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بسم الله الرحمن الرحيم

شبكة المعلومات الجامعية

التوثيق الالكتروني والميكروفيلم

شبكة المعلومات الجامعية

جامعة عين شمس

التوثيق الالكتروني والميكروفيلم

قسم

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بالرسالة صفحات لم ترد بالاصل

ANGIOGENIN LEVELS IN ATHLETIC, NON ATHLETIC AND ATHEROSCLEROTIC VASCULAR OCCLUSIVE SUBJECTS

Thesis

Submitted in Partial Fulfilment of
The Requirements of Master Degree in Clinical pathology

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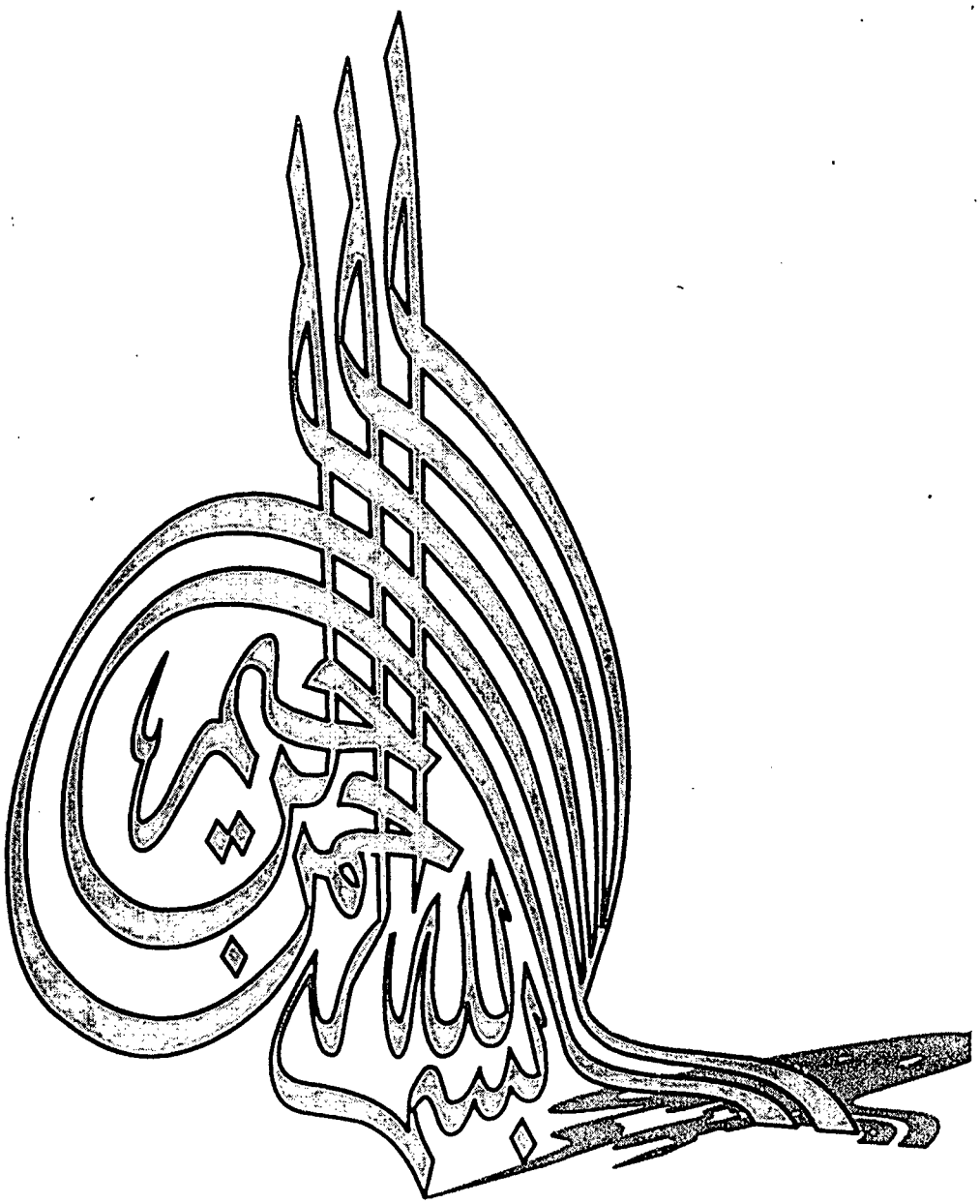
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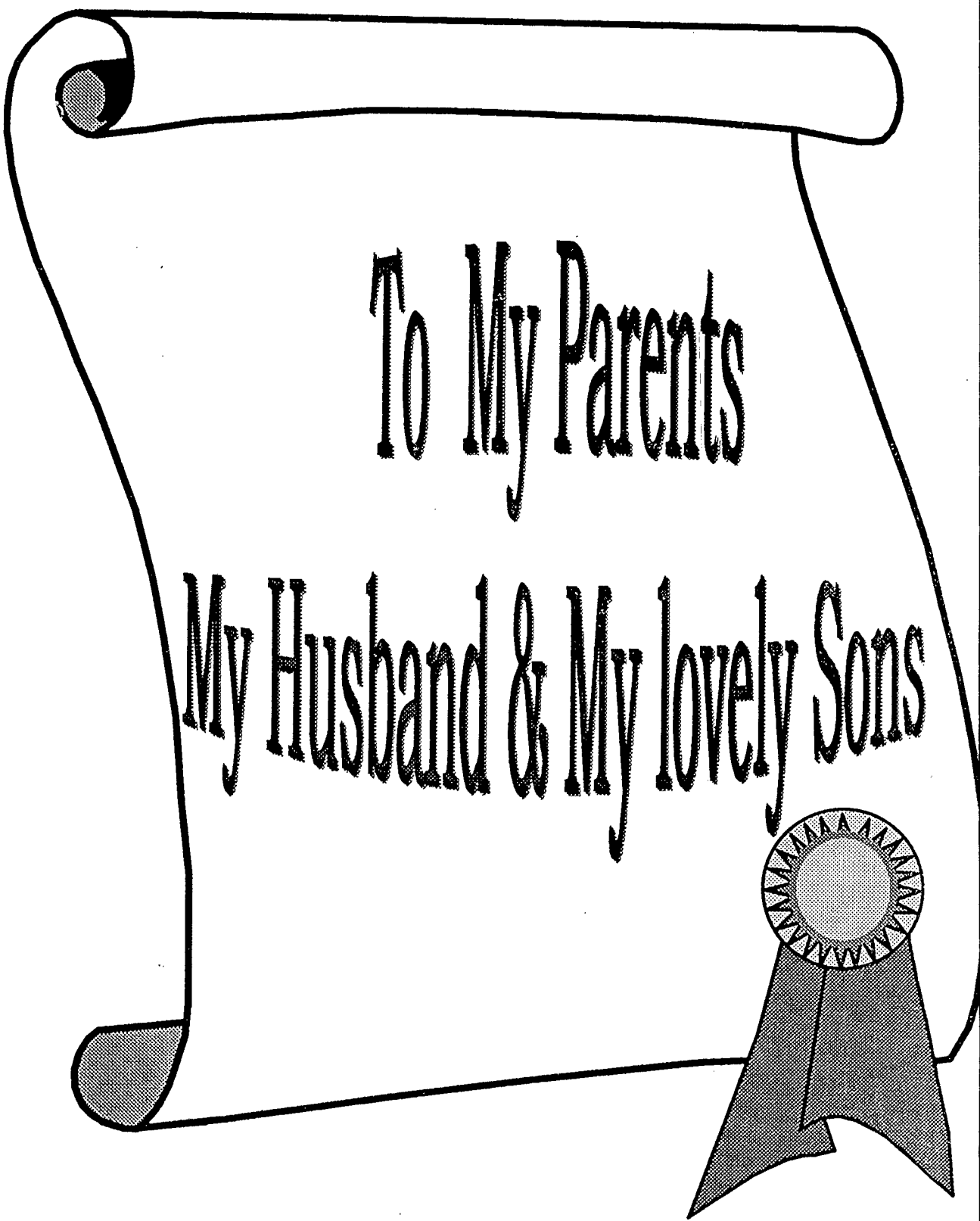
To My Parents
My Husband & My lovely Sons

ACKNOWLEDGEMENT

First of all thanks **to ALLAH**, whose magnificent help is the main factor in completing this work and in everything we can do in our lives.

I would like to express my deepest gratitude to ***Prof. Dr.*** **Nabih Helal EL-Fadali.** Prof. of Clinical pathology, Faculty of Medicine, Tanta University for his guidance, continuos encouragement, unlimited help, careful supervision and revision of the thesis.

I am deeply grateful to ***Dr.*** **Enas Arafa El-zamarany** assistant professor of Clinical pathology, Faculty of Medicine, Tanta University for her cooperation and continous help throughout this work. For her kind help I will remain always remembering.

I wish to express my sincere appreciation to ***Dr.*** **Mai Mohamed Abd EL- Moneim Salama** assistant professor of Cardiovascular Medicine, Faculty of Medicine, Tanta University for her kind supervision, continuos help and encouragement throughout this work.

LIST OF ABBREVIATIONS

AMI	:	Acute myocardial infarction.
ANG	:	Angiogenin.
APP	:	Acute phase protein.
Arg	:	Arginine .
≈	:	Approximately.
bFGF	:	Basic fibroblast growth factor.
BM	:	Basement membrane.
CK- MB	:	Creatine kinase (myocardial fraction).
CPAE	:	Calf pulmonary artery endothelial.
EC	:	Endothelial cells.
GM7373	:	Foetal bovine aortic endothelial.
His	:	Histidine.
HME	:	Human microvascular endothelial.
HT-29	:	Human colon adeno carcinoma.
HUAE	:	Human umbilical artery endothelial.
KDa	:	Kilo Dalton.
Lys	:	Lysine.
MI	:	Myocardial infarction.
m RNA	:	Messenger RNA.
PAD	:	Peripheral arterial disease.
PAOD	:	Peripheral arterial occlusive disease.
PMNL	:	Polymorphonuclear leukocytes.
RI	:	Ribonuclease inhibitors.
tPA	:	Tissue plasminogen activator.
tRNA	:	Transfer RNA.
VEGF	:	Vascular endothelial growth factor.
Vrs	:	Versus.

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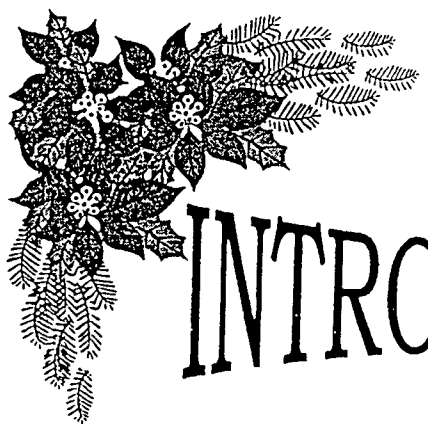

INTRODUCTION

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Atherosclerosis is not a single disease entity, the lesions of atherosclerosis take different forms, depending upon their anatomic site, the age, genetic and physiological status of the affected individual and presumably, upon the risk factors to which each individual may have been exposed ***(Gown et al., 1986).***

Atherosclerosis is the underlying substrate in nearly all patients with peripheral arterial occlusive disease (PAOD) and myocardial infarction (MI). The earliest recognized atherosclerotic lesion is the fatty-streak, the more advanced lesion is the fibrous plaque which protrude into the lumen leading to its narrowing. With progressing of disease, further cell necrosis and calcification, crack and fissure formation, ulceration and mural thrombosis, these lead to complicated lesions and give rise to obstruction and complete occlusion of the vessel lumen and ischaemia of the end organ ***(Basha and sowers., 1996)***.

Since the mid 1980s a new strategy is come from bench to practical used termed angiogenesis. This process involves the growth of new vessels from pre-existing vessels by sprouting. Ischaemia being the major stimulus of angiogenesis ***(Helisch and schaper., 2000)***. These newly formed vessels are not surrounded by mural cell and lack vascular

smooth muscle cells, these making them fragile and prone to rupture. So remodelling process (arteriogenesis) follows angiogenesis to result in fully functional and structurally vessel which may bypass sites of arterial occlusion in many regions of the body ***(Buschmann and schaper., 2000).***

Angiogenesis is induced by a variety of angiogenic molecules including angiogenin (ANG). ANG is a potent inducer of new blood vessels growth more than others angiogenic stimuli ***(Etoch et al., 2000)***. It belongs to the pancreatic R Nase superfamily of proteins, it is the only member of the superfamily able to stimulate angiogenesis ***(Wiedlocha., 1999).*** However, its mechanism of action is not known as yet, it has been demonstrated to induce most of the individual events in the process of angiogenesis ***(Strydom., 1998).***

AIM OF THE WORK