work is to evaluate the effect of good glycemic control of diabetes on gastric emptying, also, the difference in the response after metoclopramide and metopemazine compared to good glycemic control on gastroparesis and delayed gastric emptying.